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Prevalence and Associated Risk Factors of Bovine Trypanosomosis in Nono District, West Shewa Zone, Ethiopia

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Abstract

Trypanosomosis is a major constraint to livestock production due to the challenge of vector control activities and drug resistance development in Sub-Saharan Africa particularly Ethiopia. The most common trypanosome species that affects cattle in Ethiopia are *T. congolense. T. vivax* and *T. brucei.* Therefore, A cross sectional study was conducted from November 2019 to December 2020 to determine the prevalence of bovine trypanosomosis Nono district of Western Shewa zone, Ethiopia. The study district was purposively selected and PAs were randomly selected to take sample for the study. For the prevalence study, dark phase contrast buffy coat examination and Giemsa-stained thin blood smears were used and Chi- Square test was used to analysis the results. Out of a total of 384 randomly selected and examined cattle, an overall prevalence of 5.5% was recorded. Highest prevalence was recorded in Nano Halo 8(6.7%) followed by Biftu Jalala 6(5.5%) and Halo Dinki 7(4.5%) peasant associations. This study showed no significant difference (P>0.05) in trypanosomiasis infection rate among peasant associations and there was significant association between risk factors like age (X2= 6.97, P= 0.008), sex (X2= 5.38, P= 0.02), body condition (X2 = 6.09, P= 0.048) and PCV values (X2= 18.47, P= 0.000) of examined cattle. Out of species of trypanosome identified highest was *T. Congolense* was 13(61.91%), followed by *T. Vivax* was 7(33.33%) and 1(4.76%) were mixed. The present work evidenced that tsetse and trypanosomosis has continued to pose a considerable threat to cattle of the study area warranting an integrated control to safeguard cattle production and productivity.

Keywords: *Bovine, Western Shewa, Ethiopia, Nono, prevalence, Trypanosomiasis*

Introduction

Back Ground

Livestock are the main stay of the vast majority of African people. They contribute a large proportion of the continent's gross domestic product and constitute a major source of foreign currency earning for a number of countries. Ethiopia is one of African countries which has the largest livestock population in Africa and a large part of the agricultural system is not mechanized, so livestock play a crucial role in agricultural production both directly as food sources and as a source of traction power. Despite the large livestock population, Ethiopia fails to optimally utilize this resource due to different constraints such as shortage of nutrition, reproductive insufficiency, management constraints, and animal disease (Tesema and Yitayew, 2015). Among animal diseases, trypanosomiasis is one of the parasitic diseases that hampering livestock development in Ethiopia (Anderson et al., 2011; Amanuel et al., 2015).

Trypanosomosis caused by a protozoan parasite that belongs genus is Trypanosoma and is transmitted by tsetse flies (Glossina spp) and biting flies like Stomoxys and Tabanus (Alemayehu et al., 2012). The usual consequence of Trypanosoma infection is anemia, loss of body condition, decrease in fertility, and increasing calf mortality (Zubairu et al., 2013, Wanga and Munga, 2011). Trypanosoma congolense, T. vivax, and T. brucei are the most pathogenic Trypanosoma species within the country

that transmitted by tsetse flies (Magona et al., 2003). Several studies indicated that five Glossina species have existed in Ethiopia, however, only four of them (G. morsitanssubmorsitans, G. pallidipes, G. tachinoides, and G. fuscipesfuscipes) are widespread and have the economic importance (Kitila et al., 2017; Meharenet et al., 2020).

Trypanosomiasis affects directly the meat and milk productivity of cattle, increase abortion, and mortality rate (Leta et al., 2016). About 200 million US dollars were lost from the national economy due to the direct and indirect impact of trypanosomosis on agricultural and livestock production (Seyoum et al., 2013). Trypanosomosis decreased the work efficiency of oxen and hinder the introduction of drought cattle in tsetse-infested areas for crop farming (Siyum et al., 2014). Tsetse-transmitted trypanosomiasis remains one of the major production losses of cattle in Ethiopia (Kitila et al., 2017; Tulu et al., 2018). The magnitude of the problem requires a multidisciplinary approach for effectively promoting sustainable agriculture and rural development strategies (Tulu, 2019).

The principle of prevention and control of trypanosomiasis depends on reducing the contact between cattle and vectors. The control methods of trypanosomosis mainly include control of tsetse fly numbers, use of a trypanocidal drug, and use of cattle breed that tolerate the disease (Achenef and Bekele, 2013; Bouyer et al., 2014). To effectively control trypanosomosis, it is important to know the epidemiology of the disease and its vector distribution in the areas (Ebhodaghe et al., 2018).

Currently, trypanosomosis is found to be one of the factors hampering livestock production and productivity in most settlement areas of western Ethiopia and Trypanosomosis is also a neglected tropical disease that is the major constraint to agricultural activities and cattle production in the country as a whole and the Nono district in particular. However, a limited study has been done to determine the epidemiology of bovine trypanosomosis. An understanding of the prevalence of the disease plays crucial for designing appropriate control strategies. Therefore current study was planned and carried out to fill such gaps.

Objectives

- To determine the prevalence and associated risk factors of trypanosomosis in cattle of Nono district
- To identify trypanosomes species associated with bovine trypanosomosis in Nono district

Literature Review

Etiology

African animal Trypanosomosis is caused by Trypanosoma congolense, Trypanosoma vivax, and Trypanosoma brucei species. Trypanosomaevansicauses 'Surra' in camels (M baya et al., 2010). Biting flies have been reported as the major cause of Trypanosoma vivax infection in high land districts bordering Lake Tana (Sinshaw et al., 2006).TrypanosomavivaxT.congolen,T.brucei, andT.simiaeare the fourma in species responsible for African trypanosomosis affecting virtually all domestic mammals while T. evansicauses Surrain camels (Camelusdromedarieus) (M bay et al., 2010). The four species are members of the Salivariagroup of trypanosomes and are transmitted cyclically via the mouth parts of tsetse flies, hence the name salivarian trypanosomes (Abenga, 2014).

