Review On The Animal Trypanosomosis

Asmamaw Aki

Assosa, Regional Veterinary Diagnostic, Surveillance, Monitoring and Study Laboratory, P.O. Box 326, Assosa, Ethiopia; asmamawaki@gmail.com, phone: +251- 902330029

Summary: *Trypanosomosis* is a haemoprotozoan disease, mostly transmitted by the *tsetse fly (Glossina spp*.), which cause severe disease in humans and livestock in Sub-Saharan Africa (SAA). The disease results in loss of livestock and agricultural productivity with serious socio-economic consequences. Six species of trypanosomes are recorded in Ethiopia and the most important trypanosomes in terms of economic loss are the tsetse transmitted species: *Trypanosome congolense, Trypanosome vivax and T. b. brucei*. The pathogenesis of trypanosomosis depends on the pathogenicity of the strain; the host breed, genotype, age, sex, skin type etc. Besides, clinical diagnosis, parasitological, serological and molecular methods with varying degrees of sensitivity and specificity are available for the diagnosis trypanosomosis. Animal Trypanosomosis could be treated by antitrypanosomal drugs. Animal trypanosomosis can be controlled by early treatment of infected animal and vector control. Thus, it is recommended that an appropriate use of antiprotozoal drugs, integrated prevention and control program should be implemented to reduce the impact of *trypanosomosis*.

[Asmamaw Aki. **Review On The Animal Trypanosomosis.** *Rep Opinion* 2025;17(6):1-12]. ISSN 1553-9873 (print); ISSN 2375-7205 (online). http://www.sciencepub.net/report. 01. doi:10.7537/marsroj170625.01

Key words: *Animal, Trypanosomosis, Tsetse fly*

1. INTRODUCTION

Livestock is backbone of the socio economic system of most of the rural communities of Africa (Elnasri H., 2005). Ethiopia is known for its large and diverse livestock resource endowments. Livestock is primarily kept on small holdings where it provide drought power for crop production, manure for soil fertility and fuels, serves as a sources of 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 Shortage of nutrition, reproductive insufficiency, management constraints and animal disease are the major constraints (Bekele. J *et al*., 2010). One of the diseases hampering the livestock subsector is trypanosomosis (Getachew A., 2005).

Tsetse transmitted animal *Trypanosomiasis* is one of the major constraints to socio-economic development in Africa. Tsetse flies infest approximately 10 million km^2 of the continent affecting 38 countries. It is considered that 7 million km^2 of this area would otherwise be suitable for livestock and or mixed agricultural development. About 30% of the 147 million cattle in countries affected by tsetse are exposed to the disease. The situation with regard to sheep, goats, pigs, horses, donkeys and camels is probably similar but is less well documented. Data available at present indicate that the overall situation is deteriorating. Since the 1950's the areas of savanna tsetse infestation have continued to increase. As a result there is increasing pressure on tsetse-free pasturages (Getachew A., 2005).

In Ethiopia, tsetse transmitted animal *Trypanosomosis* is a serious constraint to livestock production and agricultural development, exorcising farmers and livestock keepers out of areas having very high potential for growth, and forcing them to live on a highly degraded highlands of the country (Abebe, 2005). The problem caused by tsetse and *Trypanosomosis* is not only limited to inflicting diseases but also leading to significant negative impacts such as losses due to mortality and morbidity in domestic animals, cost of livestock treatment and tsetse control, and getting rid of draught animals from their infestation areas (Juyal *et al*., 2005).

Trypanosomiasis is a devastating disease of livestock caused by protozoal parasites of the genus *trypanosoma* that inhabits blood and other tissues of vertebrates including animals, wildlife and human (Adam *et al.*, 2003; Gupta *et al*., 2009; Bal *et al*., 2014). It is a vector borne disease that is transmitted biologically by tsetse flies and mechanically by other biting flies (FAO, 2002; OIE, 2009). It is a major constraint contributing to direct and indirect economic losses to crop and livestock production (Abebe, 2005) and has a significant negative impact on economic growth in many parts of the world (Taylor *et al*., 2007; Sharma *et al*., 2013), particularly in sub-Saharan Africa (Cecchi *et al*., 2008).

The most important trypanosome species affecting livestock in Ethiopia are *Trypanosoma congolense*, *Trypanosoma vivax*, and *Trypanosoma brucei* in cattle, sheep and goats, *Trypanosoma evansi* in camels and *Trypanosoma equiperdium* in horses (Abebe G, 2005).
The Diagnosis of trypanosome infection is based on clinical signs; but the clinical signs of the African Animal trypanosomosis are indicative but are not sufficiently pathognomonic. Therefore, standard methods have been developed and applied practically to diagnose the disease in animals. The methods include: direct microscopic examination of blood, either by the wet film method; but it is insensitive (Getachew, 2005). Stained thin and thick smear techniques permit detailed morphological studies and identification of different *Trypanosoma* species by light microscopy. Sensitivity can be improved through parasitological buffy coat techniques of concentration of the parasites by centrifugation and blood inoculating into susceptible laboratory animals (Getachew, 2005).

Therefore, the objectives of this review are,

- To over view the epidemiological information of *trypanosomosis*,
- To highlight the economic impact of the disease, and
- To outline some important control and preventive measure against trypanosomosis.

2. LITERATURE REVIEW

2.1 Overview on Animal Trypanosomosis

Trypanosomes are unicellular parasites and classified as flagellated protozoa from genus trypanosomes of the family *trypanosomatid*e, which belongs to the order kinetoplastide of class *zoomastigophora*. The *zoomastigophora* is classified under the phylum *sarcomastigophora* (Radostits *et al., 2000*).
African Animal *Trypanosomosis* is disease complex caused by tsetse fly transmitted *T. congolense, T. vivax or T.brucei* or simultaneous infection with one or more of these trypanosomoses. African animal trypanosomosis is important in cattle, but can cause serious losses in pig, camels, goat, and sheep (Abebe G, 2005). Infection of animals by one or more of these three African animal trypanosomosis result in acute or chronic disease characterized by intermittent fever, anemia, occasional diarrhea, and rapid loss of condition and often terminate in death in cattle (Keno, 2005).

