American-Eurasian Journal of Scientific Research 12 (1): 13-20, 2017
ISSN 1818-6785

DOI: 10.5829/idosi.aejsr.2017.13.20

Camel Trypanosomiasis: A Review on Past and Recent Research in Africa and Middle East

[1]Kula Jilo, [1]Nejash Abdela, [2]Golo Dabasa and [3]Makida Elias

[1]Jimma University, School of Veterinary Medicine, Jimma, Ethiopia
[2]Yabello Regional Veterinary Laboratory, Yabello, Ethiopia
[3]Addis Ababa University, College of Veterinary Medicine and Agriculture, Bishoftu, Ethiopia

Abstract: Camel is comparatively less susceptible to many of the devastating diseases that affect other livestock species, such asrinderpest, contagious pleuropeumonia and foot and mouth disease but yet they are affected by many other diseases. The most important disease of camel is Trypanosomiasis, vector bornprotozoal disease caused by *Trypanosoma evansi* parasite with wide distribution throughout tropical and subtropical regions of the world. The aim of this review is to perform a comprehensive document on the prevalence of the camel trypanosomiasis mainly focusing on its current status in Africa and Middle East countries. The camel trypanosomiasis causes progressive anemia, depression, dullness, loss of condition and often rapid death. Serology is a preferable diagnostic technique while suramin, diminazeneaceturate, melarsomine and quinapyramine are drugs of choice for treatment. But due to drug resistance of the agent control of vectors transmitting the parasite is more important. Previously, *Trypanosoma evansi* was only the cause of Trypanosomiasis in animals but it is reported in human. Furthermore, *T. evansi* type B that so far only isolated from camel in Kenya is recently confirmed in Ethiopia. Beside, causing great economic losses *T. evansi* is recently emerging as zoonotic disease and unexpected new strains are being isolated from previously free areas. Therefore, routine epidemiological and biochemical studies should be performed to design and implement appropriate intervention measures.

Key words: Camel · Trypanosomiasis · Trypanosoma evansi · Surra

INTRODUCTION

Livestock contribute 40 % of the global value of agricultural output and support the livelihoods and food security of almost a 1.3 billion people. The livestock sector is one of the fastest growing parts of the agricultural economy. Livestock is the world's largest user of land resources, with grazing land and cropland dedicated to the production of feed representing almost 80 % of all agricultural land [1]. Ethiopia is known by possessing largest number of livestock population ranking 9th in the world and 1st in Africa, however; animal disease is one of the most important constraints to increase the productivity of food animals in sub-Sahara Africa [2].

The total population of camel in the world is believed to be 25.89 million, out of which 89% are one-humped dromedary camels (*Camelus dromedarius*) and the remaining 11% are the two-humped, *Camelus bactrianus* [1]. Camels are the most numerous species of animal in the arid areas of Asia and Africa, particularly in the arid lowlands of East African counties like Sudan, Ethiopia, Somalia, Kenya and Djibouti [3]. In the desert areas, camel is a vital animal to daily life of people as a source of food, means of transportation and most importantly its milk uses as medicine for diverse ailments [4]. It plays a significant multi-purpose role in the dry lands of Ethiopia such as transporting of grain, water, salt and other goods as well as for milk and meat production. Additionally, the camels are comparatively less susceptible to many of the devastating diseases that affect other livestock species, such asrinderpest, contagious pleuropneumonia and foot and mouth disease but yet they are affected many other diseases [5]. The most important and serious pathogenic disease of camel is Trypanosomiasis, protozoal disease caused by *Trypanosoma evansi* parasite which has a wide

Corresponding Author: Kula Jilo, School of Veterinary Medicine, College of Agriculture and Veterinary Medicine, Jimma University, P.O. Box: 307, Jimma, Ethiopia.

range of distribution throughout tropical and subtropical regions of the world [6]. This vector born disease is highly distributed arid areas of Northern Africa, East Africa, Asia and Arab emirates. Previously, case of camel *T. evansi* was not reported in human but recently, first laboratory-confirmed case of *T.evansi* in human is reported in Southeast Asia [7].

