

A Review of Bovine Trypanosomiasis and Tsetse Fly Density in Ethiopia

Tone Toka Tokossa

Animal Health Department, Livestock and Fishery Development Office,
Zala Woreda, Gofa Zone, Ethiopia

Abstract: Trypanosomiasis is a worldwide disease caused by the species of the genus *Trypanosoma*, which affects humans, as well as domestic and wild animals. The most important *Trypanosoma* species affecting cattle in Ethiopia are *Trypanosoma congolense*, *Trypanosoma vivax* and *Trypanosoma brucei*. Bovine trypanosomiasis is transmitted from the infected animal to susceptible host both mechanical and biological vector. Most tsetse transmission is cyclic and begins when blood from a trypanosome infected animal is ingested by the fly. Known species in Ethiopia are five in number, namely: *G. pallidipes*, *G. morsitans*, *G. fuscipes*, *G. tachinoides* and *G. longipennis*. The clinical feature of the disease follows the level or burdens of tsetse challenge species. The main feature is anemia results in a progressive drop in packed cell volume, a non-specific but useful indicator in endemic areas. The most sensitive rapid method is examining a wet mount of the buffy coat area of a PCV tube after centrifugation, looking for motile parasites. The prevalence of trypanosomiasis in enzootic area can be reduced by parasite control, vector control, host resistance protection prophylactic treatment and good husbandry management system. Trypanotolerant animals are very important in tsetse fly challenging areas, but most countries did not accept them due to their low production of milk than indigenous breed. In conclusion, prevalence of trypanosomiasis is devastating diseases of cattle in Ethiopia with both direct and indirect economic losses. Several diagnostic methods can be used in the investigation of the disease. Besides clinical diagnosis, direct (parasitological), indirect (serological) and molecular diagnostic methods with varying degrees of sensitivity and specificity are available for trypanosomiasis. Thus, it is recommended that an appropriate use of antiprotozoal drugs, integrated prevention and control program should be implemented to eradicate trypanosomiasis and other protozoan disease.

Key words: Bovine Trypanosomiasis • Diagnosis • Economic Impacts • Ethiopia

INTRODUCTION

African animal trypanosomiasis is one of the major animal health problems posing a significant effect on the settlement and socioeconomic development over large tsetse belt regions of the continent. It is a serious disease in domestic livestock that causes a significant negative impact in food production and economic growth in many parts of the world, particularly in Sub-Saharan Africa [1]. In Sub-Saharan Africa, including East Africa, the vector of a disease is distributed over 10 million km² of potential grazing lands in 37 countries, exposing the lives of around 55 million people and 160 million cattle to the risk of a disease [2].

The overall economic loss (both direct and indirect) is estimated to be about 500 billion dollars a year in terms of mortality, production, abortion, reduced fertility and ability to work as traction animals. Furthermore, the disease is responsible for an annual loss of millions of dollars in livestock health and production as a result of the cost related to treatment, prevention and vector control efforts [3].

Ethiopia has huge livestock population in Africa and the livestock sector plays a significant role in the national economy and livelihood of farmers and pastoralists [4]. The sub-sector contributes about 16.5% of the national gross domestic product (GDP) and 35.6% of the agricultural GDP. Despite this huge livestock number,

productivity is too low and even below the average for most Eastern and Sub-Saharan African countries, due to a number of complex and interrelated factors, such as inadequate feed and nutrition, widespread diseases, poor genetic potential of local breeds and inefficiency of livestock development services [5]. Among these, trypanosomosis is one of the major animal health constraints to livestock production and agricultural development [6].

Trypanosomosis is a chronic hemoprotozoan disease of domestic animals and humans caused by different species of unicellular eukaryotic parasite of the genus *Trypanosome* [6]. With an exception of *Trypanosoma equiperdum* of equines, which causes a venereal disease, all have arthropod vectors in which the transmission is either cyclically by tsetse flies of the *Glossina species* or non-cyclical by many other insects [7]. Cattle affected with trypanosomosis can show major clinical manifestations of a disease, such as intermittent fever, anemia, anorexia, dullness, apathy, watery ocular discharge, reproductive disorder and superficial lymph node enlargement. The animals progressively become emaciated and cachectic and finally die if untreated [8].

There are six pathogenic species of trypanosomes which are recorded in Ethiopia, namely *T. vivax*, *T. congolense*, *T. brucei*, *T. evansi*, *T. equiperdum* and *T. rhodesiense*. But the most important trypanosomes in the country are *T. vivax* and *T. congolense*. Both species affect a great number of cattle which are the most important species of the domestic animals in Ethiopia [9]. The prevalence varies from locality to locality depending on agro-climatic conditions, seasons and as part of activities which are intended to control the impact of the disease [10].

Trypanosome transmitted by tsetse fly continues to be a major constraint in livestock production. The disease greatly affects social, economic and agricultural development of communities in tsetse infested areas [11]. The resistance of trypanosome to existing anti-trypanosomal drugs, increasing vectors' resistance to insecticides, absence of effective vaccines and adverse effects of existing anti-trypanosomal drugs are challenges in controlling the disease. People have been using both plant and animal species for treatment and control of trypanosomosis and as tsetse fly repellent in Ethiopia [12].

Moreover, trypanosomosis is a very serious disease of cattle, which causes great socioeconomic losses in the country in general and study area in

particular. Its socioeconomic impact is reflected on direct losses due to mortality, morbidity and reduction in milk and meat production, abortion and stillbirth and also costs associated with combat of the disease are direct losses. Consequently, studying the prevalence and magnitude of the vector is inevitably important to develop appropriate control measures [9].

Over the past few decades, many efforts have been made to control tsetse and trypanosomosis in Ethiopia through coordinated action of the government, non-governmental organizations and local community. The control interventions commonly used in Ethiopia include; insecticidal pour-on, insecticide-impregnated traps and targets and use of different trypanocidal drugs [13]. However, information related to temporal and spatial dynamics of tsetse and trypanosomes remain very limited and may be a reason that control strategies are less effective and fail in endemic areas [14].

Hence, the epidemiological knowledge on bovine trypanosomosis and distribution of the tsetse fly are paramount in formulating appropriate strategies for the control of these problems [15]. Since, bovine trypanosomosis is highly devastating disease that have great economic impact on the country development. Therefore the objectives of this review were:

- To review bovine trypanosomosis and tsetse fly distribution in Ethiopia.
- To review on the effective control and preventive strategies against the disease.
- To review the epidemiological information and economic significance of bovine trypanosomosis with a particular emphasis to Ethiopia livestock subsector
- To assess potential risk factors associated with trypanosome infection.

