

A Review On Animal African Trypanosomosis And Its Control

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Summary: Animal African trypanosomosis (AAT), a disease caused by protozoan parasites of the genus *Trypanosoma*, is a major constraint on livestock and agricultural production in African countries. The disease threatens the survival of animals and reduces their production. The aim of this review is to highlight animal trypanosomosis and different methods used to control AAT. Because of the phenomenon of antigenic variation, no vaccine is available. Over the past century numerous methods of control have been developed but most heavily relied on are the trypanocidal drugs and this has led to an increasing problem with resistance in the target organisms. Current methods to control the disease, in the absence of a vaccine, directed against the parasite, the vector or the host. Unfortunately, most of these methods have also disadvantages and none has proved to be ideal. A combination of control methods is being used in order to improve the effectiveness and efficiency of control. Hence there is a great need to strengthen these integrated approaches for more effective control of trypanosomosis.

[Mesafint mitiku, Ashenafi Assefa and Askale abrhaley. **A Review On Animal African Trypanosomosis And Its Control.** *Nat Sci* 2017;15(5):101-110]. ISSN 1545-0740 (print); ISSN 2375-7167 (online). <http://www.sciencepub.net/nature>. 13. doi:[10.7537/marsnsj150517.13](https://doi.org/10.7537/marsnsj150517.13).

Key words: control, protozoa, trypanosomosis, trypanocidal drugs,

1. Introduction

Animal African Trypanosomosis (AAT) affects a wide range of hosts and is caused by several trypanosome species namely; *Trypanosoma congolense*, *Trypanosoma vivax*, *Trypanosoma evansi*, *Trypanosoma simiae* and *Trypanosoma brucei brucei*. The AAT is found mainly in those regions of Africa where its biological vector, the tsetse fly, exists. The tsetse fly infested region known as the tsetse belt covers 37 African countries including Ethiopia with an approximation of 10 million square kilometers (O'Gorman *et al.*, 2006). AAT is the cause of death of over 3 million animals per annum with 50 million animals at risk of infection (Chitanga *et al.*, 2011). The disease is thus a major constraint to livestock productivity, such as reduced milk output, reduced live animal output and reduced efficiency of animals used for cultivation, which has a significant impact on the livelihood of millions of people in African developing countries, costing several billion US dollars each year (Chitanga *et al.*, 2011).

Due to environmental changes (*i.e.* land use changes due to the increasing population and deforestation) the epidemiology of animal trypanosomosis is changing (Van den Bossche *et al.*, 2001; 2010). Because of the gradual encroachment of people and cattle, the latter are playing an increasing role as blood source for tsetse flies. This results in a change from a dominant sylvatic to a domestic cycle and it has been nicely shown that bovine trypanosomes in the latter cycle are less virulent and have less impact on production (Masumu *et al.*, 2006;

Van den Bossche *et al.*, 2011). However, although the pathogenicity of trypanosomes in certain endemic areas might decrease, this is not everywhere the case and livestock owners very often consider trypanosomosis as one of the most important disease problems.

It is nearly 100 years of tsetse and trypanosomosis control efforts, but today the problem is still far from being solved. In most African countries, tsetse distribution has remained the same and indeed in some areas, the fly has spread to new areas. The incidence of both animal and human trypanosomosis remained high with occasional endemic outbreak (Adamu *et al.*, 2011).

The fight against the disease is managed by the control of the vector, the parasite and host management. A combination of these methods in an integrated phase approach can effectively advance the control of tsetse and trypanosomosis. However, in poor rural communities, which are mostly affected by the disease, control is mainly relying on the use of trypanocidal drugs (Chitanga *et al.*, 2011).

Therefore, the objective of this seminar paper is:

➤ To review on animal trypanosomosis and the current methods used to control AAT.

