

Trypanosomiasis in Cattle and its Prevalence: Review

Yitayal Gebrie

Bahir Dar City Municipal Abattoir, Veterinary Public Health Department, Bahir Dar, Ethiopia

Abstract: Trypanosomiasis is one of the major constraints on animal production in areas of Africa that has the greatest potential for significant increases in domestic livestock populations and livestock productivity. In every case, trypanosomiasis leads to considerable under-exploitation of natural resources and to a lower level of animal production than could be achieved if the disease were eliminated. While various methods are being used by farmers to control the disease, major public efforts have been directed towards control of tsetse flies and on the use of trypanocidal drugs. Continent-wide fly eradication has recently been advocated as the ultimate solution needing public effort. Due to their nature, there are difficulties in sustaining the current methods of tsetse control. However, the efficacy of currently available trypanocidal drugs is also decreasing, due to drug resistance developing faster than generally thought. Although less attention has been focused on the use of naturally disease tolerant livestock to cope with the disease, farmers in 19 out of 40 countries in the most humid parts of West and Central African countries affected by the disease are using these livestock as a major, if not only, option to cope with the problem in an economically sustainable and environmentally friendly way. There is increasing recognition that Africa possesses animal genetic resources probably unparalleled in any other continent. The natural innate resistance possessed by breeds of cattle such as the N'Dama and the West African short horn to trypanosomiasis and to several other important infectious diseases should be an increasingly important component of national and regional disease control programmes. Researchers are providing support for this environmentally healthy solution which has been demonstrated to be economically viable at both public and private levels.

Key words: Africa • Cattle • Control • *Trypanosomiasis* • Tsetse Flies

INTRODUCTION

Trypanosomes are flagellated protozoan parasites that live in the blood and other body fluids of vertebrate hosts and the most serious problem of animal production in Sub-Saharan Africa and prevent keeping of ruminants over million square meters of potentially productive land [1]. Morbidity rates during out breaks are variable and may reach 70% in cattle infected with *T. vivax* and infection rate in cattle in endemic areas is considerable and could be over 60%. However, as a result of various control methods, the prevalence is decreasing in many African countries, particularly in west Africa and was as low as 10% in Mali in the 1980s [1]. Because of the absence of a vaccine due to the phenomenon of antigenic variation, cattle can be protected prophylactically with a few drugs. Treatment against trypanosomiasis should be given early in the disease during the initial phase of

fluctuating parasitaemia. Therefore, the control of this disease is depends against the vector, host and parasite [2].

Because of the variable responses to treatment and the ability of trypanosome organism to develop drug resistance, all control programs must include methods of controlling the tsetse fly. Current recommendations for fly control include application of insecticides with residual pyrethroids [3]. In the past, extensive tsetse control campaigns were under way in a number of African countries. These applied parasite and vector control operations on a large scale, involving some techniques that would no longer be acceptable today because of recent environment concerns. The approach usually followed a strategy that would now be call area wide and several of the companies succeeded in eliminating tsetse from large area of land, particularly at the edges of the tsetse belt [4]. The pathogenic species of tsetse

transmitted trypanosomes in terms of economic loss in domestic and agricultural productivity are found in Ethiopia about 220, 000 km² which fertile and arable land located in south western and north western part of the country more than 14 million cattle and equal number of small ruminants are at risk of the diseases [5]. In general, the most important trypanosomes in terms of economic loss in domestic livestock are the tsetse transmitted species such as *T. congolense*, *T. vivax* and *T. brucei* [6].

In every case, trypanosomiasis leads to considerable under-exploitation of natural resources and to a lower level of animal production than could be achieved if the disease were eliminated. Based on the above information and fact about control options trypanosomiasis, the objective of this paper is to review the control options of trypanosomiasis in Africa especially of Ethiopian situation.

Etiology of Trypanosomiasis: Trypanosomiasis is caused by trypanosomes. Trypanosomes are classified in the phylum Sarcomastigophora, the order Kinetoplastida and the family Trypanosomatidae [7]. The major species of tsetse transmitted bovine trypanosomes are caused by *T. congolense*, *T. vivax* and *T. brucei* [8]. They multiply in the body fluids /blood stream, lymphatic vessels and tissues, including the central nervous system (CNS) and cardiac muscle) of vertebrate hosts (mammals) and live in the digestive tract of invertebrate host which is generally a biting insect [9, 10].

Morphology: The trypanosome consists of a single cell varying in size from 8 to over 50 µm. There are distinct differences in appearance, shape and size between the various species of trypanosomes, allowing specific identification [11]. All the tsetse transmitted bovine trypanosomes are motile, extracellular, spindle shaped, flagellated protozoan parasites ranging from about 10 to 30 µm in length [12]. Trypanosome species have single flagellum, which runs to the anterior end of the body and attached along its length to form an undulating membrane [13]. The salivaria group of trypanosomes may or may not have a free flagellum, the kinetoplast is terminal or sub-terminal and the posterior end of the body is usually blunt.

Epidemiology of Tsetse Transmitted Trypanosomiasis: The occurrence of African trypanosomiasis is determined mainly by the ecology of tsetse fly which is found only in tropical Africa [1]. Based on the epidemiology of trypanosomiasis three elements must be considered.

