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Review on Camel Trypanosomosis: its Epidemiology and Economic Importance

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Abstract: Trypanosomoses causes a significant impact in food production and economic growth in many parts of the world. Camels are affected by many infectious and parasitic diseases. Among parasitic diseases, camel trypanosomosis (Surra), caused by *Trypanosoma evansi* is the most important single cause of morbidity and mortality in camels. The disease transmitted non-cyclically by haematophagus flies (eg. *Tabanus)* is endemic in Africa, Asia, central and South-America and in addition to camels other species of domesticated livestock are affected. Because of the wide spread of the disease, its control has attracted international attention, with focus on formulating and implementing effective strategies aimed at increasing productivity and achieving decrease in morbidity and mortality. Camel trypanosomosis is an acute/chronic disease of camel results progressive anaemia, anoxic condition and immunosuppression which later develops and predisposes the animal to other infections and death if untreated. It causes economic losses as a result of reduced productivity, abortion in all age groups of pregnancy period, drop in milk and meat yield morbidity up to 30% and mortality of around 3%. It has marked seasonal incidence related to wet and humid conditions and increased activities of biting flies during the wet season. The paper reviews the epidemiology of the disease and host response against the parasite. Therefore, emphasis is placed on accurate diagnosis of Surra, treatment with effective trypanocidal drugs and the use of vector control methods in the control and management of this disease.

Key words: Camel · Epidemiology · Trypanosoma evansi · Trypanosomosis · Vector control

INTRODUCTION

The livestock sector contributes 40% of the global value of agricultural output and support the livelihoods and food security of almost a billion people [1]. In many developing countries, livestock keeping is a multifunctional activity. Beyond their direct role in generating food and income, livestock are a valuable asset, serving as a store of wealth, collateral for credit and an essential safety net during times of crisis [2, 3].

Camels are the most numerous species of animal in the arid areas of Asia and Africa, particularly in east African counties (Sudan, Ethiopia, Somalia and Kenya, Djibouti). One humped camel, *Camelus dromedarius*, is an important livestock species uniquely adapted to hot and arid environment more than any other domestic animals [4]. Approximately, 11.5 million camels occur in this region and represent over 80% of camel's population of Africa and two-third of world [5-9]. In Ethiopia approximately 2.6 million camels are found in the South Eastern, North Eastern and Western parts of the country [4, 9, 10]. Camels are playing significant multi-purpose role in the dry land of about 50% of the total areas of Ethiopia [5, 11].

The commonest use of camels by pastoralists is for transportation of grain, salt and other goods and for milk production. Even during the dry season and drought period when milk from cattle and goat is scarce, camels are very reliable milk producers. Generally, camel is the most efficient domesticated animal for converting fodder into work, transport, milk and meat in arid and semi arid area [6, 9, 12].

Compared with many other species, the camel is not affected by many an acute or sub acute diseases. Outbreaks are rare; on the other hand chronic forms of bacterial and parasitic disease are frequent [13, 14]. Among many parasitic diseases known to affect camels;

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Camel trypanosomosis also known as Surra, is a disease of camels caused by *T. evansi*. The disease is the most important single cause of economic losses in camel rearing areas causing morbidity up to 30% and mortality of around 3% as reviewed by Enwezor and Sackeyp[11] and Njiru *et al*. [15] and Tekele and Abebe [16]. *Trypanosoma evansi*, the protozoan parasitic cause of camel trypanosomesis (surra), constitute one of the major veterinary problems in worldwide [15, 17- 19]. The disease is transmitted mechanically by vector mainly *Tabanus* species. Transmission enhanced when camels congregated or closely herded and when they have high numbers of parasite in their blood [20- 22].

The disease is characterized by fever, progressive emaciation, anaemia, sub-cutaneous oedema, nervous sign and death and is regarded as the most significant health problem in camels [23- 26]. It is most severe in horse, donkey, mules, camels, dogs and cats. *T. evansi*, like other pathogenic trypanosomes induce a generalized immune-suppression [4]. There were different prevalence rate of camel Trypanosomosis in some countries based on serological tests reported as 27% in Nigeria [27], 22% in India [28], 28% in Kenya [15], 10% in Iran [29] and 21% in Ethiopia [5].

Even if a numerous investigation has been carried out in different areas of the world, the prevalence rate of the disease was somewhat under reported. Moreover, research in the area of identifying the exact epidemiological surveillance systems and its associated risk factors is also at its infant stage because of lack of coordinated infrastructures. So, in countries such as Ethiopia where camels have no equal acceptance by all community and the disease expected to be prevalent, the knowledge of the diseases and factors associated to such important disease is crucial. Therefore, in line with the above introduction, the objectives of this paper are to: Review and give background information on Epidemiology of the disease and recommend modern control measures and further study on the current status of the disease in Ethiopia.

