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THE EFFECT OF MELARSENOXYDE CYSTEAMINE HYDROCHLORIDE (CYMELARSAN®) ON HAEMATOLOGICAL CHANGES IN ONE HUMPED CAMELS (*Camelus dromedarius*) EXPERIMENTALLY INFECTED WITH *Trypanosoma evansi*

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ABSTRACT

A study was conducted to determine the effect of melarsenoxyde cysteamine hydrochloride (Cymelarsan®) on the haematological changes in one humped camels (*Camelus dromedarius*) experimentally infected with a field strain (CT/29) of *Trypanosoma evansi*. Twenty camels of both sexes aged between 1 to 3 years and weighing between 160 and 400 kg were randomly dived into 6 groups (A – D) of five each. group A was infected with 0.5ml of blood containing x103/µl of *T. evansi* and treated with Cymelarsan® by day 20 post-infection (p.i.) at the peak of parasitaemia (210.2 ± 1.81). Group B and C were infected and uninfected controls, respectively while group D was uninfected but treated with Cymelarsan®. From day 4 (p.i.) packed cell volume (PCV), red blood cell (RBC), haemoglobin concentration (Hb), white blood cell (WBC), absolute lymphocytes, neutrophils and platelets counts decreased significantly ($p<0.05$) in an inverse relationship with parasitaemia. On the other hand, monocytosis was consistent while reticulocytosis occurred by days 16 and 36 (p.i.). The Mean corpuscular volume (MCV) decreased significantly ($p<0.05$) from day 24 (pi.). The erythrocyte sedimentation rate (ESR), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) increased significantly ($p<0.05$) from day 4 (p.i.). These changes were however modulated to their pre-infection values in group A, following treatment with Cymelarsan®.

Key words: Camels, cymelarsan®, haematological changes, *Trypanosoma evansi*

Camel trypanosomosis (surra) is commonly caused by the haemoflagellate (*Trypanosoma evansi*) (Mbaya *et al,* 2010). The disease is a major threat to camel production (Njiru *et al,* 2002; Enwezor and Sackey, 2005). Serious outbreaks of the disease have been reported among cattle and horses in the Pantanal, Brazil (Silva *et al,* 1995) and among captive Asian tigers (*Panthera tigris*) in Nandankanan Zoo (Parija and Bhattacharya, 2005). The first human infection of *T. evansi* has been reported in India (Joshi *et al,* 2006). Surra in camels occur in acute or chronic forms and cause significant morbidity and mortality (Aradib and Majid, 2006). It also causes immunosuppressive effects (Njiru *et al,* 2004), infertility and retarded growth (Gutierrez *et al,* 2000). The disease lasts between 7-42 days and often results in emaciation and death (Cadioli *et al,* 2006).

So far, little has been done on the complete haematology of *T. evansi* infection in camels. Few authors (Herrera *et al,* 2002; Aradib and Majid, 2006; Cadioli *et al,* 2006) have, however, worked on some of the parameters in naturally infected camels. It is against this backdrop that camels were experimentally infected with a field strain (CT/29) of *T. evansi* to monitor the effect of melarsenoxyde cysteamine hydrochloride (Cymelarsan®) a trivalent arsenical in reversing the various haematological changes that occurred in the course of the infection.

Materials and Methods

Experimental Animals

Twenty apparently healthy one humped camels (*C. dromedarius*) of both sexes aged between 1 to 3 years and weighing between 160 and 400 kg were purchased from the Cattle Market in Maiduguri. These were screened for blood, intestinal and external arthropod parasites according to standard criteria (Soulsby, 1982). These were routinely treated against ecto, endo and haemoparasites and kept for 60 days acclimatisation period before the commencement of

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the experiment. They were housed on concrete floor and fly-proof houses and fed on groundnut husks, wheat bran, leaves, chopped cucumbers, water melons, concentrates and water was provided *ad-libitum*. The experiment was approved by the Ethics Committee of the Faculty of Veterinary Medicine. All handling and experimental procedures were in accordance with the International guidelines for the use of animals for biomedical research (Broom and Johnson, 1993).

Source of trypanosomes

The *T. evansi* (CT/29) field strain used for the study was isolated by the authors from a natural outbreak of "surra" in camels at the Department of Veterinary Medicine, University of Maiduguri, Nigeria. It was identified on the basis of morphology and negative blood inhibition and infectivity test (BIIT), stabilised in Wister albino rats and sent to the Nigeria Institute for Trypanosomiasis and Onchocerciasis Research (NITOR), Vom, Nigeria where it was authenticated as *T. evansi* strain (CT/29) by DNA sequencing using polymerase chain reaction (PCR). Approximately $1x10^3$ trypanosomes per ml was injected into 6 donor Wister albino rats in order to multiply the trypanosomes which were monitored for development of parasitaemia until approximately $5x10^3$ trypanosomes per ml of blood was achieved. The infected blood was diluted serially with phosphate buffered glucose saline (PBSG, Ph 7.2) until $x10^3$ trypanosomes per 0.5ml was obtained. Each camel was inoculated intravenously via the lateral abdominal vein with a uniform dose of the inoculums (0.5 ml) of infected blood

Experimental design

The camels were randomly separated into four groups (A-D) of five each. They were weighed using a Tru-Test® multi purpose digital livestock scale (Algen Scale Corporation Bohemia, New York) model XHD with extra heavy load bars and an aluminium alloy weight platform placed inside a crush. Group A was infected but treated via the intramuscular route with a single dose of melarsenoxyde Cymelarsan® at a dose rate of 0.25mg/kg as recommended by the manufacturers (Rhone Merieux Lyon France) by day 20 (p.i.) at the peak of parasitaemia. Group B was infected untreated control while group C served as uninfected control. Group D was uninfected but treated with Cymelarsan® by day 20 (p.i.) (Table 1).