2. Animal African Trypanosomosis

2.1 Definition and etiology

Animal African Trypanosomosis (AAT) is also called Nagana, Surra, Dourine, or tsetse fly disease depending on the host or the region (Lopes *et al.*, 2010). The term Nagana is used to describe diseases

caused by animal trypanosomes (almost all animal species can be infected) of typically African origin (*Trypanosoma vivax*, *T. congolense*, *T. simiae* and *T. brucei brucei*), which are transmitted by tsetse fly. Surra (*T. evansi*) is a disease of camels, horses and dogs); which is transmitted mechanically by biting flies. Dourine is a contagious venereal trypanosomiasis of Equidae that is caused by *T. equiperdum* (Uilenberg *et al.*, 2010). *T. vivax*, *T. congolense*, *T. b. brucei*, *T. evansi* and *T. simiae* are the main species responsible for AAT affecting virtually all domestic mammals. *T. vivax* and *T. congolense* are the main pathogens of cattle (Radostits *et al.*, 2007).

Trypanosoma congolense is the major cause of AAT in livestock (McDermott *et al.*, 2003). This trypanosome is a major cause of the disease in cattle in West Africa. Sheep, goats, horses, and pigs may also be seriously affected. In domestic dogs, chronic infection often results in a carrier state. *T. vivax* is a heteroxenous parasite that can be transmitted cyclically by tsetse flies (*Glossina*) and also mechanically by hematophagous flies (Taylor *et al.*, 2007)). Its hosts include; domestic animals like the bovines, ovines where it is pathogenic and at times causes a hemorrhagic disease. The severity of *T. vivax* infection varies with host resistance and the parasite strains (Thumbi *et al.*, 2010).

Trypanosoma brucei brucei is widely distributed within the tsetse-belt region due to the distribution of the *Glossina* vector (Barrett *et al.*, 2003). *T. b. brucei* resides in the subgenus *Trypanozoon*. Horses, dogs, cats, camels and pigs are very susceptible to *T. b. brucei* infection. The parasite is restricted within the sub-Saharan Africa and causes a mild infection in cattle. Unlike the other subspecies of *T. brucei*, *T. b. brucei* is not human infective because it is susceptible to trypanosome lytic factors associated with high

density lipoproteins like the human apolipoprotein L1 in human blood (Vanhamme *et al.*, 2003).

Trypanosoma simiae is tsetse-transmitted and belongs to the subgenus *Nannomonas*. It is not known to cause disease in cattle but is severely pathogenic in pigs, goats, camels and equines (Kaufmann, 1996). *T. evansi* belongs to the *Trypanozoon* group and it is the causative agent of Surra which affects camels, horses, cattle, pigs, buffaloes and dogs (Baral, 2010).

2.2 Morphology

A sound knowledge of the basic features of the various trypanosomes enables the identification of each species and so the exact cause of the disease. Trypanosomes are classified in the phylum Sarcostigophora, the order Kinetoplastida and the family Trypanosomatidae (Taylor *et al.*, 2007). 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 (Uilenberg, 1998).

Trypanosoma evansi is long slender trypanosomes, with a prominent undulating membrane and long, free flagellum. It is monomorphic and similar to the slender form of *T. b. brucei* with centrally placed nucleus, a small sub-terminal kinetoplast, a well developed undulating membrane, a long free flagellum and a pointed posterior end (Taylor *et al.*, 2007). *Trypanosoma equiperdum* cannot be distinguished morphologically from *Trypanosoma evansi* (Womack *et al.*, 2006).

Trypanosoma vivax and *T. congolense* are identical by having monomorphic form and inconspicuous undulating membrane. But kinetoplast is terminal and posterior end in case of *T. vivax* and medium and marginal in *T. congolense* (Urquhart *et al.*, 1996).