Camel Trypanosomiasis is most prevalent in Ethiopia (22%) and an important single cause of economic losses, causing morbidity up to 30.0% and mortality around 3.0% camels in Ethiopia [3]. From two strains of *T. evansi*: type A and B; only type A strain is believed to be found in Ethiopia while type B has been isolated only from Kenyan dromedary camels a decades ago [8, 9]. However, study reported the isolation, genetic and phenotypic characterization of type B *T. evansi* stocks from camels in Northern Ethiopia [8 9]. Beside great economic losses caused by camel trypanosomiasisitis recently emerging as zoonotic disease and unexpected new strains of *T. evansi* are spreading to previously free areas. Therefore, the objective of this review is to perform a comprehensive document on prevalence of camel trypanosomiasis mainly focusing on its current status in Africa and Middle East countries and to recommend further investigation on these areas.

Historical Background and Zoonotic Importance of *T. evansi* in Human: This Trypanosoma was discovered in India more than a hundred years ago by Evans (1880) [10], who detected it in horses, mules and camels with a disease locally called "Surra" [11]. Subsequently, numerous reports of trypanosomiasis in horses and camels were recorded in North Africa, the Americas and Eurasia [12]. Many different scientific names for the parasite were used, until it was found that all these non-tsetse transmitted [13]. Trypanosoma is a genus of unicellular parasitic flagellate protozoa while *Trypanosoma brucei* species and *Trypanosoma cruz*i are the major agents of human trypanosomiasis; other Trypanosoma species can cause human disease, but rare [14]. But in a recent study in March 2015, a 38-year-old woman presented to a healthcare facility in southern Vietnam with fever, headache and arthralgia. Microscopic examination of blood revealed infection with *Trypanosoma*. Furthermore, they declared first laboratory-confirmed case of T. evansi in a previously healthy individual without APOL1 deficiency, potentially contracted via a wound while butchering raw beef and successfully treated with suramin. A linked epidemiological investigation revealed widespread and previously unidentified burden of T. evansi in local cattle, high lighting the need for surveillance of this infection in animals and the possibility of further human cases [7].

Etiology: *Trypanosoma evansi* is a species belonging to the subgenus Trypanozoon and is the causative agent of camel trypanosomosis. It is long slender trypanosomes with a prominent undulating membrane and long free flagellum [3]. It is hypothesized that *T.evansi* originated 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 [15]. Camels that came into contact with tsetse flies acquired infections and when such camels moved to non-tsetse areas, transmission were spread by other hematophagous flies, other species of trypanosomes, e.g. *T. congolense*, *T. brucei* and *T. vivax* have also been isolated from camels in Sudan, but their role in camel trypanosomiasis is insignificant [16]. But, Camel trypanosomiasis is the most important single cause of morbidity and mortality in camels [5].

Phatogenesis: Anaemia is a major component of the pathology of surra and of African trypanosomiasis generally. Anemia in *T. evansi* infections of camels is reportedly macrocytic and hypochromic [15]. In the early phases of infection the anemia is hemolytic and haemophagocytic but the mechanism (s) responsible for this increased erythrophagocytic activity are not fully understood [8]. Several have been proposed, viz, immune complexes, expanded mononuclear phagocytic system per se, hemolytic factor produced by the trypanosome, fever and disseminated intravascular coagulation [15]. In the late stages, anemia continues to be a major factor, with probably additional causes. However, irrespective of the cause of anemia the primary abnormality of function are the anoxic conditions created by the persistent anemia [17]. Following this are the signs of dysfunction which appear in the various organs. An increase in cardiac output due to increases in stroke volume and heart rate and a decrease in circulation time are obvious manifestations. The central nervous system is reported to be most susceptible to anoxia with consequent development of cerebral anoxia [18]. The marked depression observed in camel trypanosomiasis is a mental state and is a manifestation of depression of cerebral cortical function in various degrees other nervous signs reported, such as circling movement, incoordination and dullness; appear to be the results of brain tissue disturbance or damage by the parasites. Evidence of *T. evansi* being found in the cerebrospinal fluid has been presented [16].