2.2 Etiology

Trypanosomosis is a protozoan disease of both human and animals caused by different species of the genus trypanosome (Radostits *et al*., 2000). The most important trypanosomes in terms of economic loss in domestic livestock and by the way of cyclical transmission are the tsetse transmitted species such as *T.congolense, T.vivax and T.brucei* (Radostits *et al*., 2000). Closely related *T.brucei* subspecies *T.b.rhodesiense and T.b. gambiense* cause human sleeping sickness.

2.3 Classification of Trypanosomes

The modern classification of trypanosomosis is rearranged in to two sections, the stercoraria which is nonpathogenic to man and animals with few exceptions and the salivaria which is pathogenic to human & other animals. Stercoraria trypanosomes develop as epimastigotes in the midgut (posterior station). In these section of trypanosome, new host are infected by contaminative form, means through infective feces. These trypanosomes are non-pathogenic to man and his livestock and their multiplication in the trypanosome form are discontinuous in the vertebrate host (Kassa, 2005).
The salivarian trypanosomes are more pathogenic to human being and livestock as compared to stercoraria. They are important parasite that develops as trypanosomes (procyclic) in the mid gut of the tsetse flies, migrate interiorly to fly mouth parts (salivary glands) where they develop as epimastigote. They complete their development in the anterior station and the infective metacyclic trypanosomes are introduced to new host by inoculation through the insect mouth part (Radostits, 2000; FAO, 1998).

Table 1. Classification of sub- genus and species of section salivaria

Sub genus	Species
Duttonella vivax group	*T. vivax, T. uniform*
Nanomonas congolense group	*T. congolense, T. simae*
Pyconomonas suis group	*T. suis*
Trypanozoon brucei group	*T.brucei, T.rhodensies, T. gambiense, T. evansi , T. equiperdum, T. equinum*

Source: FAO, 1998, Getachew, 2005

2.4. Morphology and Motility of Trypanosome

Trypanosomes are microscopic, elongated and flattened cell which move with the help of single flagella directed towards, at the base of which is found characteristic structure, the kinetoplast. The length and the position of the trypanosomes flagellum are variable. In trypanosomes from the blood of a host, the flagellum originates near the posterior end of the cell and passes forward over the cell surface to extend freely at the anterior end. When the flagellum is adherent to the cell surface, it sheath is expanded and form away flange, called the undulating membrane (Jemere, 2004).

Trypanosomes move actively and progress by the movement of the undulating membrane and the free flagellum, when present. They are elongated spindle shaped protozoa ranging from 8-39 micro metre. They are characteristically leaf-like in shape, they have simple flagellum and this attached to organisms by undulating membrane (Jemere, 2004).

Motility of each species of parasite can be identified in fresh unfixed blood films. *T. brucei* moves rapidly with in small area of the microscopic field, *T. congolense* moves sluggishly often apparently attached to red blood cells and *T. vivax* moves rapidly across the microscope field (Getachew, 2005; FAO, 1998).

Trypanosome brucei is pleomorphic in form and ranges from long and slender to short and stumpy. The undulating membrane is conspicuous, the kinetoplast is small and sub- terminal and posterior end is pointed. The slender form has well developed free flagellum which in the stumpy form it is either short or absent. *T. congolense* is mono morphic in the form, the undulating membrane is inconspicuous, the medium sized kinetoplast is marginal, there is no free flagellum and the posterior end is blunt. *T. vivax* is monomorphic in form, the undulating membrane is inconspicuous, the largest kinetoplast is terminal and the posterior end is broad and round (Getachew, 2005 ; Bekele. J., *et al*., 2010).