The Trypanosomes: African animal trypanosomosis or Nagana is a complex chronic, debilitating and often fatal diseases of animals caused by different species of flagellated unicellular parasites belonging to the genus *Trypanosome* and found in the blood and other tissues fluids of vertebrates including livestock and wild animals [16]. It is mainly transmitted cyclically by the genus *Glossina* (Tsetse flies), but also transmitted mechanically by several biting flies like Tabanids, Stomoxys, Haematopota and Chrysops. The disease can affect various species of mammals but, from an economic point of view, tsetse-transmitted trypanosomosis, is particularly important in cattle [17].

Trypanosomes are single celled flagellated protozoan parasites that live and multiply extracellular in blood and tissue fluids of their mammalian hosts and transmitted by the bite of vector flies. The name Trypanosome is derived from Greek word *trypano* (borer) and *some* (body) because of their cork-screw like motion. The trypanosome consists of a single cell varying in size from 8 to 50 μm . The different trypanosome species differ in morphological characteristics as described by in appearance, shape and size between the various species allowing specific identification [18].

Taxonomy and Classification of Trypanosomes:

Trypanosomes are unicellular protozoan parasites of the phylum Sarcomastigophora, order Kinetoplastida, due to the presence of a kinetoplast at the base of the flagellum, family Trypanosomatidae and genus *Trypanosome*. Genus *Trypanosome* presents flagella and an organelle recognized by its kinetoplast [19]. On the basis of the site of development in the insect vector, the genus *Trypanosome* is divided into two sections: stercoraria and salivarian. In the stercoraria section the metacyclic trypanosomes develop in the hindgut of the vector and are thus transmitted to the mammalian host via faeces. Salivarian trypanosomes develop in the anterior portion of the fly's digestive tract in the salivary glands (*T. brucei*) or in the midgut, *T. congolense* and in the proboscis (*T. vivax*) and are transmitted via the saliva [20].

Etiology of Trypanosomosis: Trypanosomosis is an important protozoan disease caused by the genus *trypanosome* transmitted through bites by different species of *Glossina* and mechanically by a number of biting flies such as *Tabanus* and *Stomoxys* species. Bovine trypanosomosis is a parasitic infection caused by an extracellular hemoparasites known as trypanosomes. They swim in body fluids by flagellum, boring their way between cells. They generally, possess a kinetoplast and undergo cyclical development in an arthropod vector. Their biological adaptations, morphology and pathogenicity are fascinating and are being extensively studied [21]. Three main pathogenic species of trypanosomes are recorded in Ethiopia. These are: *T. congolense*, *T. vivax* and *T. brucei*. *T. vivax* and *T. congolense* are the main pathogens of cattle [17].

Morphological Characteristics: The different trypanosome species differ in morphological characteristics as described by Bengaly *et al.* [22]. *Trypanosome congolense* is smaller in size, usually

without free-flagellum, but has marginally located medium sized kinetoplast. It is divided into four subtypes with different distributions and pathogenicity: savannah type, forest type, Tsavo type and Kilifi type. *Trypanosome congolense* savannah type is the most pathogenic of the four and is capable of causing severe anemia and even death of infected cattle [22]. Other *T. congolense* types cause mild disease that in certain instances does self-cure.

Trypanosome vivax is a monomorphic parasite with distinct free flagellum, larger and terminal kinetoplast. In East Africa, there are two types of *T. vivax* isolates: the hemorrhagic *T. vivax* that causes an acute hemorrhagic syndrome and the mild strain [23]. Cattle infected with the hemorrhagic *T. vivax* produce auto-antibodies to red blood cells, a phenomenon that is not observed in the non-hemorrhagic *T. vivax* [24].

Parasites in *Trypanosome brucei* group show pleomorphic with slender, intermediate or stumpy forms. They have small sub-terminal kinetoplast, undulating membrane with conspicuous posterior end taper to a point except in stumpy forms. During the course of the infection, there is a change in the trypanosome population from the long thin forms, through the intermediate to the short stumpy and this altered appearance is accompanied by a change in the type of respiration, as the trypanosome prepares for its period within the tsetse fly. The short stumpy forms are adapted to living and developing in the tsetse, while long thin forms are the true mature blood forms which die in the gut of the insect. Similar metabolic changes also occur in other trypanosome species, but there are no such obvious morphological changes associated with them as in *T. brucei* [18].

Life Cycle of Trypanosomes: Most tsetse-transmission is cyclical and begins when blood from a trypanosome infected animal is ingested by the tsetse fly. The trypanosome loses its surface coat, multiplies in the fly, then reacquires a surface coat and becomes infective. *Trypanosoma brucei* species migrate from the gut to the proventriculus to the pharynx and eventually to the salivary glands; the cycle for *T. congolense* stops at the hypopharynx and the salivary glands are not invaded; the entire cycle for *T. vivax* occurs in the proboscis. The animal infective form in the tsetse salivary gland is referred to as the metacyclic form. The life cycle in the tsetse may be as short as 1 week with *T. vivax* or extend to a few weeks for *T. brucei* species [25].

The African trypanosomes have four major life cycle stages. The procyclic form (PF), epimastigote form (EMF) and metacyclic form (MCF) all develop in tsetse while the

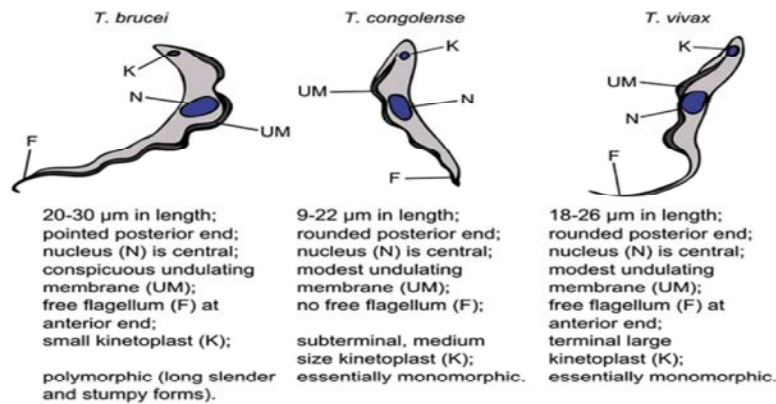


Fig. 1: Morphology of trypanosome species [25].

blood stream form (BSF) is found in the mammalian host [20]. Tsetse flies ingest infective blood stream trypomastigotes when they feed on infected hosts and may remain infected, acting as a continual source of infection. Tsetse ingests trypomastigote that found in the blood and lymph while feeding on an infected host. The trypomastigotes lose their glycoprotein surface coat and become elongated multiplying in the midgut before migrating forward to the salivary glands and proboscis. They then transform into epimastigote forms which multiply and then transform again into small typical metacyclic trypomastigotes which are the infective stages and are introduced into the vertebrate host during feeding. These metacyclic have been shown to have a small repertoire of Variable Surface Glycoprotein (VSG) genes and they multiply at the inoculation site for a few days before invading blood stream and lymphatics [26]. This life cycle has variation in reference with the *trypanosome* species involved. Development of *T. congolense* occurs in the midgut and proboscis while development of *T. brucei* takes place in the mid gut and salivary glands [20].