These are the definitive host (domestic or wild mammals, man), parasites (trypanosomes) and vector (tsetse flies and other biting flies). The role of each factors and their relationships is based on knowledge of the ecology of the mammalian hosts and vector (habits, distribution, activity), their behavior (movements, search for food, reproduction), population size, dynamics of trypanosome transmission and the role of parasite reservoirs (resistance of tolerant wild animals and domestic animals) and the virulence of the parasite [14]. The distribution of trypanosomiasis is approximately 10 million square kilometers of sub Saharan Africa between latitudes 14N° and 29°S [13]. The tsetse belt extends from the Southern part of Rift Valley around the southern low lands to the larger Rift Valley of the Abaya/Dedessa, Baro/Akobo, Ghibe /Omo and all the pathogenic trypanosome species affecting animals are reported to be found in Ethiopia (Figure 2).

The distribution of trypanosomiasis is throughout the country with the exception of the highlands where it is rare or absent in Ethiopia. The distribution of tsetse transmitted African trypanosomiasis is governed by the presence of tsetse vector that is roughly between 15° and 25° latitudes in Tropical Africa [11]. The distribution of the major *Trypanosoma* species found in Ethiopia affecting both human as well as animals is shown in Figure 3.

Vector: The vector that is responsible to transmit cattle trypanosomiasis, tsetse flies (*Glossina* spp.), are found only in Africa. They are biological or mechanical vectors of trypanosomes and constitute a potent and constant to livestock over much of Sub Saharan Africa. Thirty-one species and sub species of tsetse flies have been identified. Only a few species are causing human sleeping sickness but all are potential vector of animal trypanosomiasis [15]. The historical classification of tsetse based on morphological criteria, divides the species into three groups the fusca group (subgenus *Austina*) tend to occur in low land rain forest of West and Central Africa. The palpalis group (subgenus *Nemortinas*) is found in the riverine galleries of West and Central Africa but extend into savanna region between river systems; *G. palpalis* and *G. tachinoides* are important animal trypanosomiasis vectors in this group. The moristans group (subgenus *Glossina*) occurs in variety of savannah habitats lying between the forest edges and desert and includes several vectors of animal trypanosomiasis including *G. moristans*, *G. pallidipus* and *G. austini* [15, 16]. Of the three tsetse flies, the savannah and

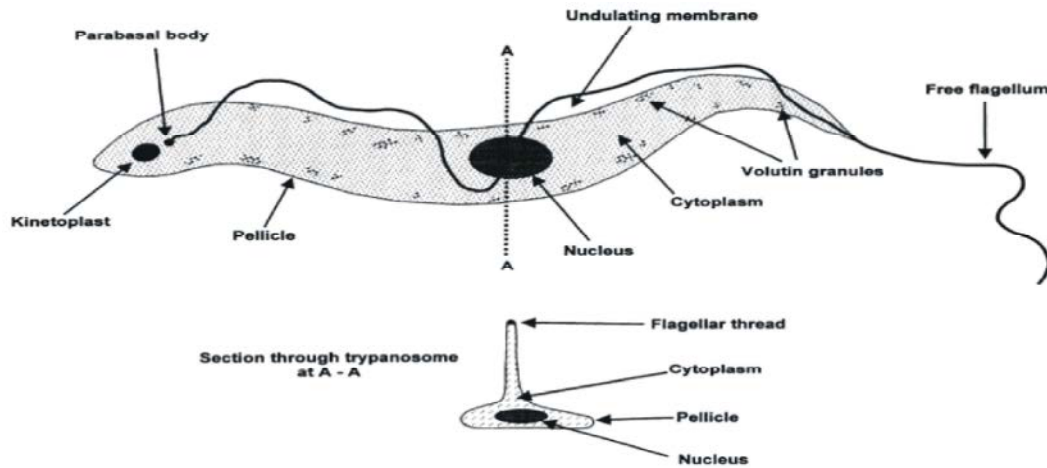


Fig. 1: Typical feature of trypanosomes, Source: [11]

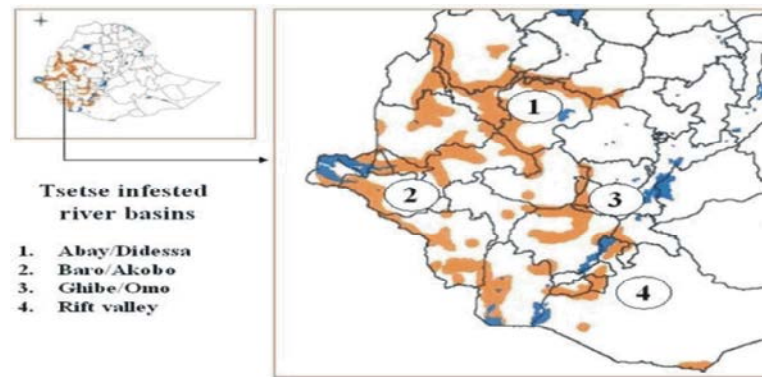


Fig. 2: Tsetse Distribution in Ethiopia, Source: [5]

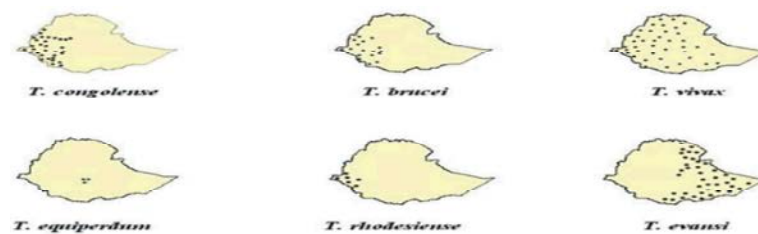


Fig. 3: Distribution of *Trypanosoma* species affecting both Humans and Animals in Ethiopia Source: [5]

riverine are most important since they inhabit areas suitable for grazing and watering. Although the infection rate of *Glossina* with trypanosomiasis is low, ranging from 1-20% of the flies, each is infected for life and their presence in any number makes the rearing of cattle extremely difficult. Biting flies may act as a mechanical vector but their significance in Africa is still undefined [9, 17].

