Acta Parasitologica Globalis 12 (2): 24-31, 2021
ISSN 2079-2018

DOI: 10.5829/idosi.apg.2021.24.31

Review on Trypanosomosis in West Gojjam Zone, Amhara Region, Ethiopia

Gizachew Nibret Tilahun and Ayana Selamu Dessie

National Institute for Tsetse Fly and Trypanosomosis Control and
Eradication Ethiopia, P.O. Box: 19917

Abstract: West Gojjam zone is one of Amhara region with sixteen woredas and three city administrations of which five woredas were infected with more than one species of tsetse flies. Known species in this zone are five in number namely *Glossina pallidipes*, *G. morsitans*, *G. fuscipes*, *G. tachinoides* and *G. longipennis*. Most tsetse transmission is mechanical and cyclic and begins when blood from a trypanosome-infected animal is ingested by the fly. 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. The methods of tsetse fly control involved bush clearing, elimination of game animals on which tsetse feed, impregnated targets, pour on and the sterile male technique (sterile insect techniques). Since female tsetse only mates once in a lifetime, this technique is theoretically able to eradicate a targeted tsetse species. Trypano-tolerant 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.

Key words: Animals · Trypanosomiasis · Tsetse Fly · Protozoa

INTRODUCTION

Trypanosomosis has long been recognized as a massive constraint on animal husbandry, livestock production and mixed farming in vast rural areas [1]. Ethiopia is known for its large and diverse livestock resource endowments. Livestock is primarily kept on small holdings where it provides drought power for crop production, manure for soil fertility and fuels, serves as a source's family diet and sources of cash income (from livestock and livestock products). Despite large livestock population, Ethiopia fails to optimally utilize this resource due to different constrains facing the livestock subsector [2]. Tsetse flies (Glossina) inhabit wide range of habitats covering over 10 million km2 representing 37% of the African continent and affecting 37 countries [3] including Ethiopia.

Currently, in Ethiopia about 220, 000 km^2 area is infested tsetse flies namely *Glossina pallidipes*, *Glossina morsitans*, *Glossina fuscipes*, *Glossina tachinoides* and *Glossina longipennis* [4]. The most important trypanosome species affecting livestock in Ethiopia are *Trypanosoma congolense*, *Trypanosoma vivax* and *Trypanosoma brucei*, in cattle sheep and goat, *Trypanosoma evansi* in camel and *Trypanosome equiperdum* in horse [5]. In west gojjam zone, trypanosomosis is considered an important disease of cattle [6- 8] but systemic studies have not yet been carried out on the epidemiology, prevalence and economic significance of trypanosomosis in this site. Since more than 90% of crop production in Ethiopia are dependent on animal draught power mainly on ploughing oxen, many large fields lie fallow due to lack of these animals in trypanosomiasis infested area [9], which worsen the food supply and living conditions in affected areas.

Corresponding Author: Gizachew Nibret Tilahun, National Institute for Tsetse Fly and Trypanosomosis Control and Eradication. Ethiopia, P.O. Box: 19917.
Tel: +251922442259/+251912146597.

Trypanosomes are flagellated protozoan parasites that live in the blood and other body fluids of vertebrate hosts [10]. Trypanosome is one of the diseases that are caused by this flagellated protozoal parasite belonging to the genus trypanosome [11]. This group of diseases caused by protozoa of the genus Trypanosoma affects all domestic animals. *T. brucei* rhodesiense and *Trypanosoma brucei* gambiense are zoonotic, with people as the predominant host. Animals are mainly affected by tsetse-transmitted trypanosomes and in geographic areas where tsetse-transmitted trypanosomiasis occurs [12]. In west gojjam zone, trypanosomosis is more widespread in domestic livestock in associated with abay river basin [13, 14]. The objective of this paper is to summarize the available research evidence on trypanosomosis and reveals the gap that needs future research attention.

Trypanosomiasis: Trypanosomosis 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 [15-17]. Animal trypanosomosis and its vectors occur in vast areas with devastating impact on livestock productivity [18].