Trypanosome vivax completes cyclical development exclusively within the mouth parts of tsetse flies [20]. The blood trypomastigote forms are taken up by tsetse along with its blood meal and undergo stages of complex biological development inside the insect host before becoming infective. Both male and female flies are capable of transmitting trypanosomes. *Trypanosome vivax* usually multiplies rapidly in blood and is evenly dispersed throughout the cardiovascular system, whereas *T. congolense* tends to aggregate in small blood vessels and capillaries of the heart, brain and skeletal muscle from where a small proportion of parasites enter the blood circulation. *Trypanosome brucei* localize in tissues aside blood vessels [27]. The parasites are then transmitted to a subsequent host at the next blood meal [28].

Mode of Transmission: Most trypanosomes must develop for one to a few weeks in tsetse flies (*Glossina spp*), which act as biological vectors before transmitted to susceptible hosts. The tsetse fly becomes infected with trypanosomes when feeding on an infected animal. When an infected tsetse fly bites an animal, the parasites are transmitted to susceptible host in the saliva [29]. Trypanosome species that commonly infect cattle in Ethiopia such as *T. congolense*, *T. vivax* and *T. brucei* are transmitted to cattle biologically via the bite of infected tsetse flies. Other studies made in different parts of Ethiopia revealed that, in addition to *Glossina spp*, other biting flies such as *tabanids*, *haemtopota* and *stomoxys* are responsible for mechanical transmission of trypanosomes to susceptible animals [30].

Pathogenesis: The pathogenesis of African animal trypanosomiasis depends on several factors, including parasite-related aspects (species and virulence), host (species, breed, age and nutritional status and physical condition), vector (species, density and infection rate and host preference) and the environment (the availability of food and water and the season [31]. During a blood meal on a mammalian host, an infected tsetse fly injects metacyclic trypanosomes in to the skin tissues [32]. Following inoculation, trypanosomes then continue to proliferate by binary fission for a few days, leading to a local inflammatory response called a chancre. The size of chancre is determined by the animal immune status, the virulence of the infecting trypanosome species and the inoculation dose [33]. The chancre disappears 3 to 15 days post-infection and trypanosomes enter to the lymph nodes and the blood stream and transform in to blood stream trypomastigotes, which are carried to other tissues. The chancre not only forms a site for the establishment of the infection but also is a focus for multiplication and persistence of trypanosomes before their dissemination in to blood stream [34].

Clinical Signs: Bovine trypanosomosis causes severe anemia, edema, immunosuppression and various neurological disorders, which may eventually produce the death of the affected animals [35]. The basic clinical syndrome appears after an incubation period of 8-20 days. There is fever, which is likely to be intermittent and to last for a long period. Affected animals are dull, anorexic and apathetic have a watery ocular discharge and lose condition. Superficial lymph nodes become visibly swollen, mucous membranes are pale, diarrhea occasionally occurs and some animals have edema of the throat and underline. Estrus cycles become irregular, pregnant animals may abort and semen quality progressively deteriorates. The animal becomes very emaciated and cachectic and dies within 2-4 months or longer. Thin, rough-coated, anemic, lethargic cattle with generalized lymph node enlargement are said to have 'fly struck' appearance. Furthermore, intercurrent bacterial, viral, or other parasitic infections may mask or complicate the basic clinical syndrome [36].

Diagnosis: Diagnosis of trypanosomosis in tsetse, humans or domestic livestock is a basic requirement for epidemiological studies as well as for planning and implementing chemotherapy and for monitoring vector control operations. Accurate diagnosis of trypanosome infection in livestock is required for a proper appreciation of the disease in any geographical locality. The general clinical picture is as follows but there are many variations determined by the level of tsetse fly challenge, the species and strain of the trypanosome and the breed and management of the host [11]. Definitive diagnosis of the disease is ultimately dependent on the detection of the trypanosome in blood samples from infected animals [9].

Serological Diagnosis: Serological methods are indirect methods for diagnosis of trypanosomosis based on detection of antibodies or circulating antigens of parasites. The most commonly used serological tests include, indirect haemagglutination test (IHT), precipitation tests, indirect fluorescent antibody test (IFAT) and ELISA. However, these serological tests are used as tools for research, monitoring and control surveys. The one exception to this generalization is in the application of the CFT for the diagnosis of dourine in horses [37].

Molecular Diagnosis: The method that is currently recommended for trypanosome diagnosis is molecular identification, due to the high sensitivity and specificity. The principle of molecular methods is to detect DNA sequences that are specific for trypanosome subgenus,

species, subspecies, type or strain. There are a number of PCR based techniques that have been developed for identification of trypanosome species. These molecular techniques rapidly detect and identify trypanosome species in both mammalian hosts and the tsetse vector with high sensitivity and specificity even in cases of low parasitaemia; however, none have been validated for routine diagnostic purposes [38].

Clinical Diagnosis: The clinical manifestation of bovine trypanosomosis is influenced by the host as well as the trypanosomes species and "strain" [39]. Infection of cattle by one or more of the three African animal trypanosomes results in subacute, acute, or chronic disease. However, under natural challenge, disease manifestation may be more complex [40].

Parasitological Diagnosis

Wet Blood Film: These are made by placing a droplet of blood (about 2 μ l) on a clean microscope slide and covering with a cover slip (22 \times 22 mm). The blood is examined microscopically at 40x total magnification with condenser aperture, phase-contrast or interference contrast. Approximately 50-100 fields are examined. Trypanosomes can be recognized by their movement among the red blood cells (RBCs). The method is simple, inexpensive and gives immediate results [41]. Final confirmation of the species is made by the examination of the stained preparation. The diagnostic sensitivity of the method is generally low, but depends on the examiner's experience and the level of parasitaemia. Sensitivity can be improved significantly by lysing the RBCs before examination using a haemolytic agent such as sodium dodecyl sulfate [42].

Thick Blood Films: These are made by placing a drop of blood (5-10 μ l) on a clean microscope slide and spreading it over an area of approximately 2 cm in diameter, using the corner of another slide. The thickness of the resultant film should be such that when dry, the figures on a wristwatch dial can just be read through it. The film is dried thoroughly by rapidly waving in the air and without fixation is de-haemoglobinised by immersion in distilled water for a few seconds and dried before staining. A dry smear should be kept dry and protected from dust, heat, flies and other insects [42]. It is stained for 30 minutes with 4% diluted Giemsa stain in phosphate buffered saline, pH 7.2. Therefore, it is important to start with the manufacturer's directions and to vary staining time and stain concentration to obtain the optimal result. The stained smear is then washed with buffered water and examined at 500 to 1000x total magnification [42].