Host: Trypanosomiasis is basically an infection of wild life i.e. wartog, buffalo, bushbuck, kudu and bush pig and may acquire prolonged trypanosome infections. Lives to

exhibit a range of susceptibility to infection, from refractory to highly vulnerable [17, 18]. However, the wild life in Africa generally tolerates infection and often serves as reservoir for human and livestock infective trypanosomes. Monkeys, rats, mice, guinea pigs and rabbits can also be infected with trypanosomes; ruminants, wild equidae, lions, leopards and wild pigs can serve as carriers [19].

Environment: Climate variations have a direct impact on tsetse distribution. The resent drought in West Africa and the ensuring migration of people towards the south

(resulting in the destruction of the vegetation and fauna) have caused a regression in tsetse: *G. tachinoides* has now reduced to the intermediate zone between the forest and the savanna in Cote d'Ivoire and *G. morsitans* sub *morsitans* and *G. tachinoides* are very rare in the Wago Park in Niger and Burkinafaso [20-23].

Life Cycle: The trypanosome has two life stages: trypomastigotes, (flagellated stage found free in blood), amastigotes (non-flagellated intracellular form) and epimastigotes (flagellated form found in the vector) and the vector is then infected with ingesting trypomastigotes during blood meal.

The life cycle of trypanosomes within the vector mid gut: the organism transform in to slender procyclic trypomastigote that enter salivary glands. Their transformation takes place into epimastigotes, then subsequently to metacyclic trypomastigote which are inoculated into uninfected susceptible host during feeding [24]. Insects are usually involved in the natural transmission of the African pathogenic trypanosomes. When this is the case, the life cycle has two phases, one in the insect vector and one in the mammalian host. Transmission by insects may be cyclical by tsetse flies, *Glossina* species, or mechanical by other biting flies (but apart from transmitting trypanosomes cyclically, tsetse flies can also act as mechanical vectors). Transmission can be occurred by ingesting the vector, by blood transfusions, by ingesting of infected tissue or milk or transplacentally. When multiplication occurs in the digestive tract and proboscis, so that the new infection is transmitted when feeding, the process is known as anterior station development and the various species of trypanosomes which use this process are often considered as a group, salivaria. All are trypanosomes transmitted by tsetse flies, the main species being *Trypanosoma congolense* (sub genus Nanomonas), *T. vivax* (sub genus Duttonella) and *T. brucei* (sub genus Trypanozoon) [25].

Pathogenesis: Initial replication of trypanosomes is at the site of inoculation in the skin; this causes a swelling and a sore (chancre). Trypanosomes then spread to the lymph nodes and blood and continue to replicate. *Trypanosoma congolense* localizes in the endothelial cells of small blood vessels and capillaries. *Trypanosoma brucei* and *T. vivax* localize in tissue. Antibody developed to the glycoprotein coat of the trypanosome kills the trypanosome and results in the development of immune complexes. When metacyclic trypomastigotes are introduced subcutaneously and multiply, in 2-3 days there is itching,

swelling, pain and redness and the earliest sign of generalized infection is fever [26]. The degree of virulence of trypanosomes has been classified as: extreme death within a week; high death within a month; moderate death within several months; low chronic infection and negligible or absence of clinical signs [24].

Generally, pathogenesis of trypanosomiasis may be considered under localized swellings (chancre), lymphadenopathys, anaemia and tissue damage [5]. The number of hematogeneous parasites is highest in animals with a dual infection by *T. congolense* and *T. brucei*, in which the clinical diagnosis of trypanosomiasis may be confirmed by inoculating blood or CSF specimen into laboratory mice and observing the recipients for parasitaemia with direct dark field examination of the patient's blood [3]. Nagana in most species is a progressive, but not always fatal disease and the main feature are anemia, tissue damage and immune suppression and their behavior there after depends largely on the species of trypanosome transmitted and the host [1]. The animals are markedly show anemia, emaciation, anasarca and enlargement of liver, spleen and lymph nodes in cattle [27].

Clinical Signs: There are no pathognomonic signs that would help in pinpointing a diagnosis. The general clinical picture is determined by the level of tsetse challenges, the species and strain of the trypanosome and the breed and management of the host [1]. In addition to this the clinical picture of cattle suffering from nagana is influenced by several factors, namely breed and health status of cattle infected, pathogenicity of infecting trypanosomes, duration of exposure to infection and level of tsetse fly challenges, which in itself is dictated by several factors. *Trypanosoma vivax* infections in cattle in West Africa are wide spread and commonly produced an acute, rapidly fatal disease in which affected cattle die during the initiation phase of fluctuating parasitaemia and fever [12].

In ruminants, the major signs are anaemia, generalised enlargement of the superficial lymph glands. Lethargy and progressive loss of bodily condition, fever and loss of appetite occur intermittently during parasitaemic peaks, the latter becoming marked in the terminal stages of the disease. Typically, the disease is chronic extending over several months and usually terminates fatally if untreated. In the terminal stages animals become extremely weak, the lymph nodes are reduced in size and there is often a jugular pulse. Death is associated with congestive heart failure due to anemia and myocarditis [13].

