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Review on Serum Biochemical Changes in Ruminants Infected with Major Trypanosome Species

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Abstract

African trypanosomes are pathogens for humans and livestock. They are single-cell, extra-cellular parasites that cause persistent infections of the blood and induce profound immunosuppression. The life cycle of the African trypanosome begins when a tsetse fly feeds from an infected mammalian and the epidemiology depends on three factors, the distribution of the vectors, and the virulence of the parasite and the response of the host.The major pathogenic tsetse-transmitted trypanosome species are *Trypanosoma congolense, T. vivax* and *T. brucei* in cattle, sheep and goats and are found in the bloodstream and tissues of vertebrates throughout the world. However, these species are of overwhelming importance as a serious cause of morbidity and mortality in animals in tropical regions. There are no pathognomonic gross or histopathological lesions found in the animals that die of trypanosomiasis. Most of the observed lesions are due to the circulatory disturbances caused by anemia. Serum biochemical changes are characteristics of trypanosome infections, the severity of which are often determined by the strain of the infecting trypanosome and the host. It is known that pathophysiological alterations occur in the cellular and plasma components of blood during infection. The disease affects different body organs of infected animals which lead to alterations in serum biochemical parameters like liver enzymes, Total Proteins, Albumin, Glucose and Cholesterol. Hypoglycemia, which has been shown to occur during trypanosomosis, is reported to be due to excessive utilization of blood glucose by trypanosomes for their metabolism.Continuous utilization from the blood stream of cholesterol, phospholipids and total lipids also are a contributory factor to lowering of the serum levels of lipids and cholesterol. Thus, this manuscript reviews the serum biochemical changes and the major *Trypanosoma* species infecting ruminants.

Keywords: Ruminants, Serum biochemical changes, *Trypanosoma brucei*, *Trypanosoma congolense*, *Trypanosoma vivax*.

1. Introduction

Trypanosomosis is among the well-known constraints to livestock production in Africa as it causes a serious and often fatal disease of livestock mainly in the rural poor community and rightfully considered as a root cause of poverty in the continent. Trypanosomosis are a group of serious, often fatal parasitic diseases that occur in large areas of Africa, Latin America, the Middle East, and Asia. These diseases affect most species of domestic livestock, many types of wild animals, and human beings (Vreysen, 2006).

In Africa the most important trypanosomes in terms of economic loss in domestic livestock, particularly in cattle, sheep and goats are the tsetse transmitted species *Trypanosoma congolense, T.vivax, and T. brucei brucei*. The closely related *T.brucei*sub-species,*T.b.rhodesiense* and *T.b.gambiense* cause human sleeping sickness. The distribution of African trypanosomiasis in domestic animals and human beings coincides with the known distribution of the tsetse fly vector. Tsetse flies reside in a wide range of

habitats, altogether covering over 10 million sq. km, representing 37% of the African continent, and affecting 38 of the total of 55 countries (Abebe*et al*., 1993).

Conventionally, *T.vivax* and *T.congolense* are considered strictly plasma parasites confined to the circulatory system, whereas *T. brucei* has been considered a parasite of intercellular fluids, connective tissue, parenchymatous tissues, and the fluids of the body cavities. However, *T. congolense* undergoes an early extra-vascular phase of development at the site of tsetse bite and in the pre-femoral lymph nodes therefore, this parasite cannot be regarded as a strict plasma resident (Abebe *et al*., 1993).

The disease, transmitted by tsetse flies, is caused by an infection with pathogenic trypanosomes. Such an infection causes an acute or chronic disease characterized by fever, anemia, loss of appetite and condition. Mortality and morbidity vary widely and depend on factors such as the host species and breed, the trypanosome species, the virulence of the parasite and the innate resistance of the host (Connor and Bossche, 2004).

Trypanosomosis is often a serious problem in Ethiopia, particularly in cattle. The disease, sometimes known as nagana, is characterized by lymph adenopathy and anaemia accompanied by progressive emaciation and, often, death. In endemic areas, trypanosomiasis reduces calving rates, milk yields, off take and animal work efficiency whereas animal mortality is increased (Dagnachew and Shibeshi, 2011).