Life Cycle

The life cycle of any one species may include more than one of these configurations; Promastigote which is elongated form with ante nuclear (in front of nucleus near the anterior end of the body) kinetoplast; flagellum arising near it and emerging from the anterior end of body e.g. Leptomonas; Epimastigote which is elongated form with ajuxta nuclear kinetoplast (between nucleus and anterior end), flagellum arising near it and emerging from the site of the body as a short undulating membrane e.g. Blastocrithidia and some Trypanosoma species; Trypomastigote which is the "true" trypanosomes type; post nuclear kinetoplast; flagellum arising near it to run a long undulating membrane; and Amastigote that is rounded or oval forms devoid of external flagellum e. g. Leishmania species (Hunt, 2010). As trypanosomes progress through their life cycle they undergo a series of morphological changes as is typical of trypanosomatids. The life cycle often consists of the trypomastigote form in the vertebrate host and the trypomastigote or promastigote form in the gut of the invertebrate host. Intra cellular life cycle stages are normally found in the amastigote form. The trypomastigote morphology is unique to species in the genus Trypanosoma (FAO, 2006).

Epidemiology

Distribution

Trypanosomosis epidemiology depends on the interaction between the ecological factors i.e. parasite, vector, and host factors. The disease severity depends on the strain and the species of the trypanosomes that has infected the animal. In West Africa, T, vivax infection predominates and T.

congolense poses a chronic disease. While in East and Central Africa most of the infection in Cattle is due to T .vivax but with a mild disease as compared to T. congolense. The bovine trypanosome species like the T .congolense, T, brucei, and T .vivax are normally associated with the humid and sub-humid areas of Africa (15oN and 25oS), that is inhabited by their intermediate hosts the Glossina (Sow, 2013).Transmission of trypanosomosis mostly depends on the distribution and the capacity of the vector Glossina species for transmission. The savannah and riverine are the most are the ones that inhabit the grazing and watering areas. The most important trypanosomes that cause economic losses in livestock are T. congolense, T. vivax, and T. brucei (Sow, 2013).

Mode of Transmission

Cyclical Transmission: When a tsetse flies hatches from its pupal case it is free from trypanosomes. Until its first blood meal, it is called a teneral fly. It acquires a trypanosomal infection when feeding on a parasitaemic (having parasites in the circulating blood) mammalian host. The trypanosomes undergo a cycle of development and multiplication in the digestive tract of the fly until the infective Met acyclic trypanosomes are produced; different trypanosome species develop in different regions of the digestive tract of the fly, and the Met trypanosomes occur either in the biting mouth parts or the salivary glands (Sinshaw et al., 2006). The period from ingesting infected blood to the appearance of these infective forms varies from one to three weeks; once infective Met trypanosomes are present the fly remains infective for the remainder of its life (Shimelis and Melkamu, 2015). During the act of feeding the fly penetrates the skin with its proboscis. By the rupture of small blood vessels a pool of blood is formed in the tissues and the fly injects saliva to prevent coagulation. Infection of the host takes place at this stage, with infective Met acyclic trypanosomes in the saliva (FAO, 2006; Shimelis and Melkamu, 2015).

Mechanical Transmission: A biting insect passes the blood forms from an infected animal to another in the course of interrupted feeding. The time between the two feeds is crucial for effective transmission because the trypanosomes die when the bloodies. The importance of this mode of transmission is variable from place to place, depending on the numbers of hosts and biting insects present, and also on the species of trypanosome. A large biting insect such as tabanids carries more blood and is more likely to act as mechanical vectors than for examples tomoxes. (Tsetse flies themselves can of course also act as mechanical vectors.) In non-cyclical transmission, trypanosomes can be transmitted in the absence of tsetse flies (Glossina). But Glossina species are also capable of transmitting mechanically, in this case the fly feeds on more than one animal before repletion and remain infective for only a short time (Levine, 1973), after trypanosomes have been introduced into a herd. Biting flies are capable of transmitting in their mouth parts if they feed on more than one host in a short interval. Trypanosomes are mechanically transmitted by blood sucking flies chieffly Tabanus striatus, Stomoxy calcitrans, Chrysops species, Haematobia irritans, Lyperosia species and Hippoboscidae. T. vivax is commonly spread by this mechanism (Parisetal., 1982; Desquesne, 2004).

Risk Factors

Host Factors: 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 (Aschalew et al., 2015). This phenomenon is called trypanotolerance, it is both genetic and environmental in origin and the level of tolerance varies. Cross breeds 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 (Loses and Ikede, 2002).

Environmental Factors: The density of tsetse population in the area and the level of their contact with the host, will determine the level of infection. 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, 2002). 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 (Aschalew et al., 2015).

Pathogen Factors: In cattle, T.vivax generally produces a higher level of parasitaemia than other

species. And since, its life cycle in the tsetse is also shorter; T. vivax is more readily transmitted than the others when animals are newly introduced into a tsetse infested area. Higher parasitaemia also facilitate mechanical transmission. On the other hand, T. brucei is rarely detectable by direct examination of cattle blood, even though infection can be confirmed through other diagnostic methods (Aschalew et al., 2015).

Pathogenesis

The precise pathogenesis of trypanosomosis remains far from clear. Four features: chancre, lymph adenopathy, anemia, andtissue damages dominate the pathology of trypanosomosis. The trypanosome species affecting man and domestic animals have been sub divided into two groups, the haematinic group (T. congolense and T .vivax) which remains in the plasma and the tissue invading group (T.brucei,T.evansi,T. b.gambiense, T. b. rhodesiense and T. equiperdum found in extra and intravascular spaces (Ngure et al., 2008). Because of their presence in the blood, these invading parasites produce numerous changes in the cellular and biochemical constituents of blood (Taiwo et al., 2003).

Meta cyclic trypanosomes are inoculated intra dermally as the fly feeds. They multiply at this site provoking a local skin reaction (chancre), which is most pronounced in a fully susceptible host and may be slight or absent with some strains or species of trypanosomes. Within the chancre, meta cyclic parasites change to trypomastigote form, enter the bloods stream directly or through the lymphatic, where they reproduce asexually by binary fission (Maudlin et al, 2004).

T. vivax and T. brucei invade tissues and result in tissue damage in several organs and initiate characteristic intermittent parasitaemia. T. vivax usually multiplies rapidly in the blood of cattle, sheep and goats, and is evenly dispersed throughout the cardiovascular system, whereasT. congolense tends to be aggregated in small blood vessels and capillaries of the heart, brain, and skeletal muscle, and rarely causes heavy parasitaemia in ruminants T. brucei also found extra viscerally, for example in the myocardium, the central nervous system and theReproductive tract (Radostits et al., 2007; Mary and David, 2009).

When ananimalis infected with trypanosomes; anti bodies against the surface coat are produced (Shimelis and Melkamu, 2015). The parasite releases toxic substance when destroyed within the circulatory system and hence damages the lining of the blood vessels (Abenga, 2014). The ability of Trypanosoma spp to change their surface coat antigen continuously leads to exhaustion of the antibody production by the host leading immunosuppression (Shimelis and Melkamu, 2015). Lymphoid enlargement, and splenomegaly development is associated with plasma cell hyperplasia and hypergamma-globulinaemia, which is primarily due to an increase in IgM (FAO, 2006).