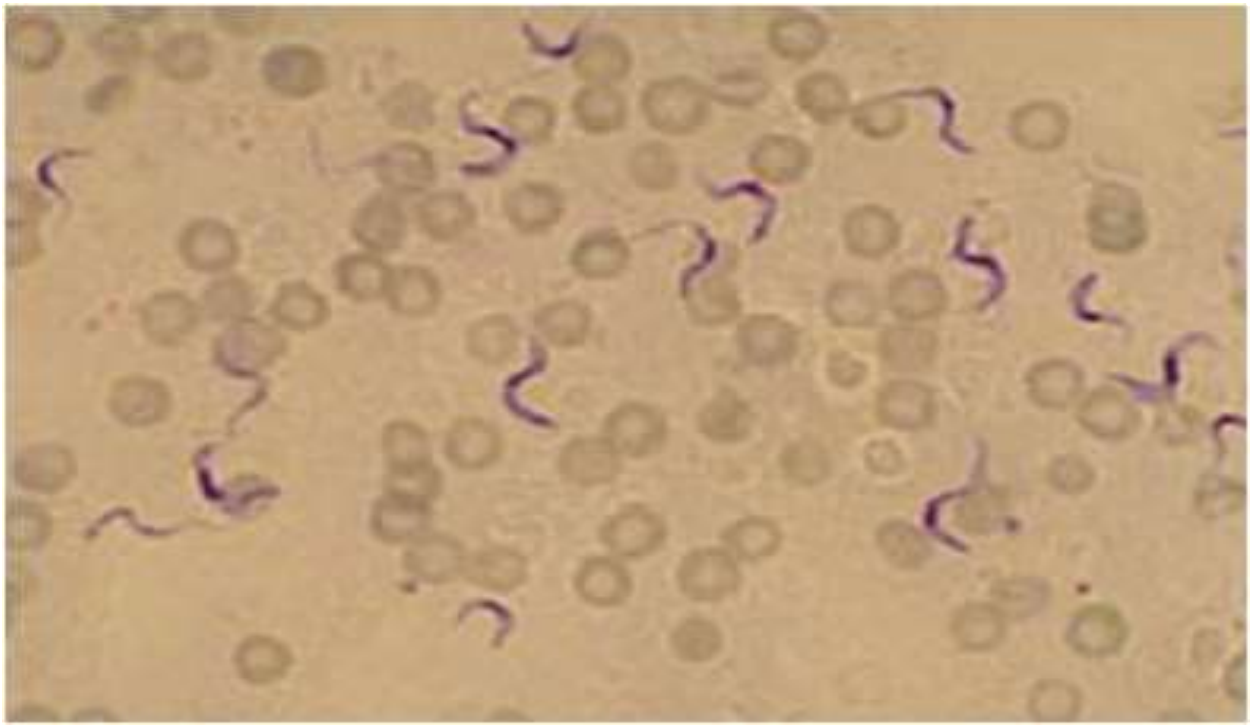


Figure 1. Shows morphology of Trypanosomes

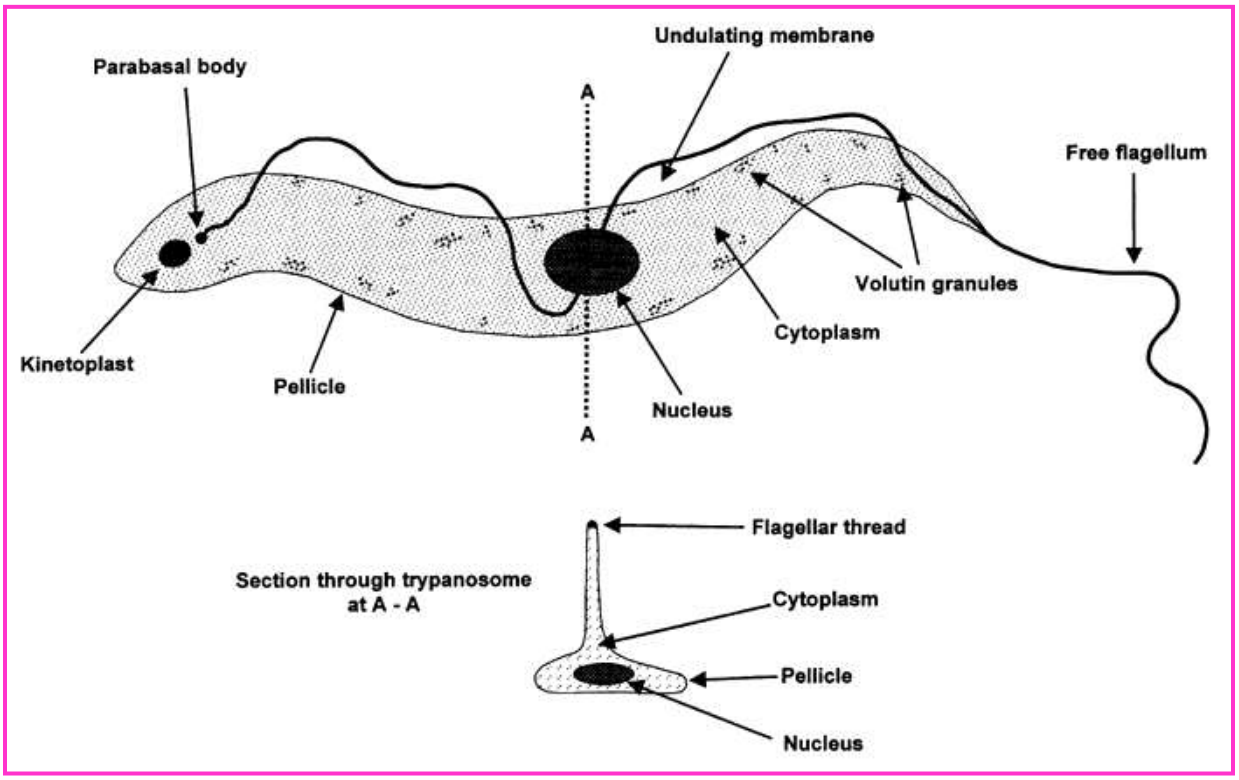


Figure 2: Illustration of the fundamental features of trypanosome.

Table 2: Morphology and Motility of each species of trypanosomes

Species	Morphology	Motility in fresh blood smear	Undulating membranes
T. brucei	Pleomorphic	Moves rapidly in small area	Pronounced
T. congolense	Monomorphic	Moves sluggishly often attached to RBC	Poorly developed
T. vivax	Monomorphic	Moves strictly across the field	Poorly developed
T. simiae	Monomorphic	-	Prominent
T. evansi	-	-	Well developed

Source: Urquhart *et al*., (1996); Getachew, 2005

LIFE CYCLE

Trypanosomes reproduce by longitudinal binary fission both in the blood stream & in the fly

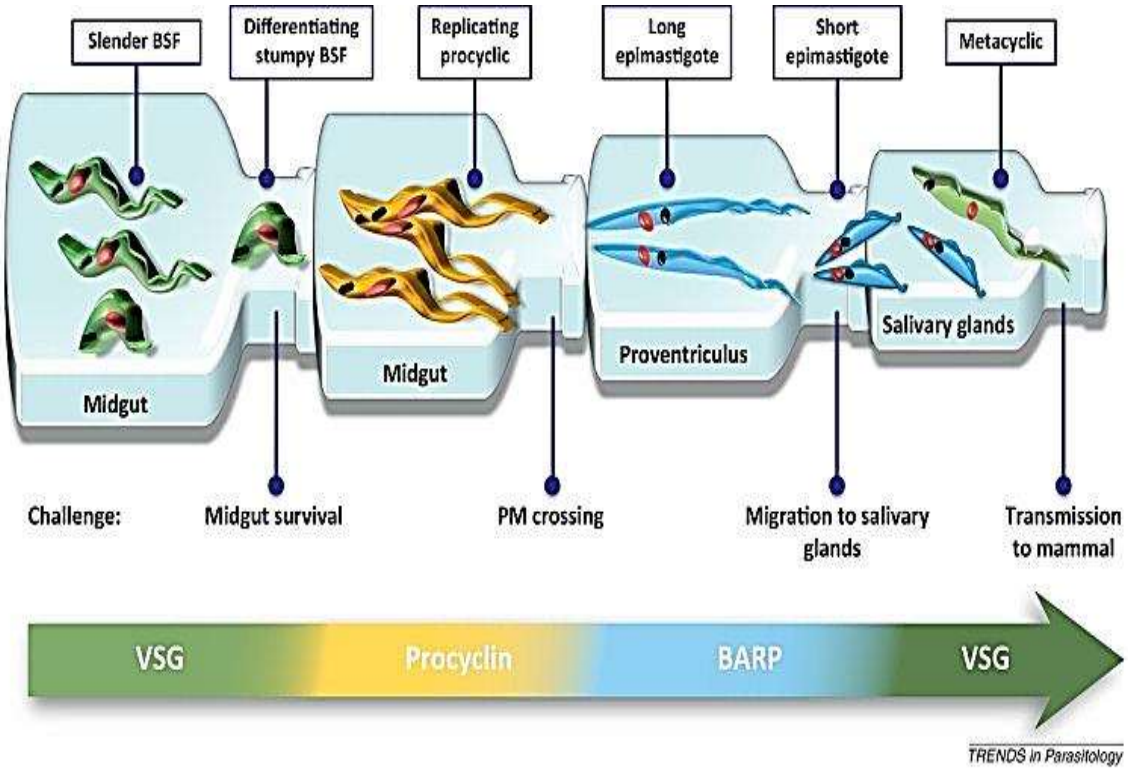
- multiplication in each host culminates in the presence of mature trypanosomes