Etiology: Trypanosomosis is a disease that affects animals resulting from infection with protozoa of the genus Trypanosoma transmitted primarily by tsetse fly and also by other haematophagous fly [19]. *T. vivax*, *T. equiperdum*, *T. congolense*, *T. brucei* brucei and *T. simiae* are the main species responsible for trypanosomes affecting virtually domestic mammals. *T. vivax* and *T. congolense* are the main pathogens of cattle [20].

Epidemiology of Trypanosomosis: Trypanosomosis is an important disease of livestock in Ethiopia [21]. There are six pathogenic *T. equiperdum* and *T. rhodesiense* but the most species of trypanosomes are discovered in Ethiopia, which are namely *T. vivax*, *T. congolense*, *T. brucei*, *T. evansi* and important trypanosomes which are found in the study area are *T. vivax* and *T. congelense* [5, 22].

Its epidemiology and impact on livestock especially cattle production are determined largely by the prevalence and distribution of the disease and its vectors in the affected area [23].

Modes of Transmission, Vectors and Ecological Preference: The epidemiology of trypanosomiasis is determined mainly by the ecology of the tsetse fly which is found only in tropical Africa [20]. Most tsetse-fly transmission is cyclic and begins when blood from a trypanosome-infected animal is ingested by the fly. The trypanosome alters its surface coat, multiplies in the fly, then alters its surface coat again and becomes infective [12]. Tsetse flies (genus Glossina) are restricted to Africa from about latitude 15° N to 29° S. 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 [12]. The riverine species (*G. palpalis*, *G. tachinoides* and *G. fuscipes*) are important as vectors of bovine [20].

The prevalence of some Tabanus spp. all year round ensures that transmission of the parasite occurs wherever reservoirs hosts, vectors and susceptible hosts co-exist. This finding may explain the sporadic occurrence of the disease during the dry season and outbreaks during the rainy season [24]. However, the efficiency of the different flies to transmit Trypanosomes appears to vary in different geographic conditions and is also dependent on the interval between two successive feeds and intensity of the fly challenge [25]. Although the mode of mechanical transmission is well established, its dynamics are not understood. Therefore, considerable experimentation is still required to attempt to define quantitatively the effect of the host species, the duration of infection in the host and the level of parasitaemia, the period between feeds and the relative efficiency of different vector species in ensuring successful transmission [26].

Pathology and Pathogenesis: Infected tsetse inoculates metacyclic trypanosomes into the skin of animals, where the trypanosomes reside for a few days and cause localized inflammation (chancres). They enter the lymph and lymph nodes, then the bloodstream, where they divide rapidly by binary fission. In T congolense infection, the organisms attach to endothelial cells and localize in capillaries and small blood vessels. *T. brucei* species and *T. vivax* invade tissues and cause tissue damage in several organs [12, 20]. Antibody developed to the glycoprotein coat of the trypanosome kills the trypanosome and results in the development of immune complexes. Antibody, however, does not clear the infection, for the trypanosome has genes that can code for many different surface coat glycoproteins and change its surface glycoprotein to evade the antibody [27].

Anemia: Anemia is a major component of the pathology of trypanosomiasis generally. Anemia in trypanosomes infections is reportedly macrocytic and hypochromic [28]. In the early phases of infection, the anemia is hemolytic and haemophagocytic. The mechanism(s) responsible for this increased erythrophagocytic activity are not fully understood. Several have been proposed, immune complexes, expanded mononuclear phagocytic system, hemolytic factor produced by the trypanosome, fever and disseminated intravascular coagulation [29]. In the late stages, anemia continues to be a major factor, with probably additional causes. However, irrespective of the cause of anemia the primary abnormality of function are the anoxic conditions created by the persistent anemia. Following this are signs of dysfunction which appear in the various organs. An increase in cardiac output due to increases in stroke volume and heart rate and a decrease in circulation time are obvious manifestations. The central nervous system is reported to be most susceptible to anoxia with consequent development of cerebral anoxia and dullness, appear to be the results of brain tissue disturbance or damage by the parasites. Evidence of Trypanosome evansi being found in the cerebrospinal fluid has been presented [30].