Diagnosis: Diagnosis of trypanosomal infection is performed based on clinical signs and on the demonstration of the parasites by direct or indirect methods. The clinical signs of animal trypanosomosis are indicative but are not sufficiently pathogenomonic and diagnosis must be confirmed by laboratory methods [28]. In tsetse infested areas of Africa, Nangana is well recognized and diagnosis is often based on a history of chronic wasting conditions of cattle in contract with the tsetse fly. Differential diagnosis are babesiosis, anaplasmosis, helminthosis and any conditions that cause anemia and emaciation, notably malnutrition [12].

Direct Methods (Parasitological Tests): The direct methods include: examination of wet blood films, Giemsa stained thick and thin fixed films, haematocrit centrifuge technique and animal inoculation. Confirmation of clinical diagnosis depends on the demonstration of trypanosomes in the blood. Occasionally, when the parasitaemia is massive it is possible to detect motile trypanosomes in fresh films of blood and microscopic examination of stained thin and thick blood smears. More sensitive techniques utilize centrifugation in a microhaematocrit tube followed by microscopic examination of the interface between the Buffy coat and the plasma [13]. Microscopic examination of wet blood films must be done at the time of sampling and cannot be used to identify different species of trypanosomiasis. Although, these techniques are not particularly sensitive and may not detect animals with low parasitaemia, such as those suffering chronic disease [12].

Serological Test: Serological test is the detection of humeral antibodies to trypanosome antigens which includes complement fixation (CFT), Precipitation and indirect haemagglutination. These are not being applied in large scale survey. More recently, indirect fluorescent antibody techniques (IFAT), ELISA and agglutination tests have been employed [29]. Both tests have high sensitivity and genus specificity, but their specificity is generally low. Antibodies persist on average 3-4 months after curative treatment or self-cure, but may last up to 13 mouths [16].

These conventional techniques of microscopic examination for the presence of trypanosomes are still widely used, but newer and far more sensitive methods are beginning to supplement them. The antigen-detecting enzyme-linked immunosorbent assay is extremely sensitive for the detection of trypanosomiasis in cattle

and goats and species-specific DNA probes have been shown to detect simultaneous infection of cattle with *T. vivax* [30].

Molecular Test: Polymerase chain reaction (PCR) with amplification of DNA samples has been developed as a diagnostic test for a number of parasites both in tsetse and cattle using PCR and DNA probes. It is highly specific and more sensitive than direct identification but in an evaluation of PCR for detecting *T. vivax* in cattle, sensitivity was not greater than direct technique and give false negative result when parasitaemia is high. In PCR technique it is possible to identify parasites at subgenus, species and subspecies level. False negative frequently occurs when cattle chronically infected, or when primers do not recognize all isolates of a particular trypanosome species [19].

Treatment and Control: Because of the phenomenon of antigenic variation, no vaccine has against trypanosomiasis and is unlikely to in the foreseeable future. In a fried, this leaves tsetse control as the main method of prevention [12]. The number of trypanocidal drugs available for treating and preventing infections in endemic areas is limited and about 6 million doses are administered yearly in Africa. Ideally, each country or region establishes a group of sanative drugs which are to be used only as a break in course of one of the more common drugs [1]. Treatment against trypanosomiasis, in order to be effective, should be given early in the disease during the initial phase of fluctuating parasitaemia. As no new drugs have been developed against the disease for nearly 30 years and some has been withdrawn because of resistance, treatment is new essentially to two compounds, diminazene aceturate and homidium (either chloride or bromide). However, resistance has been recorded against both drugs and undoubtedly will be an increasing the problem [12].

Control Options of Trypanosomiasis: Several approaches to fly control have been used with varying degrees of success. Discriminative bush clearing, extensively used in early tsetse fly eradication campaigns, has been locally useful because it eliminates the breeding places of the tsetse. But, to be completely effective, bush clearing requires ecologically unacceptable destruction of vast areas of brush and forest. It is still a useful procedure when used locally in conjunction with other control methods. Game elimination and thus elimination of the

Table 1: Regimen for treatment of trypanosomiasis to minimize development of resistance

Tsetse challenge	Drug alternative	
	Homidium	Diminazene
Very high	6 months	1 year
High	1 year	1 year
Medium	2 year	1 year
Low	As long as possible	1 year

Source: [12].

Table 2: Current techniques to control animal trypanosomiasis

Target	Technique
Vector	<ul style="list-style-type: none"> • Insecticides • Ground or aerial spraying • Pour-on • Netting • Screens and traps • Sterile insect technique (SIT)
Trypanosome	<ul style="list-style-type: none"> • Chemotherapy • Chemoprophylaxis
Host	<ul style="list-style-type: none"> • Management • Trypanotolerant breeds

Source: [29].

main source of blood meals for the tsetse, was used in early eradication campaigns. Application of the sterile male technique (as used in screwworm eradication in the United States) received considerable attention in the 1980's [31]. The control of trypanosomiasis in livestock can be directed against the parasite, the vector and host (Table 2)

Vector Control: Current vector control interventions involve the use of insecticides either through sequential aerosol spraying technique (SAT); ground spraying; insecticide-treated targets or insecticide-treated animals – live baits; the use of other-baited traps or screens and the sterile insect technique (SIT) [25]. Breeding of the male flies have been overcome and field trials have been done in both East and West Africa to determine the effectiveness of this approach in vector control. In limited trials, this procedure has reduced fly populations. Ground and aerial spraying with insecticides and the use of synthetic pyrethroids on cattle have lowered fly densities in some areas, but widespread use would require considerable international cooperation and expense. Widespread application of insecticide has the tremendous disadvantage of eradicating many other arthropods, several of which are desirable. The recent introduction of

odor-baited targets impregnated with insecticides is proving promising as a means of reducing the tsetse fly [31].