❖ **Sexual** : Through cytoplasmic exchange in the fly

❖ **Asexual**: longitudinal binary fission both in the blood stream & in the fly

Developmental stages of trypanosomes in the tsetse fly

- The tsetse fly when it emerges from pupa is always uninfected (clean) and cannot transmit disease
- Pre adapted trypanosomes in the blood vessels of the mammalian host may be taken up by the tsetse fly when it feeds.
- In this way the infection is passed on and the parasite transforms in the fly into the following stages
  - Pre adapted trypanosomes in the blood
  - Procyclics/promastigotes/
  - Epimastigotes
  - Trypamastigotes
  - Metacyclic trypamastigotes/meta trypanosomes/



2.5. Epidemiology

Tsetse transmitted trypanosomosis occurs in Africa according to the distribution of vector. Mechanically transmitted trypanosomes can occur elsewhere in Africa, large area of Asia, Middle East, and South America. Among salivarian group *T. vivax* is considered to be spread beyond the confines of tsetse fly belts by mechanically transmission (Jemere, 2004).

2.5.1 Distribution

The distribution of trypanosomosis is depending on the three factors: the distribution of vectors, the virulence of the parasite and the response of the host. Epidemiologically trypanosomes are distributed in the tropical Africa in the latitude of 15^0N to 29^0S where they are associated with their *vectors*, *Glossina*, the *tsetse fly* (Bekele. J *et al*., 2010). The *tsetse flies(vectors*), *G. fusca*; the bush fly, *G. morsitans*, which inhibit principally savannah area and *G. palpalis*; a riverine species, effectively prevent the rearing of the cattle over the large area of the Africa (Getachew , 2005).

According to Feyesa (2004), the general distribution of the tsetse flies is determined principally by climate and influenced by altitude, vegetations and the presence of suitable host animals. Population of savannah species feed mainly on mammalian host particularly antelopes, buffalo, cattle, sheep and goats, while the riverine tsetse have a very wide range of preferred host including reptile and humans (Jemere, 2004; OIE, 2009).

Table 3: Distribution of Trypanosome species in Ethiopia

Trypanosome species	Distribution
T. congolense	South western Ethiopia
T. vivax	All over Ethiopia
T. brucei	Western and south west Ethiopia
T. evansi	Camel rearing areas of Ethiopia
T. equiperdium	Arsi- Bale highland
T. rhodesiensi	Gambela region and Gilo river

Source: (FAO, 1998; Getachew, 2005

2.5.2 Transmission

Trypanosomosis is a complex disease transmitted by tsetse flies cyclically (biologically), noncyclically (mechanically) by other biting flies and by other means like venereal, Iatrogenic and by coitus of transmission (Awoke, 2000). The three main groups of tsetse flies for transmission of trypanosomes are; *Glossina morsitans*, which favors the open wood land of savanna; *G. palpalis*, which prefers the shaded habitat immediately adjacent to rivers and lakes; and *G. fusca* which favors the high dense forest areas. Trypanosomosis is transmitted by other biting flies through the transfer of blood from one animal to another. The most important mechanical vectors are flies of the genus *Tabanus*, *stomoxys*, *Haematopota*, *hiperosia* and *chrysops flies* (Bekele J *et al*., 2010). *T. vivax* and *T .brucei* have spread beyond the tsetse fly belts where transmission by biting flies (FAO, 1998). With single exception of *T. equiperdium* of equines which is venereal disease. All species have an arthropod vector, in which transmission is either cyclically or noncyclical (mechanical transmission) (Getachew, 2005; Bekele J *et al*., 2010).

2.5.3 Cyclical (biological) Transmission

Cyclical transmission during which the trypanosome actively multiply in the vectors, occurs through the intermidialy *Glossina* or tsetse flies. It requires a period of incubation of the trypanosomes within the tsetse host. The term biological is used because trypanosomes must reproduce through several generations inside the tsetse host during the period of incubation. This requires extreme adaptation of trypanosome to the tsetse host (Taylor *et al*., 2007).

Tsetse is believed to be more likely to become infected by trypanosomes during their few blood meals. Tsetse infected by trypanosomes is thought to remain infected for the remainder of the lives. Because of the adaptations required for biological transmission, trypanosomes transmitted biologically by tsetse cannot be transmitted in this manner by other insects (FAO, 1998; Getachew, 2005).

Different trypanosome species develop in different regions of the digestive tract of the fly, and the *metatrypanosomes* occur either in the biting mouth part or the salivary glands. The period from ingesting infected blood to the appearance of these infective forms varies from one to the three weeks. Once infective *metatrypanosomes* are present the fly remains infective for the remainder of its life. During the act of feeding the fly penetrate their skin with its proboscis. By the reparture of small blood vessels a pool of blood is formed in the tissue and the fly injects saliva to prevent coagulation(Getachew, 2005).

Salivarian group: When multiplication occurs in the digestive tract and proboscis of the vector, new infection is transmitted by feeding on the host. The various species of trypanosomes which use this process are often considered as a group of the salivarian trypanosome species. All are trypanosomes transmitted by tsetse flies the main species are *T.congolense*, *T.vivax*, *T. brucei* and *T. simie* is the minor species (Getachew, 2005).

Stercorarian group: In the other trypanosomes multiplication and transformation occurs in the gut and the infective forms migrate in to the rectum and are passed with the feces and the trypanosomes species are grouped together as the stercorarian. In domestic animals these are all non-pathogenic trypanosomes such as *T. theleria* and *T. melophogium* (Getachew, 2005).