Camel Trypanosomosis (SURA)

Historical Background and Description of the Disease: The disease is caused by *T. evansi* which was discovered by Griffith Evans, in 1880 in infected camels and equids in the Dara Ismail Khan District of Punjab [10, 27, 30, 31]. The local Indians had a local name for the disease-Surra, meaning emaciated [32, 33]. These Trypanosomes are microscopic, elongated unicellular organisms, that move with the help of single flagellum directed forwards, at the base of which is found characteristics structure, the kinetoplast. The disease is commonly called as camel trypanosomosis (Surra); in addition to that locally known as "Dhukane"(emaciation) in Ethiopia,"Debaba"(fly) in United Arabi-Emirate and "Salaf "(rotten) in Somalia [33-3].

It is thought that *T. evansi* evolved from its ancestors along the edges of the tsetse fly belt in Africa and from there was spread via infected camels used for trade with India [36]. Continuous mechanical transmission by blood-sucking flies in the absence of *Glossina* caused the loss of cyclic transmissibility and gave rise to a predominance of slender parasite forms [4]. But in addition the name Surra, other names such as "Murrina" "Malde-Caderas "or "Derrengadera" are used to describe similar disease caused by trypanosomes indistinguishable from *T. evansi* in South America. The collective name American Surra has since proposed for adoption of uniformity in nomenclature in the South America [27, 37].

Etiology and Morphology: Trypanosomosis in *Camelus dromedarius* is commonly caused by *T. evansi* which is belonging to sub-genus Trypanozoon. *T. evansi* is long slender trypanosomes with a prominent undulating membrane and long free flagellum as shown in Figure 1 [35]. Morphologically, *T. evansi* is monomorphic and similar to the slender form of *Trypanosoma brucei* with centrally placed nucleus, small sub terminal kinetoplast, a well developed undulating membrane, a long free flagellum and a blunt or truncated from *T. brucei* by adaptation to a non-cyclical mode of transmission and loss of ability to undergo growth and differentiation in the fly vector [22, 15].

This happens when camels that came to contact with tsetse fly vector acquired infections and when such camels moved to non tsetse areas, transmission was spread by other haematophagus flies [5, 38]. However, other species of *T. brucei, T. congolense* and *T. vivax* as reviewed by Enwezor and Sackeyp [11] are also a causative agent, but their role in camel trypanosomosis is insignificant [21].

Evidences from isoenzyme studies and characterization of nuclear Kintoplast DNA (KDNA) support this hypothesis and suggest only limited evolutionary origin of

Trypanosoma evansi [7, 39]. The most notable molecular difference is that while the kinetoplast DNA (KDNA) of *T. evansi* lacks maxicircle DNAs and posses only inter locked 1-kb minicircle DNAs, the KNDA networks of *T. brucei* has both [11].

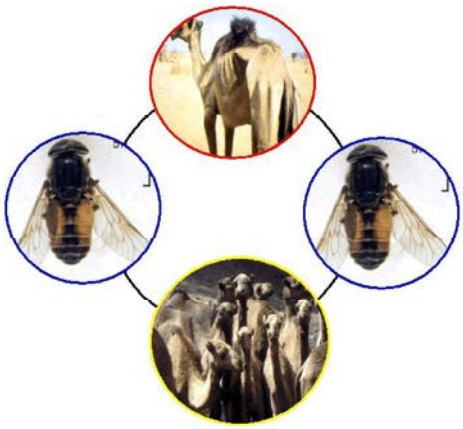

Fig. 1: Haematophagus flies (Tabanus)
Source for both figures: [35]

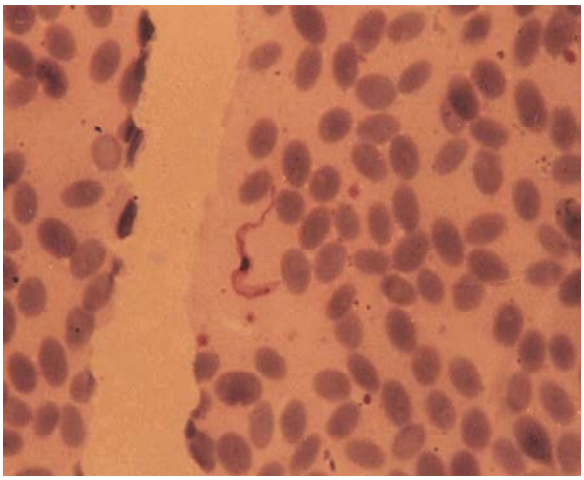

Fig. 2: Morphology of *T. evansi* in camel blood smears

Epidemiolgy

Life Cycle: Replication of the trypanosome occurs by longitudinal binary fission both in the host and in the vector with the flagellum and kinetoplast dividing together [40], but in the non-cyclically transmuted *T. evansi* developmental stages were not observed in any of the mechanical vectors [17, 41].