Tissue Damages: The atypical lesions of multiple necrotic foci found in the liver and spleen, as well as generalized lymphoid tissue hyperplasia suffering from trypanosomiasis on post mortem, could be attributed to pathological events that occur in the tissues of animals infected with causative agent. The degenerative changes thus observed could be due to tissue anoxia, possibly caused by anemia, which results in a fall in tissue pH and vascular damage. Other mechanisms may also be involved.

It is known that *T. evansi* is a member of the brucei group of trypanosomes, which have a known preference for connective tissues of a host, where they disrupt the collagen bundles and destroy the fibroblasts which produce and maintain the collagen. This disruption of host connective tissues, along with the vascular damage attributable to brucei group trypanosomes [31], would be expected to release large quantities of cytoplasmic and mitochondrial enzymes into the serum, thereby causing further tissue damage.

Indeed, a two-step process in the pathology of infection with trypanosomes based on studies of changes in serum enzymes has been proposed [31]. The first step coincides with the appearance of trypanosomes in the host bloodstream and is characterized by a sharp and as yet unexplained rise in sorbitol dehydrogenase (SDH) activity. The second step occurs later in the infection and is characterized by a large increase in serum levels of glutamic oxaloacetic transaminase (GOT) now known as aspartate alanine transferase (AST) [32] and a smaller rise in glutamic pyruvic transaminase (GPT), now known as alanine amine transferase (ALT) [32].

The rise in AST level can be attributed partly to cellular damage caused by the trypanosomes lysis, while the increase in ALT probably results from host destruction of trypanosomes. AST is found mostly in cell organelles and rises when there is a great damage to the heart, kidney, skeletal muscles and liver. ALT is a specific liver enzyme found in the cell cytoplasm and its rise is associated with cell membrane damage. The reported increases in these enzymes, especially AST, is not surprising as it is indicative of organ damage and supports the post mortem reports of necrotic foci in the liver and spleen of animals suffering from trypanosomosis. The fever characterized by high temperature might be due to the effects of toxic metabolites produced by dying trypanosomes [33]. In addition, the edema reported in the dependent parts of the body during the chronic stage could be due to a significant decrease in the albumin levels, resulting in alterations in osmotic pressure of the blood. This leads to excessive accumulation of fluid in tissue spaces caused by a disturbance in the mechanism of fluid interchange between capillaries, the tissue spaces and the lymphatic vessels. All this possibly indicates great liver damage [32]. The hemorrhage and serous exudates that occurred could be caused by hemolysis involving the expanded mononuclear phagocytic system. This has also been observed in Trypanosoma brucei-infected donkeys, while the frequent abortions reported may be attributed to endocrine dysfunction [34].

Clinical Signs: 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 [20]. Severity of disease varies with species, age of the animal infected and the species of trypanosome involved. The incubation period is usually 1 to 4 weeks. The primary clinical signs are intermittent fever, anemia and weight loss. Cattle usually have a chronic course with high mortality, especially if there is poor nutrition or other stress factors [12]. The anemia results in a progressive drop in packed cell volume, a non-specific but useful indicator in endemic areas [20]. Necropsy findings vary

and are nonspecific. In acute and fatal cases, extensive petechiation of the serosal membranes, especially in the peritoneal cavity, may occur. Also, the lymph nodes and spleen are usually swollen [12].