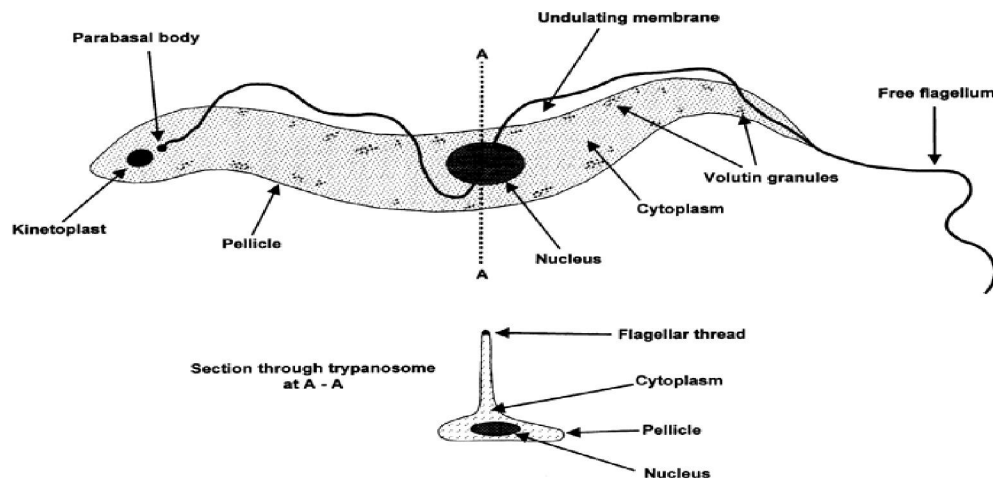


Figure 1: Diagrammatic illustration of the fundamental features of a trypanosome

Source: Uilenberg, 1998

2.3. Epidemiology

The epidemiology of AAT depends on three factors, which are the distribution of the vectors, the virulence of the parasite and the response of the host (Urquhart *et al.*, 1996).

2.3.1 The vectors

The savannah and riverine varieties, from the group of *Glossina* flies, are the most important since they inhabit areas suitable for grazing and watering. Although the infection rate of *Glossina* with trypanosomes is usually low, ranging from 1 to 20% of the flies, each is infected for life, and their presence in any number makes the rearing of cattle, pigs and horses extremely difficult. Biting flies may act as mechanical vectors but their significance in Africa is still undefined (Taylor *et al.*, 2007). However, in Central and South America, *T. vivax* is thought to be transmitted readily by such flies (Radostits *et al.*, 2007). Tsetse fly density is the most variable factor in the transmission of trypanosomosis. Climate affects tsetse abundance *via* one or more of four demographically important rates namely of birth, mortality, immigration and emigration (Rogers, 1991).

2.3.2 The parasites

The parasite virulence, immunogenicity and response to chemotherapeutics are important factors in the epidemiology of trypanosomosis as the trypanosome species occur in a remarkable variety of genotypes. Since parasitaemic animals commonly survive for prolonged periods, there are ample opportunities for fly transmission, especially of *T. b. brucei* and *T. congolense*. In contrast, some strains of *T. vivax* in cattle and *T. simiae* in domestic pigs kill their hosts within 1 – 2 weeks, so that the chances of fly infection are more limited. Perhaps the most important aspect of trypanosomosis which accounts for the persistent parasitaemia is the way in which the parasite evades the immune response of the host through antigenic variation. When an animal is infected with trypanosomes; antibodies against the surface coat are produced. The problem is that these trypanosomes have multiple genes, which code for different surface proteins; this allows organisms with a new coat glycoprotein to escape the immune response. This process is called antigenic variation (Urquhart, 1996; Taylor *et al.*, 2007).

Table 1: Tsetse transmitted animal trypanosomes

Trypanosome species	Animals mainly affected	Major geographic distribution
<i>T. congolense</i>	Cattle, sheep, goats, dogs, pigs, camels, horses, most wild animals	Tsetse region of Africa
<i>T. vivax</i>	Cattle, sheep, camels, goats, horses, various wild animals	Tsetse region of Africa, Central and South America, West Indies
<i>T. b. brucei</i>	All domestic and various wild animals; most severe in dogs, horses and cats	Tsetse region of Africa
<i>T. simiae</i>	Domestic and wild pigs, camels	Tsetse region of Africa

Source: Khan, 2005

2.3.3 The hosts

The mechanisms underlying bovine trypanotolerance remains mostly unknown however, immune response to trypanosomes greatly differs between susceptible and resistant cattle. Whereas the immunity has a genetic basis, the intensity of the tsetse challenge has a strong influence on the degree of tolerance. Trypanotolerance has been defined as the relative capacity of an animal to control the intensity, prevalence and duration of parasitaemia and to limit the pathological effect of the parasites, the most prominent of which is anemia (Uienberg *et al.*, 2010).