Thin Blood Smear: These are made as in the case of blood smears to detect on the blood parasites like trypanosomes. They are fixed by methanol and stained with Giemsa stain, or with one of the more recent test stains such as Diff-Quik, field's stain, which have the advantage of acting much faster than Giemsa. They are read using oil immersion objectives, for identification of trypanosomes. Hence, what is most important thing of using such a method is that specific diagnosis of trypanosomes is possible. Nevertheless, the sensitivity is extremely low and the main use of thin smear is in fact the specific identification of trypanosomes found in wet or thick [43].

Parasite Concentration Techniques: In this procedure, heparinized capillary tubes are three quarters filled with the suspected blood sample containing an anticoagulant. The dry ends of the capillary tubes are sealed with cristaseal and centrifuged at 12, 000 rpm for 5 minutes. After centrifugation, the buffy coat/plasma junction is located between the plasma and the red blood cells and contains white blood cells as well as the parasites. The capillary tubes are then mounted on a woo chamber and can then be directly viewed at low magnification for mobile parasites [44]. The analytical sensitivity of BCT depends on the species of trypanosome as has been demonstrated by Paris [45], with the smallest numbers detectable per milliliter of blood being 2.5×10^2 , 5×10^2 and 5×10^3 , for *T. congolense*, *T. vivax* and *T. brucei*, respectively.

Epidemiology of Trypanosomosis: The epidemiology of African trypanosomosis is determined mainly by the ecology of the tsetse fly which is found only in tropical Africa [11]. Tsetse flies (*genus Glossina*) are restricted to Africa from about latitude 15° N to 29° S. Ethiopia is situated at the East end of the African tsetse belt and in Ethiopia, tsetse flies are confined to south western and north western regions between longitude 33° and 38° E and latitude 5° and 12° N of an area covers 220,000 km². The three main species that inhabit relatively distinct environments are: *G. morsitans* usually found in savanna country, *G. palpalis* prefers areas around rivers and lakes and *G. fusca* lives in high forest areas. All three species transmit trypanosomes and all feed on various mammals. The riverine species (*G. palpalis*, *G. tachinoides* and *G. fuscipes*) are important as vectors of bovine [11].

Geographical Distribution: Bovine trypanosomosis (Nagana) is found in the low lands of Ethiopia, especially in the "tsetse belt". For example, rift valley, Omo, Borena, Metekel Zone of Benshangul Gumuz region. The most important trypanosomes affecting cattle in Ethiopia are: - *Trypanosoma congolense*, *T. vivax* and *T. brucei* [46].

The general distribution of tsetse flies is determined principally by climate and influenced by altitude, vegetation and presence of suitable host animals. Out of the nine regions of Ethiopia, five (Amhara, Benishangul Gumuz, Gambella, Oromia and Southern Nation Nationalities and peoples region) are infested with more than one species of tsetse flies. To date, five

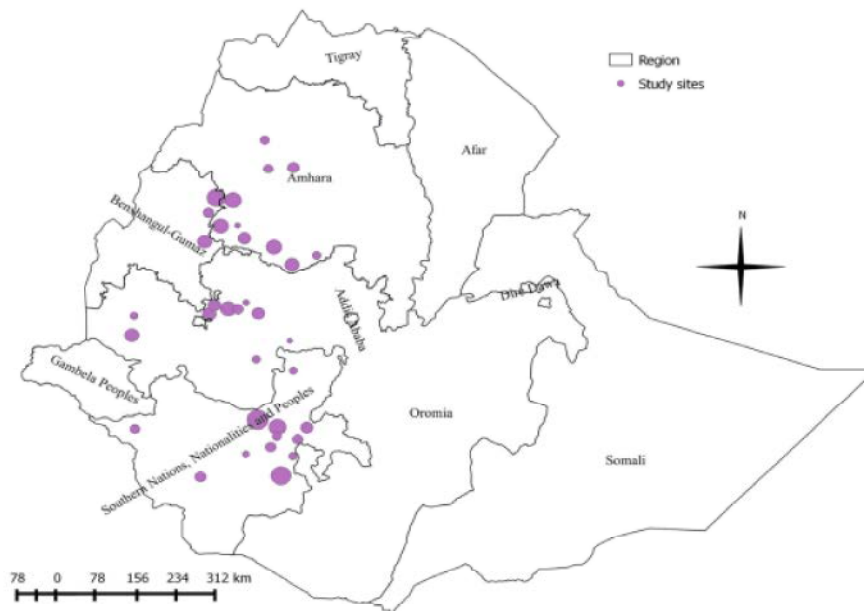


Fig. 2: Observed spatial distribution of bovine trypanosomosis in Ethiopia [48].

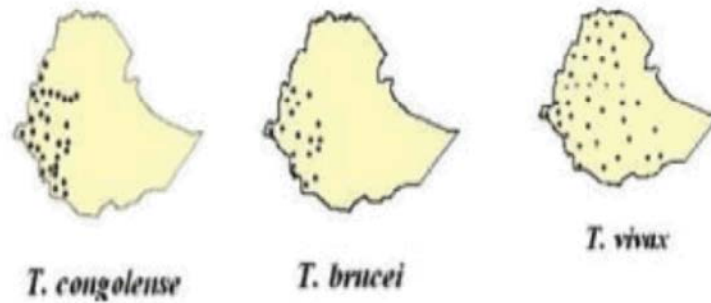


Fig. 3: Distribution of pathogenic trypanosomes in Ethiopia [49].

species of *Glossina* (*Glossina morsitans submorsitans*, *G. Pallidipes*, *G. tachinoides*, *G. f. fuscipes* and *G. longipennis*) have been recorded from Ethiopia [47].

Risk Factors Affecting Bovine Trypanosomosis

Host Related Factors: Trypanosomes can infect all domesticated animals; clinical cases have been described in cattle, sheep, goats, camels, horses, donkeys and other species of animals. In parts of Africa including Ethiopia, cattle are the main species affected due to the feeding preferences of tsetse flies [29]. However, the effect of infection varies with the host in that in most wild animals, such as warthogs, bushbucks, kudus or buffalos, trypanosomes become established but do not produce the disease. This is because these animals and the parasites have evolved for many years resulting in a balanced host/parasite relationship. In domestic animals the relationship with the parasite has not fully developed leading to development of the disease [50]. The level of trypano-tolerance varies; depend on both genetic and environmental in origin. The indigenous zebu cattle are trypano-susceptible and West African *Bos Taurus* breeds are trypano-tolerant, i.e. they can survive and be productive without treatment under trypanosomosis risk. Exotic imported ruminants (improved dairy cattle) are more severely affected than local genotypes. In Ethiopian, four cattle breeds namely Abigar, Gurage, Horror and sheko have been thought to be relatively trypano-tolerant when compared to the indigenous zebu cattle [51].