Tsetse flies are the main vectors of animal trypanosomiasis, but some trypanosomes can also be transmitted mechanically by biting flies such as tabanids and *Stomoxys* spp. Large areas can be cleared from tsetse flies by using ground and/or aerial spraying of insecticides [33]. Ground spraying uses residual insecticides (e.g. Dichloro Diphenyl Trichloro Ethane, dieldrin, endosulfan) which target the tsetse resting sites. Because of the negative effects on the environment these persistent insecticides are more and more replaced by the less toxic synthetic pyrethroids. These products are also used for aerial spraying with fixed wing aircrafts i.e. the sequential aerosol technique (SAT) [34].

Nowadays many African farmers use pour-on insecticides because they can be easily and rapidly applied without any sophisticated equipment. Furthermore, the insecticides kill also biting flies and ticks resulting in less nuisance for the animals and higher productivity [35]. Applications can be restricted to the preferred biting sites of tsetse flies allowing a reduction of up to 90% of the amount of insecticide needed. Consequently, this kind of treatment reduces the cost to less than 1 US\$ per head of cattle per year [36]. A large variety of traps and targets (impregnated with pyrethroid insecticides) has been developed to attract and kill tsetse flies. Especially for the savannah tsetse species the efficacy of these traps/targets can be improved by using odour attractants (such as octenol and phenols) [37]. At a density of 1 to 4 targets per square km, certain tsetse fly populations can be suppressed to low numbers in a short time period. Although this technology is not sophisticated and environment friendly, it is labour intensive and too expensive for most African peasants [36].

Sterile Insect Technique (SIT) has been used successfully in several African countries, i.e. Botswana, where the Okavango delta was cleared from tsetse flies without negative impact on the environment. The SIT consists of the release of irradiated sterile male flies at a proportion of at least 10 sterile to one wild male so that they are able to compete with the wild male flies. When a sterile male mates with a virgin female fly, this results in no offspring because female tsetse usually mates only once in their life. SIT is often needed for the final eradication of tsetse flies. It is a very expensive technique because mass-rearing of tsetse flies is necessary to provide huge amounts of sterile males which have to be



Fig. 4: Morphology of tsetse fly, Source: [32]



Spraying (A)



Aerial spraying (B)

Fig. 5: Ground spraying with portable prepressurized Source [6]



Fig. 6: Targets (impregnated with pyrethroid insecticides)
Source: Bahr Dar regional laboratory, 2011

released (preferably aurally) on a weekly basis for a period of 15 to 18 months. SIT is only effective when the population density of the target flies is very low which implies prior suppression of the flies using other techniques [34].

Control of the Parasites: This consists of the use of trypanocidal drugs on infected animals. The method aims first at limiting losses caused by the disease and second at eliminating trypanosome reservoirs. Thus, detection and treatment of infected animals can be considered to be both a curative and a prophylactic procedure [32].

Chemotherapy: A limited number of drugs are available to treat animal trypanosomiasis. For the treatment of cattle three products are on the market since more than 50 years. Diminazene aceturate has curative properties whereas isometamidium chloride and the homidium salts (ethidium and novidium) have both curative and prophylactic activities. Although ethidium is mutagenic and should be

withdrawn from the market, it is still widely used in East Africa [33].

Currently the treatment of affected animals with trypanocidal drugs still remains the most frequently applied measure to control trypanosomiasis. Treatment is mainly carried out by the livestock owners themselves without any supervision by veterinary personnel. It has been observed that under dosing occurs very frequently, which is an important risk factor for the development of drug resistance [38].

Chemoprophylaxis: The use of drugs for the prevention and treatment of trypanosomiasis has been important for many decades, but the rapidity with which the trypanosomes have developed resistance to each drug introduced has tremendously complicated this approach to controlling the disease. Infections in cattle, sheep and goats can give protection for up to 6 months. The most widely used of the newer chemoprophylactic drugs (and also the least expensive) is isometamidium chloride [40].

Table 3: List of some of the curative trypanocidal drugs for the control of trypanosomiasis

Drugs	Synonyms	Dose(mg/kg)	Host	Indications
Diminazen acetate	Berenil	3.5 (IM)	Cattle	<i>T. vivax</i> <i>T. congolense</i> <i>T. brucei</i>
Quinapyramine sulphate	Antrycid Sulphate	2.2-4.4 (SC)	Cattle	<i>T. vivax</i> <i>T. congolense</i> <i>T. brucei</i>
Homidium chloride	Ethidine	1-2 (IM)	Cattle	<i>T. vivax</i> <i>T. congolense</i> <i>T. brucei</i>
Isometamidium chloride	Trypamidium	0.25-1 (IM)	cattle	<i>T. vivax</i> <i>T. congolense</i> <i>T. brucei</i>

Source: [39]

Table 4: List of some of the prophylactic drugs used for control of trypanosomiasis in cattle

Drugs	Synonyms	Dosage(mg/kg)	Host	Indications
Quinapyramine sulphate	Antrycidal (prosalt)	2-4 (SC)	Cattle	<i>T. vivax</i> <i>T. congolense</i> <i>T. brucei</i>
Isometamidium chloride	Trypamidine	1-2 (IM)	Cattle	<i>T. vivax</i> <i>T. congolense</i> <i>T. brucei</i>

Source: [39]