The disease is manifested by elevation of body temperature which is directly associated with parasitaemia. Infected animals show progressive anemia, marked depression, dullness, loss of condition, rough hair coat, emaciated, swollen superficial lymph nodes and often rapid death. Anemia was observed to be a major clinical finding in animals'trypanosomisis [35]. Milder cases develop recurrent episodes of fever. Some animals develop edema in their dependent parts of the body, urticaria plaques and petechial hemorrhages in serous membranes. Death finally ensues if untreated. However, some may harbor trypanosomes for 2-3 years thus constituting reservoirs of infection to susceptible camels and hosts. Other well documented field reports are death [36]; abortion [37]; weight loss, reduced draught power [25] and nervous signs like circling movement and trembling, unusual aggressiveness, running aimlessly and sudden collapse in severely stressed and over worked animals [38]. At post mortem, necrotic foci in the liver and spleen as well as generalized lymphoid tissue and hyperplasia are common findings [30].

Diagnosis: Definitive diagnosis of the disease is ultimately dependent on the detection of the trypanosome in blood samples from infected animals [5, 39].

Parasitological Diagnosis: A presumptive diagnosis is based on finding an anemic animal in poor condition in an endemic area. 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, looking for motile parasites. Other infections that cause anemia and weight loss, such as babesiosis, anaplasmosis, theileriosis and haemonchosis, should be excluded by examining a stained blood smear [12].

Serological Diagnosis: Various serologic tests measure antibody to trypanosomes, but their use is more suitable for herd and area screening than for individual diagnosis. Rapid agglutination tests to detect circulating trypanosome species-specific antigens in peripheral blood are available for both individual and herd diagnosis, although their reliability remains varied [12]. Another alternative is a series of standard serological tests to detect anti-trypanosome antibodies in sera or other body fluids. The three tests used most often are the indirect immuno-fluorescent antibody test (IFAT), the capillary agglutination test (CAT) and the ELISA [20].

Molecular Technique: Molecular techniques for trypanosome detection and differentiation have been developed, but they are not generally available for routine field use [12]. Dried blood spots on filter papers are also a useful source of DNA for the detection of *T. congolense* and *T. brucei* by Trypanosome and Trypanosomiasis PCR but the test is expensive and can only be done in specialized laboratories [20].

Treatment: Therapeutic drugs for the treatment of trypanosomiasis includes diminazen aceturate, homidium bromide and homidium chloride. Prophylactic drugs for cattle include homidium bromide, homidium chloride and isometamidium [40].Treatment with trypanocidal drugs such as quinapyramine and Cymelarsen are effective against *T. evansi* infections in cattle and horses [41]. Trypan, which is a Formulation, containing diminazenediaceturate (diamidinophenyltriazene diaceturatetetrahydrate), phenazone and procaine hydrochloride is effective against *T. evansi* infections, as well as infections with Trypanosoma Congolese, *Trypanosoma vivax* and *Trypanosoma brucei*.

The drug is also effective against Babesia bigemina, Babesia canis or other Babesia and Theileri annulata [42]. It has a synergistic and an additive effect in comparison with other trypanocidal drugs and is reported to have a painless, antipyretic and long-lasting effect. It has also been adjudged as being the most effective trypanocidal drug to date [42]. Trypan can be used for curative and preventive treatment. On the whole, control of trypanosomiasis requires treatment of infected animals with effective drugs and reducing blood sucking flies by regular insecticide treatment.

Prevention and Control of Trypanosomosis

Parasite Control: The control of trypanosomosis in enzootic countries involves control of tsetse fly population, prophylactic treatment, good husbandry of animals at risk and use of trypano-tolerant animals [20]. Most have a narrow therapeutic index, which makes administration of the correct dose essential [12]. Therapeutic drugs for the treatment of trypanosomiasis includes diminazen aceturate, homidium bromide and homidium chloride. Prophylactic drugs for cattle include homidium bromide, homidium chloride and isometamidium [40].