Serum biochemical changes are characteristics of trypanosome infections, the severity of which are often determined by the strain of the infecting trypanosome and the host. It is known that pathophysiological alterations occur in the cellular and plasma components of blood during infection. The disease affects different body organs of infected animals which lead to alterations in serum biochemical parameters like liver enzymes, total proteins, glucose, cholesterol, albumin, urea, creatinine and minerals. Total proteins and gamma globulins increase while serum albumin decreases in several Trypanosomes infections (Herrera *et al*., 2002). Nevertheless, Katunguka-Rwakishaya *et al*. (1999) and Osaer*et al*. (2000) reported significant decline in total protein due to decreased albumin in sheep and ewes experimentally infected with *T. congolense*. Trypanosomosis is known to cause anaemia, hypoproteinemia, leukocytosis, immunosuppression, hypoglycemia and changes in serum enzyme and cholesterol levels.

Therefore the objectives of this seminar are:-

- ✓ To review the serum biochemical changes in ruminants infected with major trypanosome species (*T.brucei*, *T.vivax* and *T.congolense*),
- ✓ To describe the major *Trypanosoma* species infecting ruminants, and
- ✓ Highlighting their biology, life cycle, epidemiology, pathogenesis and relationship with their hosts.

2. TRYPANOSOME

The *Trypanosoma* is classified as a Flagellate Protozoa from the genus *Trypanosoma* of the Family *Trypanosomatidae* which Belong to the Order *Kinetoplastida*of the class *Zoomastigophora*. There are many species of *Trypanosoma*. Three species, *Trypanosoma congolense*, *T. vivax* and *T. brucei* cause animal African trypanosomiasis or nagana disease. Only two subspecies of *Trypanosoma brucei*, *T. brucei gambiense* and *T. brucei rhodesiense*, which are morphologically indistinguishable, is infectious to humans (Soulsby, 1982).

Members of genus *Trypanosoma* are found in the bloodstream and tissues of vertebrates throughout the world. However, a few species are of overwhelming importance as a serious cause of morbidity and mortality in animals and man in tropical regions (Urquhart *et al*.,1992). Species of trypanosome infecting mammals fall into two distinct groups and accordingly, have been divided into two sections: (A) the Stercoraria (subgenera *Schizotrypanum*, *Megatrypanum*and *Herpetosoma*), in which trypanosomes are typically produced in the hindgut and are then passed on by contaminative transmission from the posterior; and (B) the Salivaria (subgenera *Duttonella*, *Nannomonas*,*Trypanozoon*), in which transmission occurs by the anterior station and is inoculative; characteristically, salivarian species, by virtue of variant surface glycoprotein(VSG) genes, are the only trypanosomes to exhibit antigenic variation (Hoarse, 1972).

With the exception of *T.equiperdum* of equines which is venereal disease;all have arthropod vector in which transmission is either cyclical or non-cyclical. In cyclical transmission the arthropod is a necessary intermediate host in which the trypanosomes multiply

undergoing a series of morphological transformations before forms infective for the next mammalian host are produced. So that the new infection is transmitted when feeding, the process is known as anterior station development and the various species of trypanosomes which use this process are often considered as a group, the Salivaria. All are trypanosomes transmitted by tsetse flies, the main species being *T congolense, T.vivax and T.brucei*. In other trypanosomes, multiplication and transformation occurs in the gut and the infective forms migrate to the rectum and are passed with the faeces: this is posterior station development and the trypanosomes species are grouped together as the Stercoraria. In domestic animals these are all relatively non-pathogenic trypanosomes such as *T.theileri* and *T.melophagium* transmitted by *Tabanid* flies and sheep keds respectively, but this is certainly not the case in man in which *T. cruzi*, the cause of the serious Chagas' disease in South America, is transmitted in the faeces of reduviid bugs. Non-cyclical transmission is essentially mechanical transmission in which the trypanosomes are transferred from one mammalian host to another by the interrupted feeding of biting insects, notably *Tabanids* and *Stomoxys*(Urquhart *et al.*, 1992).

2.1. Major Trypanosomes of Ruminants

Trypanosomosis is a major constraint on ruminant livestock production in many areas of Africa, Asia, and South America. Many animal species can be affected by the different trypanosomes, thus severely impairing the economic efficiency in endemic areas (Gutierrez *et al.*,2006).The major pathogenic tsetse-transmitted trypanosome species are *T. congolense*, *T. vivax* and *T. brucei* in cattle, sheep and goats (Nantulya,1990).