The response of antibodies developed to the glycoprotein coat of the trypanosomes kills the parasite and results in the development of immune complexes (FAO, 2006; Hamilton et al., 2007). Immunologic lesions are significant in trypanosomosis and it has been suggested that many of the lesions (e.g. anemia and glomerulo nephritis) in this disease may be the result of deposition of immune complexes that interfere with, or prevent, normal organ function. Profound immune suppression occurs following infection and this lowers the hosts‟ resistance to other infections and thus results in secondary reservoir, able to re infection of the blood stream (Mogk et al., 2014).

Most trypanosomes have to survive within two hosts, mammalian and insect, necessitating adaption to differing nutritional environments, and remodeling of their surface coat (Gadelha et al., 2011); and must also live within two specialized environments in their mammalian host. In the blood stream and lymphatic system the parasites evade both the acquired and innate immune systems, predominantly by antigenic variation, changing the variant surface glycoprotein (VSG) expressed on their surface to avoid anti body mediated responses (Mansfield et al., 2014). During the second stage of infection, in the CNS, they are more protected from the immune system, and may exist as reservoir, able to re infect the blood stream (Mogk et al., 2014).

Clinical Signs

Trypanosoma infection display typical signs depending on the species and strain of the trypanosome vector and resistance of the affected breed of animal (Teka et al., 2012).Clinical sign includes fever, anemia due to the destruction of blood cell which occurs intravascular in the acute phase and also extra vascular in sub-acute and chronic stages. The anemia is caused by multi factorial factors like by directly inserting hemolytic action by producing potentially hemolytic factors on autolysis (Biryomumaisho and Rwakishaya,2012), poor body condition, animals imported from infested area with tsetse flies can be sub clinical carriers and may become ill when they are stressed, localized swelling,

lymph adenopathy (Wobo et al., 2010),weight loss, dairy animals there is decrease in milk yield, neurological signs are diarrhea , keratitis, lacrimation, loss in appetite ,abortion, premature birth ,and perinatal losses as its effect in reproduction(CFSPH,2009).

Diagnosis

Trypanosomosis can be diagnosed based on either detection of the parasite by the light microscope or demonstration of the circulating antibody (serological) in conjunction with clinical observation (Mezene et al, 2014). The stained thin blood smears afford the best means of identifying species of trypanosomes (Efrem et al., 2013).

Wet Blood Films

These were made by placing a drop of blood on a clean microscope slide. Using a drop per tip of a drop per, the blood were be spread to about 2mm in diameter on the slide to avoid over flow, then the blood can be covered with a cover-slip (22 22mm). The blood can be viewed microscopically at 40 total magnifications with condenser aperture-phase-contrast or interference contrast. Approximately 50-100 fields were examined. Trypanosomes were recognized by their movement among the red blood cells (RBCs) (OIE, 2013; Zubairu et al., 2013).

Thin Blood Smear

Thin blood smears are made by placing a small drop of blood(about5µl),for example from a micro hematocrit capillary tube, on a clean microscope slide approximately 20mm from one end (allowing for space to apply the thin smear) and spreading with the edge of another slide (Nakayima, 2016). This slide is placed at an angle of approximately 30° to the first slide and drawn back to make contact with the blood droplet. The blood is allowed to run along the edge of the spreader, which is then pushed to the other end of the slide in a fairly rapid but smooth motion (Kemal, 2014). If the correct amount of blood is used, the slide should be covered with a thin film of blood with no surplus before the end of the slide is reached, and the smear should take the shape of a bullet. Ideally, thin films should be prepared so that the RBCs are fairly close to each other but not over lapping. The slide is dried quickly by waving in the air and protected from dust, flies and other insects (OIE, 2013).

Treatment

Trypanocidal drugs are the most widely applied method that farmers use to treat and prevent trypanosomosis in sub-Saharan Africa. It has been estimated that about 35million doses of trypanocides are administered each year to approximately 45-60 million cattle at risk of trypanosomosis. Trypanocides are popular because farmers can directly treat and, if successful, cure their animals without relying on the efforts of others (Holmes et al., 2004).

Despite livestock keepers "dependence on trypanocides only three compounds namely isometamidium chloride, homidium (bromide and chloride), and diminazene aceturate, are currently available for treating cattle. All these drugs have been on the market for over 40years and several generic forms of them from a wide range of companies have become available on the African market (Dabo and Maigari, 2017). Isometamidium is principally used as a prophylactic drug and can provide up to 6 months of protection against trypanosomosis, homidium has limited prophylactic properties, but it is primarily used as a therapeutic agent. Whilst diminazene provides also short-term protection of 2 to 3weeks, it is mainly used for therapeutic purposes (Onono et al., 2013).

Prevention and Control

The control of trypanosomosis in enzootic countries involves control of tsetse fly population, prophylactic treatment, and good husbandry of animals at risk and use of trypanotolerant animals. Control of tsetse has been successfully attempted, 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 fly feed. More recent methods involved the use of insecticides applied strategically in the form of ground and aerial spraying over large expanses of land (Boulange et al., 2002).

The sterile insect technique as recently been used in the coast of East Africa including Ethiopia, since females only mate a few times in their life, generally only once, and mating with a sterile male prevents that female from giving birth to any off spring (Maudlin et al., 2004). Other effective methods involve targets impregnated with insecticides and traps that attract and catch tsetse. These are simple and cheap and can be constructed and maintained by local communities. Furthermore, they do not pollute the environment and are suitable for both small- and large-scale fanning (Boulange et al., 2002).

Economic Significance

Tsetse flies infect 10 million square kilometers of Africa involving 37 countries. 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. The disease in Africa costs livestock producers and consumers an estimated US$1340 million each year. The anticipated losses due to T. vivax in South America exceed $160 million.1 Furthermore, the disease may impact on various immunization campaigns in endemic areas due to the fact that it can cause immunosuppression (Aschalew et al., 2015).