Vector Control: Till date five species of Glossina namely *G. morsitans* submorsitans, *G. pallidipes*, *G. fuscipes* fuscipes, *G. tachinoides* and G. longipennis are known to exist in Ethiopia. These vectors cyclically transmit four species of trypanosomes (*T. congolense*, *T. vivax* and *T. brucei* of livestock and *T. rhodesiense* of human). The occurrence of trypanosomosis in the region was attributed to the existence of cyclical vectors, *G. pallidipes* and *G.f. fuscipes*. However, *G. pallidipes* was the predominant and most widely distributed vector [43]. Control of tsetse fly by impregnated target and pour on has been attempted in the study areas, but reinvasion is frequent if the land is not properly utilized. The earliest methods involved bush clearing and elimination of game animals on which tsetse feed [20]. Another method is the sterile male technique. Since the female tsetse only mates once in a lifetime, this technique is theoretically able to eradicate a targeted tsetse species in areas where other methods have been used to reduce its density, but it is expensive [19].

Host Resistance Protection: Trypano-tolerant animals are being used to establish ranches in areas where tsetse challenge is not too heavy, but they have not been readily accepted in some countries, supposedly because they are smaller in size and they produce less milk than other indigenous breeds and crosses with Exotic breeds [20]. They are infected by tsetse flies but do not show clinical disease. Cross breeding is however a common practice [10]. The four Ethiopian cattle breeds Abigar, Gurage, Horro and Sheko in aspects are related to trypano-tolerance [27].

Public Health Importance of Trypanosomiasis: This protozoan is the cause of sleeping sickness in infected individuals and is fatal if left untreated [44]. The effects of this disease are recognized by the World Health Organization (WHO) and several programs and campaigns have been formed to eradicate this disease [45]. Nevertheless, this disease remains one of the most regulated tropical diseases and steps should be taken to prevent transmission of the disease and treat infected individuals with better treatment options [46].

The evolution of the human Trypanosomiasis is likely an even more recent event since humans are resistant to the older animal infectious strains [47]. In 1901, Robert Michael Forde was the first to observe trypanosomes in the blood of humans. This was identified and classified as Trypanosoma brucei gambiense in 1902 by Joseph Everett Dutton. In 1910, the other human disease causing strain, (4) Trypanosoma brucei rhodesiense, was identified by John William Watson Stephens and Harold Benjamin Fantham [47].

The two different strains of the same species (gambiense and rhodesiense) infect human populations only, but are found in slightly different habitats and reservoirs. The gambiense strain is an anthroponotic strain causing the chronic form of sleeping sickness that transmits from human-to human and humans function as the main reservoir [44]. The rhodesiense strain on the other hand is a zoonotic strain and causes the acute form of sleeping sickness that transmits from animal to human [44]. Many animals (in particular the bush pig) can act as reservoirs for this strain [48].

Symptoms of the disease occur according to the phase of the trypanosome within the infected individual. In phase one (haemolymphatic stage), the patient may exhibit some of the following symptoms: fevers, skin eruptions, irregular febrile episodes, headaches, malaise, exhaustion, anorexia, extreme thirst, muscle and joint pains, anemia, rash, pruritus, deep hyperesthesia, feelings of coldness, lack of appetite, hyperplasia, polydipsia and impotence, amenorrhea and day time somnolence or night-time insomnia [49]. In the second stage (meningoencephalitic stage), when the patient will exhibit the symptoms depends on the subspecies of Trypanosomiasis brucei infection. The following symptoms can appear within months or over years: the symptoms observed during the haemolymphatic stage, headaches, sensory disturbances and display of primitive reflexes, exaggerated deep tendon reflexes, psychiatric disorders (e.g., aggression, euphoria and mutism), tremor, disruption of body temperature regulation and cortisol and prolactin or growth hormone secretion disruptions [49]. At the terminal stage of illness, CNS demyelination and atrophy, dementia, epileptic fits and finally death, are observed [49].

Economic Significance of Trypanosomiasis: Trypanosomiasis has long been recognized as a massive constraint on animal husbandry, livestock production and mixed farming in vast rural areas [1]. Ethiopia is known for its large and diverse livestock resource endowments. Livestock is primarily kept on small holdings where it provides drought power for crop production, manure for soil fertility and fuels, serves as a source's family diet and sources of cash income (from livestock and livestock products). Despite large livestock population, Ethiopia fails to optimally utilize this resource due to different constrains facing the livestock subsector [2].