Environmental Factors: Trypanosomosis maintains large area of Africa (so called “fly belts”) and it is suppose that wildlife have contributed a lot in the maintenance of the diseases in a relatively defined ecosystem [52]. The environment allows for the interaction between the *Glossina species*, vertebrate hosts and the trypanosomes in order for trypanosomosis to be occurred. In West Africa, tsetse habitats have been sub-divided along distinct North-South climatic gradients,

with predominantly riverine tsetse species in the North and a mixture in the South [53]. In the North, arid conditions prevent fly spread and riparian vegetation constitutes suitable niches for the localized, well-demarcated pockets of tsetse populations. Outside these favorable micro-climates, tsetse hardly survives and it would appear that no links exist between pockets, except occasionally and in spatially limited neighboring areas during the rainy seasons. In the intermediary band, climatic conditions and vegetation become slowly more suitable. Distinct fly pockets tend to merge and tsetse distribution patterns become more linear along main streams. Tsetse population still remain concentrated in pockets during dry season, but spreading [54], during the rainy season over large parts of the river systems, including important tributaries and savannah buffers. In the humid South, there are no climatic limitations to fly distribution and flies are present along river systems and even around humid woodlands and forests.

Due to increasing human population and as a result the opening up of more land for crops, the *morsitans* group is disappearing in most places of Africa [55]. Riparian tsetse species on the other hand are more versatile and can co-exist with human development. They are opportunistic feeders; where agricultural intensity is low; they feed on wild reptiles and rarely carry pathogenic trypanosomes [56].

Pathogen Factors: In cattle, *T. vivax* generally produces a higher level of parasitemias than other species since its life cycle in the tsetse is also shorter. Parasite virulence is also an important factor influencing the epidemiology of African animal trypanosomosis. The pathogenicity appears to vary depending on which type or strain of *Trypanosome* is involved [22]. Within *T. congolense*, different types exist (savannah, forest, kilifi and Tsavo) that have a different pathogenicity. *Trypanosome congolense* Savannah type is the most pathogenic and is responsible for acute infection and death of diseased

animal. However, *T. congolense* forest and Kilifi types cause mild infections [22]. Apart from *T. congolense*, other members of sub genus *Nannomonas* causing AAT include: - *T. simiae* (affecting domestic suids) and *T. Godfreyi* [57].

Economic Impacts of Bovine Trypanosomosis: In domestic animals, trypanosomosis is a disease with a great economic impact, affecting not only the wellbeing of the livestock population, but also efficient food production in crop-livestock production systems [58]. African animal trypanosomosis puts 50 million cattle at risk and leads to the death of three million animals every year, inflicting a direct annual loss of US\$ 1.0-1.2 billion in cattle production [59]. Hence, nagana is today the most important disease of livestock in the continent. Since nagana is a wasting disease, affected animals are chronically unproductive in terms of milk, meat, manure and traction and the mortality rate can be high. Furthermore, the disease may impact on various immunization campaigns in endemic areas due to the fact that it can cause immunosuppression [60].

Trypanosomosis directly affects the milk and meat productivity of animals, reduces birth rates, increases abortion as well as mortality rates and all of these reduce the herd size and herd composition. The indirect impact of the disease mostly lies on crop production through the availability and cost of animals that provide traction power [61]. The overall negative impact extends to the access and availability of cultivable areas, changes in land use and exploitation of natural resources, restraint of opportunities for diversification and intensification of agricultural activity.

Control of Bovine Trypanosomosis: There has been a long history of tsetse and trypanosomosis control in Africa, but today the problem is still far from being solved and there is no control method that can fully eradicate African animal trypanosomosis and the incidence of both animal and human trypanosomosis remained high with occasional endemic outbreak [62].

Despite, extraordinary research efforts directed at the development of vaccines against trypanosomes, no vaccine has so far has been developed in the near future [63]. Hence, control of animal trypanosomosis relies primarily on control of the vector, farming of trypano-tolerant breeds and use of trypanocidal drugs [64]. Prevention of successful establishment and/or maturation of trypanosomes within the tsetse fly have been proposed as possible future control method [65].

The Tsetse Vector: Tsetse fly (Diptera, Glossinidae) is large biting fly that inhabit about 10 million km² of area in 37 sub-Saharan Africa countries and are distributed discontinuously throughout their range and each taxon is restricted to a relatively specific habitat [66].

Depending on the environmental type, there are three main subgroups of Tsetse flies: Palpalis (riverine), Morsitans (savannah) and Fusca (forest-dwelling) [67]. Approximately one-third of Africa's total landmass is infested by these flies [48]. Vector distribution mainly confined to the Southern and Western regions between longitude of 33° and 38°E and latitude of 5° and 12°N [68]. Among 31 species of tsetse flies, five species: - *Glossina pallidipes*, *G. morsitans*, *G. fuscipes*, *G. tachinoides* and *G. longipennis* are known in different regions of Ethiopia (Amhara, Benishangul Gumuz, Gambella, Oromia and Southern Ethiopia [69].

Morsitans Group: Are also called the savannah flies due to their preference to this environment and the most important vectors as the African savannah is a vast area and the flies come into contact with man, livestock and wild game animals. In Ethiopia, this group is distributed in Didessa valley near the village of Wonago and Lado on the eastern side of Lake Abaya, Shambo, on the Muger River, on the Dabous River (Wollega), on the Baro and Gilo Rivers (Gambella district), Illubabor associated with Akobo river, in the Savannah near Turmi and near Mizan Teferi [70]. All species belonging to this group are restricted to savannah wood lands and their distribution and abundance is tied with wild animal's distribution. During dry season, they are concentrated near the source of water courses and spread out in wooden savanna during the rainy season. The species under these groups are; *G. morsitans* and *G. pallidipes* identified from Ethiopia. *G. pallidipes* is highland species being present in some coastal areas and rift valley [71].

Palpalis Group: The distribution of the palpalis group species is likewise associated with lowland rain forest [72], specific vegetation like riparian forests that line the hydrographical network or plantations of certain crops and extended along river systems in the humid savanna. They are also called riverine tsetse fly groups and can tolerate a wide range of climatic conditions [72]. There are two species of palpalis group in Ethiopia; *G. tachinoides* and *G. fuscipes*. *G. fuscipes* is found in Maze, Gorgora, Bazo and Cuccia Rivers (Gamo Gofa), on the Ketto tributary and at Degen of the Birbir (Wellega), on the tributary of the Gojeb (Kaffa) and near the bridge on the Omo River and Addis to Jimma high way [71].

Fusca Group: They are forest tsetse flies and are densely colonized where vegetation are found [71]. In transition zones between true forest and wooded lands, they prefer dense shade and riverine thickets. These mainly forest-dwelling groups consequently have little epidemiological significance. In Ethiopia, the distribution of this vector is along the Walmal River (Bale), at tributary of Wabe Shebele in (Daghatto River) in the Ogaden and near Lake Abaya, Gamo goffa and Keffa. Under this group, there are two species of tsetse flies i.e. *G. brevipalpis* and *G. longipennis*. *G. brevipalpis* is found only at the lower part of the Omo River [71]. Both males and females are blood eaters and therefore, both sexes play a role as potential vector for trypanosome. In addition to that, once a tsetse fly has been infected, they remain infected throughout its life [73].