2.1.1. T.brucei

The African parasites of the species *Trypanosoma brucei* are extracellular hemoflagellate protozoa transmitted by the bite of the tsetse fly, capable of infecting both humans and livestock. *T. b. brucei* is one of the chief pathogens infecting livestock, producing a disease known as nagana (De Sousa *et al*., 2011).From the experimental view point, *T. brucei* infection has been reproduced in Nigerian West African Dwarf goats using a local virulent strain (Chiejina *et al*.,2005).

2.1.2. T.vivax

T. vivax accounts for up to half of total *Trypanosoma* prevalence in West Africa where it is considered the major pathogen for livestock and small ruminants (Njiokou*et al.*, 2004). Outside tsetse endemic areas, West African *T. vivax* isolates were introduced long ago into South American countries where it represents a real threat since it can be efficiently transmitted across vertebrate hosts by mechanical means and by various biting flies and *Tabanids* (Osorio *et al*.,2008). The severity of the disease depends on parasite strain, endemicity and host species, but the key steps in the *T. vivax* host interactions are still largely unknown. Several pieces of evidence point to the importance of host genetic factors in determining individual susceptibility and/or resistance to this infection (Njiokou *et al*.,2004).

T. vivax is the second most common trypanosome of goats in Africa. *Trypanosoma vivax* can be transmitted to domestic livestock by tsetse flies (*Glossina* species cyclical transmission) as well as directly (mechanical transmission) by other blood sucking insects thus allowing *T. vivax* to extend its distribution beyond tropical Africa. *T. vivax* was introduced in South America in 1830 by a shipment of zebu cattle from Senegal (Davila and Silva, 2000).

2.1.3. T.congolense

T. congolense is the most common trypanosome of goats in Africa. Goats can also act as a reservoir of *T. congolense* for other species. In the Sudan, goats infected with *T. congolense* developed a chronic form of disease from which many spontaneously recovered. When the organism was passage from goats into calves, however, acute fatal bovine trypanosomosis occurred (Gutierrez *et al.*, 2006).

Infection of cattle with *Trypanosoma vivax* or *Trypanosoma congolense* causes lesions in the male reproductive organs of cattle. *T. congolense* appears to cause more severe effects than *T. vivax* and caused highly significance and drastic decrease in sperm concentration and volume and also increases in sperm morphological defects which results in complete infertility of bulls in the late stages of infection (Sekoniand Rekwot, 2004).

2.2. Biology and Life Cycle of Trypanosomes

2.2.1. Biology of Trypanosomes

Regardless of whether they are mainly tissue-fluid dwelling, as is the casefor *T. brucei*, or blood dwelling, such as for *T. vivax* and *T. congolense*, the level of trypanosomes in the blood fluctuates with time, due to antigenic variation. This survival process shares a number of features with evasion systems in many other micro pathogens, including viruses and bacteria. There are general logistic similarities among these systems, which collectively are known as contingency gene systems (Moxon *et al.*, 1994).

The basis of the trypanosome system of antigenic variation is the protective coat on the parasite. The entire cell surface of bloodstream and metacyclic form trypanosomes, including the flagellum, is covered with a coat that is thought to provide general protection against non-specific host resistance mechanisms. The coat is a prominent immunogenic and elicits high titers of antibodies that are lytic to the parasite. Through antigenic variation, which operates simply by rare individuals changing to another coat, some parasites survive and can produce a new wave of growth. Each variant is termed a distinct variable antigen type (VAT). The different VATs retain the general protectiveness of the coat, while providing the variation enabling avoidance of specific antibodies. As antigenic variation is centrally linked to the growth and transmission of parasites, it is important to understand its underlying organismal, molecular and genetic mechanisms (Borst, 2002).

The population biology of trypanosomes has developed as a major subject of research since the early 1980s. The major thrust of the research during that period has been directed at three main, interrelated topics: taxonomy, genetic exchange and molecular epidemiology. For disease control, it is crucially important to define and identify species, subspecies, strains and populations of trypanosomes to enable epidemiological investigation (Tait, 2000).