2.5.4 Non cyclical (Mechanical) Transmission

Mechanical transmission involves the direct transmission of the same individual trypanosomes taken from an infected host in an uninfected host. The name mechanical reflects the similarity of this mode of transmission to mechanical injection with a syringe. Mechanical transmission requires that tsetse feed on infected host and acquire the trypanosome in the blood meal, and then with in a relatively short period for tsetse to feed on an uninfected and regurgitate some of the infected blood from the first blood meal in to the tissue of the uninfected animal. This type of transmission occurs frequently when tsetse are interrupted during a blood meal and attempt to satiate themselves with another meal. Other biting flies such as horse flies also can cause mechanical transmission of trypanosome. Mechanical transmission is particularly important in relation to *T. vivax* and *T. envansi* in free of tsetse area. Iatrogenic means of infection also can occur when using the same needle or surgical instrument on more than one animal, at sufficiently short interval, the blood on the needle or instrument does not dry (Chernet *et al*., 2004; FAO, 1998).

2.5.5 Risk Factor

2.5.5.1 Host factor

The effect of infection varies with the host in that most wild animal and some domestic ones, establish a balance with the parasite and remain as clinically normal carriers for long periods. Specifically, some breeds of cattle indigenous to Africa can tolerate light to moderate challenge with tsetse flies by limiting the multiplication of trypanosomes in their blood and by apparently warding off the infection, especially *T. vivax* (NTTICC, 2004).

This phenomenon is called trypanotolerance. It is both genetic and environmental in origin and the

level of tolerance varies. Crossbreeds of indigenous Taurine and Zebu animals are also more tolerant than pure breed zebu. However, due to the uncertain genetic makeup of animals within these so-called breeds and crossbreeds, the level of trypanotolerance may also vary with individual animals within a given category and it can be overcome by heavy tsetse challenge, malnutrition, or other stress factors (Moor, N and J. Messina, 2010). The Sheko breed classified as a humpless Short horn is the only known breed of Taurine type in eastern Africa which exhibit trypanotolerance (NTTICC, 2004). The breed is found in the Bench-Maji zone of southern Region in the south- western parts of Ethiopia (Stein, J., 2011).

2.5.5.2 Environmental Factor

The density of tsetse population in the area and the level of their contact with the host, will determine the level of infection. This is further influenced by the vectorial capacity of the fly and the availability of its preferred host, which may not be livestock. Trekking of cattle through tsetse-infested vegetation is a risk nomadic farmer's face from time to time and the risk is even greater where cattle routes converge, for example, at major bridges or watering holes (NTTICC, 2004).

Agricultural and industrial developments generally lead to a lowering of tsetse density by destroying its habitat, whereas the establishment of game or forest reserves provides large numbers of preferred hosts or a suitable habitat for tsetse, respectively. Herds located near such reserves are therefore at a higher risk (Stein, J., 2011).

The vector for trypanosomosis, the tsetse fly (Glossina spp), requires a habitat that strongly influenced by ecological and climatic features particularly rainfall, soil type, temperature and vegetation type. Fly larvae can die as a result of drying soils. Temperature extremes, particularly above 36 °C and below 10°C also lead to adult fly mortality through starvation and water loss via respiration. Moisture levels directly related to precipitation is also involved in fly mortality, though the exact mechanism is not clear (NTTICC, 2004).

Cumulative effects of long rainy season or dry season are thought to have been important in influencing advances and recession in tsetse population (Moore, N. and J. Messina, 2010). The effect of altitude on tsetse distribution is through its effect on climate, mainly temperature. As temperature fall with increasing altitude the geographic limitations of different species may be due to their inactivity in lower temperature (NTTICC, 2004). Different species of tsetse flies require particular vegetation type that would provide an optimal condition for growth and survival and vegetation is also important that provides shelter for their hosts, all environmental factors that affects the tsetse fly indirectly affects the occurrence of trypanosomosis (Moore, N. and J. Messina, 2010).

2.5.5.3 Pathogen Factor

Living and dead trypanosomes produce a number of biologically active substances which are involved in the causation of trypanosomosis. These include variant surface glycoproteins (VSG), enzymes, Bcell mitogen and T lymphocyte triggering factor (TLTF) (NTTICC, 2004). Variant surface glycoproteins: In the mammalian host, the whole parasite is covered with a glycoprotein coat of a single molecular species, called the variant surface lycoprotein (VSG). The surface coat of one trypanosome consists of about 10 VSG molecules (Moore, N. and J. Messina, 2010).

It is the predominant surface antigen of African trypanosomes (Vincendeau, P. and B. Bouteille, 2006). African trypanosomes undergo antigenic variation of their VSG coat to avoid immune system-mediated killing by their mammalian host. The VSG of trypanosomes is attached to the cell surface by means of a phosphatidylinositol containing glycolipid membrane anchor. The membrane form of the variant surface glycoprotein (mfVSG) of live trypanosomes can be transferred from the parasite plasma membrane to that of erythrocytes. This transfer of mfVSG may sensitize the erythrocyte cells to immune destruction (anti-VSG antibody-mediated complement lysis) and contribute to the development of anemia (Vincendeau, P. and B. Bouteille, 2006).

2.6. Pathogenesis of Trypanosomosis

The Pathogenesis of tsetse-transmitted trypanosomosis can be categorized in to four groups according to the site of host- parasite interaction.

Chancre: The first interaction between trypanosomes and host occur in the skin following a successful feed by an infected tsetse fly. Within a few days of bite, cattle develop a raised cutaneous swelling called a chancre, which is caused by the reaction to multiplying trypanosomes (Getachew, 2005; Elnasri H., 2005).

Lymphadenopathy: Following enlargement of the lymph node draining the chancre, generalized enlargement of lymph nodes and splenomegaly develop. This is associated with marked proliferation of lymphoied cells in the organs. In the medullary cords of lymph nodes and splenic red pulp there are increases in plasma cells and numerous large active germinal centers are also present. In addition, the red pulp of the spleen, there is an increase in the number

of activated macrophages, some of which are engaged in erythrophagocytosis.

Anaemia: Plays the major role of Pathogenesis of bovine trypanosomosis. The development of anaemia is well recognized sign of trypanosome infection in cattle. The anaemia in bovine trypanosomosis can be divided in to two phases based on the presence or absence of trypanosomes, response to trypanocidal drug treatment and pathological findings. These are referred as acute and chronic phases of anaemia. The acute phase anaemia is characterized by progressive anaemia accompanied by parasitaemia. The initial fall in PCV value is associated with the first wave of parasitaemia in the blood. During this period the anaemia is extravascular and is possibly the result of increased red cell destraction by phagocytosis in the spleen, lung, lymph nodes and bone marrow. Progressive decrease in PCV takes place over a period of 4 to 12 weeks after infection and may result in death (Murray and Dexter, 1988; Juyal *et al*., 2005).

