

Description for five different Forms of *Trypanosoma evansi* infecting blood of *Camelus dromedarius*.

Barakat Shehata Abd-Elmaleck*, Gama Hassan Abed

Department of Science, University of Assiut, Assiut, Egypt

Abstract

Only five specimens (2.5%) from 195 *Camelus dromedarius* were infected with *Trypanosoma evansi*. Light microscopy was showed different developmental stages.

At the same time five different forms were distinguished beside that, Scanning electron microscopy supported these results by revealed different stages such as binary fission.

Also appears of different stages of trypomastigote, epimastigote and metacycling stage.

Keywords: *Camelus dromedaries*, Epimastigote, Scanning, Different forms, Metacycling.

Accepted on 06 August, 2021

Introduction

Hemoparasitic diseases like trypanosomiasis and dipetalonemiasis have adverse impact on health, productivity and working capacity of camel. Local studies on hemoparasites in camel with particular relation to hema to chemical and biochemical dimensions are limited. *T. evansi* is notorious in developing resistance to drugs and infection popularly called “surra” is probably the most serious protozoan disease of camel and is widespread throughout camel rearing areas of the world. This disease adversely affects the growth, reproduction as well as trekking potential of the camel. Trypanosomiasis in camel is usually chronic but can be acute with 90% mortality, if not treated. Chronically infected animals may survive for 3-4 years. The disease in this form is characterized by anemia, emaciation, recurrent fever, atrophy of the thigh muscles, edema of the dependent parts, corneal opacity and diarrhea.

Other clinical changes recorded in experimentally induced chronic infections include debility alopecia, keratinization and depletion of subcutaneous fat and facial edema. Most of the work on the affection until now is done on the incidence, diagnosis and treatment of the affection in different animals. There are only few reports on the pathology of the disease. Very little work have been done on biochemical and hematological alterations induced by natural trypanosomosis in racing camels [1].

Tsetse flies (Diptera: Glossinidae) are only cyclical vectors of many trypanosome species in Africa. Human sleeping sickness is endemic to 36 countries in Sub-Saharan Africa with 70 million of the 400 million inhabitants at risk. The continent is recovering from an epidemic that there currently 50-70000 people infected per year. Sleeping sickness is fatal if left untreated. The related disease (nagana) in domesticated animals causes estimated losses to Africa agriculture, and had profound effect of the development of the continent. The evidence suggests that all 31 species of Tsetse flies can transmit trypanosomes to greater or lesser extent. Most economically important African trypanosomes are transmitted during the bite of tsetse fly. Humans are only infected *T. brucei rhodesiense* and *T. b. gambiense*. The closely related parasite *T. brucei* is not

pathogenic for humans, although it infects all livestock; as a consequence, this species is used most frequently in laboratory studies [2].

Other major pathogens of domesticated animals that are transmitted by tsetse are *T. congolens*, *T. vivax* and *T. simiae*, while *T. evansi*, which is most closely related to the brucei group, is transmitted by biting flies. In recent *T. evansi* in mainland Spain, several forms of the parasite were commonly observed in blood smear examinations, some of them resembling *Trypanosoma vivax*. The molecular techniques confirmed that *Trypanosoma vivax* was the causal agent, discarding other African trypanosomes.

Materials and Methods

Out of 195 blood samples of camels (*Camelus dromedarius*) examined for blood protozoan parasites collected from different localities of Slaughter houses at Assiut city, Egypt (Dairout, Beni ady, Elethamna). These freshly collected blood samples were divided in two groups one in a tube coated with EDTA, and the other in a test tube for Centrifugation to obtain sera. Thick and thin blood smears were made for morphological examination of *Trypanosoma evansi*. Electron microscopic studies.

TEM

Few drops from blood which is highly infected with *Trypanosoma evansi* immediately fixed in 3 ml. of 3% glutaraldehyde solution in phosphate buffer (PH 7.2), for 24 hours and Kept at 4°C in refrigerator. The samples were post fixed in 1% Osmium tetra-oxide in phosphate buffer (PH 7.2, 300 mom), for 30 minutes.

They were washed several times with phosphate buffer solution. The samples were then embedded in Epon which can preserve in structure from distortion during processing then ultra-thin sections were cut by an Ultra microtome and examined by JEOL, 100 CXII operating at 80 KV (TEM) [3].

SEM

For scanning electron microscope of blood; few drops were fixed in 3% glutaraldehyde in buffer for 24 hours. Specimens were washed three times in Phosphate buffer and post fixed in 1% Osmium tetra-oxide for 2 hours and then washed in the same buffer. They were Dehydrated in different grades of ethyl alcohol and then mounted on special holders and coated with gold. Then they were examined in a JSM-T 200 L.V. 5400 Scanning Electron Microscopy (SEM).