2.2.2. Life Cycle of Trypanosomes

The life cycle of the African trypanosome begins when a tsetse fly feeds from an infected mammalian host. The disease is maintained in ecological system which includes tsetse flies, woody vegetation and game or wild life. It is only when livestock is introduced into this system that tsetse fly will use the livestock as their food source and infect them with trypanosome. Additionally, it is believed that biting flies including *Tabanidae* and *Stomoxys* also transmit the parasite mechanically. This activity is responsible for the persistence of *T. vivax* in areas of Africa free from tsetse flies as wells in several South American countries like Brazil, Colombia, and Guyana (Ugochukwu, 2008).

The life cycle of the single-celled trypanosome is complex in both the tsetse fly vector and mammalian host, trypanosomes undergo a series of transformations into different forms as flies feed on animals infected with the parasite, they take up blood containing trypanosomes which then completes the life cycle (Ilard, 1990). After ingestion of a blood meal from an infected host, tsetse fly becomes infected. In the insect vector mid-gut, the trypanosomes develop into procyclic trypomastigotes and divide for approximately 10 days and then they obtain a functional cytochrome system and TCA cycle. After division cycles, the parasites migrate to the salivary glands and transform into epimastigotes. These forms divide and transform into metacyclic trypomastigotes, the infective stage for mammalian hosts. The life cycle of trypanosome in the insect is 25-50days, which depends on the fly species, the trypanosome strain and the temperature. If tsetse flies infected by more than one strain of trypanosome, genetic exchange between the two strains happens and it increases genetic diversity (Khosravi, 2010).

2.3. Epidemiology

Three elements influence the epidemiology of African animal trypanosomiasis, namely: the trypanosome, the tsetse fly and the animal itself. Many studies have been done and are presently underway on each of these elements. Any discovery, even if it is only partial, leads not only to a better understanding of this complex group (parasite-vector-host and their multiple interactions) but also to a better control of the disease. Further, despite immense progress, the exact epidemiology of animal trypanosomiasis is still poorly understood for several reasons. Clinical diagnosis is difficult as there may be no pathognomonic signs. Detection of trypanosomes is the only proof of the disease (Bauer *et al*., 1987).The epidemiology depends on three factors, the distribution of the vectors, and the virulence of the parasite and the response of the host (Urquhart, 1992).

2.3.1. Vectors

Of the three groups of *Glossina* the savannah and riverine are the most important since they inhabit areas suitable for grazing and watering. Although the infection rate of *Glossina* with trypanosomes is usually low, ranging from 1 to 20% of the flies, each is infected for life, and their presence in any number makes the rearing of cattle extremely difficult. Biting flies may act as mechanical vectors, but their significance in Africa is still undefined. However in Central and South America, *T.vivax* is thought to be transmitted readily by such flies (Urquhart, 1992).

2.3.2. Parasites

The parasite virulence, immunogenecity and response to chemotherapeutics are also important factors in the epidemiology of trypanosomosis as the trypanosome species occur in a remarkable variety of genotypes. Since parasitaemic animals commonly survive for prolonged periods, there are ample opportunities for fly transmission, especially of *T. brucei*and *T. congolense*. In contrast, some strains of *T.vivax* in cattle kill their hosts within 1-2 weeks so that the chances of fly infection are more limited (Urquhart, 1992).

2.3.3. Hosts

Trypanosomiasis is basically an infection of wildlife in which, by and large, it has achieved a *modus Vivendi* in that the animal hosts are parasitaemic for prolonged periods. But generally remain in good health. Thissituation is known as trypano-tolerance. In contrast, rearing of domestic livestock in endemic areas has always been associated with excessive morbidity and mortality although there is evidence that a degree of adaptation or selection has occurred in several breeds. Thus in West Africa small humpless cattle of the *Bostalirus* type, notably the N'dama**,** survive and breed in areas of heavy trypanosome challenge despite the absence of control measures. However, their resistance is not absolute and trypanosomoses exerts a heavy toll, particularly in productivity. In other areas of Africa, indigenous breeds of sheep's and goats are known to be trypano-tolerant, although this may be partly due to their being relatively unattractive hosts for *Glossina.* Precisely how trypano-tolerant animals cope with antigenic variation is unknown. It is thought that the control and gradual elimination of their parasitaemias may depend on the possession of a particularly rapid and effective antibody response, although other factors may also be involved (Urquhart, 1992).