The effects of trypanotolerant cattle on trypanosomosis transmission have not been investigated. It might be expected that both the probability of a fly becoming infected from an infected cow, and cows becoming infected from an infected fly would decrease (McDermott and

Coleman, 2001). The fact that the parasite affects not only cattle but also wild animals which constitute the reservoirs of the disease makes the epidemiology of animal trypanosomosis extremely complicated (Uilenberg *et al.*, 2010).

2.4. Transmission

Adult tsetse male and female feed exclusively on vertebrate blood; the immature stages do not feed. Adult flies can become infected with trypanosomes by taking a blood meal from an infected host. Once the infection is established, the flies appear to remain infected for life. These trypanosomes replicate in the tsetse fly and are transmitted through tsetse fly saliva when the fly feeds on an animal. However, cyclical development - the establishment and development of the trypanosomes in the tsetse vectors - is a complex process, involving a series of vector and parasite defense and counter-defense mechanisms (Wellburn

and Maudlin, 1999), and there is evidence that tsetse become harder to infect as they mature. Often therefore, natural populations of tsetse show relatively low infection rates – generally less than 0.1% in the case of human-infective forms, but often as high as 10-15% in the case of cattle – infective forms.

Mechanical transmission can occur through tsetse or other biting flies. In case of *T. vivax*, *Tabanus* species and other biting flies seem to be the primary mechanical vectors outside the tsetse areas, as in Central and South America. Mechanical transmission requires only that blood containing infectious trypanosomes be transferred from one animal to another (Khan, 2005).

2.5. Life cycle

The tsetse fly gets ingests the trypanosome blood form trypanosomes in the blood or lymph while feeding on infected hosts, thereafter the trypanosomes lose their glycoprotein surface coats and in the case of *brucei* and *congolense* become elongated and multiply in the mid gut, are broad with a kinetoplast midway

between nucleus and posterior end before migrating forward to the salivary glands (*brucei*) and proboscis (*congolense*). There they undergo a transformation losing their typical trypomastigote form and acquire an epimastigote form characterized by the fact that the kinetoplast lies just in front of the nucleus. After further multiplication of epimastigote they transform against into small, typically trypomastigote forms with a glycoprotein surface coat. These are the infective forms for the next hosts and are called metacyclic trypomastigote (Mullen, Gary, 2002). The entire process takes at least 2-3 weeks and the metacyclic trypomastigote are inoculated into the new host when the tsetse fly feeds.

With *vivax* a similar process of cyclic development takes place except that it occurs entirely within the proboscis (Radostits, 2007; Uilenberg, 2010). In case of *equiperdum* the organism divides by longitudinal binary fission in various tissue fluids, particularly in subcutaneous urticarial plaques and in the reproductive system (Taylor *et al.*, 2007).