Clinical Sign: In camels, the disease is manifested by elevation of body temperature which is directly associated with parasitaemia. Infected animals show progressive anemia, marked depression, dullness, loss of condition and often rapid death [17]. In a typical case, the dromedary loses weight, develops a drooping hump, is unable to walk long distances and may or may not develop edema of the feet, brisket, underbelly and eyelids; the coat becomes rough. In the initial attack of fever there may be lacrimation, shivering, reduced appetite and mild diarrhea. The animal always shows progressive anemia and fluctuating body temperature with initial peaks of fever up to 41°C. Later, the appetite is relatively unimpaired and the temperature may become normal or slightly elevated. Clinically it is manifested by weakness, lethargy, tachycardia, fever, pale mucosa, subcutaneous edema in brisket and eyelids, nasal and ocular discharges, abortion in pregnant camel and weight loss [19].

Diagnosis: Trypanosomiasis is diagnosed by demonstrating the parasite and tentatively can be diagnosed by owner's observations and clinical examination of camels in the field [20]. The chronic form of trypanosomiasis is most common in camel and may present an association with secondary infections due to immunosuppressant caused by *T. evansi* infection which may complicates clinical diagnosis [6] The parasites can be detected in blood 13 to 16 days after an infective fly has had a meal to confirm infection. Parasitological diagnosis is mainly carried out by the direct microscopic examination of wet or stained blood films. However, the test has a poor sensitivity. One often less than 50% due to parasitaemia is intermittent. Post-mortem examination reveals no absolutely typical signs, but some degree of anemia is often visible. Skeleton and heart muscles are pale and there are signs of dehydration, pericardial effusion, enlarged lymph nodes and splenomegaly [15].

Life Cycle and Transmission: *Trypanosoma evansi* is transmitted mechanically by hematophagous biting flies [21]. No developmental stage in a vector has been demonstrated which differentiates the parasite from *T. brucei* but Tabanids (Horseflies) play the major role in transmission, while *Stomoxys* spp and *Lyperosia* spp. may also transmit it Nasir [22]. An interrupted feed upon an infected host leaves the fly hungry. Whenever it moves to another host, it can establish a new infection through its trypanosome-contaminated mouthparts. Trypanosomes remain infective on the proboscis for a short period only. The parasite replicates in camels, horses, donkeys, dogs, cattle, water buffaloes and even elephants. Equines and dogs are very susceptible and usually die after an acute course of the disease. Dogs may also become infected by eating meat from a trypanosome-infected carcass. Cattle, sheep, goats and antelopes often carry the parasite sub clinically, acting as asymptomatic reservoirs [23].

Trypanosoma evansi cannot undergo growth and differentiation in the insect vector because it lacks the genes necessary for mitochondrial development [24]. It is transmitted mechanically by the bites of hematophagous flies, such as *Tabanus* and *Stomoxys* [5]. Replication of the trypanosome occurs by longitudinal binary fission both in the host and in the vector with the flagellum and kinetoplast dividing together [25].

Epidemiolgy: According to Van Hennekeler [26] *T. evansi* has a wide host range. In some countries incidence of surra increases significantly furring the rainy season when biting fly populations have greatly increased. Surra affects mainly camels and horses but buffaloes and cattle are also affected. Other species that develop severe disease include donkeys, mules, deer, llamas, dogs, cats, cattle and buffalo. Sheep, goats, pigs and elephants may occasional develop mild or chronic disease. Camel rising in Africa and buffalo production in Asia is severely affected. *Trypanosoma evansi* is pathogenic in most domesticated animals and some wild animals [Domesticated animals: horses, mules, donkeys, cattle, buffalo, camels (Dromedary and Bactrian), llamas, pigs, sheep, goats, dogs and cats] [22]. It is a most important single cause of morbidity and mortality in camels. Additionally, wild animals: deer, capybara (reservoir host) and other species are susceptible. Furthermore new world camelids in South America are experimentally susceptible but natural disease has not been reported despite presence in cattle and horses. There are a reservoir host to camels and horses: cattle, buffalo, capybara and vampire bat. Moreover, Rats and mice are highly susceptible as experimental hosts for detecting subclinical (Nonparent) infections [15].