Zoonotic Importance

The animal pathogens do not infect humans, but animals can serve as reservoirs of T. brucei rhodesiense and T. brucei gambiense, the causes of human sleeping sickness, which are morphologically indistinguishable from T.bruceibrucei. Human infections result from tsetse bites, generally in game parks forest reserves and along streams or other rural setting (Aschalew et al., 2015)

Materials and Methods

Study Area

The study was conducted selected peasant associations (PAs) of Nono districts in the South West Shewa Zone of Oromia Region, Ethiopia. The district is situated at about 230km southwest of Addis Ababa with the altitude range of 1500-1600 meters above sea level bordering the Gibe river system. The district is located at latitude 80o 50'N and longitude 37o45'E. The total area coverage of the district is about 50,000 hectares and the weather condition is characterized by a sub-humid climate and a moderately hot temperature with a mean annual temperature of 20oC. The highest average monthly temperature occurs in January with a mean maximum temperature of 28oC.The lowest monthly temperature occurs in August with an average monthly minimum temperature of 12oc. It receives high and reliable annual rain fall averaging 1100mm/annum with a low inter-annual variation. The livestock crop (mixed) farming system is the dominant farming system in the area. The livestock population in the study area includes, cattle 230, 000, sheeps 65,000, goats 36,000 and equine 55,127 are the predominant species in this district. Those animals have been dependent on communal grazing and watering points (NALDFB, 2020).

Study Animal

The study population was cattle of different ages, body condition, and sex kept under extensive management system in selected PA of Nono district. Study units were local cattle breeds with one year age and above included in the samples. A cross-sectional study design was conducted to determine the prevalence and associated risk factors of bovine trypanosomosis in the Nono district. The study was conducted from Nomber 2019 to April 2020 in the study area. The age of the cattle was estimated using their dentition as described by Pasquini et al. (2003) and information from owners of the cattle. The body condition was scored using the method described by Nicholson and Butterworth (1986).

Sample Size Determination and Sampling strategies

The multistage sampling method was conducted with the district as the highest and individual animals as the lowest sampling stage, peasant association (PA), village, and herd in between two stages. Nono district was selected purposively based on number of cattle and infrastructure. From a total of 33 PAs in the Nono district, three PAs were selected by the lottery method, namely, Nano Halo, Halo Dinki, and BiftuJalala. The sampling frames of the individual cattle were obtained from each respective village. A simple random sampling method was conducted to sample individual cattle from each herd. The sample sizes selected from each herd could vary based on the number of animals in each herd. The sample size was determined according to the formula given by Thrusfield (2005).

$$N = \frac{1.962\ Pexp\ (1-Pexp)}{d2}$$

Where, N = required sample size, Pexp = expected prevalence, d = desired absolute precision. The sample size determination was using 95% level of confidence, 50% expected prevalence since there was no previous study conducted in Nono districts, and 0.05 desired absolute precision. Thus, a total of 384 cattle were required for the demonstration of the study.

Study Methodology and Procedures

Buffy Coat Technique

Blood was collected from an ear vein using a heparinized micro hematocrit capillary tube which was sealed. A heparinized capillary tube containing blood was centrifuged for 5 min at 12,000 rpm. After the centrifugation, trypanosomes were usually found in or just above the buffy coat layer. The capillary tube was cut using a diamond-tipped pen 1 mm below the buffy coat to include the uppermost layers of the red blood cells and 3 mm above to include the plasma. The content of the capillary tube was expressed on to slide, homogenized onto a clean glass slide, and covered with a cover slip. The slide was examined under a 40X objective and 10X eyepieces for the

movement of the parasite (Thrusfield, 2005).

Thin Blood Smear

The trypanosome species were identified using Giemsa-stained thin blood films. A small drop of blood from a micro hematocrit capillary tube to the slide was applied to a clean slide and spread by using another clean slide at an angle of 45°, air-dried and fixed for 2 min in methyl alcohol, then immersed in Giemsa stain (1:10 solution) for 50 min. Drain and wash off the excess stain using distilled water, allowed to dry by standing upright on the rock and examined under the microscope with oil immersion objective lens. This technique is the most sensitive of the parasitological tests for the detection of T. vivax and T. Congolese (Nakayima, 2016).

Thin blood smears were made by placing a small drop of blood (about 5µl), for example from a micro hematocrit capillary tube, on a clean microscope slide approximately 20 mm from one end (allowing for space to apply the thin smear) and spreading with the edge of another slide (Nakayima, 2016). This slide is placed at an angle of approximately 30° to the first slide and drawn back to make contact with the blood droplet. The blood is allowed to run along the edge of the spreader, which is then pushed to the other end of the slide in a fairly rapid but smooth motion (Kemal, 2014). If the correct amount of blood is used, the slide should be covered with a thin film of blood with no surplus before the end of the slide is reached, and the smear should take the shape of a bullet. Ideally, thin films should be prepared so that the RBCs are fairly close to each other but not over lapping. The slide is dried quickly by waving in the air and protected from dust, flies and other insects (OIE, 2013).

The slide is fixed for 3minutes in methanol, and stained as for thick blood smears. After staining, the slide is washed gently under tap water and allowed to dry (Dabo and Maigari, 2017). A variation of this method is to fix in methanol for 2minutes, apply May–Grünwaldstain for 2 minutes, then add an equal volume of buffered water, PH 7.2, and leave for a further 8 minutes and drain off. Approximately 50–100 fields of the stained thin blood smear are examined, with a ×100 oil immersion objective lens, before the specimen is considered to be negative. Even after a trypanosome has been detected, approximately 20 extra fields are investigated to determine if more than one species is present (Nakayima, 2016). The sharp extremity of the smear must be extensively explored as, because of their capillary properties, trypanosomes may be concentrated at this place (especially true for large species like T. brucei and T. vivax) (OIE, 2013; Jamonneau et al., 2015).

Measuring of Packed Cell Volume (PCV)

Blood samples were obtained by puncturing the marginal ear vein with a lancet and collected directly into a capillary tube. The capillary tubes were placed in a micro hematocrit centrifuge with its sealed end outermost. The tube was loaded symmetrically to ensure good balance. After screwing the rotary cover and closing the centrifuge lid, the specimens were allowed to centrifuge at 12,000 rpm for 5 min. Tubes were then placed in hematocrit and the readings were expressed as a percentage of packed red cells to the total volume of whole blood. Animals with PCV < 24>et al., 2000).

Data Analysis

Data recorded during the study were stored in Microsoft Excel® window 2010. The prevalence of the parasite was computed by dividing the number of positive samples by total samples. Chi-square test was used to test the association between the prevalence of trypanosomosis and its associated risk factors such as PAs, age, body condition, sex, anemic status of the studied animals and PCV values.