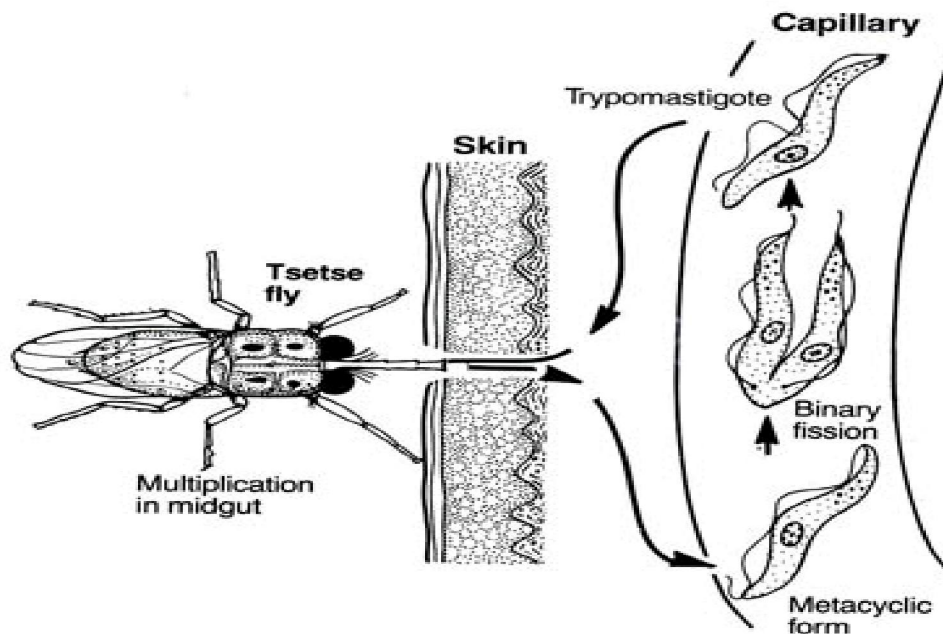


Figure 2: Generalized life cycle of a fly-transmitted trypanosome
Source: Gardiner *et al.*, 1989

2.6. Pathogenesis

Initial replication of trypanosomes happens at the site of inoculation in the skin; this causes a swelling and a sore (chancre). They enter the lymph nodes, then the blood stream, where they divide rapidly by binary fission. *T. congolense* localizes in small blood vessels and capillaries. *T. b. brucei* and *T. vivax* invade tissues and cause tissue damage in several organs (Khan, 2005).

T. congolense and *T. vivax* exert their effect mainly by causing severe anemia and mild to moderate organ damage. The anemia has a complex pathogenesis involving mainly increased erythrophagocytosis, some hemolysis, and dyshemolysis. Very acute infections with *T. vivax* in cattle or *T. simiae* in pigs result in fulminating parasitaemia and disseminated intravascular coagulation with hemorrhages. Such syndromes

resemble a septicemia, and anemia may not be severe (Radostits *et al.*, 2007).

T. b. brucei and, rarely *T. vivax*, have the added capability of escaping from capillaries into interstitial tissues and serous cavities where they continue to multiply. Such infections result in more severe organ damage in horses, sheep, and goats, in addition to anemia. The cerebrospinal fluid is often invaded by *T. b. brucei* alone or mixed with other species or a relapse after an apparently successful treatment (Radostits *et al.*, 2007).

2.7. Clinical sign

Severity of disease varies with species and age of the animal infected and the species of trypanosome involved. The incubation period is usually 1-4 weeks. Clinical signs of tsetse transmitted trypanosomosis, causing acute and chronic forms. Acute forms include fever, parasitaemia and enlarged lymph nodes while chronic form includes intermittent fever, anemia and progressive loss of condition (Khan, 2005).

In cattle, the major signs are anemia, generalized enlargement of the superficial lymph glands, lethargy and progressive loss of body condition. Fever and loss of appetite occur intermittently during parasitaemic peaks, the latter becoming marked in the terminal stages of the disease. As a herd phenomenon, the growth of young animals is stunted, while adults show decreased fertility, and if pregnant, may abort or give birth to weak offspring. 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 (Taylor *et al.*, 2007).

2.8. Diagnosis

The definite diagnosis of trypanosomosis depends on the detection of the parasite which can be accomplished through direct (parasitological) and/or indirect (serological) demonstration of the parasite, clinical signs and molecular diagnosis (William *et al.*, 2000).

2.8.1. Clinical diagnosis

Clinical diagnosis based on an examination of animals that are suspected of being infected with trypanosomosis is often uncertain as the signs are not specific and may resemble another parasitic or infectious disease that results in emaciation. Clinical diagnosis based on the association of symptoms specific to each animal species and to each *trypanosome* species can sometimes lead to a strong presumption of disease, particularly in enzootic areas. However, trypanosomosis should be suspected when an animal in an endemic area is febrile, anemic and in poor condition (Uilenberg *et al.*, 2010).

