INFECTIVITY OF TRYPANOSOMA EVANSI IN KANO BROWN GOATS INOCULATED WITH POOLED CAMEL BLOOD FROM KANO ABATTOIR

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Abstract
This study was conducted to isolate and determine the infectivity of T. evansi in Kano Brown goats (KBG) with a view to observe the clinical signs base on the previous report on the existence of variations in the infectivity and pathogenicity of the parasite. Six KBG, aged between 2 – 3 years were experimentally infected intravenously (IV) with 2.0 x 10^6, Trypánosoma evansi, Kano isolate. Clinical signs, temperature changes in responses to the experimental infection were determined in the goats. All the infected goats developed parasitaemia 7 days post infection (PI). There was a slight rise in the mean rectal temperature (38.90 ± 0.15) between days 2 - 3, in some of the infected goats especially those in group 1. It was discovered that the parasites disappeared from the blood of the goats in all the groups, 3 days PI. There was resurgence of parasitemia on day 6 PI in all the infected goats. It was concluded that, the parasites can be establish in KBG, as such periodic checking of T. evansi in small ruminants (sheeps and goats) particularly in areas where camels are being raised or slaughtered should be carried out.

Key words: Infectivity, *Trypanosoma evansi*, Kano Brown goats, Pooled camel blood, Abattoir

Introduction

Trypanosoma evansi is responsible for a disease called "Surra" and is the most widespread pathogenic trypanosome globally (Luckins, 1988). It is the most important factors limiting livestock production in areas of humid and sub-humid Africa. The parasite has wide range of hosts and is pathogenic to many species of domestic and wild animals (Franke *et al.*, 1994; Herrera *et al.*, 2002). It is primarily transmitted mechanically by biting flies like tabanus, stomoxys, haematopota, lyperosia and chrysops (Luckins, 1999a). In Africa camel is the most important host (Dia *et al.*, 1997). But cattle have also been reported as the next most highly susceptible animal (Mahmoud and Gray, 1980). Pathology of the trypanosomiasis in different domestic animals has been well documented (Raisingham *et al*, 1980). However, most of the reports were on *T. vivax, T. brucei* and *T. congolense*. In goat infected with *T. evansi*, lesions observed were hyperplasia of the lymphatic tissue, muscular atrophy, and necrotic foci in the liver, kidneys, spleen and lungs with bronchopneumonia (Ngeranwa *et al.*, 1993).

Since *T. evansi* is primarily a parasite of camel, there is like likelihood that animals herded together with camel, especially small ruminants may become infected with the parasites (Ngeranwa *et al.*, 1993).In Nigeria, extensive work has been done on animal trypanosomiasis (Anosa 1977; Saror, 1975;Sackey, 1998), but little has been done on infection due to *T. evansi* especially in goats. Hence, the present study was carried to investigate the infectivity in Kano Brown goats following infection with *T. evansi* Kano isolate.

Materials and Methods

Study Area

The study was conducted at the Teaching and Research Farm of the Department of Animal Science, faculty of Agriculture and Agricultural Technology, Kano University of Science and Technology Wudil. The farm lies between longitude 8°58' East and latitude 11° 35' North at a altitude of 403m above sea level. The average annual rainfall ranges from 850 – 870mm, minimum and maximum temperature is 26°C and 33°C, respectively (Olofin *et al.*, 2008).

Source of Trpanosoma evansi

The parasites were isolated from naturally infected camels at Kano abattoir. Twenty (20) blood samples were collected from slaughtered camels using sample bottle that contained ethylene diamine tetracetic acid (EDTA) and then kept in cold conditions. The blood samples were pooled together and then 2ml of pooled blood were inoculated into three white albino rats intraperitoneally to multiply and preserve the parasites.