Since more than 90% of crop production in Ethiopia are dependent on animal draught power mainly on ploughing oxen, many large fields lie fallow due to lack of these animals in trypanosomiasis infested area [9], which worsen the food supply and living conditions in affected areas. Tsetse flies infest 10 million square kilometers of Africa involving 37 countries. Hence, nagana is today the most important disease of livestock in the continent [39]. 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 [20, 50]. The disease in Africa costs livestock producers and consumers an estimated US$1340 million each year [40].

CONCLUSSION AND RECOMMENDATIONS

Trypanosomiasis is an important disease and a potential threat affecting the health and productivity of cattle in fertile areas of jabitehinan, womberma, dembecha with and some parts of buries district in west gojjam zone which is caused by *T. congolense* and *T. vivax* was found to be an important disease of cattle. Prevalence of trypanosomiasis progress is high in bovine and impact of the disease on productivity of infected animals. The prevalence of the disease varies from site to site. Infection with trypanosomiasis negatively affects PCV and body condition. This indicated that trypanosome infection of cattle in the study areas causes loss of body weight and production. Further study on the occurrence of tsetse and trypanosomiasis at different season of the year, at different altitude and different species of animals should be conducted. Since trypanosomiasis 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 and restriction movement density population of tsetse.
- Increase awareness creation those animals rearing society especially pastoral community.
- Importing modern and latest drugs for trypanosomosis.
- Application of validated, standardized diagnostic tests (particularly penside tests) at both local and regional levels.
- Identification of the principal vector species responsible for transmission in different ecological situations.
- Investigating the potential for vector control in the management of this disease.
- Determining which factors that precipitate epidemic outbreaks of disease.

REFERENCES

1. Oluwafemi, R.A., 2014. Tsetse/trypanosomosis challenges and rural poverty in Africa: Implications for food security. Res. J. Agric. Environ. Manage., 3(3): 158-161.
2. Bezabih, M., Z. Shabula and N. Beyene, 2015. Prevalence of bovine trypanosomiasis in Dara District Sidama Zone, Southern Ethiopia. J. Parasitol. Vector Biol., 9(9): 132-136.
3. Finelle, P., 1980. Programme for the control of African Trypanosomiasis and related development. In: Isotope and Radiation Research on Animal Disease and their Vectors, IAEA. Vienna, pp: 3-14.
4. MOA, 1995. Ruminant Livestock Development Strategy (RLDS), Ministry of Agriculture, Addis Ababa, Ethiopia, pp: 10.
5. Abebe, G., 2005. Current situation of Trypanosomosis, in: review article on: Trypanosomosis in Ethiopia. Ethiop. J. Biol. Sci., 4: 75-121.
6. Cherenet, T., R.A. Sani, J.M. Panandam, S. Nadzr, N. Speybroeck and P. Van Den Bossche, 2004. Seasonal prevalence of bovine Trypanosomosis in a tsetse infested zone and a tsetse-free zone of the Amhara Region, north-west Ethiopia. Onderstepoort J. Vet. Res., 71(4): 307-12.
7. Shimelis, D., A.K. Sangwan and A. Getachew, 2005. Epidemiology of tsetse transmitted Trypanosomosis in Abay (Blue Nile) basin of North West Ethiopia. Revue Elev. Vets. Pays Trop., 58(3): 151-157.
8. Sinshaw, A., 2004. Prevalence of trypanosomosis of cattle in three woreda of Amhara Region. Msc thesis, FVM, AAU, DebreZeit.
9. Kenaw, B., G. Dinede and T. Tollosa, 2015. Bovine Trypanosomosis in Asossa District, Benishangul Gumuz Regional State, Western Ethiopia: Prevalence and Associated Risk Factors. European J. Appl. Sci., 7(4): 171-175.
10. Office Internationaledes Epizooties (OIE), 2008. “Standardized techniques for the diagnosis of tsetse transmitted trypanosomosis”. OIE Terrestrial Manual, Rome, Italy.
11. Jember, T. and B. Mitiku, 2013. Prevalence of Bovine Trypanosomosis and Distribution of Vectors in HawaGelan District, Oromia Region, Ethiopia. J. Vet. Adv., pp: 238-244.