Tissue damage: Organs are damaged during the course of infection, some consistently more severely than others. Even though necrosis is a major feature of bovine trypanosomosis, tissue cell damage and degeneration may be marked (Morrison *et al*., 1981a). The heart is constantly damaged by all three species of trypanosomes. Other vital organs or systems, which are commonely affected, include the skeletal muscle, central nerves system, endocrine organs, reproductive systems (Cecchi *et al*., 2008; OIE, 2009).

When an animal is infected with trypanosomes, antibodies agianst the surface coat are produced. However, trypanosomes have multiple genes, which code for different surface proteins; allowing organisms with new surface coat glycoproteins to include the immune response. This process is called antigenic variation and results in the persistence of these organisms. Antigenic variation has thus far prevented development of a vaccine and permits reinfection when animals are exposed to tsetse carrying trypanosomes with surface coat glycoproteins of a new antigenic type (OIE, 2009; Abebe G.2005).

The pathogenesis of trypanosomosis is however, rather complex and depends on the trypanosome species and the species of the transmitting vector as well as on the resistance of the host. Genetic resistance to animal trypanosomosis has been attributed to certain breeds of livestock, most notably to the N`dama`s ability to prevent or reduce the rate and degree of development of anaemia (Keno, 2005; Kassa, 2005).

2.7 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 epidemiology of the disease in any geographical locality. Besides clinical diagnosis, parasitological, serological and molecular methods with varying degrees of sensitivity and specificity are available for the diagnosis of trypanosomosis (Dagnechew, S., 2004).

Thick blood smears

- Place drop of blood on slide
- Spread it to a size of about 2 cm in diameter
- Air dry quickly
- Immerse the smear in distilled water 5-10 min
- Fix with methanol alcohol of 75 % for three minutes
- Stain with Giemsa diluted in distilled water 1:10 for 30 min
- Examine under the microscope

Thin blood smear

- Put drop of blood on slide
- Spread the blood on the slide using a cover slip or another slide at an angle of 45 degree
- Dry with air
- Fix with methanol for three minutes
- Stain with Giemsa
- Wash with phosphate buffer at PH 6.8-7.2
- Allow to dry
- Examine under the microscope 1x100 magnification

Examination of tissue for the presence of trypanosomes

- Lymph nodes, brain crash smear(*T.brucei* group) oedematous fluid for *T.equiperdum*
- Concentration techniques

Microhaematocrit centrifugation (Woo method)

- ❑ Ear vein blood is collected in heparinized capillary tube of 75x1.5 mm
- ❑ seal one end with crista seal
- ❑ Place in microhaematocrit centrifuges symmetrically
- ❑ Close the rotor cup
- ❑ Allow to rotate 12,000 rpm for 5 min

- Put in tube carrier
- Examine the buffy coat by slowly rotating the tube

Dark ground /phase contrast BCT *(Murray method)*

- Collect blood in heparinisd capilary tube
- Seal one end with crystaseal
- Place in microhaematocrit centrifuge
- Allow to spin 12,000 rpm for 5 min
- Cut the capillary tube with diamond pencil 1 mm below the buffy coat to include RBC
- Extrude on slide & cover with cover slip
- Examine in dark ground phas-contrast microscope with x40 objective

2.8 Clinical signs

The disease shows a variety of clinical manifestation, which is also common to other diseases. The fact that the disease may run an acute, chronic or sub- clinical course further complicates the diagnosis of trypanosome infections on the basis of clinical signs. In general, fever can be observed which may be intermittent due to the variation in parasitaemia, and if the animal survives the disease becomes chronic and there is development of anaemia and emaciated (Kenow, 2005; kassa, 2005). This therefore, makes fever, anaemia and body condition important parameters that are routinely used for the tentative diagnosis of trypanosomosis in areas where this disease is endemic and laboratory services are not available. Definitive diagnosis of the disease is ultimately dependent on the detection of the trypanosome in blood samples from infected animals.

2.8 Treatment and Control of Trypanosomosis

Treatment and control of trypanosomosis in order to be effective treatment should be given early in the initially phase of parasitaemia. As no new drugs have been withdrawn because of resistance; treatment is now essentially limited to two compounds, diminazene aceturate and homidium salts and Isometamidium chloride (ISMM) (either chloride or bromide) (IAEA, 2002).

Control is aimed at interrupting the cycle of development of the protozoa either within the mammalian host or the insect vector. Control of the trypanosomosis can be based on control of the parasite (trypanosomosis), control of the vectors (tsetse or biting flies), Use of innate resistance (trypanotolerant) and integrated approach combing other methods (Getachew, 2005; Bal *et al*., 2014).

Parasite control involves the application of trypanocidal drugs (curative and prophylactic drugs). Even though the exact action of trypanocidal is unknown, they disrupt or block one or more of the vital process of metabolic pathways essentially to the embedding micro organisms and toxic to the trypanosomes. It is important to realize that drug alone will not cure trypanosomosis. Trypanosomes overwhelm the immune system of the host. Chemotherapy stop the multiplication of the trypanosomes, helps the immune system to overcome the infection Traps, targets, pour on, insecticides, etc used to control and kill the vector (Adam *et al*., 2003; Gupta *et al*., 2009).

Use of trypano tolerant breed is the other way of controlling trypanosomosis. Based on the actual experience in the field, the introduction and keeping of trypanotolerant, West African taurine cattle breeds seem to be an alternative method to prevent clinical trypanosomosis and thus economic loss for animal holders. Such taurine (hump less) breeds are now mainly confined to West Africa, from Senegal to Nigeria, but also occur in East and Central Sudan and even Western Ethiopia (Cecchi *et al*., 2008; Juyal *et al*., 2005).

2.9 Economic Importance

According to the Food and Agricultural Organization of the United Nations, trypanosomosis is probably the only disease which has profoundly affected the settlement and economic development of a major part of SSA of the approximately 7-10 million km of land that are infested by tsetse fly, only 20 million cattle are raised. Under different circumstances, this land could support more than 140 million cattle and increase meat production by 1.5 million tons (Reid R.S., 1997).