Status of Tsetse Fly and Trypanosomosis in Ethiopia:

Unfortunately, the development and intensification of livestock productivity in Ethiopia is hampered among others by cross-border epizootic diseases such as African animal trypanosomosis. Out of the nine regions of Ethiopia (Oromia, Benishangul Gumuz, Amhara, Gambella and SNNPR) are infested with more than one species of tsetse flies [9].

In tsetse infested areas 14 million of cattle, equivalent number of small ruminants and more than 7.5 million equines and 1.2 million of camels are at risk of contracting trypanosomosis. Trypanosomosis also prevents full use of land and the introduction of highly productive exotic dairy animals and draught oxen to low land areas [74].

The prevalence of trypanosomosis in tsetse infested areas range from 11.85-37% [75]. According to Geja *et al.* [76], the fly density, trypanosomosis prevalence and mortality due to trypanosomosis have been significantly reduced and the government of Ethiopia has conducted a massive settlement program in early 2000s to the tsetse and trypanosomosis belt.

CONCLUSION AND RECOMMENDATIONS

Trypanosomosis is a chronic hemoprotozoan disease of domestic animals and humans caused by different species of unicellular eukaryotic parasite of the genus *Trypanosome*. There are six pathogenic species of trypanosomes which are recorded in Ethiopia, namely *T. vivax*, *T. congolense*, *T. brucei*, *T. evansi*, *T. equiperdum* and *T. rhodesiense*. But the most important trypanosomes in the country are *T. vivax* and *T. congolense*. Prevalence of trypanosomosis progress is high in bovine and impact of the disease on productivity of infected animals. Since

trypanosomosis is worldwide problem and causes great economic lose due to infectious and death of animals as well as medication costs, agricultural and loss of production. To reduce its effects, the following measures are recommended:

- Improve management practices such as rearing, feeding, housing, medication.
- Increase awareness creation those animal rearing society especially pastoral community.
- The need for strengthening the vector and parasite control interventions.
- Government and non-government organization should conduct community based integrated tsetse fly control strategy in order to bring sustainable solution for livestock producing community.
- Extend and strengthen the national tsetse and trypanosomosis control scheme in tsetse infested areas.

REFERANCES

1. Cecchi, G., R. Mattioli, J. Slingenbergh and S. De La Rocque, 2008. Land cover and tsetse fly distributions in sub-Saharan Africa. *Med. Vet. Entomol*, 22(4): 364-373.
2. Stijlemans, B., P. De Baetselier, S. Magez, A. Van Ginderachter and C. De Trez, 2018. African trypanosomiasis associated anemia: the contribution of the interplay between parasites and the mononuclear phagocyte system. *Front Immunol*, 9:218. doi:10.3389/fimmu.00218.
3. Assefa, E. and G. Abebe, 2001. Drug-resistant *Trypanosoma congolense* naturally infected donkeys in North Omo Zone, Southern Ethiopia. *Vet. Parasitol.*, 99: 261-71.
4. Central Statistical Agency, 2020. Agricultural Sample Survey, Volume II report on livestock and livestock characteristics (private peasant holdings: Addis Ababa Ethiopia, pp: 183.
5. Leta, S. and F. Mesele, 2014. Spatial analysis of cattle and shoat population in Ethiopia: growth trend, distribution and market access. *Springer Plus*. 3:310.
6. Abebe, R., S. Gure and J. Simon, 2017. Bovine trypanosomosis and vector density in Omo Ghibe tsetse belt, Southern Ethiopia. *Acta tropica*, 167: 79-85.
7. Gebisa, G., K. Beriso, B. Bogale, O. Gizaw and D. Chala, 2020. Bovine trypanosomosis and its vectors in three selected districts of Buno Bedele Zone of Oromia Region, Ethiopia. *Vet. Med. Int.*, 1-8. doi: 10.1155/2020/1571947.

8. Constable, D., W. Hinchcliff, H. Done and W. Grünberg, 2017. Veterinary Medicine: A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs and Goats. 11th ed. St. Louis, MO: Elsevier Ltd, pp: 2150-6.
9. Abebe, G., 2005. Review article: trypanosomosis in Ethiopia. Ethiop. J. Biol. Sci., 9: 75-121.
10. Tadesse, A. and B. Tsegaye, 2010. Bovine trypanosomosis and its vectors in the two villages of Benchi Maji Zone: South-Western Ethiopia. Trop. Animal. Health Prod, 42: 1757-1762.
11. Radostitis, M., C. Gay, W. Hinch and C. Cliff, 2007. Disease associated with Trypanosomes. In: Veterinary Medicine, Textbook of disease of cattle, horses, pigs and goats, 10thed. Elsevier, UK, pp: 1531-1554.
12. Abdeta, D., M. Amante and Y. Tamiru, 2020. Survey on Ethno-Botany and Medicinal Animals at Sayoand Hawa Gelan Districts of Kelem Wollega Zone, Western Ethiopia. Prev- Med, 28(2): 21408.
13. Duguma, R., S. Tasew, A. Olani, D. Damena, D. Alemu and T. Mulatu, 2015. Spatial distribution of Glossina spp. and Trypanosoma spp. in south-western Ethiopia. Parasites Vectors, 8: 2-10. doi: 10.1186/s13071-015-1041-9.
14. Eyasu, T., S. Mekuria and D. Sheferaw, 2021. Seasonal prevalence of trypanosomosis, Glossina density and infection along the escarpment of Omo River, Loma district, Southern Ethiopia. Heliyon, 7: 66-67. doi: 10.1016/j.heliyon.e06667.
15. Van Den Bossche, P. and R. De Deken, 2002. Seasonal variations in the distribution and abundance of the tsetse fly, Glossina morsitans in eastern Zambia. Med. Vet. Entomol, 16: 170-6.
16. Singh, V. and L.D. Singla, 2012. Trypanosomosis in cattle and buffaloes from latent carrier status to clinical form of disease: Indian scenario. In: Integrated Research Approaches in Veterinary Parasitology, Shanker, D., J. Tiwari, A.K. Jaiswal and V. Sudan, (Eds), Bytes and Bytes Printers, Bareilly, pp: 10-18.
17. Desquesnes, M. and A. Davila, 2002. Applications of PCR-based tools for detection and easy method for species-specific diagnosis of Trypanosoma species in cattle. Vet. Parasitol, 110: 171-180.
18. Maudlin, I., H. Holmes and A. Miles, 2004. The Trypanosomiasis. Wallingford, UK: CABI International Publishing, pp: 1-634.
19. Eloy, J. and B. Lucheis, 2009. Canine trypanosomiasis, etiology of infection and implications for public health. Anim. Toxins. Incl. Trop. Dis., 15: 589-611.
20. Peacock, L., C. Simon, F. Vanessa, B. Mick and G. Wendy, 2012. The life cycle of T. congolense in the tsetse fly. Parasite and Vectors, 5:109, doi: 10.1186/1756-3305-5-109.
21. Magona, J., J. Mayende, W. Olaho-Mukani, P. Coleman, N. Jonsson, S. Welburn and M. Eisler, 2003. Comparative study on the clinical, parasitological and molecular diagnosis of bovine trypanosomosis in Uganda, Derstepoort. J. Vet. Res., 70(3): 213-218.
22. Bengaly, Z., I. Sidibe, R. Ganaba, M. Desquesnes, H. Boly and L. Sawadogo, 2002. Comparative pathogenicity of three genetically distinct types of *T. congolense* in cattle: clinical observations and haematological changes. Vet. Parasitol, 108: 1-19.
23. Magona, W., J. Walubengo and T. Odimin, 2008. Acute haemorrhagic syndrome of bovine trypanosomosis in Uganda. Acta. Trop., 107: 186-191.
24. Bett, B., C. Orenge, P. Irungu and K. Munga, 2004. Epidemiological factors that influence time to treatment of trypanosomosis in Orma Boran cattle raised at Galana Ranch, Kenya. Vet. Parasitol, 120: 43-53.
25. OIE, 1982. Report on the 8 meeting of the international working group Trypanosoma evansi. Paris.
26. Pays, E., S. Lips, D. Nolan, L. Vanhamme and D. Perez-Morga, 2001. The VSG expression sites of Trypanosoma brucei: Multipurpose tools for the adaptation of the parasite to mammalian hosts. Mol. Biochem. Parasitol, 114: 1-16.
27. Langousis, G. and L. Hill, 2014. Motility and more: the flagellum of T. brucei. Nat. Rev. Microbiol., 12(7): 505-518.
28. Brun, R., J. Blum, F. Chappuis and C. Burri, 2010. Human African trypanosomiasis. Lancet, 375: 148-159.
29. CFSPH, 2009. African animal trypanosomosis, Nagana, Tsetse disease, Tsetse fly Disease, Iowa State University and Ames, Iowa, 5: 11.
30. Worku, Z., B. Eticha, D. Tesfaye, T. Kifile, K. Gurmesa and N. Ibrahim, 2017. A study on prevalence of bovine trypanosomosis and associated risk factors in Mao Komo special district of Benishangul Gumuz regional state, Western Ethiopia. European Journal of Biomedical Science, 9(2): 85-92.
31. Van den Bossche, P. and V. Delespau, 2011. Options for the control of tsetse transmitted livestock trypanosomosis. An epidemiological perspective. Vet. Parasitol, 181: 37-42.