Consequently a procyclic or insect stage (epimastigotes) does not exist in *T. evansi* which is attributed to lack of maxi circles in the kinetoplast DNA [42]. The non-cyclical transmis-sion of trypanosomes is aided by biting flies and thus, in the absence of *Glossina,* the transmission is maintained in the ecosystem. Biting flies, such as *Tabanids* (horse flies), *Stomoxys* and *Hippoboscids* transmit *T. evansi* mechanically through their mouthparts when they feed on more than one host within a short interval because the trypanosomes remain infective for only a short period [43].

Vectors: Trypanosoma evansi lacks the genes necessary for mitochondrial development [11] and is therefore unable to undergo growth and differentiation in the vector [44]. Camel trypanosomosis have been reported to be mechanically transmitted by number of species of haematophagus biting flies including *Tabanus*, *Stomoxys* and *Haematobia* species which are present around river banks and watering places in arid zones [4, 15 and 19]. In Central and Southern America the vampire bats also act as a vector (Soulsby, 1982). More than 20 different species of *Tabanus* have been shown experimentally to transmit *T. evansi* [7,37].

Disease outbreak occurs in Surra in a seasonal pattern associated with increasing number of biting flies during or shortly after the rainy season [6]. No cyclical development occurs in the fly vector and the trypanosomes survive few minute outside the host in the mouth of fly vector [31, 37]. The prevalence of some *Tabanus* species, all year round that transmission of the parasite occurs wherever reservoir host, vector and susceptible host co-exist. This finding may explain the sporadic occurrence of the disease during the dry season and outbreaks during the rainy season [11]. However, the efficiency of the different flies to transmit *T. evansi* appears to vary depending on different geographic conditions, interval between two successive feeds and intensity of the fly challenge [37, 45].

Transmission: Trypanosomosis undergo two ways of transmission that means cyclical and non-cyclical way of transmission. Of the two ways of transmission as [36] reported that the only method of transmission of *T. evansi* both under experiment and natural condition is mechanical with various blood sucking Diptera: *Tabanus, Stomoxys* and *Hypobusca* plays important role [16, 46].

Transmission of *T. evansi* both under experiment and natural condition is mechanical with various bloodsucking [10]. Transmission by biting flies is not the sole means by which infection is perpetuated; ingestion of meat from infected carcasses by carnivores can result infection and in South America, vampire bats are said to be of importance both as reservoirs of infections and as vectors [37].

However, there is no definitive study has ever been conducted to confirm their role in epidemiology of trypanosomosis and it is therefore not really clear how important they are. Mechanical transmission requires only that blood containing infectious trypanosomes be transferred from one animal to another animal [7, 35]. Although the mode of mechanical transmission is well

established, its dynamics is not understood. Therefore, considerable experimentation is still required to attempt to define quantitatively the effect of the host species, the duration of infection in the host and the level of parasitaemia, the period between feeds and the relative efficiency of different vector species insuring successful transmission [11].

Host: *Trypanosoma evansi* affects a wide range of host including horse, camel, dog, cattle, buffalo elephant, pig and also deer. Laboratory rodents, rabbit, rat and guinea pigs are readily affected. But depending on the virulence of the strain and the susceptibility of the individual host the disease may be acute in horses, camels and dogs, but in other domestic animals such as cattle, buffalo and pigs overt disease is uncommon and their main significance is as a reservoir host for infection of others [30]. Nevertheless, in these species occasional outbreaks of acute disease with sudden death may occur [31, 37].

Even though *T. evansi* is pathogenic to most domestic animals, camels are highly susceptible to infection. In Camels, the disease is running an acute or chronic course terminating fatally in untreated cases but, its effect on host species varies according to the virulence of the parasite, the susceptibility of the host and the local epidemiological conditions such as the presence of carrier animals and the vector. All age groups are susceptible, but immature, stressed and immune compromised animals as well as lactating animals are extremely vulnerable [7, 45].