2.4. Host parasite Interaction

Interaction refers to the interdependent operation of factors to produce effect. Following the bite of the mammalian host by a trypanosome infected tsetse fly, the parasites multiply locally in the skin and provoke a local host response that manifests itself as an indurated skin lesion called the chancre. The parasites enter the blood circulation via lymph vessels and can survive in the blood circulation throughout the infection of the host and thus, the parasite remains continually exposed to the host's immune system. *T. brucei* species have the ability to penetrate the walls of capillaries, invade interstitial tissues, but always remain extracellular. *T. congolense* is an extracellular, intravascular blood parasite that is unable to leave the circulation and has a tendency to bind to walls of capillaries and small vessels of infected cattle and mice. African trypanosomes have evolved very sophisticated evasion mechanisms to survive in the chronically infected host. Well-documented evasion mechanisms include antigenic variation of the VSG and the induction of alterations in the host's defense system, such as excessive activation of the complement system leading to persistent hypocomplementemia, down regulation of nitric oxide production, polyclonal B-lymphocyte activation, and marked immunosuppression (Tabel *et al*., 2008).

2.5. Pathogenesis

Even though the mechanisms of pathogenesis of trypanosomiasis is not clearly understood, trypanosome derived substances and immune complexes comprising trypanosome antigens and parasite-specific IgM and/or IgG antibodies plus complement were shown to be involved in the pathogenesis (Mansfield, 1990). Immune complexes have been detected in several tissues of infected animals and humans including heart, brain, kidney and skeletal muscles. It has been suggested that trypanosomes generate toxic catabolites and biologically active metabolites with complement activating and inflammatory properties, such as cytokines, vascular amines and plasma proteases which can contribute to the pathogenesis of the disease (Liu *et al*., 1993). Highly susceptible mice infected with *T. congolense* die of a systemic inflammatory response syndrome (SIRS) that is mediated by IFN-γ (Shi *et al*., 2005). This SIRS was associated with elevated plasma levels of IL-6, IL-12p40, IL-10, and

IFN-γ. Focal liver lesions of apoptotic parenchymal cells, 5-fold enlargement of Kupffer cells, and apoptosis of 10% of Kupffer cells, enlarged capillary bed, hypotension, decreased body temperature, piloerection, hypomotility and death were also associated with SIRS (Shi *et al*., 2006).

3. SERUM BIOCHEMICAL CHANGES

Serum biochemical and hematological aberrations are characteristics of trypanosome infections, the severity of which are often determined by the strain of the infecting trypanosome and the host. It is known that pathophysiological alterations occur in the cellular and plasma components of blood during infection. Serum protein changes have been reported in West African dwarf sheep experimentally infected with *T.brucei*, in *T. congolense* infected Scottish Blackface sheep and *T. congolense* infected Zambian goats. Immunosuppresion is a frequent accompaniment of African trypanosome infections. Also serum protein changes have been reported in trypanosomosis due to experimental *T. vivax* infection in sheep and *T. congolense* infected sheep (Takeet *et al*., 2009).

Tissue activities of *T. brucei* subspecies evoke marked serum biochemical derangements. Serum total proteins had been found to be generally increased accompanied by increase in serum globulins and decrease in albumin levels with resultant fall in the albumin: globulin ratio. However, normal or decreased total proteins had also been reported in infections due to other trypanosome species. Increases in total proteins in African trypanosomiasis is believed to arise from hyperglobulinaemia and especially hypergammaglobulinemia and has been associated with increase in immunoglobulin M (IgM) as well as dehydration which are consistent finding in trypanosomiasis of man and animals. Albumin is produced entirely in the liver and plays important roles in the regulation of flow of water between the plasma and tissue fluids by its effect on colloid osmotic pressure. Hypoalbuminaemia on the other hand is therefore the result of decreased protein synthesis due to hepatic dysfunction in trypanosomiasis (GARJMMS, 2014).