Experimental Goats

Six Kano brown goats, aged between 2 – 3 years were used for the study. The goats were treated against internal and external parasites with albendazole (10mg/kg body weight) and diazintol (25ml/50L), respectively. Goats were screened for haemoparasites using thin blood smear and buffy coat methods and vaccinated against pestes des

petit ruminants (PPR) at a dose of 0.5ml/animal, subcutaneously. They were also given antibiotic prophylaxis using long active oxytetracycline (Tridox L.A., Farvet Bladel Holland) at a dose of 20mg/kg body weight. The animals were kept in an individual arthropod – proof pen and fed with grasses and concentrate diet. Water and mineral lick were given *ad-libitum*.

Post-Infection Monitoring

All the experimental goats were monitored daily through the observation of physical appearances, gait, posture, discharges from eyes and nose, cutaneous edema, size of superficial lymph nodes and appetite.The following parameters of all the goats were monitored. Daily rectal temperature was taken using clinical digital thermometer, parasitaemia was determined every day using wet mount and buffy coat examination as described by Murray *et al.* (1983).

Data Analysis

Data generated were analyzed using simple descriptive statistics, such as graph presentation and histogram using spread sheet.

Results and Discussion

The result of the post infection (PI) temperature (Fig: 1) revealed a slight increased (38.90±0.15) in the mean rectal temperature between 2- 4 days PI in some of the infected goats especially, those in group 1. Those in groups 2 and 3, maintained rectal temperature that is within the normal range (37.38±0.15).This conquered with the report of Ogbaje *et al.* (2011) who reported that a slight rise (39.03±0.23) in the mean rectal temperature between days 3-6 PI in West African Dwarf (WAD) goats. Most of the goats maintained rectal temperature that was within normal range (37.50±0.12 to 38.00±0.15). Also Murray *et al.* (1998) reported that parasitaemia may remain high for 4 to 6 days after which it declines with remission of the temperature. This slight variations and maintenance of rectal temperature within the normal range may be attributed to the nature of the parasite (tissue parasite). The reasons for this fluctuation of body temperature could be as a result of the parasites being killed by antibodies in the circulatory system, but some may escape the defense mechanism of the body and hide in the spleen, bone marrow and lymph nodes and multiply again, there by changing their body surface and re-appear in to the blood stream thereby causing the pyrexia for the second time, which lead to the fluctuation of the body temperature. All the infected goats developed parasitaemia 7 days PI (Figs: 2, 3, 4 and 5). The parasitaemia disappeared from the blood especially from animals in groups one and two 3 days PI. This was at variance with the report of Ogbaje *et al.* (2011) who reported that parasitemia developed in WAD 3 days post infection and the parasites disappeared from peripheral circulation of all infected goats 7 days PI. There was resurgence on day 6 PI in all of the infected goats. The disappearance and resurgence of parasitaemia at different intervals may be as a result of dynamic of antigenic variation activity of the parasites as earlier reported (Jones and Mckinell, 1984).

From the result of thin blood smear (Figs: 2 and 3), the parasites were scanty when compared with Buffy coat smear (Figs: 4 and 5).This may be attributed to the fact that, the parasites were concentrated in buffy coat as a result of centrifugation. This agreed with the finding of Reid *et al.* (2001) who reported that buffy coat or haemotocrit centrifugation technique are necessary as they increase the sensitivity of microscopic examination. However, the pre-patent period was shorter than what was reported by Sharma *et al.* (2000) and Shehu *et al.* (2006).