Trypanosomosis threatens 50 million head of cattle in SSA. Every year, trypanosomosis causes about 3 million deaths in cattle while approximately 35 million doses of trypanocidal drugs are administered to enable livestock to survive in tsetse-infested areas. While the economic losses in cattle production alone are in the range ofUS$1.0-1.2 billion, the indirect impact engendered by the disease on the total agriculture-livestock production is estimated at US$4.5 billion a year. The overall negative impact extends to the access and availability of cultivable areas, changes in land use and exploitation of natural resources, restriction of opportunities for diversification and intensification of agricultural activity. The magnitude of the problem requires a multidisciplinary approach for effectively promoting sustainable agriculture and rural development strategies (Mattioli R.C & J. Slingenberh, 2013).

The disease directly affect the milk and meat. The disease directly affect the milk and meat productivity of animals, reduces birth rates, increases the abortion rates as well as mortality rate; all of these affect the herd size and herd composition (Swallow, B.M.,1999). Indirect impact of trypanosomosis mostly lies on crop production through the availability and cost of animals that provide traction power (Swallow, B.M., 2000). Animal trypanosomosis reduces work efficiency of oxen for cultivation, reducing access to animal traction or discourages the introduction of drought animals in to crop farming (Omotainse, S.O *et al*., 2004).

Evaluation on effect of trypanosomosis incidence on the productivity of oxen used for traction showed that relative inefficiency in the high risk area was38% less efficient than oxen in the low risk area (Swallow, B.M., 2000). Additional traction capacity allows farmers expand the area that they cultivate, increase yield of existing crops; grow different mix of crops or allocated labour land and fertilizer more efficiently. In other study (Shaw, A.P *et al*., 2014) discussed the economic benefits from intervening against bovine trypanosomosis. These authors reported significant benefits especially for Ethiopia, because of its very high livestock densities and the importance of animal traction. The estimated maximum benefit per square kilometer of tsetse infested area is US$ 10,000. Consequently, the total maximum benefits from dealing with bovine trypanosomosis in Ethiopia could be as much as US$ 1billion (Shaw, A.P *et al*., 2014).

3. CONCLUSIONS AND RECOMMENDATION

The disease resulted in serious economic losses specially western and southwestern parts, posing a significant impact on the country development. Handfuls of options are available for the diagnosis of Animal *Trypanosomosis*; however, in Ethiopian practical situation the diagnosis of Animal *Trypanosomosis* is mainly relies on the less sensitive parasitological diagnosis techniques. Earlier tsetse and Trypanosomosis strategies relied bush clearing and elimination of wild animals on which tsetse feed. These methods are environmentally unfriendly and less effective. The current initiatives to control *trypanosomosis* are mainly based on tsetse fly control (area-wide integrated pest management, using traps and targets, deltamethrine pour on techniques). Animal *Trypanosomosis* can be treated by both the prophylactic and curative drugs. The extensive and uncontrolled use of trypanocidal drug in tsetse infested areas resulted in trypanocidal drug resistance. Trypanocidal drug resistance is reported from different African countries including Ethiopia. Based on this conclusion, the following recommendations are forwarded.

- Integrated control strategy, proper management (restriction of pasture grazing in the tsetse belt), vector control (control of tsetse fly) and treatment of the infected animal should be practiced in tsetse infected areas to reduce the economic impact of animal trypanosomosis,
- The government and concerned animal health professionals should monitor the use of trypanocidal drugs to avoid further drug resistances,
- Government and other non-governmental organizations, should provide financial support for researches on new and alternative drugs,
- Restriction of cattle movement from an infected area to the disease free area and vice versal to prevent and control of further expansion of animal trypanosomosis.
- Appropriate control measures have to be designed to lessen the undesirable impact of the disease in the Region.

4. REFERENCE

1. Abebe G., (2005): Current situation of Trypanosomosis. In: review article on: Trypanosomosis in Ethiopia. Ethiop. *J Biol.med. Sci* **4(**1**)**: 75-121
2. Adam KMG, Paul J, Zaman V (2003): Medical and Veterinary Protozoology. Churchill living stone Edinburgh and London.
3. Abraham ZA, and Zeryehun T (2012): Prevalence of Bovine Trypanosomosis in Selected District of Arba Minch, Snnpr, Southern Ethiopia, *Global Veterinaria* **8**(2): 168-173.
4. Bal MS, Sharma A, Ashuma Bath BK, Kaur P and Singla LD (2014): Detection and management of latent infection of Trypanosoma evansi in a cattle herd. Ind. *J. Anim. Res.* **48**(1): 31-37.
5. Cecchi G, Mattioli RC, Slingenbergh J, de la Rocque S (2008): Land cover and tsetse fly distributions in sub-Saharan Africa. *Med. Vet. Entom.* 22: 364-373.
6. Cherenet T, Sani RA, Panandam JM, Nadzr S, Speybroeck N, Van Den Bossche P (2004): Seasonal prevalence of bovine trypanosomiasis in a tsetse-infested zone and a tsetse-free zone of the Amhara region, north-west Ethiopia. Onderstepoort *J. Vet. Res.* **71**(4): 307-312.
7. Dagnachew, S., (2004): Epidemiology of bovine trypanosomosis in the Abay basin areas of northwest Ethiopia, MSc thesis, Addis Ababa

University, Faculty of Veterinary Medicine, Debre zeit.