Distribution: Originally, the distribution of *T. evansi* coincided with that of camel [31]. The diseases were further disseminated by camel caravans traveling to North Africa, the Middle East and further more East and South Asia. In similar manner, horses were probably the means by which Surra reached America, principally by movement of the animals from West Africa in the 16th century [11]. Now days camel trypanosomosis is reported from all areas where camels are kept [44]. *Trypanosoma evansi* does not require the presence of flies (*Glossina* species) to ensure its maintenance. This makes unique from other trypanosomes and they also accounts for its wide spread distribution in areas far removed from tsetse infestation area [47, 12]. The prevalence of infection among camel herd is related to the size of the vector population, of course in rainy season and around wet, swampy places including watering points, the prevalence rate is likely to be high [18, 48].

Table 1: Prevalence of surra caused by *Trypanosoma evansi* in some countries of the world

Author	County	Prevalence (%)	Trypanosoma species
[48, 5]	Ethiopia	21	*T. evansi*
[21]	Sudan	33	*T. evansi*
[15]	Kenya	28	*T. evansi*
[11]	Nigeria	27	*T. evansi*
[27]	Chad	30	*T. evansi*
[47]	Mauritania	24	*T. evansi*
[49]	Niger	29	*T. evansi*
[23]	India	22	*T. evansi*
[33]	Jordan	33	*T. evansi*
[29]	Iran	10	*T. evansi*

Source: [11]

Surra Distribution in Some African and World Counties: Surra is widespread in different parts of the world and poses a major constraint to camel productivity [21].

Camel Trypanosomes Distribution and Prevalence in Ethiopia: Among many disease that affect camels in Ethiopia, Trypanosomosis (Surra) is the most important. The prevalence of *T. evansi* in Ethiopia was 21% [5, 6]. The disease which is widely distributed throughout camel rearing areas of Ogaden, Borena and Afar regions and causes considerable economic effects due to loss of beast burden and food [35]. *Trypanosoma evansi* is said to be the only agent incriminated for camel trypanosomosis in Ethiopia [4, 50, 18].

Nevertheless, [51] reported mixed infection of *T. vivax* with *T. evansi* in eastern part of Ethiopia. [18] also reported infection of *T. vivax* in Liben district, Borena zone of Oromiya. However, *T. brucei, T. congolense* [44, 12] have been reported also as a causative agent. The disease is endemic in low lands areas of the country.

Stomoxys, Tabanus, Haematobia and *Hippobusca* were identified as possible vector in eastern Ethiopia [52]. The involvement of *Hypobusca* in the transmission of the disease was also reported by different researchers. In Ethiopia Surra is observed in more of its chronic form [4, 53, 15]. Little research was done on camel trypanosomoses in Ethiopia.

However according to so far available information and also different surveys conducted on camel trypanosomoses [54, 52, 53] the disease is found to be most important disease problem in camels in almost camel rearing areas of Ethiopia and results indicated prevalence range of 0.3 up to 31.9% with tendencies towards increasing some areas. Recent epidemiological studies on camel Trypanosomosis in Borena regions indicated a prevalence of 10 and 50 % using parasitological tests and serological tests respectively [35].

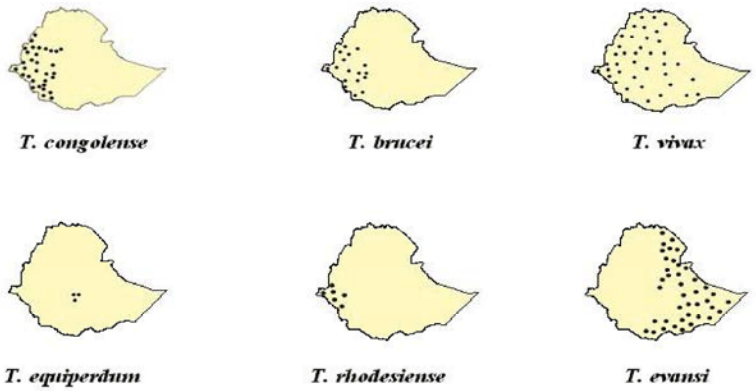

Fig. 2: Distribution of pathogenic trypanosomes in Ethiopia. Source: [35].

Table 2: Retrospective data on the prevalence of camel Trypanosomosis in Ethiopia

Study Areas	Trypanosoma species	Prevalence %	Source
Borena	*T. evansi*	10.9	[16]
Dire Dawa	*T. evansi*	12	[48]
Dire dawa , East Hararghe	*T. evansi*	7.7	[32]
Afar and Tigray	*T. evansi*	5	[53]
Leben, Borena	*T. evansi*	10.2	[18]
Southern range land of Ethiopia, Yabelo moyale	*T. evansi*	45.9	[6]

Source: [35]

Clinical Manifestations: *Trypanosoma evansi* can infect a variety of hosts and cause species specific pathology [55] confirmed that camel trypanosomosis is slow wasting disease. The animal become thin, weak and eventually dies. The first signs of the disease are a drop in production (milk and meat yield) and the tendency of pregnant females to abort. In the acute form trypanosomes are invariably present in the blood and the disease is almost always fatal [26]. In atypical case the dromedary losses weight, develops dropping hump, unable to walk long distances and may develops oedema of the feet, brisket, under belly and eyelids [25].