Results

Overall Prevalence of Trypanosomosis

Out of 384 cattle of examined in Nono District an overall prevalence of 21(5.5%) was recorded. In this area highest prevalence was recorded in Nano Halo 8(6.7%) followed by Biftu Jalala 6(5.5%) and Halo Dinki 7(4.5%) peasant associations which have no statistically significant association observed between study Pas (X2 = 0.59, P= 0.746) (Table 1).

Table 1: Prevalence of Trypanosomosis based on Origin

Variables	Categories	No Sampled	Positive	Prevalence%	X^2	P – Value
Origin	*Nano Halo*	*120*	*8*	*6.7*	*0.59*	*0.746*
	Halo Dinki	*154*	*7*	*4.5*		
	BiftuJalala	*110*	*6*	*5.5*		
Total		*384*	*21*	*5.5*		

In this area higher prevalence was detected in young age group 13(9.6%) than in adult 8(3.2%) which has statistically significance difference (X2= 6.97, P= 0.008) (Table 2);

Table 2: Prevalence of Trypanosomosis based on Age

Variables	Categories	No Sampled	No Positive	Prevalence %	X^2	P – Value
Age	*Young*	*135*	*13*	*9.6*	*6.97*	*0.008*
	Adult	*249*	*8*	*3.2*		
Total		*384*	*21*	*5.5*		

higher prevalence was recorded in male 15(8.3%) than in female 6(2.94%) which has statistically significance difference (X2= 5.38, P= 0.02) (Table 3);

Table 3: Prevalence of Trypanosomiasis based on Sex

Variables	categories	No Sampled	No Positive	Prevalence %	X^2	P – Value
Sex	*Male*	*180*	*15*	*8.3*	*5.38*	*0.02*
	Female	*204*	*6*	*2.94*		
Total		*384*	*21*	*5.5*		

highest prevalence was recorded in poor 13(9.2%) followed by medium 7(3.3%) and good 1(3.1%) body condition which has statistically significant association observed between study PAs (X2 = 6.09, P= 0.048) (Table 4),

Table 4: Prevalence of Trypanosomosis based on body condition score

Variables	Categories	No Sampled	No Positive	Prevalence %	X2	P - Value
Body Condition	*Good*	*32*	*1*	*3.1*	*6.07*	*0.048*
	Medium	*211*	*7*	*3.3*		
	Poor	*141*	*13*	*9.2*		
Total		*384*	*21*	*5.5*		

higher prevalence was recorded in anemic 18(11.5%) than in non-anemic (normal) 3(1.3%) which has statistically significance difference (X2= 18.47, P= 0.000) (Table 5) and out of 21 species of trypanosome identified *T. Congolense* was 13(61.91%) , *T. Vivax* was 7(33.33%) and 1(4.76%) were mixed (Table 6).

Table 5: Prevalence of Trypanosomosis based on PCV

Variables	Categories	No Sampled	No Positive	Prevalence %	X^2	P – Value
PCV	*Anemic*	*157*	*18*	*11.5*	*18.47*	*0.000*
	Normal	*227*	*3*	*1.3*		
Total		*384*	*21*	*5.5*		

Table 6: Distribution of Trypanosomosis species in the study Pas

Species	PAs			Total Prevalence%
	Nano Halo	***Halo Dinki***	***Biftu Jalala***	
T. Congolense	*6(75%)*	*5(71.4%)*	*2(33.33%)*	*13(61.91)*
T. Vivax	*1(12.5%)*	*2(28.6%)*	*4(66.67%)*	*7(33.33)*
Mixed	*1(12.5%)*	-	-	*1(4.76)*
Total	*8(38.1%)*	*7(33.33%)*	*6(28.57%)*	*21(100)*

Discussion

The present study revealed that trypanosomosis is a major constraint to the utilization of large land resources and also affect livestock in the Nono district. The determined overall prevalence was 21(5.5%), where 8(6.7%), 6(5.5%), 7(4.5%) prevalence was seen in Nano Halo, Biftu Jalala and Halo Dinki peasant associations respectively. These differences in prevalence between PAs had no statistically significant association (P< 0>et al. (2014) who reported (4.43%) in the selected villages of Arbaminch, southern Ethiopia, the reports of Adale and Yassine (2004) who reported 6.3% in Wolaita zone of Kindo Koish district, Southern Ehiopia. Higher than reports of Ayana et al. (2012) who reported prevalence of (2.1%) from Amhara region, Northwest Ethiopia.

However the present result was lower than reports of Muluwa et al. (2011) who reported prevalence of 28.1% from Asosa District of Benishangul Gumuz Regional State, Western Ethiopia; Fentahun and Tekeba (2013) reported prevalence of (12.4%) from Hawa Gelan District, Oromia Region, Ethiopia and Mulugeta (2013) who reported (13.24%) from Didessa valley western Ethiopia. A significant reduction of the Trypanomes infection in the current study area could be considerable suppression of tsetse flies' population by the use of strategic application of insecticide impregnated targets, spot on (deltametrin 1%) and use of strategic prophylactic and curative Trypanocidal drug treatment of livestock in the area based on the package prepared by Oromia Regional state animal and Fisheries Health and Development Agency to control livestock disease and trypanosomosis in the western Oromia settlement areas.

The current study indicates that the higher prevalence was recorded in young age group 13 (9.6%) than in adult 8(3.2%) which has statistically significance association (P= 0.008). This agrees with reports of Gemeda (2015), prevalence of 13.79% in young, 9.40% in adult and 4.0% in old in and around Nekemte Areas, East Wollega Zone, Ethiopia.

Present study revealed that higher prevalence was recorded in male 8.3% than female 2.94%. These has significant association with prevalence of trypanosomiasis in cattle of the study area (P=0.02). This result agrees with Mulugeta (2014) who reported higher prevalence in male (12.67%) than female (12.22%) and Girma et al. (2014) indicating higher prevalence in male cattle than in female. This is a clear testimony of un stability of male animals. The higher infection rate in males compared to females in this study may be due to management related risk factors that male animals are mostly managed in extensive grazing fields where there is a high risk of tsetse challenge for longer hours than the female ones which are managed around house for milk yield especially during rainy season where oxen are which could decrease sent desert/ lowland areas which facilitates exposure to fly bites and there by the chance of getting infected by Trypanosomosis in the study area.

The study done in different body conditions revealed highest prevalence of Trypanosomosis in poor (9.3%) followed by medium (3.3%) and lower in good (3.1%) body condition. The difference in body condition is statistically significantly associated with prevalence of trypanosomiasis (P= 0. 048). This finding is in line with the report of Girma et al. (2014) who stated that, there is a significant difference (p< 0>et al. (2012) reports of higher Trypanosome infection prevalence in poor body conditioned animals than in good and medium ones, this might be attributed to immune-suppression and stress in poor body condition animals.