Distribution: Originally, the distribution of *T. evansi* was restricted to camel rearing areas but through time diseases were further disseminated by camel caravans traveling to North Africa, the Middle East and East and South Asia [3]. In similar manner, horses were probably the means by which Surra reached America, principally by movement of the animals from West Africa in the 16thcentury [15].

T. evansi is unique from other trypanosomes because it does not require the presence of flies (*Glossina* species) to ensure its maintenance as a result, it is widely

distributed in areas far removed from tsetse infestation [27]. The prevalence of *T. evansi* among camel herd is depends on the size of the vector population, rainy and wet season and swampy city [28].

The ability to be transmitted by blood-sucking insects other than *Glossina*, has enabled *T. evansi* to extend its range into African areas north of the Sahara desert, into Asia Minor, Pakistan, India, the USSR, China, Sumatra, Java, the Philippines, Mauritius, Madagascar and South and Central America. It was introduced by camels into Australia, North America and South-West Africa [6].

Treatment: Treatment of surra depends largely on four drugs: suramin, diminazeneaceturate (Berenil), melarsomine (Cymelarsan) and quinapyramine. Suramin and quinapyramine have been used for the treatment of *T. evansi* infection in camels and only recently melarsomine (Cymelarsen) was introduced for the treatment of surra in camels because of the problem of drug resistance. Most drugs are either not curative such as homidium bromide, or are too toxic for camels such as diminazeneaceturate [6].

Prevention and Control: According to Abera *et al.* [3] camel rising in Africa and buffalo production in Asia is severely affected by surra. As in tsetse-transmitted trypanosomiasis, losses are due to reduced productivity, mortality and cost of treatment. Control of surra can be difficult as there is no vector specificity and a wide range of hosts. Control of camel trypanosomiasis involves parasite control, vector control and retreatment with trypanocidal drug is the usual method of control of *T. evansi*. This trypanocidal drug have curative and prophylactic role.

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

Country	Prevalence (%)	References
Jordan	33	[29]
Sudan	33	[16]
Niger	29	[30]
Kenya	28	[31]
Nigeria	27	[15]
Mauritania	24	[27]
India	22	[32]
Ethiopia	21	[33]
Iran	10	[34]
Chad	30	[35]
Saudi Arabia	13.2	[36]
Pakistan	10	[37]
Morocco	43.3	[38]
Egypt	65.9	[39]

Sanitary Prophylaxis: Control measures are aimed at the host rather than vector, unlike Nagana control measures include detection and treatment of infected animals, prophylactic treatment of susceptible animals and protection of animals from biting flies and vampire bats [11].

Medical Prophylaxis: Drugs such as suramin, prothridium and isometamidium chloride (As a prophylactic) and diminazeneaceturate (Curative) can be used although drug resistance has been reported. For camels melarsomine (Cymelarsan) is very effective (Curative) against *T. evansi*. So far this drug is only registered for use in camels. No vaccines are available or likely in the near future because of the ability of trypanosomes to rapidly change their surface glycoproteins to avoid the immune response [6].

Current Status of Camel Trypanosomiasis in Ethiopia: Previously, *T. evansi* type A is the most abundant and found in Africa, Asia and Latin America while type B has been isolated only from Kenyan dromedary camels [15]. But a recent study was conducted by Birhanu *et al*. [9] and reported the isolation and the genetic and phenotypic characterization of *T. evansi* type B stocks from camel in Northern Ethiopia. According to that study, 30 cryopreserved buffy coat specimens from parasitologically positive dromedary camels were inoculated in immunosuppressed Swiss albino mice. In total, 22 parasite stocks originating from 22 different animals isolated and cryopreserved after 2 to 5 sub passages in mice. Then based on positivity in RoTat 1.2 PCR and EVAB PCR of the corresponding cryopreserved buffy coats, 20 of these stocks were *T. evansi* type A and 2 were *T. evansi* type B and they were labelled as MCAM/ET/2013/MU/01 to MCAM/ET/2013/MU/22.