2.8.2. Parasitological diagnosis

The easiest technique for detection of trypanosomes in peripheral blood is by direct

microscopic examination of blood, either by the wet film method to detect motile trypanosomes or, as stained thick and thin smears, when parasites are identified on the basis of their morphology by light microscopy. The most sensitive rapid method is to examine a wet mount of the Buffy coat area of a packed cell volume (PCV) tube after centrifugation (Khan, 2005). *T. b. brucei* and *T. vivax* are larger and the latter more mobile, they are more seen more easily than *T. congolense*. This method enables tentative identification of the parasite on morphology and motility criteria (Uilenberg *et al.*, 2010). Parasitological tools are continually relied on in the field as they can be carried out directly and give results as well as being practical in resource poor settings (Cox *et al.*, 2010).

2.8.3. Serological diagnosis

Various serological tests such as indirect Fluorescent antibody test (IFAT) and ELISA measure antibody to trypanosomes, but their use is more suitable for herd and area screening than for individual diagnosis. Tests for detection of circulating trypanosome species-specific antigens in peripheral blood are becoming available for both individual and herd diagnosis, although their reliability require further evaluation and standardization (Taylor *et al.*, 2007; Khan, 2005).

2.8.4. Molecular diagnosis

Molecular diagnostic tools have been developed with high sensitivity and specificity (Cox *et al.*, 2005) can detect trypanosomes even in cases of low parasitaemia. Until now molecular techniques were not easily applicable to field conditions owing to the amount of time required to process samples by conventional methods like the phenol chloroform extraction method prior to analysis and the equipments needed. Accurate diagnosis of animal trypanosome is and definitive identification of causative trypanosome species is based on the detection and amplification of nucleic acid (DNA and RNA). The technical potential to achieve their detection and identification of trypanosomes by molecular means should be based on stable parasite, specific genetic characteristics and the parasite that can withstand environmental influences exerted by either the host or vector (Ian Maudlin *et al.*, 2004).

2.8.5. Differential diagnosis

- Anaplasmosis
- Babesiosis
- Theileriosis
- Brucellosis
- Helminthosis
- Malnutrition
- Other haemoparasitoses

3. Control Of Trypanosomosis

The control of trypanosomosis in livestock can be directed against the parasite, the vector or can also involve the host (Geerts, 2011).

Table 2: Current techniques to control animal trypanosomosis

Target	Technique
Vector	BiologicalChemicalMechanical
Parasite	ChemotherapyChemoprophylaxis
Host	ManagementTrypanotolerant breeds

Source: Geerts, 2011

3.1. Methods to control the vector

Tsetse flies are the main vectors of animal trypanosomosis, but some trypanosomes can also be transmitted mechanically by biting flies such as *Tabanus* and *Stomoxys species* (Geerts, 2011). The vector of tsetse fly can be controlled through biological, mechanical or chemical methods.

3.1.1. Biological control

Biological control is the use of living creatures or organisms such as parasites and predators, to control or eradicate other creatures, which are harmful. The most important parasites of tsetse in this regard are those that attack the pupae and the main ones are small flies such as *Mutilla glossinae* (Ahmed, 2015).

The sterile insect technique (SIT) is another method of biological control, whereby overwhelming numbers of sterile male insects are released that competes with the wild males for female insects (Dyck *et al.*, 2005). The SIT is the release of a large number of reproductively sterile male insects into a wild population of the same species. When a sterile male mates with a virgin female fly, this results in no offspring, because female tsetse usually mate only once in their life time (Feldman, 2004).

According to (FAO, 1992), in order to control any pest species, it is necessary to have a good understanding of its biology, behavior and population dynamics that is the natural factors, which affect distribution and abundance. Here the means are provided by which the tsetse can eradicate itself; it may also be called an autocidal (self-killing) method of control (Ahmed, 2015). SIT relies on the rearing of the target insect in large numbers in specialized production centers, the sterilization with ionizing radiation and the sustained sequential release of the sterilized insects over the target area (Vreysen, 2001).