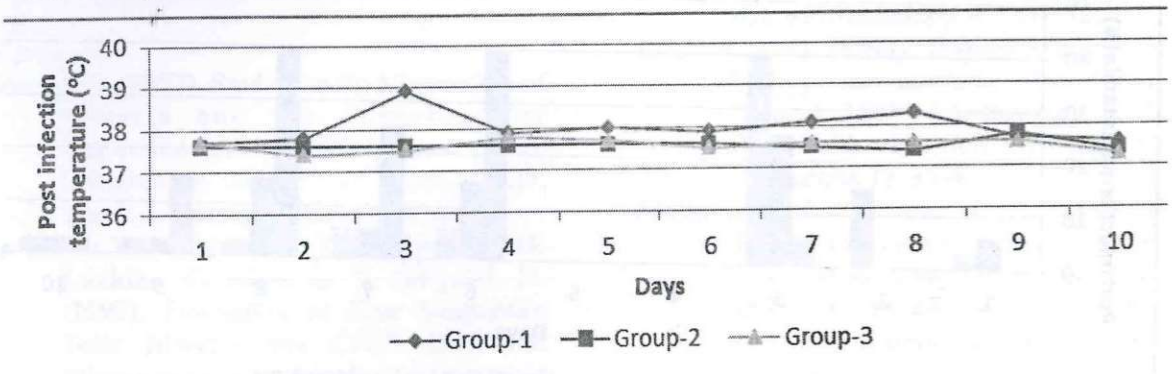


Fig. 1: post-infection d aily temperature (°C) of Kano Brown goat experimentally infected with 2.0X10 ^{6} *T. evansi* (Kano isolate).

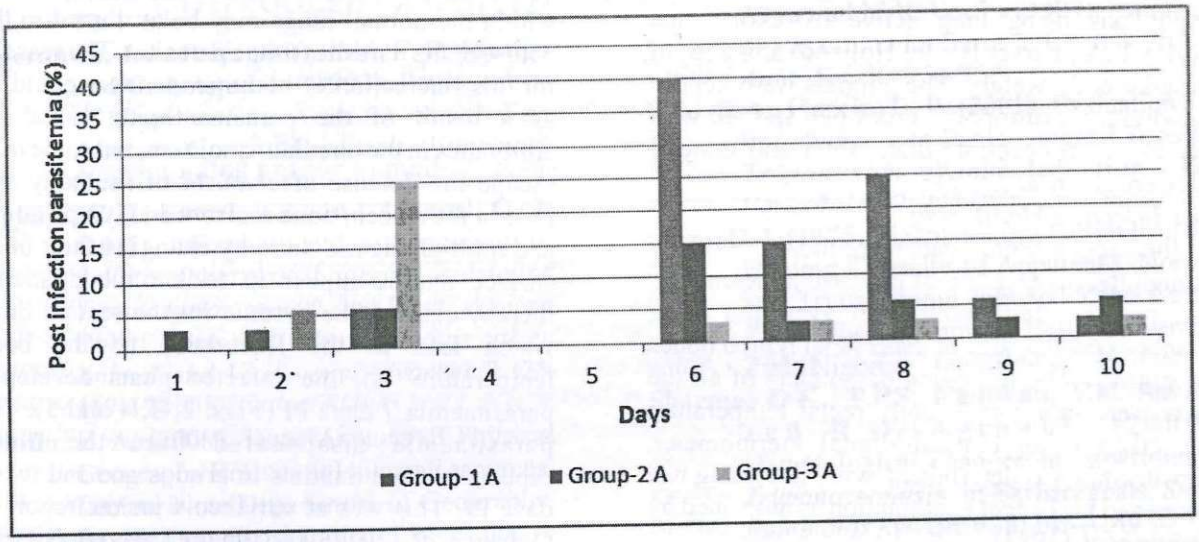


Fig. 2: Post-infection daily parasitaemia (thin blood smear) of Kano Brown goat infected with 2.0X10 ^{6} *T. evansi* (Kano isolate).