In Ethiopia, the distribution of *T. evansi* strain A coincides with the distribution of camels in the semi-desert environment of the country. Surra in camel which caused by *T. evansi* is common in the southern, eastern regions of the country and rift valley areas (Table 2). This trypanosome also occurs in the dry country of the North West near the Sudan border and In Southern Ethiopia (Borena).

Cross sectional study conducted by Kassa *et al*. [5] in Fantale district (Dry season) to determine the prevalence of camel trypanosomiasis and assess the distribution and dynamics of the vectors responsible for transmission of the disease reported overall prevalence of 4.4%, calves (Less than 2 years of age) were negative and the high prevalence is recorded (7.7%) in young camels

Table 2: Prevalence of *T.evansi* in different areas of Ethiopia

Zones	Prevalence (%)	Diagnosis Technique	Reference
Fantale	4.7	MHCT	
	4.4	Blood smear	[5]
Tigray and Afar Jijiga	13.7	CATT	
	11.7	PCR	[9]
	3.9	Blood smear	[19]
Borena	2.33	Serology:	[40]
		✓ thinsmear	
Afar Region	5.5	HCT	[41]
	23.77	CATT	
Bale	12.12	Parasitology:	[42]
	24.88	✓ BCM	
		Serology:	
		✓ CATT	
East Haraghe	8.1	blood smear	[43]

MHCT= Microhaematocrit Test; CATT=Card Agglutination Test;PCR=Polymerase Chain Reaction;HCT= Haematocrit Test;BCM= Blood Mononuclear Cell

(Between 3-4 years of age). A tendency of infection rate to increase with age this is mainly due to the fact of larger scale movement, which increases the risk of infection, by the adult camels than the younger [19].

Seasonal outbreaks of *T. evansi* infections and the increase in number of *Tabanus* occur during the rainy season. In this study, it is suggested that very few *Tabanus* were collected, which may probably be attributed to the low prevalence of *T. evansi* in the study area during the study period. Additionally, according to these researchers the possible reason why calves were less infected than other age groups could be due to the fact that pastoralists keep them in the residence area and they do not go to distant areas where the fly burden is high [31].

Another cross-sectional study conducted by Tadesse *et al.* [19] to estimate the prevalence of camel trypanosomiasis (Surra) and identifying the species of trypanosome involved in Jijiga Zone reported overall prevalence of camel trypanosomiasis as 3.9% and identified *T. evansi* while higher prevalence of trypanosomiasis is reported in adult camels (4.5%) and no young camel (0 to 4 years) found positive. Study revealed that the prevalence of camel trypanosomiasis was higher (4.49%) in adult compared to young camels. This finding is in a general agreement with Dial *et al*. [27], Atarhouch *et al*. [44] and Gutierrez *et al.* [45] who reported that a tendency for infection rate to increase with age. This is mainly due to the fact of larger scale movement, which increases the risk of infection, by the adult camels than the younger.

Study made by Olani *et al*. [40] in Borena zone, southern Ethiopia to determine the prevalence of camel trypanosomiasis (Surra) and its associated risk factors reported the overall prevalence of camel trypanosomiasis in the area to be 2.33 % and *T. evansi* is identified with laboratory examination. Additionally, significant prevalence difference among the surveyed districts is revealed as the highest prevalence, 12.2 and 10.6 % was observed in Cheri-leche and Magado-sake pastoral associations, respectively. Furthermore, the prevalence of trypanosomiasis in female camels (2.51 %) is higher than that of male animals (1.77 %) but difference was not statistically significant. Moreover, this study reported that the camels residing in lowland areas, below 1000 m above sea level, exhibited a prevalence of 7.23 % which is higher as compared to those dwelling at elevated areas with altitudes of 1000–1499 and above 1500 m which showed prevalence rates of 1.9 and 2.14 %, respectively.