During the initial attack there may be lacrimation, shivering, reduce appetite and mild diarrhea, progressive anaemia and elevated body temperature up to 41°c which is directly associated with parasitaemia. Later, in the chronic stage of infection, the appetite is relatively unaffected and the temperature may become normal or slightly elevated. The mucus membranes are pale and the PCV drops [25, 24]. In the more chronic form which may last up to a year, there is a continuation of anaemia and progressive emaciation and weakness, often accompanied by the development of skin abscess [12, 31].

As reported by [42] also the PCV of diseased animal was decreased between 18-20% (normal 24-49% with an average of 30%). The herders may notice a characteristic odour of the camel's urine and may identify infected animals by this sign alone. Abortion in all stage of pregnancy is common. Reduced milk productions, cases of blindness and nervous signs like circling movements and trembling, unusual aggressiveness, running aimlessly and sudden collapse in severely stressed and over worked animal [56, 25].

Generally, infected animals show progressive anaemia, marked depression, dullness, loss of condition and often rapid death [24]. The clinical signs observed in the acute cases of the disease were hyperlacrimation, rough hair coat, weakness and depression. The camel owner also reports that sharp decline in milk production in lactating and abortion in pregnant animals. *Trypanosomes* were observed in the smear of the animals for acute of the disease. In its chronic form the disease was maintained by decreased milk production and long calving interval as stated by the camel owner [35].

Pathogenesis: During blood sucking the fly inoculates trypanosomes into the animal skin. After a few days trypanosomes invade the lymph and blood stream, where they multiply through binary fission. *Trypanosoma evansi* is not restricted to blood stream and may enter the joint fluids and other tissue compartments. It may cross the blood brain barrier. Because this, less accessible to treatment and to clinical diagnosis by demonstration of the parasite in the peripheral blood stream [25, 45].

Anaemia is a major component of pathology of Surra. Anemia in *T. evansi* infections of camel is reported to be macrocytic and hypochromic [57]. In early phases of infection the anemia is haemolytic and haemophagocytic. The mechanism responsible for increased erythrophagocytic activity is not fully understood.

Food and Agriculture Organization [50] proposed that immune complexes, expanded mononuclear phagocytic system, haemolytic factor produced by the trypanosome, fever and disseminated intravascular coagulation are some of the factors. In later stages, anemia continues to be major factor, with probably additional causes such as anoxic condition created by the persistent anaemia, signs of dysfunction which appear in the various organs [10, 48].

An increase in cardiac output due to increases in stroke volume and heart rate and decrease in circulation time are obvious manifestations. The central nerves system is reported to be most susceptible to anoxia with consequent development of cerebral anoxia. The marked depression observed in camel trypanosomosis is mental state and is a manifestation depression of cerebral cortical function [11].

Tissue damage also happens due to pathogenesis of the disease. The typical lesions of multiple necrotic foci found in the liver and spleen, as well as generalized lymphoid tissue hyperplasia in camels suffering from Surra on post mortem examination could be attributed to pathological events that occur in the tissues of animals infected with *T. evansi* [20, 4]. The degenerative changes thus observed could be due to tissue anoxia, which results in a fall in tissue P^H and vascular damage [16].

It is known that *T. evansi* is a member of *Brucei* group of trypanosomes, which have a known preference for connective tissue of a host, where they disrupt the collagen bundles and destroy the fibroblasts which produce and maintain the collagen. This disruption of host connective tissues, along with vascular damage attributable to *Brucei* group trypanosomes [58, 22] would be expected to release large quantities of cytoplasmic and mitochondrial enzymes into the serum, thereby causing further tissue damage [22].

Indeed, a two-stage process in the pathology of infection with *T. evansi* in camels based on studies of serum enzymes has been proposed [58]. The first steps coincide with the appearance of trypanosomes in the host blood stream in which there is rise in sorbitol dehydrogenase (SDH) activity [27, 25]. The second occur later in the infection and is characterized by a large increase in serum levels of glutamic oxaloacetic transaminase (GOT). Now, known as aspartate alanine transferase (AST) and smaller rise in glutamic).