3.1. Glucose

Glucose is the only sugar found in blood, which is stored in the form of polymer glycogen. The level of glucose is maintained within a relatively narrow range and is controlled by several factors such as hepatic and renal uptake and release of glucose; glucose removal by the peripheral tissue; effects of hormonal influences on these processes; and intestinal absorption of glucose, which has only temporary effect on blood glucose levels (Coles, 1986).

However during a disease situation there is a decrease in glucose level in the blood. For instance, according to Kadima *et al*., (2000) serum glucose levels were significantly low on days 3, 4 and 5 post infections of cattle with *T. vivax*, corresponding with the first parasitaemia build up. Hypoglycemia was also observed in groups of infected sheep and this became apparent during the period of first wave of parasitaemia (14 days post infection). Hypoglycemia, which has been shown to occur during trypanosomosis, is reported to be due to excessive utilization of blood glucose by trypanosomes for their metabolism through their glycolytic pathway (Takeet *et al*., 2009).

3.2. Albumin

The degree of Hypoalbuminaemia was related to the level of parasitaemia and/or severity of the disease (Katunguka-Rwakishaya *et al*, 1997). Albumin is required by trypanosomes for growth and multiplication, hence development of high parasitaemia particularly in the early stages of infection (high parasitaemia) may lead to increased utilization of albumin and this coupled with heamodilution may lead to a reduction in the concentration of albumin. In parasitic gastroenteritis, excessive amounts of serum proteins leak into the parasitized stomach and intestines as a result of increased mucosal permeability, thereby resulting in severe Hypoalbuminaemia. The severity depends on the magnitude of the protein leakage, the animal protein intake and the ability to synthesize more serum proteins in order to maintain homeostasis (Chiejina, 1987). The Hypoalbuminaemia and the decreased protein retention common in parasitic gastroenteritis contribute to the increased water retention and the tissue oedema in parasitized animals (Hammerberg, 1986).

3.3. Total protein

The fall in total serum protein level may also be due to reduced protein synthesis arising from damaged liver or as a result of excessive protein breakdown arising from reduced feed intake as observed in all the infected animals. It could also arise from low level of albumin caused by catabolism, uptake of albumin by

trypanosomes or heamodilution (Katunguka-Rwakishaya*et al*., 1997) which is contributory factor to plasma total protein level. In sheep infected with *T. brucei* no change in levels of total plasma proteins from pre-infected values at the initial stage of the infection, but in the later stage the levels increased significantly above pre-infection levels (Taiwo *et al*., 2003). Protein levels usually drop in trypanosome infections as a result of depressed albumin levels. The increase in protein levels during the chronic phase of the infection is usually due to the increase in globulin levels. This is as a result of the immune response by the animals to the infection (Rajora *et al*., 1986).

3.4. Cholesterol

There had been conflicting reports on the serum biochemical changes in animals infected with trypanosomes. Nakamura (1998) reported increase in the plasma cholesterol level and all lipid forms except HDL- cholesterol in *T. brucei* infected rabbits while Biryomumaisho *et al*., (2003) observed decrease in plasma level of cholesterol and the HDL-cholesterol following experimental infection of goats with *T. brucei* and *T. congolense.* It is known that lipid is an important macromolecule in the body serving as hormone and or hormone precursor, aiding in digestion, providing energy storage and metabolic fuel, acting as functional and structural component in biomembrane, forming insulation to allow nerve conduction and prevent heat loss. Therefore, alteration of these lipid levels in the plasma could cause a number of clinical disorders (Takeet *et al*., 2009).

Although the cause of disparities in the serum levels of lipids and cholesterol in some of the previous reports was not investigated, differences in species or strain of trypanosomes or of the host animal breed and species used in the different studies might have contributed to the variable results obtained. Irrespective of the host and parasite species involved, the clinical disorders inflicted by trypanosomes are generally the same and most of the times are consistent. The alterations observed in the serum concentrations of the triglyceride, high density lipoprotein and cholesterol could involve many pathophysiological mechanisms (Adamu *et al*., 2008). It has been reported that trypanosomes require lipoproteins for them to multiply under axenic culture. Thus, the lowering of the serum the lipids and cholesterol as observed in the present and previous studies (Biryomumaisho *et al*., 2003; Adamu *et al*., 2008) could, partly, be the result of trypanosomal utilization of the molecules. Also, blood-stream forms of trypanosomes are unable to synthesize cholesterol and so require it along with phospholipids and total lipids for synthesis of their membranes and growth. Thus, the continuous utilization, from the blood stream, of these molecules could therefore be a contributory factor to lowering of the serum levels of lipids and cholesterol (Green *et al*., 2003).