32. Hunt, R., 2010. Microbiology and Immunology: Parasitology. The University of South Carolina, pp: 1-6.
33. Nwoha, O., O. Eze and M. Anene, 2013. Serum biochemical and liver enzymes changes in dogs with single and conjunct experimental infections of *T. brucei* and *A. caninum*. *Afr. J. Biotechnol.*, 12(6): 618-624.
34. Elnasri, H., 2005. Prevalence and Ranking of Bovine trypanosomiasis in Unity State, Sudan, University of Khartoum, Faculty of Veterinary Medicine, Unity State, Sudan, pp: 1-76.
35. Gonzatti, M., B. González, P. Aso and A. Reyna-Bello, 2014. *Trypanosoma vivax* and Trypanosomosis in Latin America: secadera/huequera/cacho hueco. In: Magez, S., Radwanska, M. (Eds.), *Trypanosomes and Trypanosomiasis*. Springer, pp: 261-285.
36. Cannor, R., P. Vanden Bossche, J. Coetzer and R. Tustin, 2004. *Infectious diseases of livestock*, 2nd (Edn), Cape Town: Oxford University Press, 1: 251.
37. Cauchard J., A. Soldan, A. Madeline, P. Johnson, Büscher, P. and S. Petry, 2014. Interlaboratory ring trials to evaluate serological methods for dourine diagnosis. *Vet. Parasitol.*, 205: 70-76.
38. Deborggraeve S. and P. Buscher, 2010. Molecular diagnostics for sleeping sickness: what is the benefit for the patient? *Lancet. Infect. Dis.*, 10: 433-9.
39. Bezie, M., M. Girma, S. Dagnachew, D. Tadesse and G. Tadesse, 2014. African trypanosomes: virulence factors, pathogenicity and host responses. *J. Vet. Adv.*, 4(11): 732-745.
40. Taylor, A. and E. Authié, 2004. Pathogenesis of animal trypanosomiasis. In: I. Maudlin, P. Holmes and M. Miles (eds): *The Trypanosomiasis*. CABI Publishing. Wallingford, UK, 331- 353. Taylor, M.A. Coop R.L and. Wall, R.L., 2007. *Vet. Parasitol.* 3rd ed. UK: Blackwell publishing, pp: 787-788.
41. Organization of Internal Des Epizootics, 2021. Nagana infections with salivarian Trypanosomes (excluding *T. evansi* and *T. equiperdum*). OIE Terrestrial Manual, 4-10. *Parasitol.* 3rd ed. UK: Blackwell publishing, pp: 787-788.
42. Ndao, M., V. Pandey, J. Zinsstag, K. Pfister and N. Vanmeirven, 1995. Evaluation of Sodium Dodecyl-Sulfate (SDS) as a Hemolytic Agent for the Detection of Microfilariae and Trypanosomes in the Blood of Cattle. *Annalesde la Societe Belge de Med. Trop.*, 75: 145-148.
43. Geysen, D., V. Delespoux and S. Geerts, 2003. PCR-RFLP using *Ssu-rDNA* amplification as identification of animal trypanosomes. A review and perspectives. *Vet. Parasitol.*, (109): 213-231.
44. Chappuis, F., L. Loutan, P. Simarro, V. Lejon and P. Buscher, 2005. Options for the field diagnosis of human African trypanosomiasis. *Clinic. Microbiol. Rev.*, (10): 133-146.
45. Salas, J., M. González, M. Noa, N. Pérez and G. Díaz, 2003. Organophosphorus pesticide residues in Mexican commercial pasteurized milk. *J. Agric. Food Chemistry*, 51(15): 4468-4471.
46. Mekuria, S. and F. Gadissa, 2015. Survey on bovine trypanosomosis and its vector in Metekel and Awi Zones of Northwest Ethiopia. *Acta. Tropica*, 117(2): 146-151.
47. Keno, M., 2005. The current situation of tsetse and trypanosomosis in Ethiopia, Ministry of Agriculture and Rural Development, Veterinary service department, in proceeding of 28th meeting of International Scientific Council for Trypanosomosis Research and Control. Kenya. *PLoS*; 5:e8628. doi.org/10.1371/journal.pone.0008628.
48. Leta, S., G. Alemayehu, Z. Seyoum and M. Bezie, 2016. Prevalence of bovine trypanosomosis in Ethiopia: a meta-analysis. *Parasites and Vectors*, 9: 139.
49. Uilenberg, G., 1998. A field guide for diagnosis Treatment and Prevention of African animal trypanosomosis; W.P. FAD, Rome, pp: 43-35.
50. Namangala, B., 2011. How the African trypanosomes evade host immune killing. *Parasite and Immunology*, 33(8): 430-437.
51. Desta, T., W. Ayalew and B. Hegde, 2011. Breed and trait preferences of Sheko cattle Keepers in South-Western Ethiopia. *Tropical Animal Health and Production*, 43(4): 851-856.
52. Reichard, E., 2002. Area wide biological control of disease vectors and agents affecting wildlife. *Rev. Sci. Tech. Off. Int. Epiz.*, 21(1): 179-185.
53. Hendrickx, G., S. de la Rocque and R.C. Mattioli, 2004. Long-term tsetse and trypanosomiasis management options in West Africa. PAAT technical series, pp: 6.
54. Bouyer, J., F. Stachurski, A.S. Gouro and R. Lancelot, 2009. Control of bovine trypanosomosis by restricted application of insecticides to cattle using foot baths. *Vet. Parasitol.*, 161: 187-193.
55. Djiteye, A., K. Moloo, K. Foua, M. Touré, S. Boiré, S. Bengaly, E. Coulibaly, M. Diarra, D. Traore, I. Ouattara and Z. Coulibaly, 1997. Réactualisation des données sur la répartition des glossines au Mali. *Rev. Elev. Méd. Vét. Pays Trop.*, 50: 126-132.