The current study also indicated that the prevalence value appeared to be higher in anemic (11.5%) than in non-anemic 1.3% animals which agrees with reports of Mulalem (2014). The difference between PCV values of anemic and non- anemic cattle of the study area was significant (P= 0.000). In fact the difference in mean PCV between anemic and non-anemic cattle indicated that trypanosomosis may be involved in adversely lowering the PCV values of infected animals. Regarding the case of apparently trypanosome free cattle with low PCV could be due to various concurrent disease and nutritional interference with development of anemia, conversely many cattle having high PCV also show to be infected in which it may be occurred due to recent infection.

According to the result obtained, T. congolense 13(61.91%) was the predominant species and found to be a major cause of infection in the study area followed by T. vivax 7(33.33%) and mixed 1(4.76%) infection of T. congolense and T. Vivax which agree with reports of this finding agrees with Abiy (2002) who reported the higher prevalence of T. Congolense than T. Vivax in Goro district, south Ethiopia. This may be due to suppression of tsetse flies which resulted in lower T. Vivax prevalence as seen in the current study area.

Conclusion and Recommendations

The present study indicated that Trypanosomosis is an important disease limiting livestock rearing and

agricultural activity in the Nono district of Western Shewa, Oromia, Ethiopia. The overall prevalence of Bovine Trypanosome infection in the study area was 5.5%. In this study, T. congolense, T. vivax and mixed infection of T. Congolonse and T. vivax are trypanosomes species identified. Higher prevalence of Trypanosomosis infection was observed in males, young cattle with poor body condition, anemic animal. There is statically significant association between body condition, sex and age, PCV values with infection. The current situation may get not worse as the prevention and control of Trypanosomosis is practicing in the area and that is limiting the vector and also chemotherapy.

Based above conclusion the following recommendations are recommended:

- A progressive integrated control campaign is quite necessary to minimize trypanosomosis prevalence and tsetse densities
- Strategic control of Bovine Trypanosomosis including vector control should be strengthened
- Further studies should be carried out on drug resistance which have essential roles for overall control of tsetse transmitted trypanosomosis.

Conflict Of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper

References

1. Abenga, J. N. (2014). Review: A comparative pathology of Trypanosomabrucei infections. Global Advanced Research Journal of Medicine and Medical Science, 3(12): 390-399.
2. Abiy M. 2002. Prevalence of Bovine Trypanosomosis in Goroworeda, southwest Ethiopia. DVM Thesis Abstract, Debre Zeit, 344.
3. Achenef, M., Bekele, B., 2013.Drug and Drug Resistance in Africa Animal Trypanosomosis. European Journal of Applied Science, 5(3), 84-91.
4. AlemayehuB, Basaznew B, Tewodros Fentahun, Mersha Chanie(2012).Bovine trypanosomosis;athreat to cattle production in Chena district, South west Ethiopia.OpenJournalofAnimalSciences.vol.2,No .4,287-291(2012).
5. Amanuel, D., Tadele, A., Kassahun, B., Tilahun, Z., Girma, K. And Tadele, K. (2015).Prevalence of Bovine Trypanosomosis, and it's Associated Risk Factors in Abaya District, Borena Zone, Ethiopia. Nature and Science, 13(10): 64-70.
6. Anderson, N. E., Mubanga, J., Fevre, E. M., Picozzi, K., Eisler, M. C., Thomas, R., & Welburn, S. C. (2011). Characterisation of the wildlife reservoir community for human and animal trypanosomiasis in the Luangwa Valley, Zambia. PLoS neglected tropical diseases, 5(6), e1211.
7. AschalewAyisheshim, ShimelsAbegaze, Samuel Derso, Shewatatek Melaku, Hailemariam Hailu, ShiretBelete and Natnael Mekonnen, 2015. Review on BovineTrypanosomosis in Ethiopia. Department of Veterinary Clinical Medicine, Faculty of Veterinary Medicine, University of Gonda, Gondar, Ethiopia. Acta Parasitologica Globalis6 (3): 136-146.
8. Ayana, M., Z. Tesfaheywet and F. Getnet, 2012. A cross-sectional study on the prevalence of bovine Trypanosomosis in Amhara region, Northwest Ethiopia Department of Parasitology and Pathology.
9. Azene, M., Workneh, T. S., & Woldetsadik, K. (2014). Effect of packaging materials and storage environment on postharvest quality of papaya fruit. Journal of food science and technology, 51, 1041-1055.
10. Biryomumaisho, S., &Katunguka-Rwakishaya,E. (2012). The Pathogenesis of Anemia in African Animal Trypanosomosis. Anemia, 259.
11. Boulangé, A., Katende, J., & Authié, E. (2002). Trypanosoma congolense: expression of a heat shock protein 70 and initial evaluation as a diagnostic antigen for bovine trypanosomosis. Experimental parasitology, 100(1), 6-11.
12. Bouyer, F., Seck, M.T., Dicko, A.H. Sall, B., Lo M., Vreysen, M.J.B., Chia E., Bouyer, J., Wane, A., 2014. Ex-ante Benefit-Cost Analysis of the Elimination of a Glossina palpalis gambiensis Population in the Niayes of Senegal. PLOS Neglected Tropical Diseases, e3112.
13. College of Veterinary Medicine, Haramaya University Dire Dawa, Ethiopia.
14. Dabo, N.T.and Maigari A.K.(2017).Soft Options for Effective Diagnosis of African Animal Trypanosomiasis: A Review .International Journal of Medical Evaluation and Physical Report,2(2):1-9.
15. Desquesnes, M. (2004). Livestock trypanosomes and their vectors in Latin America (Vol. 8). Paris,

France: OIE.