3.5. Aspartate Aminotransferase (AST), Alkaline Phosphatase (ALP), and Alanine Aminotransferase (ALT)

Enzymes are protein catalysts synthesized by all living things. As catalysts their only biologic activity is to alter the rate at which equilibrium is established between reactants and their products. Enzymes of cellular metabolism are located within tissue cells and are present there in high concentrations as long as the cell remains healthy and the membrane is intact. However during infection the level of these enzymes in extracellular fluids and plasma become abnormally high. Alterations in serum enzymes activity due to malfunctioning indicate damage to the different vital organs. The increase in serum ALT and AST and reduced cholesterol levels may be associated with liver damage, while the elevated serum urea and creatinine levels may probably be due to renal damage(Egbe-Nwiyi *et al*., 2005).

The result of enzyme assays showed elevation of both ALP and AST in *T. brucei* infected sheep, while these enzymes remained unchanged in *T. congolense* infected sheep. This has been reported to be due to tissue breakdown (necrosis) and inflammation in the host, particularly of the liver, heart, muscle and kidney (Lososand Ikede, 1972). Another possibility is the increase induced by the lysed trypanosomes at different stages of the infection. It is possible to suggest that in this case, increases seen only in *T. brucei* infected sheep were due to the fact that *T brucei* has the ability to invade solid tissue, especially the liver, kidney and heart, thereby localizing and causing tissue damage. This leads to the release of these enzymes from the damaged tissues and are measurable in the plasma (Whitela *et al*., 1980).

The increase in AST values in the disease on days 3 to 5 post infection could not have been due to any tissue damage, but rather due to parasites secreting it as part of their metabolites into blood circulation, since ALT and AST have been observed in homogenates and suspensions of trypanosome. However, the decreases following the second parasitemic build-up suggest a

possible progressive liver fibrosis occurring as the disease progresses(Kadima *et al.,* 2000).

Alkaline phosphatase is an enzyme that encompasses a family of phosphatases that carry out their enzymatic activities in an alkaline environment (Bain, 2003). The elevation of serum enzymes are usually due to tissue damages resulting in the leakage of these enzymes from intracellular stores into the plasma (Orhue *et al.,* 2005). The elevation of ALP in the serum is associated with liver damage (Egbe-Nwiy *et al.*, 2010).

4. Conclusion and Recommendations

Trypanosomosis is a major constraint on ruminant livestock production in many areas of Africa, Asia, and South America. Many animal species can be affected by the different trypanosomes, thus severely impairing the economic efficiency in endemic areas. The major pathogenic tsetse-transmitted trypanosome species are *Trypanosoma congolense, T. vivax* and *T. brucei* in cattle, sheep and goats. Serum biochemical aberrations are characteristics of trypanosome infections, the severity of which are often determined by the strain of the infecting trypanosome and the host. It is known that pathophysiological alterations occur in the cellular and plasma components of blood during infection. The increase in serum ALT and AST and reduced cholesterol levels may be associated with liver damage. The elevation of serum enzymes are usually due to tissue damages resulting in the leakage of these enzymes from intracellular stores into the plasma. The elevation of ALP in the serum is associated with liver damages. On top of this there is a decrease in the serum level of glucose due to excessive utilization of blood glucose by trypanosomes for their metabolism through their glycolytic pathway and albumin as it is required by trypanosomes for growth. Most likely African trypanosomes induce other, yet undiscovered, changes in the physiology of the infected host, which might interfere with effective control of the parasite.

Therefore, based on the above conclusion the following recommendations are forwarded:

- Knowing the level of serum biochemical changes is important to predict the prognosis of the disease, to determine pathological changes in the body; thus further investigation must be done on the level of serum biochemical changes.
- The parasite induce other undiscovered changes in the physiology of the infected host, which might interfere with effective control of the parasite
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and this undiscovered changes in the physiology of the infected host must be investigated.

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