12. Merck Veterinary Manual, 2005. Kahn and Line ed, (9th edition), Wiley Publishers, Merck and Co., Inc: White house station, USA, pp: 18-35.
13. Tekle, Y., 2012. Prevalence of bovine trypanosomosis in tsetse controlled and uncontrolled areas of Eastern Wollega, Ethiopia, Priory Lodge Education Limited, www.priory.com.
14. Langridge, W.P., 1976. A tsetse and trypanosomiasis survey of Ethiopia, Addis Ababa, Ethiopia, Ministry of Overseas Development of British and Ministry of Agriculture of Ethiopia, pp: 97.
15. Taylor, K.A., 1998. Immune responses of cattle to African trypanosomes: protective or pathogenic? Int. J. Parasitol., 28: 219-240.
16. Uilenberge, G., 998. A field guide for diagnosis, treatment and prevention of African animal Trypanosomosis. Adopted from the original edition by Boyt W.P. FAO, Rome, pp: 43-135.
17. Tesfaye, M., 2002. Report of Trypanosome infection rate in G. m. morstans and *G. tachninoides* in Didessa Valley from July 29 to Sept. 26/2002. Bedelle.
18. ILRAD, 1994. Annual Report of the International Laboratory for Research on Animal Disease. Nairobi, Kenya, pp: 21-30.
19. Urquart, G.M., J. Armour, J.L. Dunncan, A.M. Dunn and F.W. Jennings, 1995. Veterinary Parasitology, the University of Glasgow, Elbs ed, pp: 203-212.
20. Radostitis, O.M., C.C. Gay, K.W. Hinch and P.D.C. Cliff, 2007. Disease associated with Trypanosomes. In: Veterinary Medicine, Textbook of disease of cattle, horses, pigs and goats 10th ed. Elsevier, UK, pp: 1531-1554.
21. Alemayehu, B., B. Bogale, T. Fentahun and M. Chanie, 2012. Bovine trypanosomosis: A threat to cattle production in Chena district, Southwest Ethiopia. J. Anim. Sci., 2(4): 287-291.
22. Eticha, B. and A. Aki, 2016. Prevalence of Cattle Trypanosomiasis, Apparent vector density and Associated Risk Factors in Dibate District, Western Ethiopia. Biomed. Nursing 2(4) http://www.nbmedicine.org.
23. PATTEC, 2001. PAN African Tsetse and Trypanosomosis Eradication (PATTEC) Plan of action. June, 28-37.
24. Njiru, Z.K., O. Bett, I.M. Ole-mapeny, J.B. Githiori and J.M. Ndung'u, 2002. Trypanosomosis and helminthosis in camels: comparison of ranch and traditional camel management systems in Kenya. J. Camel Practice Research, 55: 67-71.
25. Luckins, A.G., 1998. Epidemiology of Surra: Unanswered Questions. J. Protozool. Res., 8: 106-119.
26. Obihiro, 1998. Proceedings of RCPMI-Obihiro/OIE Paris International Symposium on Strategies for Research and Control of SurraTrypanosomaevansi infection. J. Protozool. Res., 8: 1-15.
27. Eshetu, E. and B. Begejo, 2015. The Current Situation and Diagnostic Approach of Nagana in Africa: A review. Online (5) 17, 117. Ethiopia J. Nat. Sci. Res. www.iiste.org.
28. Jatkar, P.R. and M.S. Purohit, 1971. Pathogenesis of anaemia in Trypanosomaevansi infection.1. Heamatology. Indian Vet. J., 48: 239-244.
29. Food, Agriculture, Organization, 1979. Pathology and Immunopathology In: The African trypanosomiases. Food Agricultural Organization Animal Production and Health Paper (14).
30. Rottcher, D., D. Schillinger and E. Zwegarth, 1987. Trypanosomiasis in the camel (Camelusdromedarius). Rev. Scientifique Technique, 6: 463-470.
31. Boid, R., 1980. Changes in the levels of some serum enzymes in dromedary camels infected with Trypanosomaevansi. Res. Vet. Sci., 28: 336-340.
32. De La Rue, M.L., G.A. De carli, H.M. Herrera and R.A. M.S. Silva, 1997. Biochemical changes in acute infection of dogs with Trypanosomaevansi. J. Protozool. Res., 7: 28-35.
33. Tizard, I.R., K.H. Nielsen, J.R. Seed and J.E. Hall, 1978. Biologically active products from African trypanosomes. Microbiol. Rev., 42: 661-681.
34. Losos, G.J., 1980. Diseases caused by Trypanosomaevansi: A Review. Vet. Res. Comm., 4: 165-181.
35. Rami, M., T. Atarhouch, M.N. Bendahman, R. Azlaf, R. Kechna and A. Dakkak, 2003. Camel's trypanosomiasis in Morocco 2. A pilot disease control trial. Vet. Parasitol., 115: 223-231.
36. Tuntasuvan, D., N. Sarataphan and H. Nishikawa, 1997. Cerebral trypanosomiasis in native cattle. Vet. Parasitol., 73: 357-363.
37. Lohr, K.F., S. Pholpark, P. Siriwan, N. Leesirikul, N. Srikitjakarn and L.C. Staak, 1986. Trypanosomaevansi infection in buffaloes in north east Thailand. 2. Abortions. Trop. Anim. Hlth. Prod., 18: 103-108.
38. Manuel, M. F., 1998. Sporadic outbreaks of surra in the Philippines and its economic impact. J. Protozool. Res., 8: 131-138.
39. Diseases of Cattle, 1981. Economic and zoonotic relevance. ISBN.978015-689.