The rise in AST level can be attributed partly to cellular damage caused by the trypanosomes lysis, while the increase in Alanine- amine Transferase probably results from host destruction of trypanosomes. AST is found mostly in cell organelles and rises when there is a great damage to the heart, kidney, skeletal muscles and liver. ALT is a specific liver enzyme found in the cell cytoplasm and its rise is associated with cell membrane damage [59].

The reported increases in these enzymes, especially AST is not surprising as it is indicative of organ damage and supports the post mortem reports of necrotic in the liver and spleen of camels suffering from Surra [11].

The fever characterized by high temperature might be due to the effects of toxic metabolites produced by died trypanosomes [60]. In addition, the oedema reported in the different parts of the body during the chronic stage could be due to a significant decrease in the albumin levels, resulting in alterations in osmotic pressure of the blood. This leads to excessive accumulation of fluid in tissues spaces caused by a disturbance in the mechanism of fluid interchange between capillaries, the tissues spaces and the lymphatic vessels [39].

The haemorrhage and serous exudates that occurred could be caused by haemolysis involving the expanded mononuclear phagocytic system. This has also been observed in *T. brucei* infected donkeys, while the frequent abortions reported may be attributed to endocrine dysfunctions [27].

Diagnosis: The diagnosis of camel trypanosomosis follows the classical diagnostic methods trypanosomoses involving clinical diagnosis, parasitological, serological as well as molecular techniques [60].

Clinical Diagnosis: The clinical diagnosis of Surra the disease caused by *T. evansi* in susceptible animal is based on pyrexia, progressive anaemia and loss of condition. Oedema, particularly of the lower parts of the body, urticarial plagues and petechial haemorrhages of serous membranes, conjunctivas are often observed. Lymph node hypertrophy, conjunctivitis or keratitis is also seen [24].

However, these symptoms are often not sufficiently pathognomonic signs of trypanosomosis which especially in their chronic forms resembles any other parasitic or infections condition, so the diagnosis has to be supported by laboratory methods [38, 62].

Parasitological Diagnosis: Parasitological diagnosis involves both direct and indirect methods.

Direct Methods: The usual field methods involve sampling from blood vessels and tissue since *T. evansi* inhabits the deep blood vessels and tissues. The direct methods include wet blood films, thick/ thin blood smears,

Lymph node biopsies, concentration methods such as buffy coat dark ground microscopy, haematocrit centrifugation techniques (HCT) as animal inoculation [63].

Wet blood films and thin blood smears are relatively insensitive techniques and in most instances reveal acute cases. Thin smears particularly permit morphological studies and the identification of the agent. Lymph node biopsies usually obtained from the prescapular and prefemoral lymph nodes may also assist in the diagnosis [62].

Concentration methods such as the haematocrit centrifugation and mini anion exchange techniques are used to detect low level parasitaemia in the case of mild, sub clinical or chronic infection. Laboratory animal inoculations are employed to reveal sub clinical parasitemia in domestic animals [64]. However, this method is not very useful for diagnosis due to the long prepatent period in commonly used laboratory animals and the expense involved. Nevertheless, it is said to be extremely sensitive in the diagnosis of camel trypanosomosis [65].

The Buffy coat dark ground phase contrast techniques found to be a more sensitive technique than thick, thin and wet smears and the haematocrit centrifugation technique. In addition, identification of the trypanosome species can be made on the basis of behavioral patterns observed in the buffy coat dark ground illuminated preparations [63]. However, the test has a poor sensitivity, often less than 50%. The implication of this is that in most situations *T. evansi* is under-diagnosed and the level of infection is greater than frequently reported [11].

Indirect Methods: Indirect methods involve haematological, biochemical test and serological test to demonstrate the effect of the parasite on its host rather than directly detecting parasite itself [12].

Serological techniques, e.g. Immuno Fluorescents Antibody Test (IFAT), Enzyme Linked Immuno Sorbent Assay (ELISA) and the Card Agglutination Test for Trypanosomosis (CATT), although sensitive, cannot distinguish current from cured infections [66, 67] the Complement Fixation Test (CFT) and the passive haemagglutination test [68].

Enzyme Linked Immuno Sorbent Assay test (ELISA) has two parts, the antibody detection and antigen detection part. For use in a field surveys in Indonesia the two Ag-ELISA and Ab-ELISA and the CATT were fully validated in terms of their diagnostic sensitivity and specificity [10].

Serological tests are used to detect specific humoral antibodies and circulating antigen [44]. Nevertheless, none of the above test could differentiate between past and present infections as antibodies persist for up to hundred days or more after chemotherapy has cleared trypanosomes from the circulation [67].