16. Ebhodaghe, F., Isaac C., Ohiolei, J.A., 2018.A meta-analysis of the prevalence of bovine trypanosomiasis in some African countries from 2000 to 2018.Preventive Veterinary Medicine, 160, 35-46.
17. Efrem, D., F. Bashatu, B. Balachew, H. Addisalem and D. Misgana, 2013.Prevallence of Bovine Trypanosomosis in Lalo Kile District, Kelem Wollega Zone, Oromia Regional State, Western Ethiopia, Acta Parasitologica. Globalis, 4 (2): 34-40.
18. Fentahun T. and Tekeba M. 2013.Prevalenceof Bovine Trypanosomosis and Distribution of Vectors in Hawa Gelan District, Oromia Region, Ethiopia.
19. Food and Agricultural Organization (FAO),(2006).A field guide for diagnosis ,treatment and prevention of African Animal Trypanosomosis. Corporate document repository. Food and Agriculture Organization of the United Nations.
20. Gadelha,C., Holden, J. M., Allison, H. C., and Field, M. C. (2011).Specializations in a successful parasite: what makes the blood stream-form African trypanosomes odeadly? Molecular Biochemistry and Parasitology, 179:51–58.
21. Geerts, S., Holmes, P. H., Eisler, M. C., & Diall, O. (2001). African bovine trypanosomiasis: the problem of drug resistance. Trends in parasitology, 17(1), 25-28.
22. Gemeda, F., 2015. Prevalence of Bovine Trypanosomosis in and around Nekemte Areas, East Wollega Zone, Ethiopia. Open Access Lib. J., 2: 1521.
23. Girma K, Meseret T, Tilahun Z, Haimanot D, Firew L, Tadele K 2014; Prevalence of Bovine Trypanosomosis, its Vector Density and Distribution in and Around Arbaminch, Gamogofa Zone, Ethiopia. Wollega University, School of Veterinary Medicine, Nekemte, Ethiopia .Acta Parasitologica Globalis. 5(3):169-176.
24. Gouteux, J.P., Lancien, J., 1986. The pyramidal trap for collecting and controlling tsetse flies (Diptera: Glossinidae). Comparative trials and description of new collecting technics. Tropical Medicine Parasitology, 37(1), 61–66.
25. Hamilton, P. B., Gibson, W. C., & Stevens, J. R. (2007). Patterns of co-evolution between trypanosomes and their hosts deduced from ribosomal RNA and protein-coding gene phylogenies. Molecular phylogenetics and evolution, 44(1), 15–25.
26. Holmes, P. H., Eisler, M. C. and Geerts, S. (2004). Current chemotherapy of animal trypanosomiasis. In The Trypanosomiases. In: Maudlin, I., Holmes, P. H. and Miles, M.A. (eds.). Walling ford, UK: CABI International.
27. Hunt, R. C. (2010). Molecular parasitology: trypanosomes eucaryotic cells with a different way of doing things. Microbiology and Immunology. Online.
28. Jamonneau, V., Camara, O., Ilboudo, H., Peyl hard, M., Koffi, M., Sakande, H., N"Dri, L., Sanou, D., Dama, E. and Camara, M. (2015). Accuracy of individual rapid tests for sero diagnosis of gambiense sleeping sickness in West Africa. PLoS Neglected Tropical Diseases 9, e0003480.
29. Jornal of Veternary and Advanced science, 3(8): 238-244.
30. Kemal, J. (2014). Laboratory Manual and Review on Clinical Pathology. OMICS Groups eBooks. Foster City, USA.
31. Kitila, G.,Kebede, B., Guta, D., Bekele, F., Wagari, M., Tilahun, B., Tadesse, A., 2017. Epidemiological investigation of bovine trypanosomosis and its vector apparent densities in Yayo District Illuababora Zone, Western Oromia, Ethiopia. Austin Journal of Veterinary Science & Animal Husbandry, 4(1), 1031.
32. Leak, S.G.A., 1999. Tsetse biology and ecology: their role in the epidemiology and control of trypanosomiasis. Wallingford: CAB International Publishing.
33. Leta, S., Alemayehu, G., Seyoum, Z., Bezie, M., 2016. Prevalence of bovine trypanosomosis in Ethiopia: a meta-analysis. Parasites & Vectors, 9, 139.
34. Levine, N. D. (1973). The haemo flagellate. In Levine and, protozoa parasites of domestic animal and man 2nd Ed. Burgess Publishing, Minneapolis, 36-78.
35. Loses, G. and Ikede, B., 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. Path. (Suppl), 9: 1-71.
36. Magona, J.W.,Mayende,J.S.P.,Olaho-Mukani,W.,Coleman P.G Jonsson, N.N., Welburn, S.C. & Eisler, M.C. (2003). A comparative study on the clinical, parasitological

and molecular diagnosis of bovine trypanosomosis In Uganda. Onderstepoort Journal/ of Veterinary Research. 70:213-218.