Another cross sectional study conducted on camel trypanosomiasis in Dello- Mena and Sawena districts of Bale Zone Oromyia Region, Ethiopia comparing two areas of different ecological characteristics (Wet and dry) reported the overall parasitological and serological prevalence of camel trypanosomiasis as 12.12% and 24.88%, respectively [42]. But recently, study in the same area reported 17.9% prevalence of *T. evansi* [3].

Conclusion and Recommendations: Camel trypanosomiasis is a disease of major economic importance in many countries in Africa, Asia and South America and now emerging as zoonotic as case is confirmed in human. A new strain of *T. evansi* type B is reported in Ethiopia. Accurate prevalence of *T. evansi* in Ethiopia is not yet known as disease reveals seasonal and geographical variations. In addition, difference in sensitivity to the different diagnosis techniques has a major effect on estimation of true prevalence. The chemo-therapeutic drugs in current use for the treatment of surra are toxic and problems of resistance are increasing.

So, in line with the above outlines the following recommendations are forwarded:

- More sophisticated diagnostic techniques should be used in prevalence estimation and strain identification.
- Care should be taken when managing clinically sick camel with trypanosomiasis since *T. evansi* is emerging as zoonotic disease.
- Biology of the parasite and interaction with host-pathogen should be studied for each specific geographical area.
- The dynamics of mechanical transmission of camel trypanosomiasis in endemic areas has to be thoroughly studied by including those factors contributing to occasional outbreaks.

REFERENCES

1. FAO, 2013. Statistical year book.Food and Agriculture Organization of the United Nations, Rome, Italy, 2013.
2. Yilma, M., 2016. Rose Bengal Plate Test (RBPT) Based Sero-prevalence of *Bovine Brucellosis* in and around Chench, Gama Goffa, Southern Ethiopia. Immunome Res., 12: 105.
3. Abera, Z., A. Usmane and Z. Ayana, 2015. Review on Camel *Trypanosomosis:* its Epidemiology and Economic Importance. Acta Parasitologica Globalis, 6(2): 117-128.
4. Kula, J. and D. Tegegne, 2016. Chemical Composition and Medicinal Values of Camel Milk. A review. International Journal of Research Studies in Biosciences (IJRSB), 4(4): 13-25. ISSN 2349-0357 (Print) & ISSN 2349-0365 (Online) www.arcjournals. org.
5. Kassa, T., E. Tadesse and C. Hassen, 2011. "Prevalence of camel trypanosomosis and its vectors in Fentale district, South East Shoa Zone, Ethiopia." Vet. Arhiv, 81: 611-621. Kenya. J. Camel Pract. Res., 55: 67-71.
6. Eyob, E. and L. Matios, 2013. Review on camel trypanosomosis (surra) due to *Trypanosoma evansi*: Epidemiology and host response. J. Veterinary Medicine and Animal Health, 5(12): 334-343.
7. Chau, N.V.V., M. Desquesnes, S. Herder, N.P.H. Lan, J.I. Campbell, N. Van Cuong and N.T. Tue, 2016. A clinical and epidemiological investigation of the first reported human infection with the zoonotic parasite *Trypanosoma evansi* in Southeast Asia. Clinical Infectious Diseases, ciw052.
8. Bornstein, S. and M. Younan, 2013. Significant veterinary research on the dromedary camels in Kenya: Past and present. Journal of Camelid Science, 6: 1-48.
9. Birhanu, H., R. Fikru, M. Said, W. Kidane, T. Gebrehiwot, A. Hagos and P. Büscher, 2015. Epidemiology of *Trypanosoma evansi* and *Trypanosoma vivax* in domestic animals from selected districts of Tigray and Afar regions, Northern Ethiopia. Parasit Vectors, 8: 212.
10. Evans, G., 1880. Report on “surra” disease in the Dera Ismail Khan district. Punjab Government Military Department, 493: 446.
11. Elhaig, M.M., A.I. Youssef and A.K. El-Gayar, 2013. Molecular and parasitological detection of *Trypanosoma evansi* in Camels in Ismailia, Egypt. Veterinary Parasitology, 198(1): 214-218.
12. Prasad, K.J., 2010. Emerging and re-emerging parasitic diseases. J. Int. Med. Sci. Acad., 23: 45-50.
13. Gibson, W., 2007. Resolution of the species problem in African trypanosomes. International Journal for Parasitology, 37(8): 829-838.
14. Kirchhoff, L.V., 2000. American Trypanosomiasis. Principles and Practise of Clinical Parasitology, pp: 335-353.
15. Enwezor, Felicia Nneka Chizoba and Anthony Kojo Bedu Sackey, 2005. "Camel *trypanosomosis*-a review." Veterinarskiarhiv, 75(5): 439-452.
16. Elamin, E.A., M.O.A. EL-Bashir and E.M.A. Baheed, 1999. Prevalence and infection pattern of *T. evansi* in camels in mid-eastern Sudan. Tropical. Animal Health and Production., 30: 107-114.
17. Kingsley Onyekachi, M.O.H., 2014. Antitrypanosomal effects of *brassica oleracea* (Cabbage) methanolic and aqueous extracts on *rattusnorvegicus* (Albino rats) infected with *trypanosome evansi.* Diss. Department of biological sciences, faculty of science, ahmadu Bello University, Zaria.
18. Brun, R., J. Blum, F. Chappuis and C. Burri, 2010. Human African trypanosomiasis. The Lancet, 375(9709): 148-159.
19. Tadesse, A., A. Omar, K. Aragaw, B. Mekbib and D. Sheferaw, 2012. A study on camel *trypanosomosis* in Jijiga zone, Eastern Ethiopia. Journal Veterinary Advance, 2(5): 216-219.
20. Adam, S., 2015. Incidence of camel Trypansomosis in camel herds restricted to Nyala rural suburbs, South Darfur state-Sudan (Doctoral dissertation, UOFK).