Recent tests, e.g. latex agglutination test (LAT) or Surratex based on trypanosome antigen detection in blood or serum, are more reliable and have shown a high correlation with patent or sub patent disease in camels. A comparative sensitivity test for *T. evansi* in camels in Kenya revealed 68.8% sensitivity for CATT and 58.8% for Surratex, although the two techniques were more sensitive than parasitological methods in revealing the true extent of trypanosomosis in camel herds as researched by Enwezor and Sackey [11].

Biochemical test, including the mercuric chloride test, thymus turbidity test and formal gel test depend on increase in serum immuno globulins following infection. These tests used were used in all diagnosis of *T. evansi* in camels but are not specific for trypanosomes [54]. Hematological examination specially the PCV determination is often a reliable indicator of trypanosome infection. However, camels with sub clinical disease can have parasitaemia without any evidence of anaemia [12].

Molecular Diagnosis: Diagnose using PCR could offer a very precise method for detecting infection and discriminating between infected and non-infected animals. The analytical sensitivity of such tests is high but in experimental situation they have not always high diagnostic sensitivity [10].

Under experimental conditions the technique provide consistently more sensitive than parasitological techniques in detecting parasite DNA in dried blood samples. However, the primers used for PCR do not amplify the DNA of all Duttonella stocks circulating in a particular region. But in some cases, enhanced parasitological tests could provide more effective diagnosis than PCR, example: in *T. evansi* infected water buffalo, PCR was found to have sensitivity, similar to that of mouse inoculation [10].

Immune Response: Pronounced immune response and changes occur in Africa trypanosomes. An increase in immuno globulin M (IgM) during both acute and chronic *T. evansi* infections in camels has been reported [69], but this is not protective, as the majority of the antibodies are auto antibodies.

Table 3: Therapeutic and prophylactic drugs used against *T. evansi* infection

Therapeutic drug	Aqueous solution	Route of injection	Dose	Volume of injection solution	Duration of protection
Quinapyramine	10% cold water	Sc	5mg/kg	5ml/100kg	-
Suramine sodium	10% cold water	Im/Iv	3-4 gm/kg	30-40 ml/kg	-
Prophylactic drug					
Quniapyramin prophylaxis	3.5/15 ml cold water	Sc	7.4 mg/kg	5ml/100kg	2 month
Suraminsodium Quinapyramine complex	5% cold water	Sc	40 mg/kg	4ml/5kg	3 month for calf 6 month for adult

Source: [38].

Leucocytosis, neutrophilia and eosinophilia have been reported in *T. evansi* infections of the mononuclear phogocytic system. The eosinophilia observed is a feature of parasitic infections and is associated with immediate type hypersensitivity reactions. The cells expected to accumulate in tissue in response to tissue injury. In the acute phase of the disease, lymph nodes and spleen are remarkably reactive, with plasma cells predominating. This may account for the generalized lymphoid tissue hyperplasia characteristic of *T. evansi* infections, while in the later stages the immune system becomes depleted of lymphoid cells [27, 48].

Circulating and tissue mediated immune complexes have been demonstrated in laboratory animals infected with *T. brucei* species and much of the antibody found in them is directed against the trypanosome (FAO, 1979). These immune complexes are likely to have wide varying pathological effects including anaemia, complement activation, tissue damage and interference with both induction and effecter mechanisms of the immune response [50, 53].

Hypocomplementaemia (decreased alteration of the complement system) has been reported, including elevated levels of immunoconglutinin and deposition of complement in the tissues. Further possible evidence of complement reactivity is found in the demonstration that the kinin system becomes activated with the release of pharmacologically active substances, thus causing micro vascular dilatation and increased permeability [18, 50]. Several hypotheses have been put forward to explain trypanosome induced immuno suppression and the most favoured to be the action of trypanosome enzymes, such as phospholipases neuraminidases and proteases have all been implicated in membrane fluidity and cellular damage [11].

Moreover, the phyospholipases generate free fatty acids (FFAs) and these have been reported to not only have a haemolytic effect but also to control lymphocyte reactivity through their role as prostaglandin precursors. The net effect of immuno suppression is lack of pre-immunity to other diseases [11].

Treatment and Control: Treatment against trypanosomes involves the use of the therapeutic drugs, injected parentally into the infected animal that aimed to limit losses due to this disease and reduce the risks of its propagation [22]. The treatment of choice for trypanosomosis of dromedaries due to *T. evansi* is Quinapyramine, but Suramin may become recommended in spite of its high cost [38]. As it is explained previously *T. evansi* is not restricted to blood stream in which they are less accessible to chemotherapy [25].