40. Ayisheshim, A., S. Abegaz, S. Derso, M. Shewatatek, H. Hailemariam, B. Shiret and M. Natnael, 2015. Review on bovine Trypanosomosis in Ethiopia, European J. Biol. Sci., 8(1): 01-07.
41. Raynaud, J.P., P.L. Toutain T. Blaitz and K.R. Sones, 1989. Plasma kinetics, toxicity and tolerance of Cymelarsin in horses, cattle and camels. 20th ISCTRC Meeting, Mombasa, Kenya, April, 10-14, 1120-1124.
42. Bourdichon, A.J., 1998. Report on the use of the trypanocidal drug 'trypan'. J. Protozool. Res., 8: 258-262.
43. Abebe, R., S. Gute and I. Simon, 2017. Bovine trypanosomosis and vector density in Omo-Ghibe tsetse belt, South Ethiopia. Acta Trop., 167: 79-85.
44. Brun, R. and J. Blum, 2009. Human African Trypanosomiasis. Lancet, 375: 148-159.
45. Simarro, P.P. and A. Diarra, 2011. The Human African Trypanosomiasis Control and Surveillance Programme of the World Health Organization 2000-2009: The Way Forward. PLoSNegl Trop Dis 5, e1007.
46. Schofield, C.J. and J.P. Kabayo, 2008. Trypanosomiasis vector control in Africa and Latin America. Parasites & Vectors, 1: 1-7.
47. Steverding, D., 2008. The History of African trypanosomiasis. Parasites & Vectors, 1: 1-8.
48. World Health Organization (WHO). Tsetse Flies. WHO Biology, 2: 178-192.
49. Vincendeau, P. and B. Bouteille, 2006. Immunology and immunopathology of African trypanosomiasis. Anais da Academia Brasileira de Ciências, 78: 645-665.
50. Jano, A.A., 2016. Study on the Prevalence of Trypanosomes Affecting Bovine in Tsetse Infested Asossa District of BenishangulGumuz Regional State, Western Ethiopia, World Rural Observations, 8(3) http://www.sciencepub.net/rural.