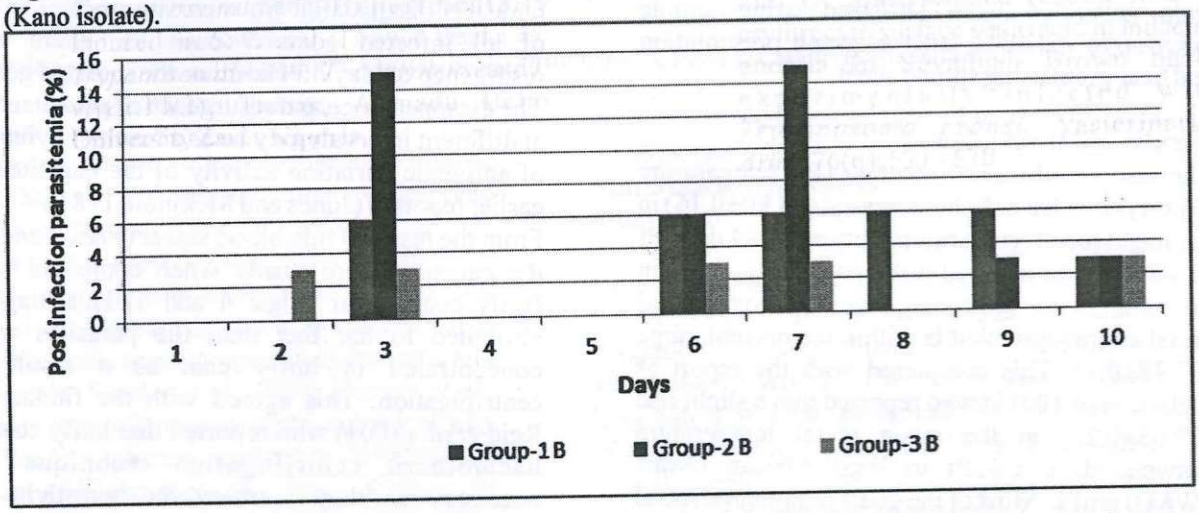


Fig. 3: Post infection daily parasitaemia (thin blood smear) of Kano Brown goat infected with 2.0X10 ^{6} *T. evansi* (Kano isolate)

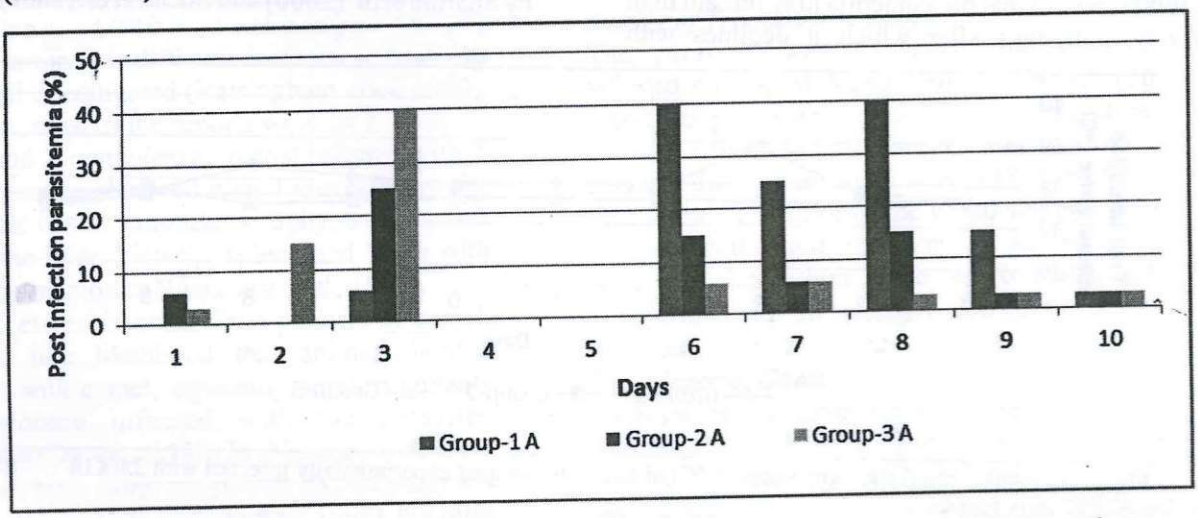


Fig. 4: Post infection daily parasitaemia (buffy coat) of Kano Brown goat infected with 2.0X10 ^{6} *T. evansi* (Kano isolate)