Results

Microscopy of the examined trypanosomes from dromedary camel in Assiut were morphologically consistent with *T. evansi* where they were slender with either tapered or blunt ends, Only five specimens (2.5%) from 195 *Camelus dromedarius* were infected with *Trypanosoma evansi* [4].

Light microscopy was showed different developmental stages (Figure 1).

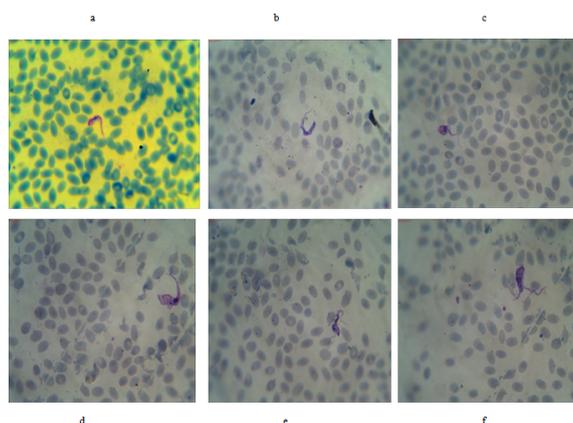


Figure 1. (a) Photomicrograph showing *Trypanosoma evansi* with an axostyle shaped flagellum in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (b) Photomicrograph showing *Trypanosoma evansi* with granulated cytoplasm in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (c) Photomicrograph showing *Trypanosoma evansi* with kinetoplast similar to the nucleus in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (d) Photomicrograph showing *Trypanosoma evansi* with clear undulating membrane in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (e) Photomicrograph showing symmetric binary fission of *Trypanosoma evansi* in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (f) Photomicrograph showing asymmetric binary fission of *Trypanosoma evansi* in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m.

At the same time five different forms were distinguished as in figure (2).

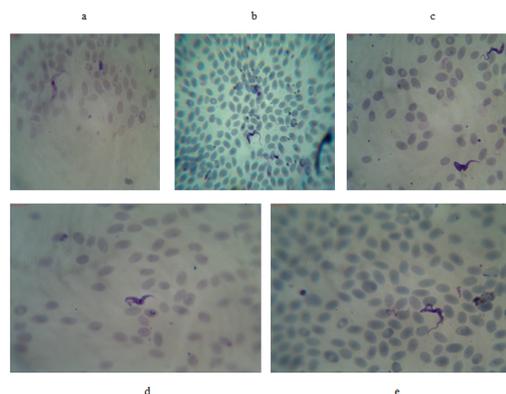


Figure 2. (a): Photomicrograph of *Trypanosoma evansi* in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (b) Photomicrograph of *Trypanosoma evansi* in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (c) Photomicrograph of *Trypanosoma evansi* in the blood of *Camelus dromedarius* stained with Geimsa \times bar=20 μ m. (d) Photomicrograph showing of *Trypanosoma evansi* in the blood of *Camelus dromedarius* stained with Geimsa stain \times bar=20 μ m. (e) Photomicrograph of *Trypanosoma evansi* in the blood of *Camelus dromedarius* stained with Geimsa stain \times bar=20 μ m.

They were slender with either tapered or blunt ends, with long free flagellum and well-developed undulating membrane as in figure 2(a), with two small folded and sharp posterior end as on figure 2(b), or no folds and truncated posterior end as in figure 2(c), or with pulp body shape and Vacuolated like structure with granules as in figure 2(d) and with well-developed undulating membrane with two to four folds and Normal posterior end as in figure 2(e) [5].

This indicated that the high prevalence infection in the blood in the same infected specimen.

Scanning electron microscopy supported these results by revealed different stages such as binary fission, elongated long trypomastigote, short epimastigote and metacyclic form (Figure 3-6) [6].

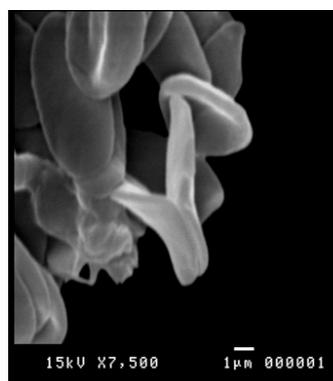


Figure 3. Scanning electron micrograph showing binary fission of *Trypanosoma evansi* in the blood of *Camelus dromedarius* \times bar=1 μ m.

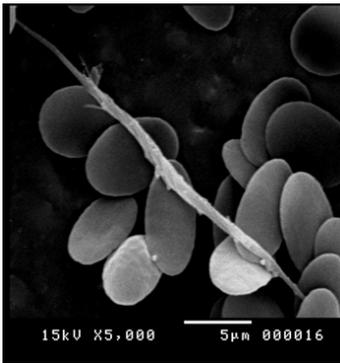


Figure 4. Scanning electron micrograph showing elongated long trypomastigote of *Trypanosoma evansi* in the blood of *Camelus dromedaries* × bar=5 µm.