8. Elnasri, H., (2005). Prevalence and Ranking of Bovine trypanosomiasis in Unity State, Sudan, MSc thesis University of Khartoum, *Faculty of Veterinary Medicine, Unity State, Sudanpp1*-76.
9. Bekele, J., K. Asmare, G. Abebe, G. Ayelet and G. Esayas, (2010): Evaluation of Deltamethrin applications in the control of tsetse and trypanosomosis in the southern rift valley areas of Ethiopia. *Vete Parasitol.*, **168**: 177-184.
10. Getechew, A., (2005): Review Article trypanasomasis in Ethiopia. Ethiopia. *J. Biol. Sci.*, **27**(1): 1-8.
11. Getachew A. (2005): Trypanosomosis in Ethiopia, A.A.U, *Faculty of Veterinary Medicine.* Debre zeit, pp 18-20.
12. Gupta MP, Kumar H and Singla LD (2009): Trypanosomiasis concurrent to tuberculosis in black bucks. *Ind. Veter. J.* **86**: 727-728.
13. 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.*
14. Mulaw S, Addis M, and Fromsa A. (2011): Study on the Prevalence of Major Trypanosomes Affecting Bovine in Tsetse Infested Asossa District of Benishangul Gumuz Regional State, Western Ethiopia. *Global Veterinaria* ***7*** *(4): 330- 336.*
15. Mekuria S, and Gadissa F (2011): Survey on bovine trypanosomosis and its vector in Metekeland Awi zones of North west Ethiopia. *Acta Tropica*, **117**: 146-151.
16. Stein, J., (2011): Trypanotolerance and Phenotypic Characteristics of Four Ethiopian Cattle Breeds. Doctoral Thesis, Swedish University of Agricultural Sciences, F*aculty of. Vet. Med. Anim. Sci., UppsalaSweden*. pp1-63.
17. NTTICC, (2004): National tsetse and Trypanomososis Investigation and Control Center report for the period7 July 2001 – 6 July 2002 *Badelle, Ethiopia*, pp: 3.
18. Loses, G. and B. Ikede, (2002): Review on pathology of diseases in domestic and laboratory animals caused by Trypanosoma congolense, T. vivax, T. brucei, T. rhodesiense and T. gambiense. *Vet Pathol*, 9: 1-71.
19. Moore, N. and J. Messina, (2010): A Landscape and Climate Data Logistic Model of Tsetse Distribution in Kenya. PLoS ONE **5**(7): e11809. Doi: 10.1371 Ethiopia, pp: 151-157.
20. Swallow, B.M., (2000):Impact of Trypanosomiasis on African Agriculture. Vol. 2, PAAT Technical and Scientific Series, FAO. Rome. *Int. J. Anim. Veter. Adv*., **2**(2): 47-50, 201.
21. Omotainse, S.O., J.O. Kalejaiye, P. Dede and A.J. Dada, (2004): The current status of tsetse and animal trypanosomiasis in Nigeria. *Vom. J. Vet. Sci*., 1: 1-9.
22. Shaw, A.P., G. Cecchi, G.R. Wint, R.C. Mattioli and T.P. Robinson, (2014): Mapping the economic benefits to livestock keepers from intervening against bovine trypanosomosis in Eastern Africa. *Prev. Vet. Med.*, 113: 197-210
23. NTTICC (National Tsetse and Trypanosomosis Investigation and Control Centre), (1996): *Annual Report on Tsetse and Trypanosomosis, Survey, Bedele, Ethiopia*. Pp.11-15.
24. NTTICC (2004): National Tsetse and Trypanosomosis Investigation and Control Center. Report for the period 7th June 2003-6th July 2004. Bedelle, p. 3.
25. OIE (2009): Manual of standards for diagnostic tests and vaccines for terrestrial animals, 6th ed. Paris. pp: 813-2008
26. OIE (Office of Internationale des Epizootics), (2008): "Standardized Techniques for the Diagnosis of Tsetse transmitted Trypanosomosis," in *OIE Terrestrial Manual*, p. 49, Rome, Italy.
27. OIE (2009): Manual of Diagnostic Tests and Vaccines for Terrestrial Animal health code http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/ pp 1-5.
28. FAO (1998): A field guide for the diagnosis, treatment and prevention of African Trypanosomes: In Food and Agriculture organization of the United Nations Rome FAO. http://www. Fao.org/ DOCREP/ 006/ X0413/ X0413EOO. HTM. Pp.170.
29. Jemere BH. (2004): Control of Tsetse and Trypanosomiasis in the southern Rift valley (step area): Evaluation of Deltamethrin Applications, Msc thesis , faculty of vet. Medicine AAU., Debrezeit, Ethiopia.
30. IAEA (2002): Campaign launched to eliminate Tsetse fly; which has turned much of Africa in to a Green Desert Press Reports on PATTEC.
31. Thrusfield M. (2005): Veterinary Epidemiology, 3rd edition, Blackwell Science Ltd, Oxford, UK. Pp.233-250.
32. Kassa B. (2005): Standard Veterinary Laboratory, Diagnostic manual, and volume 3 Parasitology. Minister of Agriculture and Rrural Development, Animal Health Department. Addis Ababa, Ethiopia. Pp 32-37.
33. Singla LD, Aulakh GS, Juyal PD, Singh J (2004): Bovine trypanosomosis in Punjab, India.

Proceeding of The 11th International Conference of the Association of Institutions for Tropical Veterinary Medicine and 16th Veterinary Association Malaysia Congress, 23-27 August 2004, Pet aling Jaya, Malaysia, pp: 283-285. 4.

34. Jemere BH (2004): Control of Tsetse and Trypanosomiasis in the southern Rift valley(step area): Evaluation of Deltamethrin Applications, Msc thesis , faculty of vet. Medicine AAU, Debrezeit, Ethiopia.
35. Juyal PD, Singla LD, Kaur P (2005): Management of surra due to Trypanosoma evansi in India: an overview. Infectious diseases of domestic animals and zoonosis in India. **75**:109-120.
36. Radostits OM, Gay CC,Blood DC, and Hinchelift KW (2000): Disease caused by protozoa – *Trypanosomes*. Veterinary Medicine: *A Text Book of Disease of Cattle, Sheep, Pig, Goat and Horses.* 9th ed. *Harcourt Publisher Ltd. London*. Pp 1531-1541.
37. OAU (Organization of African Union) (2001): Trypanosomosis, Tsetse and Africa. The year book report (2001).
38. Sharma A, Singla LD, Ashuma, Batth BK, Kaur P, Javed M, Juyal PD (2013): Molecular prevalence of Babesia bigemina and Trypanosoma evansi in dairy animals from Punjab, India by duplex PCR: A step forward to detection and management of concurrent latent infections. Biomed. Res. Int. Article ID 893862, 8 pp.
39. Taylor MA, RL coop and RL wall (2007): Veterinary Parasitology 3rd ed. Block Well publishing Ltd, oxford. Uk, Pp 96-102, 212-214.
40. Vincendeau, P. and B. Bouteille, (2006): Immunology and Immunopathology of African Trypanosomiasis. Ann. Braz. Acad. Sci., **78**: 645-665.

1/26/2025