56. De la Rocque, S., F. Michel, D. Cuissance, G. de Wispeleare, X. Augusseau, S. Guillobez and M. Arnaud, 2001. La risquetrypanosomien, une approche globale pour une décision locale. Montpellier, Edition du CIRAD, Pp, 152.
57. Van den Bossche, P., S. Chitanga, J. Masumu, T. Marcotty and V. Delespaux, 2011. Virulence in *T. congolense* Savannah subgroup. A comparison between strains and transmission cycles. *Parasit. Immunol.*, 33: 456-460.
58. Shaw, P., G. Cecchi, R. Wint, C. Mattioli and P. Robinson, 2014. Mapping the economic benefits to livestock keepers from intervening against bovine trypanosomosis in Eastern Africa. *Prev. Vet. Med.*, 113(2):197-210.
59. Cecchi, G., M. Paone, U. Feldmann, M.J. Vreysen, O. Dially and R.C. Mattioli, 2014. Assembling a geospatial database of tsetse-transmitted animal trypanosomosis for Africa. *Parasites and Vectors*, 7: 1-39.
60. Kukla, B., P. Majiwa, J. Young, S. Molloo and O. Olemoiyoi, 1999. Use of Species- Specific DNA Probes for Detection and Identification of Trypanosome Infection in Tsetse-Flies. *Parasitol.*, 95: 1-16.
61. Swallow, B., 2000. Impact of Trypanosomiasis on African Agriculture. PAAT Technical and Scientific Series, FAO. Rome. *Int. J. Anim. Vet. Ad.*, 2: 47-50.
62. Achukwi, M.D. and G.A. Musongong, 2009. Trypanosomosis in the Doayo/Namchi (*Bos Taurus*) and zebu White Fulani (*Bos indicus*) cattle in Faro Division, North Cameroon. *J. App. Biosc.*, 15: 807-814.
63. Magez, S., G. Caljon, T. Tran, B. Stijlemans and M. Radwanska, 2010. Current status of vaccination against African trypanosomiasis. *Parasitol.*, 137: 2017-2027.
64. Holmes, P., 2013. Tsetse-transmitted trypanosomes, their biology, disease impact and control. *J. Inverteb. Pathol.*, 112: 11-14.
65. Aksoy, S., W.C. Gibson and M.J. Lehane, 2003. Interactions between tsetse and trypanosomes with implications for the control of trypanosomiasis. *Adv. Parasitol.*, 53: 1-83.
66. Gooding, R. and E. Krasfur, 2005. Tsetse Genetics: Contributions to Biology, Systematics and Control of Tsetse Flies. *Tsetse Genetics: Contributions to Biology, Systematics and Control of,* Annu. Rev. Entomol, (50): 101-123.
67. Wamwiri, N. and E. Changasi, 2016. Tsetse Flies (*Glossina*) as Vectors of Human African Trypanosomiasis: A Review, *Biomed Res. Int.*, 2016: 8.
68. Kotye, D., 2006. A comparative study on the ecology of tsetse flies (diptera: Glossinidae) in the wabe and walga river systems, Addis Ababa University.
69. Bangu, B. and E. Eyob, 2017. The Distribution of Tsetse Flies Species and other Biting Flies in Mareka District of Dawuro Zone, Southern Ethiopia, *Int. J. Adv. Res. Biol. Sci.*, 4(10): 10-14.
70. Shumago, N. and W. Tekalign, 2016. Distribution of Tsetse Fly in Selected Sites of Upper Omo Belt, *Adv. Life Sci. Technol.*, 44: 30-37.
71. Hordofa, K. and G. Haile, 2017. A review on epidemiological distribution, impacts and integrated control approach of tsetse fly, *J. Parasitol. Vector Biol.*, 9(9): 122-131.
72. Vreysen, J., M. Talla, B. Sall and J. Bouyer, 2012. Tsetse flies: Their biology and control using area wide integrated pest management approaches, *J. Invertebr. Pathol.*, 112: 15-25.
73. Krasfur, S., 2010. Tsetse flies: Genetics, evolution and role as vectors. *Infection, Genetics and Evolution*, 9(1): 124-141. [https:// doi.org/ 10.1016/ j.meegid.2008.09.010](https://doi.org/10.1016/j.meegid.2008.09.010).
74. Taye, M., K. Belihu, M. Bekana and D. Sheferaw, 2012. Assessment of impacts of tsetse and trypanosomosis control measures on cattle herds? composition and performance in Southern Region, Ethiopia. *Trop. Anim. Health. Prod.*, 44: 1759-1763.
75. Fikru, R., B. Goddeeris, V. Delespaux, Y. Moti and A. Tadesse, 2012. Wide spread occurrence of *Trypanosoma vivax* in bovines of tsetse as well as non-tsetse infested regions of Ethiopia. *Vet. Parasitol.*, 190: 355-361.
76. Geja, G., G. Terefe and K. Belihu, 2012. Impact of tsetse and trypanosomiasis control on cattle herd composition and calf growth and mortality at Arba-Minch District (Southern Rift Valley, Ethiopia). *Trop. Anim Health Prod.*, 44(7): 1745-50.