21. Desquesnes, M., P. Holzmuller, D.H. Lai, A. Dargantes, Z.R. Lun and S. Jittaplapong, 2013. *Trypanosoma evansi* and surra: a review and perspectives on origin, history, distribution, taxonomy, morphology, hosts and pathogenic effects. Bio Medical research international, 2013.
22. Nasir, K.A., 2015. Prevalence and Risk factors of camel trypanosomosis in Butana area Gedarrif State (Doctoral dissertation, Sudan University of Science and Technology).
23. Röttcher, D., D. Schillinger and E. Zweygarth, 1987. Trypanosomiasis in the camel (*Camelus dromedarius)*. Rev. Sci. Tech. Off. Int. Epiz., 6(2): 463-470.
24. Schnaufer, A., J. Domingo and K. Stuart, 2002. Natural and induced dyskinetoplastictrypanosomatids: how to live without mitochondrial DNA. International Journal for Parasitology, 32(9): 1071-1084.
25. Liu, B. and Y. Liu, 2005. Fellowship of the rings: the replication of kinetoplast D N A. Tr. Parasitol., 21(8): 363-9.
26. Van Hennekeler, K., 2007. Aspects of the ecology of tabanid flies (Family *Tabinidae*) in North Queensland and their potential to transmit *Trypanosoma evansi (*Doctoral dissertation, James Cook University).
27. Dia, M.L., Meirvenne, N. Van, E. Magnus, A.G. Luckins, C. Diop, A. Thiam, A. Jacqiet and D. Harmers, 1997. Evaluation of four diagnosis test, blood smears CATT. IFAT and ELISA- Ag in study of the epidemiology of *T.evansi* camel trypanosomosis in Mauritania, Revue and Elev. Med. Vet. Pays. Trop., 50: 29-36.
28. Mohammed, R., 1999. Camel Trypanosomosis: Prevalence and Drug Sensitivity Test in Dire Dawa Administrative Council Eastern Ethiopia. DVM Thesis, FVM, AAU, Debre Zeit Ethiopia.
29. Al-Rawashdeh, O.F., L.A. Sharif, K.M. Al-Qudah and F.K. Al-Ani., 2000. *Trypanosoma evansi* infection in camels in Jordan. Revue. Elev. Med. Vet. Pays Trop., 20: 233-237.
30. Pacholek, X., D. Gamatic, S.G. Franek and R. Tibayrene, 2001. Prevalence of *Trypanosoma evansi* trypanosomosis in young camels in west Niger. Revue. Elev. Med. Vet. Pays. Trop., 44: 177-182.
31. Njiru, Z.K., B. Brett, I.M. Ole-Mapeny, J.B. Githiori and J.M. Ndung'u, 2002. Trypanosomosis and helminthosis in camels: comparison of ranch and traditional camel management systems in Kenya. Journal of Camel Practice and Research, 9(1): 67-71.
32. Radostits, D.M., D. Blood and C.C. Gry, 1994. Veterinary medicine a text book of the disease of cattle, sheep, goats, pips and equines, 8th ed. ELBS and Bailleir Tindall., pp: 1218-1219.