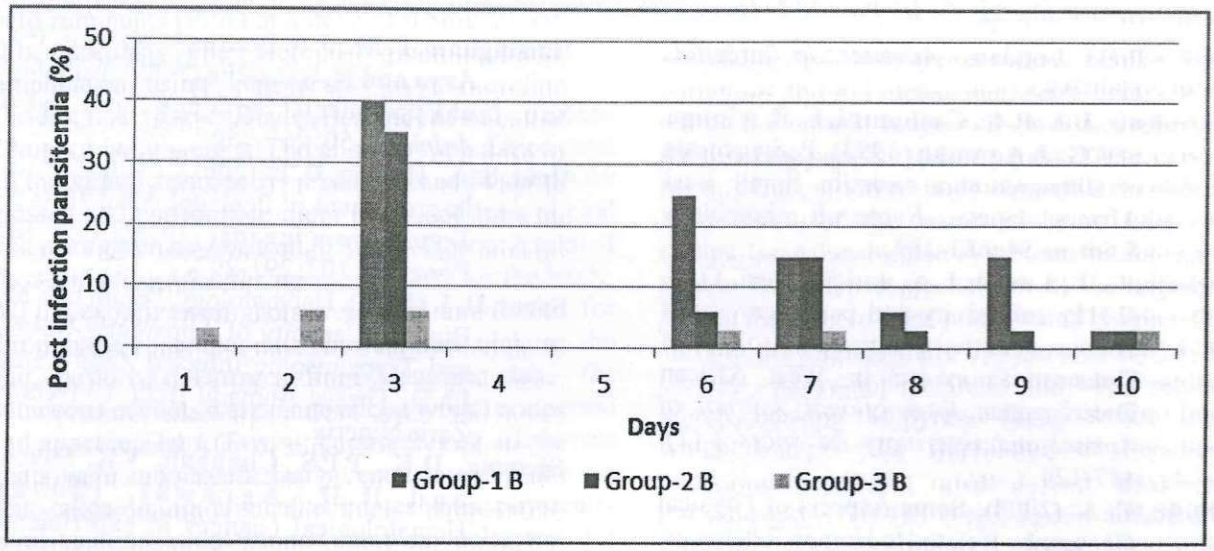


Fig. 5: Post infection d aily parasitaemia (buffy coat) of Kano Brown goat infected with 2.0X10 ^{6} *T. evansi* (Kano isolate)

Conclusion

It was concluded from the result of this study that infection of KBG with *Typonosoma evansi* (Kano Isolate) ɩ. ɔduce mild clinical signs. All infected goats developed parasitaemia within a short period time, indicating that the animals were highly susceptible to the parasites. Therefore, animals herded together with Camels may become infected.

Recommendation

It was recommended that, periodic checking of *T. evansi* in small ruminants (sheep and goats) particularly in areas where camels are being raised or slaughtered should be carried out, anti-trypanosomal drugs should be routinely administrated to small ruminants so as to guard against the menance of the disease (Surra) particularly in Kano metropolis, since camels are being consumed on daily basis.

References

Anosa, V. O. (1977). Studies on the Mechanism of Anemia and the Pathology of Experimental *Trypanosoma vivax* Infection in Sheep and Goats. Ph.D. Thesis, University of Ibadan, Nigeria.

Dia, M. L. Van Meirvenne Emmagnus, A. G. Luckins, C. Diop, A. T. Jacquiet, D. (1997). Evaluation of Four Diagnosits Tests: Blood Smear, CATT, IFAT and Elisaag in a Study of the Epidemiology of *Trypanosomaevansi*. Camel *Trypanosomiasis* in Mauritania Rev. HN. *Med. Vet. Pays. Trop.* 50: 29-36.