Selection of Trypanotolerant Breed: It has long been recognized that certain breeds of African cattle are considerably more resistant to African trypanosomiasis than others. This is especially true of the West African short-horned cattle (Muturu, Baoule, Laguna, Samba and Dahomey) and the N'Dama, which is also of West Africa. These cattle have existed in the region for over 5, 000 years. Susceptibility studies have shown the N'Dama to be the most resistant breed followed by the smaller West African short-horned cattle, but the large and more recently introduced zebu is the most susceptible [41]. The mechanisms of trypanotolerance have been extensively studied and it is now well established that trypanotolerance has a genetic basis [42]. The horns of the N'dama cattle are lyre-shaped and the tawny coat is characteristic. The other breed, according to the region in which it is found, is called Baoule, Laguna, Samba, Muturu, Dahomey are smaller than the N'dama and more powerfully built; their coats are usually black or piebald black and they have short pointed horns [32]

Prevalence of Tsetse Transmitted Trypanosomiasis:

Some of the factors that affect the prevalence of trypanosomiasis includes; animal breed, type of management, season of the year and the type of vegetation. Some riverine and forest species of tsetse fly are poorer vectors of trypanosomes than the savanna species. It is also known that nomadism tends to expose animals to high tsetse challenge and hence trypanosome infection [43].

Prevalence of *T. vivax* infections in cattle has been noted during rainy season attributed to higher density of tsetse flies and/or the abundant presence of mechanical vectors, such as tabanids and *Stomoxys* spp [22]. It was also reported that *T. vivax* is found in the entire country except in the highlands, which are 2500 meters above sea level as the acyclical transmission of the disease is effected by means of blood sucking flies which include

Glossina, *Tabanus* and *Stomoxys* species. The prevalence of trypanosomiasis infection was compared with different altitude, however, when the different spp. was considered. *T. congolense* infection was observed only in lowland areas of the district [44] (Table7).

Economic Importance of Tsetse Transmitted Animal

Trypanosomiasis: Trypanosomiasis can cause direct and indirect loss. It has direct impact on livestock productivity, reducing meat and milk off take by 20%, calving rate by 20% increase, calf mortality by 20%, decreases both lambing and kidding rates in sheep and goat and livestock management especially the number of livestock kept by farmers, the breed and species composition of the livestock herd, the way the livestock are grazed, cost of trypanocidal drugs and cost of insecticides [45]. Trypanosomiasis also has direct impacts on human settlement in a considerable part of Sub-Saharan Africa [46]. The main factors leading to changes in land use and land cover is migration. In an outbreak of trypanosomiasis that cause large losses in livestock numbers, people leave tsetse affected area to controlled area there by changes settlement patterns and increase population density of the area (Swallow, 2000). In the case of human African trypanosomiasis, it reduces productivity of the people with the disease, family members who care for the ill and the rural residents fear that they might contact the disease becomes burden to the family and the resources [7].

Indirect impact of trypanosomiasis mostly lies on crop production; through the availability and cost of animals that provide traction power [45] Animal trypanosomiasis reduces work efficiency of oxen for cultivation, reducing access to animal traction or discourages the introduction of drought animals in to crop farming. Evaluation on effect of trypanosomiasis incidence on the productivity of oxen used for traction show that relative inefficiency in the high risk area was

Table 5: The prevalence of trypanosomosis and its association with various risk factors in Asosa district (Ethiopia)

Risk factor	No. examined	No. positive	% positive \pm 95% CI	2 (P value)
Pas		15	26.8 \pm 11.6	4.460
Amba-4	56	16	24.2 \pm 10.3	
Amba-12	66	31	34.8 \pm 9.9	
Asosa	89	16	32.7 \pm 13.1	
Megele-29	49			
Amba-8	61	13	12.0 \pm 8.2	
Amba-3	63	17	27.0 \pm 11	
Sex				
Male	185	51	13.3 \pm 4.	0.055 (P>0.05)
Female	199	57	14.8 \pm 4.9	
Body condition				
Good	268	71	1.8 \pm 1.6	2.857 (P<0.05)
Poor	116	116	26.3 \pm 8.0	
Total	Total 384	108	28.1 \pm 4.5	

Source: [44]

Table 6: The prevalence of single and mixed trypanosome infection in Asosa district (Ethiopia)

Species	No. positive	% positive \pm 95% CI	χ^2 (P-value)
<i>T. congolense</i>	72	18.8 \pm 3.9	3.352 (P=0.000)
Mixed <i>T. congolense</i> and <i>T. vivax</i>	10	5.5 \pm 2.3	
<i>T. vivax</i>	21	2.6 \pm 1.6	
<i>T. brucei</i>	5	1.3 \pm 1.1	
Total	108	28.1 \pm 4.5	

Source:[44]

Table 7: The prevalence of trypanosoma spp. at different altitude in Wemberma (Amhara Region)

Altitude	Species of trypanosome		Total prevalence	P value
	<i>T. vivax</i>	<i>T. congolense</i>		
Midland	6(4.5%)	0	4.72%	O=1070
Lowland	18(7%)	6(2.3%)	11.72%	
	24(6.7%)	6(1.5%)	7.81%	

Table 8: Estimated costs of tsetse eradication or control

Technique	Costs (US\$) per km ²	Objective and location
Ground spraying	265 – 315	Eradication (Zimbabwe, Zambia)
Targets	220 – 290	Flat terrain
Aerial spraying	345 – 535	Various locations
Cattle treatment	50 – 120	Pour-on, 15 cattle/km ²
	60	Pour-on, 44 cattle/km ²
Linear km of barrier using targets	2000 – 1600	Zimbabwe, Zambia
? Barrier establishment	265 – 275	Eradication, Botswana
? Annual barrier maintenance	800	Eradication, Eastern Africa
Aerial spraying (SAT)	250-400	Eradication, West Africa
Sterile Insect Technique (SIT) Low density monopyramidal	26	Annual cost of control, Ivory Coast

Source: [45]

38% less efficient than oxen in the low risk area. Additional traction capacity allows farmers expand the area that they cultivate, increase yield of existing crops; grow different mix of crops or allocated labour, land and fertilizer more efficiently [35]. Estimating the cost of controlling trypanosomiasis is no easier than the other estimates of the cost of the disease, as some

expenses cannot be accurately quantified. The figures available are not always comparable because they are calculated according to criteria that vary with each country. The estimated costs of various methods to control tsetse flies depend heavily on the terrain conditions and on the final objective (control or eradication) [47].