33. Zeleke, M. and T. Bekele, 2001. Camel herd health and productivity in eastern Ethiopia selected seminomadic households. Revue. Elev. Med. Vet. Pays. Trop., 55: 213-217.
34. Zarif-Fard, M.R. and R. Hashemi-Fesharki, 2001. Study on tissue and blood protozoa of camels in southern Iran. J. Camel Practice Res., 35: 193-194.
35. Delafosse, Arnaud and Abdesalam Adoum Doutoum, 2004. "Prevalence of *Trypanosoma evansi* infection and associated risk factors in camels in eastern Chad." Veterinary Parasitology, 119(2): 155-164.
36. Hussain, H.S., N.A. Al-Asgah, M.S. AI-Khalifa and F.M. Diab, 1991. The blood parasites of indigenous livestock in Saudi Arabia. Arab-Gulf Journal of Scientific Research, 9: 143-160.
37. Shah, S.R., M.S. Phulan, M.A. Memon, R. Rind and W.M. Bhatti, 2004. Trypanosomes infection in camels. Pakistan Veterinary Journal, 24: 209-210.
38. Rami, M., T. Atarhouch, M.N. Bendahman, R. Azlaf, R. Kechna and A. Dakkak, 2003. Camels trypanosomosis in Morocco. A pilot disease control trial. Veterinary Parasitology, 115: 223-231.
39. El-Naga, Tarek R. Abouand Safaa M. Barghash, 2016. "Blood Parasites in Camels (Camelus dromedarius) in Northern West Coast of Egypt." Journal of Bacteriology & Parasitology, 2016.
40. Olani, A., Y. Habtamu, T. Wegayehu and M. Anberber, 2015. Prevalence of camel trypanosomosis (surra) and associated risk factors in Borena zone, southern Ethiopia. Parasitology Research, pp: 1-7.
41. Aregawi, W.G., S.T. Kassa, K.D. Tarekegn, W.T. Brehanu, S.T. Haile and F.Z. Kiflewahid, 2015. Parasitological and serological study of camel trypanosomosis (Surra) and associated risk factors in Gabi Rasu Zone, Afar, Ethiopia. Journal of Veterinary Medicine and Animal Health, 7(6): 234-240.
42. Hagos, G., 2009. Parasitological and serological survey on trypa-nosomis (Surra) in camels in dry and wet areas of Bale Zone, Oromyia Region, Ethiopia. Revue Méd. Vét., 160(12): 569-573.
43. Mohammed, T., M. Woyessa, T. Brihanu and A. Hiko, 2015. Camel Trypanosomosis in Babile District, Eastern Hararghe Zone, Oromia Regional State, Eastern Ethiopia.

44. Atarhouch, T., M. Rami, M.N. Bendahman and A. Dakkak, 2003. "Camel trypanosomosis in Morocco 1: results of a first epidemiological survey." Veterinary Parasitology, 111(4): 277-286.

45. Gutierrez, C., M.C. Juste, J.A. Corbera, E. Magnus, D. Verloo and J.A. Montoya, 2000. Camel trypanosomosis in the Canary Islands: assessment of seroprevalence and infection rates using the card agglutination test (CATT/*T. evansi*) and parasite detection tests. Veterinary Parasitology, 90(1): 155-159.