SIT introduced on to an area covered with *Glossina palpalis palpalis* and *G. tachinoides* as a target tsetse species, using a combination with fly reduction, achieved its main objective to reduce tsetse population virtually to zero have been reported. There is absence of reinvasion of tsetse flies after eradication using SIT (Oluwafemi, 2008).

The SIT to eradicate tsetse flies and trypanosomosis from the island of Zanzibar, the coast of Tanzania is one of the successful attempts (Rechard, 2002). In preparation, widespread application of insecticide, as well as fly trapping, should be used to suppress the tsetse population to the point where SIT is considered feasible. A sterile fly plant producing 70,000 pupae weekly was developed and a total of eight million sterile male flies were dispersed over time to overwhelm the residual wild tsetse population. The wild fly was not detected on the island after two years and the last case of trypanosomosis diagnosed only a year later after the program (Anon, 2000).

Efforts by the FAO International Atomic Energy Agency Division to scale sterile tsetse fly production systems to semi industrial levels are beginning in several countries of Africa. Among these is Ethiopia where eradication activities including the SIT begin in the southern Rift Valley area in which *Glossina pallipides* inhibits agricultural development (Rechard, 2002).

The Ethiopian government in collaboration with the International Atomic Energy Agency (IAEA) established in 1997/98 as the Southern Tsetse Eradication Project (STEP) a pilot project under the Ministry of Science and Technology to eliminate the disease from 25,000km² area of the Southern Rift Valley of Ethiopia, which was later expanded to other trypanosomosis affected parts of the country based on the achievements and lessons learned at initial stage of the project. The main activities of the project are tsetse suppression as well as tsetse fly mass rearing and release of sterile males (Shushay, 2012).

The two major drawbacks to current SIT methods result from the negative effects of sterilization by irradiation and difficulties in sex-separation procedures. A sterilizing dose of irradiation (or chemicals) unavoidably reduces the fitness and competitive mating ability of the irradiated males relative to wild males. Furthermore, the costs of constructing, operating, and decommissioning the radiation source adds significantly to the cost of the control program (Alphey, 2000).

3.1.2. Chemical control

Large areas can be cleared from tsetse flies by using ground and/or aerial spraying of insecticides. Ground spraying uses residual insecticides (e.g. DDT, dieldrin, and 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). SAT 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 (Kgori *et al.*, 2006).

Insecticides can also be applied on live animals by spraying or pour-on. 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 (Torr *et al.*, 2007). Consequently, this kind of treatment reduces the cost to less than 1 US\$ per head of cattle per year (Torr *et al.*, 2005). 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 fewer nuisances for the animals and higher productivity (Leak *et al.*, 1995). In spite of this, because of their detrimental effect on naturally occurring fauna and flora, the use of insecticides is limited (Allsopp *et al.*, 2004).

3.1.3. Mechanical control

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) (Green, 1994). 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. In case of zero grazing animals insecticide impregnated mosquito nets (about 1 meter high) have been used successfully around the stable in order to protect the cattle against tsetse flies (Bauer *et al.*, 2005). The use of improved traps impregnated with insecticide could develop into a simple and relatively cheap method of control. However, traps can only be applied in some types of areas and are species specific to an extent, this technology is not sophisticated and environment friendly (Allsopp, 2001; Vale and Torr, 2004).

3.2. Methods to control the parasite

A limited number of drugs are available to treat animal trypanosomosis. 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 (Geerts *et al.*, 2010). Whereas, quinapyramine, suramine and melarsomine are primarily used as therapeutic drugs for infections caused by *T. evansi* in Equidae, camels and buffaloes, although quinapyramine is also used for prophylactic purpose.