Franke, C. R., M. Greiner and D. Mehlitz (1994). Investigations on Naturally Occurring *Trypanosoma evansi* Infections in Horse, Cattle, Dogs and Capybara *(Hydrochaeris* hydrochaeris*)*in Pantanl De Pocone (Mato Grosso; Brazil). *Act. Tropical* 58: 159-169.

Herrera, H. M., L. P. C. T. Aquino and R. F. Menezes (2002). *Trypanosomaevansi* Experimental Infection in the South American Coati *(Nasunanasua);* Hematological, Biochemical and Histopathological Changes. *Act. Trop.* 81: 203-210.

Jones, T.W. and C.D. Mckinell, (1984). Antigenic Variation in *Trypanosoma evansi;* Isolation and characterization of variable antigen type population from rabbits infected with a stock of *T. evansi. Tropenmedizin Parasitol.* 35: 337–241.

Luckins, A. G. (1988). *Trypanosoma evansi. Asian Parasitology* 4: 137-142.

Luckins, A. G. (1999). *Trypanosomiasis* Caused by *Trypanosomaevansi* (Steel, 1885). Balbani 1888. (A Review of Recent Research). *Tropical Animal Health Production* 12: 35-47.

Mahmoud, M. M. and A. R. Gray (1980). Trypanosomiasis due to *Trpanosomaevansi* (Steel, 1885). Balbani 1888. A review of recent research. *Tropical Animal Health Production* 12:35-47.

Murray, M., W.I. Morrison and D.D. Whitelaw (1983). Host Susceptibility to African Trypanosomiasis: Trypanotolerance. In: Baker, J. and R. Muller, (Eds.). Academic

Press, London. *Advances in Parasitol.* 12: 1-68.

Ngeranwa, J. J., P. K. Gathumbi, E. R. Mutiga and G. J. Agumka (1993). Pathogenesis of *Trypanosoma evansi*in Small East African Goats. *Research Veterinary Sciences* 54: 283-289.

Ogbaje, C. I., Lawal, I. A. and Ajanusi, O. J. (2011). Infectivity and pathogenivity of Sokoto (Northern Nigeria) isolate of *Typanosoma evansi* in West African Dwarf goats. *International Journal of Animal and Veterinary Advances* 3 (3): 117-124.

Olofin, E. A. (2008). Some Aspects of Physical Geography Related to Human Resources. Lecture Note Department of Geography, Bayero University, Kano.

Sackey, A. K., (1998). Comparative Study of *Trypanosomiasis* Experimentally Induced in Savannah Brown Bucks by *Trypanosomabrucei, T. congolense*and*T. vivax*.Ph.D. Thesis, Ahmadu Bello University Zaria, Nigeria.

Raisingham, P. M., J. S. Bhatia, V. K. Vyas, P. L. Arya and K. R. Lodha (1980). Pathology of Experimental Surra in Camels. *Ind. J. Anim. Sci.* 50: 966-969.

Reid, S. A., Husein, D. B. (2001). Evaluation and Improvement of Parasitological Tests for *Trypanosomaevansi* Infection. *Vet. Parasitol.* 102: 291-297.

Saror, D. I. (1975). Haematology, Serum Iron and Binding Capacity of Apparently Normal and Trypanosome Infected Zebu Cattle. Ph.D. Thesis, Ahmadu Bello University, Zaria Nigeria.

Sharma, D.K., P.P.S. Chauhan, V.K. Saxena and R.D. Agrawal, (2000). Hematological changes in experimental *Trypanosomiasis* in Barbari goats. *Small Ruminants Research* 38: 145 – 149

Shehu, S.A., N.D.G. Ibrahim, K.A.N. Esievo and G. Mohammaed (2006). Role of erythrocyte surface sialic acid in inducing anemia in Savannah Brown bucks experimentally infected with *Trypanosoma evansi.* Vasterinarki. *Arhiv.*76(6): 521 - 530