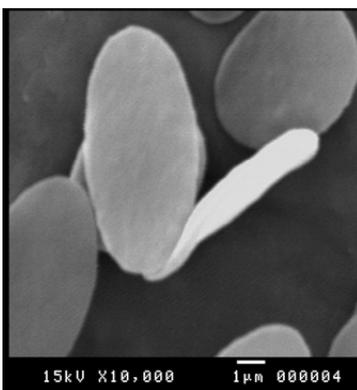


Figure 5. Scanning electron micrograph showing short epimastigote of *Trypanosoma evansi* in the blood of *Camelus dromedaries* × bar=1 µm.

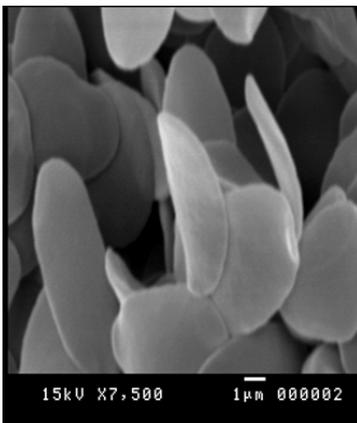


Figure 6. Scanning electron micrograph showing metacyclic form of *Trypanosoma evansi* in the blood of *Camelus dromedaries* × bar=1 µm.

Discussion

The incidence of the infection with *Tr. evansi* in the present study was found (2.5%). It was a lower as compared to who reported that, the infection rate (13.7%). The lower incidence may due to the high awareness of *Tr. evansi* effects and treatment means in slaughter houses [7].

Trypanosomes of the section *Salivaria* might or might not possess a free flagellum, their kinetoplast is terminal or subterminal in position and the posterior end of the parasite is usually rounded, but in the present study the posterior end vary between rounded to truncated or pointed. The polymorphism of the subgenus *Trypanozoon* has been indicated in some studies, which agree with these present study but some authors considered that, *T. evansi* has been monomorphic and exclusively in the slender intermediate form.

Conclusion

Several studies have observed significant morphological differences in some *T. evansi* isolate; some have even reported a relationship between the parasite's measurable morphology and the host's physical condition. Hoare was described all isolates of *T. evansi* ranged 15-34 µm in length. The mean morphological parameters (Length, Posterior end to nucleus, Nucleus to anterior end, Flagellum width and nuclear index) were compatible with the data obtained for *T. evansi* by other authors. However, the presence of several morphological types observed in the present study could suggest presence of different trypanosome species. However, the total length of *T. brucei* varies from 14 to 39 µm, width mean values between 27 and 29 µm. The longest mean length values obtained in some types which in the range of *T. brucei*. Also there is some information on the morphological differences of *T. evansi* from different hosts in the same area, as well as differences observed in the same animal hosts but located in different geographical areas

References

1. An introduction to the camel in health and disease. Food and Agriculture Organization of the United Nation. 1992.
2. Ibadullaev FI, Khalikov FR. Pathological changes caused by experimental trypanosomiasis in mice, rats and guinea pigs. Trudy Uzbekskogo Nauchno issledovatel'skog Vet Inst. 1972; 20:70-72.
3. Boid R, Jones TW, Luckins AG. 3. Protozoal diseases of camels. Br Vet J. 1985;141:87-105.
4. Löhr KF, Pholpark S, Siriwan P, et al. Trypanosoma evansi infection in buffaloes in North-East Thailand. II. Abortions. Trop Anim Health Prod. 1986;18:103-8.
5. Pathology of experimental surra in camels [India]. Food and Agriculture Organization of the United Nation. 1980.
6. Dias JC, Coura JR. Clínica e terapêutica da doença de Chagas: uma abordagem prática para o clínico geral. SciELO-Editora Fiocruz. 1997.
7. Elamin EA, El Bashir MO, Saeed EM. Prevalence and infection pattern of Trypanosoma evansi in camels in mid-eastern Sudan. Trop Anim Health Prod. 1998;30:107-14.

*Correspondence to

Barakat Shehata Abd-Elmaleck
Department of Science

Citation: Barakat Shehata Abd-Elmaleck, Gama Hassan Abed. Description for five different Forms of Trypanosoma evansi infecting blood of Camelus dromedarius. *J Bacteriol Infect Dis* 2021;9(5):1-2.

University of Assiut
Assiut, Egypt

Tel no: 0201113532752
E-mail: barakatshehata@yahoo.com