CONCLUSION AND RECOMMENDATIONS

Tsetse transmitted African animal trypanosomosis have been considered as major obstacle for sustainable development of agriculture in sub-Saharan regions that have greatest opportunity for expanded livestock production including Ethiopia. In trypanosomiasis endemic areas of sub-Saharan regions, the majority control measures have been primarily protecting the animal using suitable chemotherapeutic and chemoprophylactic drugs. Despite the repeated use of trypanocidal to control trypanosomosis emergency of multiple drug resistance created and jeopardize the prophylactic and /or therapeutic activity of available trypanocidal drugs. Based on the above conclusion the following recommendations are forwarded:

- In trypanosomiasis endemic areas both the vector and parasite control methods involving an integrated approach should be applied.
- All veterinarians' animal health technicians should aware the public on risk of trypanocidal drug resistance.
- Detail laboratory and field studies on extents of trypanocidal resistant in Ethiopia should be conducted.

REFERENCES

1. Radostitis, M.O., C. Gay, D.C. Blood and K.W. Hinch Elieff, 2007. Veterinary medicine, A Text Book of the Disease of Cattle, Sheep, Pigs, Goats and Horses. 10th ed. London: Sounders. 1531-1556.
2. Seifert, H.S.H., 1996. Tropical Animal Health. Kluwer Academic Publishers. Dorrech, Bonston, London, pp: 53-260.
3. Smith, B.P.D., 2009. Large animal internal medicine. 4th ed. St. Louis, Mosby Elsevier, pp: 324-335.
4. Dutoit, R., 1984. Trypanosomosis in Zululand and the control tsetse by chemical means. Onderstepoort J.vet. Res., 26: 317-387.
5. Abebe, G., 2005. Trypanosomosis in Ethiopia. Ethiopian Journal of Biological Science, 4: 75-121
6. Mulligan, H.W., 1970. The African Trypanosomiasis. 1st ed. London. George Allen and Unwin Ltd, pp: 950.
7. WHO, 1998. Control and surveillance of African trypanosomiasis. A report of WHO expert committee. WHO Technical Report Series, pp: 881.
8. Kahn, H.C. and S. Line, 2005. The Merck Veterinary manual. 9th ed. Philadelphia. National publishing Inc, pp: 32-34.
9. Bowman, D.D., R.C. Lynn, M.L.Emberhara and A. Alcoraz, 2003. Parasitology for veterinarian. 8th ed. Soundes, pp: 83-84.
10. Fisher, M.S. and D.R.R. San, 1989. Manual of Tropical Parasitology. 1st ed. C.A.B. I, UK, pp: 136-178.
11. Uilenberg, G., 1998. A field guide for diagnosis, treatment and African animal trypanosomosis. Adapted from the original edition by. W.P. Boyd, FAO, Rome. Under trypanosomiasis risk. Journal of Agricultural Science, 105: 147-166.
12. Andrews, A.H., R.W. Blowey, H. Boyd and R.G. Eddy, 2003. Bovine Medicine diseases and Husbandary of cattle. 2nd ed. Black Well Science, pp: 755-759.
13. Urquhart, G.M., J.L. Armour, J.L. Duncan, A.M. Durn and F.W. Jennings, 1996. Veterinary Parasitology. 2nd ed. Black well science. UK, pp: 214-218.
14. Mira Shah, F. and R. Ralphsay, 1989. Manual of tropical Veterinary Parasitology. 1st ed. England, C.A.B., pp: 181-278.
15. Ouma, E.M., 2010. Managment of trypanocidal drug resistance in cattle identified chemoresistant hot spots in the administrative Districts of sikkaso, south east Mali. MSc thesis. Free University, Berlin.
16. Mamoudou, A., V. Delespoux, A.V. Chepnd, Z. Hachmou, J.P. Rikaye and S. Geerts, 2009. Assesment of the occurrence of trypanocidal drug resistance in trypanosomes of naturally infected cattle in the Adamoua region of Cameroon using the standared mouse test and molecular tool. Acta Tropica, 106: 115-118.
17. Taylor, A.M., L.R. Coop and L.R. Wall, 2007. Veterinary Parasitology. 3rd ed. Black Well Publishing, pp: 42-43.
18. Blood, D.C., O.M. Radostits and C.C. Gay, 1989. Veterinary medicine. A Text Book of the Disease of Cattle, Sheep, Pig, Goat and Horses. 7th ed. Biallier Tindall, pp: 1209-1226.
19. OIE, 2008. Manual of standards for diagnostic test and vaccines for terrestrial animals. 6th ed. Paris, pp: 831-1008.
20. Clair, M. and G. Lamarque, 1984. Repartition des glossines dans le nord de la cote d'Ivoire. Revue Elev.med.vet. Pay Trops, pp: 60-83.
21. Clair, M., 1986. Glosines, et trypanosomes au Niger In Ellevage et potentialities pastorals saheilennes. Synthesis cartographics. Atlas du Niger C.T.A.wageningers, pays Bas. Maisons-A lfort, IEMVT. CIRAD. 170-172.