37. Mary,C.S.andDavid,M.S.(2009).Goatmedicine,2 ndEd.Wiley-Blackwell,pp.296-302.
38. Maudlin,I.,Holmes,P.H.andMiles,M.A.(2004).The Trypanosomiasis. Wallingford,UK: CAB InternationalPublishing; 1–634.
39. Mbaya, A. W., Ibrahim, U. I., & Apagu, S. T. (2010). Trypanosomosis of the dromedary camel (Camelus dromedarius) and its vectors in the tsetse-free arid zone of North Eastern, Nigeria. Nigerian Veterinary Journal, 31(3).
40. Meharenet, B., Desta, T., Lelisa, K., Tsegaye, M., Jula, D., 2020.Epidemiology of Bovine Trypanosomosis and Vector Distribution in Didessa River Basin, Ethiopia. Acta Parasitologica, 65,743–749.
41. Mezene, W., Ahimedine, B., Moti, Y., Efrem, D. and Kumela, L., 2014. Bovine Trypanasomosis and Tsetse Fly Survey in Bure District, Western Ethiopia. Acta Parasitol. Glob., 5, 95.
42. Mogk, S., Meiwes, A., Shtopel, S., Schraermeyer, U., Lazarus, M. and Kubata, B. (2014). Cyclical appearance of A fricantry panoso mesin the cere brospin alfluid: new insights in how trypanosomes enter the CNS. PLoS ONE 9:e91372.
43. Mulaw, Sh., M. Addis and A. Fromsa, 2011. Study on the Prevalence of Major Trypanosomes Affecting Bovine in Tsetse Infested Asosa District of Benishangul Gumuz Regional State, Western Ethiopia, Global Veterinaria, 7(4): 330-336.
44. Mulgeta D.,2013.Trypanosome infection rate of Glossina morsitans and trypanosomosis prevalence in cattle in upper Didessa valley western Ethiopia. International journal of current microbiology and applied science 3(3): 378-388.
45. Murray, M., Trail, J. C., Turner, D. A. and Wissocq, Y. 1983. Livestock productivity and Trypanosomosis network manual, ILCA, Addis Ababa.Pp.127.
46. Mussa A (2002). Prevalence of Bovine Trypanosomiasis in Goro wereda, Southwest Ethiopia. DVM Thesis, FVM, AAU, Debre Zeit.
47. Nakayima, J(2016).DiagnosticMethodsfor African Trypanosomiasis.,September,2016.
48. Ngure, R. M., Ndungu, J. M., Ngotho, J. M., Nancy, M. K., Maathai, R. G., & Gateri, L. M. (2008). Biochemical changes in the plasma of vervet monkeys (Chlorocebus aethiops) experimentally infected with Trypanosoma brucei rhodesiense.
49. Nicholson, M. J., & Butterworth, M. H. (1986).A guide to condition scoring of zebu cattle.ILRI (aka ILCA and ILRAD).
50. NMSA (National Meteorological Services Agency), 2013. Monthly report on temperature and Rainfall Distribution for Eastern Wollega Zone, Regional Metrological Office, Nekemte, Ethiopia
51. Nono Agricultural and Livestock andFishery Bureau 2020.
52. NTTICC, 2002. National tsetse and Trypanosomosis Investigation and control center report for the period 7 June 2003 -6 July 2004, Bedelle, Ethiopia, pp: 1-4
53. NTTICC, 2004. National Tsetse and Trypanosomosis Investigation and Control Centre (NTTICC), Annual Report, Bedelle, Ethiopia.
54. Onono, J. O., Wieland, B., & Rushton, J. (2013). Factors influencing choice of veterinary service provider by pastoralist in Kenya. Tropical animal health and production, 45(6), 1439–1445.
55. Paris, J.J. (1982).Comfort measures only for'DNR'orders. Connecticut medicine, 46(4), 195-199.
56. Pasquini, C., T. Spurgeno and S. Pasquini, 2003. Anatomy of domestic animals: Systematic and regional approach. 10th edn.Sudz.Publishin, USA, pp: 255.
57. Radostits, O., Gay, M., Hinchcliff, C.C. and Constable, P.D., 2007. Veterinary Medicine: A Text Book of Disease of Cattle, Sheep, Pigs and Goats. 10th Ed. Saunders Ltd., 1531-1540.
58. Seyoum, Z., Terefe, G., Ashenafi, H., 2013.Farmers' perception of impacts of bovine trypanosomosis and tsetse fly in selected districts in Baro-Akobo and Gojeb river basins, Southwestern Ethiopia.BMC Veterinary Research, 9, 214.
59. Shimelis, D. and Melkamu, B. (2015). Review on Trypanosoma vivax. African Journal of Basic and Applied Science, 7(1): 41-64.
60. Sinshaw, A., Abébé, G., Desquesnes, M., & Yoni, W. (2006). Biting flies and Trypanosomavivax infection in three highland districts bordering lakeTana, Ethiopia. Veterinary Parasitology, 142(1-2), 35-46.
61. Siyum, G., Tadele, K., Zelalem, A., Benti, D., 2014. Epidemiological survey of bovine trypanosomosis in Sayo District of Kellem

Wollega Zone, Western Ethiopia. American-Eurasian Journal of Scientific Research, 9, 67-75.

62. Sow A. (2013). Trypanosomosis risk factors and impact assessment of a tstse and trypanosomosis eradication campaign in Burkina Faso. PATTEC (PhD thesis). Universiteit Gent.
63. Taiwo, V.O., Olaniyi, M.O. and Ogunsanmi, A.O. (2003). Comparative lasma biochemical changes and susceptibility of erythrocytes to in vitro peroxidation during experimental Trypanosoma congolense and Trypanosoma brucei infections in sheep. Journal of Isreal Veterinary Medicine, 58: 435-443.
64. Teka W, Terefa D, Wondimu A (2012).Prevalence study of bovine trypanosomiasis and tsetse density in selected villages of Arbaminch, Ethiopia. J. Vet. Med. Anim. Health. 4(3):36-41.
65. Tesema, D. and Yitayew, D., 2015. Review on tsetse transmitted Bovine Trypanosomosis in Ethiopia. Eu. J. Appl. Sci. 7(6), 255-267.
66. Thrusfield, M. 2005. Veterinary Epidemiology; 3rd edition Black well science, Oxford, pp: 233
67. Tulu, D., 2019. Epidemiology of Bovine Trypanosomosis in Ethiopia. Epidemiology International Journal, 3(1), 000118.
68. Tulu, D., Gebeyehu, S., Aseffa, N., Negara, C., 2018. Prevalence of bovine trypanosomosis and associated risk factor in Jimma Horro District, Kellem Wollega Zone, Western Ethiopia. Journal of Veterinary Medicine and Animal Health, 10(8), 185-191.
69. Urquhart, G. M., Armour, J., Dunkan, J. L., Dunn, A. M. and Jennings, F. W. (1996). Veterinary Parasitology. 2nd Edition, Long English Language Society, 212-216.
70. Van den Bossche, P., Shumba, W. and Makhambera, P. 2000. The distribution and Epidemiology of bovinetrypanosomosis in Malawi. Veterinary Parasitology, 88:163-176.
71. Waktole, T.E., 2008.Studies on bovine Trypanosomiasis and therapeutic efficacy of selected trypanocidal drugs in Birbir valley of Gawo-Dalle district, West Oromia, MSc Thesis. Addis Ababa University, Faculty of Veterinary Medicine Debre Zeit, Ethiopia, 1-134.
72. Wanga, C. H., & Munga, L. (2011). The effective eradication and control strategies for tsetse and Trypanosomiasis the Kenyan experience. The effective eradication and control strategies for tsetse and trypanosomiasis the Kenyan experience., 27(2), 93-100.
73. WoboS.O.S,IgenezoaA.J,IdowuO.A,OtesileE.B, EkpoU.FandKehindeO.O(2010).Bovine Trypanosomiasis and its impact on cattle in derived Savanna areas of Ogun State, Nigeria. Journal of public health and epidemiology Vol1 (3), pp.43-47, .ISSN 2141-2316.
74. Zenebe, T., J. Dugassa, S. Kebede, H. Disassa, G. Kebede, O. Abebe and T. Sanbata, 2014. Prevalence of Bovine Trypanosomosis, its Vector Density and Distribution in Dale Sadi District, KellemWollega Zone, Ethiopia.Acta Parasitological Globalisa, 5(2): 107-114.
75. Zubairu A.I, Midau A, Dazala I.U, Yaha M.M, Buba Z.M.(2013). The prevalence of Bovine Trypanosomiasis in Song Local Government Area of Adamawa State, Nigeria. Global veterinarian 11 (3)310-313, 2013, (ISSN) 1992-6197,