Table 3: Drugs commonly used for trypanosomosis in domestic animals

Drug	Animal	Trypanosome	Main action
Diminazene aceturate	Cattle	Vivax, congolence, brucei	Curative
Homidium chloride	Cattle Equids	Vivax, congolense, brucei Vivax	Curative, some prophylactic activity
Isometamidium chloride	Cattle	Vivax, congolense	Curative and prophylactic
Quinapyramine sulfate	Horses, camels, pigs, dogs	Vivax, congolense, brucei, evansi, equiperdum, simiae	Prophylactic
Suramin	Horses, camels, dogs	brucei, evansi	Curative, some prophylactic activity
Melarsomine dichlorhydrate	Camels	Evansi	Curative

Source: Khan, 2005

Currently the treatment of affected animals with trypanocidal drugs still remains the most frequently applied measure to control trypanosomosis. 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 (Delespau *et al.*, 2002). Trypanocidal drug resistance is increasingly reported all over Africa and is now present in 21 sub-Saharan countries (Chitanga *et al.*, 2011; Geerts *et al.*, 2010). However, farmers continue to use the drugs because alternative products are not available. Fortunately, it has been observed at several occasions that –even when drug resistance is present at

high levels – treatment remains beneficial and allows the animal to survive and to be productive (Chitanga *et al.*, 2011; Delespau *et al.*, 2010; Geerts and Holmes, 1998).

3.3. Host management

Most livestock owners in Africa know very well the sites where tsetse flies are present. They are quite often able to manage their herds and flocks in such a way as to avoid contact with the bites of the flies. However, especially in the dry season when there is a lack of grass, the farmers are forced to bring their herds to wetter places with more grass but often also infested with tsetse flies (Geerts, 2011).

Trypanotolerance is another host-related characteristic of some livestock breeds allowing them

to survive, reproduce and remain productive under trypanosomosis risk without the aid of trypanocidal drugs (d'Ieteren *et al.*, 1998). The use of trypanotolerant breeds is a highly sustainable approach to control trypanosomosis in low or medium tsetse infested regions. However, unfortunately only about 6 % of the current cattle population in West and Central Africa consists of N'Dama, Baoule and other trypanotolerant breeds. Trypanotolerant Djallonke sheep and West African dwarf goats account for 32 and 47 %, respectively of the sheep and goat population in West and Central Africa. In East and Southern Africa there are even much smaller numbers of trypanotolerant livestock breeds (Geerts *et al.*, 2009).

4. Conclusion And Recommendations

Trypanosomosis is considered the most important protozoan disease and probably the most important health constraints to livestock leading to production losses. In most African countries, tsetse distribution has increased and spread to new areas. The incidence of the disease also remained high with occasional endemic outbreak. The methods of controlling animal trypanosomosis are numerous and varied. However, each possesses its advantages and disadvantages. All the currently available and environmentally acceptable methods for tsetse and trypanosomosis control have their specific limitations. Only a combination of the numerous methods in an integrated phase approach can effectively advance the control of tsetse and trypanosomosis.

Based on the above conclusion the following recommendations have been forwarded.

➤ Trypanotolerant livestock breeds should be improved in areas where there is a heavy tsetse challenge.

➤ In case of SIT, the life span of irradiated males in laboratory should be reduced, since irradiation reduces the fitness and competitive mating ability of the irradiated male. Although, irradiated males have lesser chance to stay alive.

➤ Trypanocidal drugs should be given to the animal by veterinary professionals in order to reduce drug resistance.

➤ When there are indications of drug resistance, it is essential to try to maintain the efficacy of the currently available drugs. The most important and most efficient measure is to adopt an integrated disease management strategy which includes preventing and the control of the agent and vector.

Acknowledgements

I would like to thank the almighty GOD who enables me to do this paper and to all my lovely parents for their moral and support.

I would like to express my gratitude and special thanks to my advisor Dr.Ashenafi Assefa for his support in supplying the materials and his valuable effort in preparing this paper.

Lastly, my deepest thanks go to the University of Gondar, faculty of veterinary medicine and the seminar coordinators for facilitation of the required materials and support in preparing the seminar paper.

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