22. Cuglovici, D.A., D.C. Bartholomeu, J.L. Reis-Cunha, A.U. Carvalho and M.F.B. Ribeiro, 2010: Epidemiologic aspects of an outbreak of *Trypanosoma vivax* in a dairy cattle herd in Minas Gerais, State, Brazil, pp: 320-326.
23. Katundo, K.M., 1984. Revision of second edition tsetse distribution Maps. Insect. Sci. Applic, pp: 381-384.
24. Bengaly, Z., I. Sidibe, H. Boly, L. Sawadogo and M. Desquesnes, 2002. Comparative Pathogenesis of three genetical distinct *Trypanosoma congolense*-Types inbred. Veterinary Parasitology Journal, 105: 111-118.
25. WHO, 2013. Human African trypanosomiasis, Eradication of tsetse in Zanzibar, pp: 1-2.
26. Morrison, W.I., M. Murray, D.D. Whitelaw and P.D. Sayer, 1983. Pathology of infection with *Trypanosome brucei* disease syndromes in dog and cattle resulting from severe tissue damage. London, Kluwer Academic, pp: 103-119.
27. Samual, W.M., J.J. Margo and A.A. Kocan, 2001. Parasitic Disease of Wild Mammals. 4th ed. Black well publishing company, USA, pp: 520-522.
28. Elnarsi, O.M., 2005. Prevalence and ranking of bovine trypanosomiasis in the Unity State by participatory epidemiological, clinical and laboratory testing. MSc thesis, University of Khartoum, Department of Veterinary Medicine, Sudan.
29. Greets and Holmes, P.H., 1998. Drug management and parasite resistance in anial trypanosomiasis in Africa. Position paper-program against African trypanosomiasis (PAAT), Technical series, FAO. Rome, Italy, pp: 22.
30. Masake, R.A. and V.M. Nantulya, 1991. Sensitivity of an antigen-detecting enzyme immunoassay for diagnosis of *Tyrypanosoma congolense* infections in goats and cattle. J. Parasitol., 77: 231-236.
31. AAT, 2004. Veterinary science/microbiology, university of Arizona, tuson AZ, 12.
32. Finelle, P., 1964: Animal Health Officer, Animal Production and Health Division, Fao, Rome, 61: 373-384.
33. Geerts, S., V. Delespaulx and P. Van Den Bossche, 2010. Drug resistance in trypanosomes of goats and cattle. J. Parasitol., 77: 231-236.
34. Feldman, U., 2004. Guide line for the raring of tsetse flies using the membrane feeding. In J.B.R. Oching-oder, ed. Techniques of insects raring for the development of integrated pest and vector management strategies. Nairobi, ICIPE science press, 1: 450-463.
35. Leek, S.G.A., 1995. Tsetse Biology and Ecology, Their role in Epidemiology and control of trypanosomiasis. CABI publishers. New York, NY, USA, pp: 5-7.
36. Torr, S.J., D.R. Hall and J.L. Smith, 2007. Response of tsetse flies to natural and synthetic oudour. Bull. Ent. Res., pp: 158-167.
37. Getachew, A., 2005. Trypanosomiasis in Ethiopia. Ethiopian Journal of Biological Science, 4: 75-121.
38. Delespaulx, V., D. Geysen, P.V.D. Bossche and S. Geart, 2008. Molecular tool for rapid detection of drug resistance in animal trypanosomosis. Trend in Parasitology, 24: 236-242.
39. Abebe, G., 1983. Trypanosome and Trypanosomiasis teaching material. Faculty of Veterinary Medicine, Debre Zeit, Ethiopia, pp: 19-21.
40. Ogunyemi, O. and A.A. Ilembade, 1989. Prophylaxis of African Trypanosmiasis; A review of some factors that may influence the duration of Isometamidium chloride prophylaxis. Vet. Bul., 59: 1-4.
41. Murray, M., W.I. Morrison, P.K. Murray, D.J. Clifford and J.C.M. Trail, 1979. Trypanotolerance. A review. World Animal Review, 31: 2-12.
42. Murray, M., J.C.M. Trail, C.E. Davis and S.J. Black, 1984. Genetic Resistance to African trypanosomiasis. J. Inf. Dis., 149: 311-319.
43. Ikeda, B.O., L. Reynolds, A.O. Ogunsanmi, M.K. Fawunmi, J.O. Ekwuruke and V.O. Taiwo, 1986. The epizootiology of bovine trypanosomiasis in the derived savanna zone of Nigeria. A preliminary report. Proceedings of the 19th Meeting of the ISCTRC/OAU, Loma, pp: 1- 6.
44. Shimelis, M., A. Mekonen and F. Abebe, 2011. Study on the prevalence of major trypanosome affecting bovine in the infested Assosa, Benshangule gumze, Western Ethiopia, Jima University, pp: 334.
45. Swallow, B.M. and M. Woudyalew, 2000. Evaluating willingness to contribute to a local public good. Application of contingent evaluation to tsetse control in Ethiopia. Ecological-Economics- Amsterdam, 11: 153-161.
46. Takele, A. and G. Abebe, 1985. A survey of trypanosomiasis and its economic impact in GamuGofa region. DVM thesis, Addis Ababa University, FVM, Debre Zeit, Ethiopia.
47. FAO, 2000. A field guide for diagnosis, treatment and prevention of African animal trypanosomiasis. 2nd ed. FAO, ROME, pp: 27-34.