Control of camel trypanosomosis involves parasite control and vector control. Treatment with trypanocidal drug is the usual method of control of *T. evansi*. This trypanocidal drug have curative and prophylactic role [38] as indicated in Table 3 below.

A serious problem for control of the disease is drug resistance development among *T. evansi* this is due to repeated administration of low doses, absence of new trypanocide drug and generation of drug tolerance strains of trypanosomes [45, 38]. Even though Quinapyramine is a drug of choice for treatment of Surra recently melarsomine (cymelarsen) was introduced for the treatment of Surra in camels because of this drug resistance development against it. Resistance up to 500ng/ml has been found in camels against Quina pyramine sulphate. Cymelarsen is also effective against *T. evansi* infections in cattle and horses and animals with surra commonly treated different stages of the disease. However, relapses in camels after treatment have been reported [11].

Another drug trypan, which is a formulation containing diaminazene-di-aceturate, phenazone and procaine hydrochloride, is effective against *T. evansi* infection, as well as infections with *T. congolense*, *T. vivax* and *T. brucei* [70]. It has a synergistic and additive effect in comparison with other trypanocidal drugs and is reported to have painless, anti pyretic and long lasting effect. It has also been recommended as being the most effective trypanocidal drug to date [70, 48]. With regard to prevention, it has been confirmed that a single injection of 15 ml of Trypan affords an animal protection against a new reinfection over a period of 3 month [70].

Vaccination with killed or irradiated trypanosomes, but this method is unreliable due to the complex host immune reaction to antigenic variation of the parasite. Immunization has been attempted by inoculating animals with virulent trypanosomes strain, then treating them with drugs [38]. Control of the vector is very difficult. Drainage is possible in the breeding places which may destroy the vectors habitat. The flies have a habit of skimming over water and occasionally dipping their bodies into it; this led to the practice of pouring kerosene onto water which kills the flies when they deep into it. Animals should be kept away from places where the flies bound during the hot part of the day [5].

Residual sprays for the inside walls of animals houses including chlorinated hydrocarbon insecticides, organophosphates such as Malathion can be used as a residual spray on the animal [31]. If not feasible watering at night or at noon, Further more watering camels in small groups limits exposure time since they spent less time at the watering site [45].

Economic Impact of Camel Trypanosomosis: The camel is an important livestock species uniquely adapted to hot and arid environments. It produces milk, wool, hair and hides, serve for riding as beast of burden and draft animals for agriculture and long distance transport [45]. However, non-tsetse transmitted Surra is the most important disease of camel that can induce losses as a result of reduced productivity, mortality and cost of treatment [23], abortion in all ages of pregnancy period [25], drop in milk and meat yield [55], weight losses in camel rearing areas causing morbidity up to 30 % and mortality of around 3% [11].

There have been few studies on the effect of non-tsetse-transmitted trypanosomosis on productivity but the information available shows that infections with both *T. evansi* and *T.vivax* have serious economic implications for livestock and farmers in south America and south east Africa [10]. There are different hypotheses those explain as the parasites (trypanosome) induces immune suppression, in which Camels lacks pre immunity to other disease. Little wonder then that in most cases secondary infections, e.g. bacteria/ broncho pneumonia, often sets in and death may ensure in untreated cases [11].

CONCLUSION

Camel trypanosomosis (surra) is a disease of major economic importance in many countries of Africa, Asia and South America. The problem of the disease was reported from different areas of the world several years back and is devastating epidemics which is not frequently seen nowadays, but they do still occur. An infection rate about 30% is common in enzootic area. So, because of the wide geographic range of surra, its control has attracted international attention, vector control seems not the solution for surra as a range of non-related biting flies should be targeted, each with its own biology, while unlike tsetse flies most other flies are proliferate breeders and as such vector populations are difficult to control.

In most cases, control is limited to treating those animals that are considered to be infected to the basis of unreliable clinical signs. Diagnosis of the disease can be achieved by different methods such as clinical diagnosis, parasitological diagnosis, serological diagnosis and molecular diagnosis. Although camel trypanosomosis has been recognized as the most important single cause of economic losses in camel rearing areas, yet there have been no planned campaigns to control *T. evansi* using modern methods of fly control or chemotherapy. Taking into accounts the above conclusion, the following recommendations are forwarded:

- The role of wild animals in the epidemiology of the camel trypanosomosis should be determined.
- Factors those precipitate epidemic out breaks of the disease should be studied in detail.
- Further epidemiological studies should be conducted in areas where sura or Camel trypanosomosis is endemic in order to determine the impact of the disease on the productivity as well as its economic impact at large.
- An integrated control measures, using modern methods of fly control and management of this disease should be implemented to improve the Camel productivity.

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