Detection of parasitaemia

Parasitaemia were detected every 4 days by the wet mount and haematocrit buffy coat microscopy (Soulsby, 1982) for 36 days (p.i.) after which, they were monitored for a period of two months for the possibility of a relapse parasitaemia. The degree of parasitaemia was estimated by the rapid matching technique (Herbert and Lumsden, 1976).

Haematological analysis

Blood samples were collected via the lateral abdominal vein into vacutainers containing anticoagulant (ethylenediaminetetracetate) every 4 days for a period of 36 days. Packed cell volume (PCV), red blood cells (RBC), haemoglobin concentration (Hb), erythrocyte sedimentation rate (ESR), reticulocytes, platelets, total white blood cells (WBC) and differential leucocytic counts (DLC) were estimated as described by Shalm *et al* (1995). The red cell indices; mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC) were calculated using standard formulae (Coles, 1986).

Table 1. Experimental protocol and mortality pattern of one humped camels (*C. dromedarius*) experimentally infected with *T. evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls.

Groups/Treatment regimes	Dose of inoculums ($x10^3/\mu L$)	Pre-patent period (Days)	Route/Duration of treatment	Day of treatment (p.i.)	Mortality rate (%)
Group A: Infected but Treated with Cymelarsan ® (n = 5)	0.5ml	4	Deep i/m/ Single dose	20	0(0)a
Group B: Infected Control (n = 5)	0.5ml	4	Nil	Nil	5(100)b
Group C: Uninfected Control (n = 5)	Nil	Nil	Nil	Nil	0(0)a
Group D: Uninfected but Treated with Cymelarsan (n = 5)	Nil	Nil	Deep i/m/ Single dose	20	0(0)a

Numbers with different superscripts in 6th column differed significantly ($p<0.05$)

Keys: p.i. = Post-infection n = No of camels per group i/m = Intramuscular route

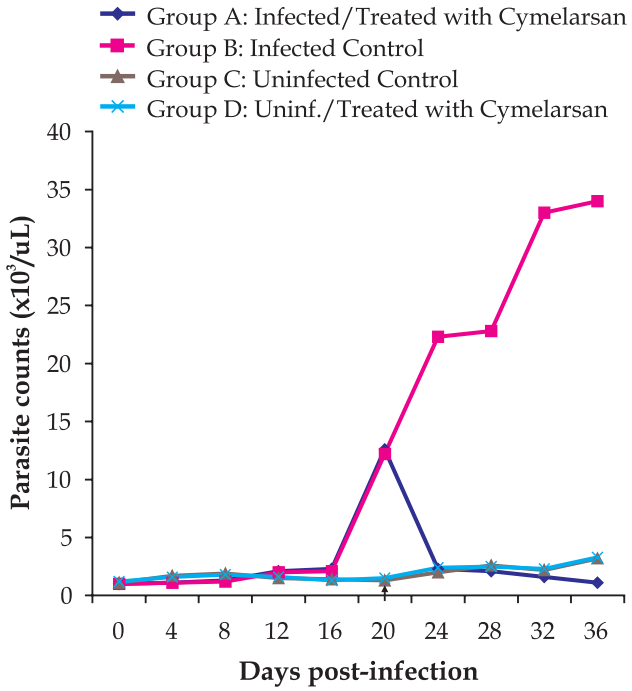

Fig 1. Mean parasite counts (x10^3/µL) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

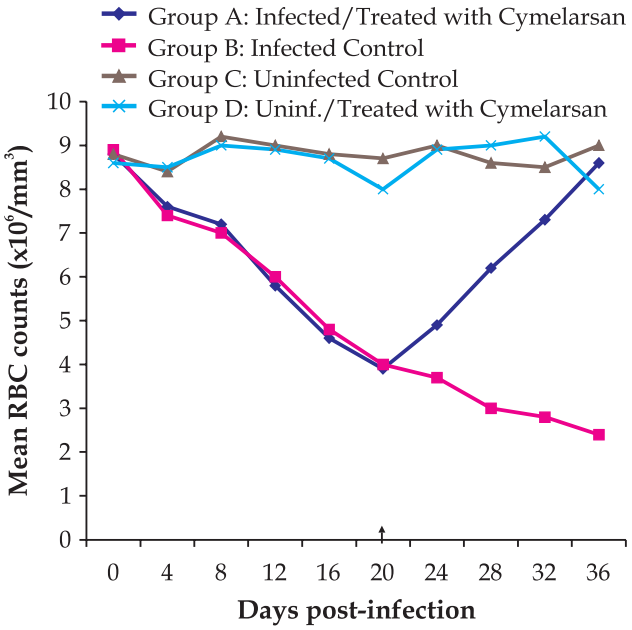

Fig 3. Mean red blood cell counts (x10^3/mm^3) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

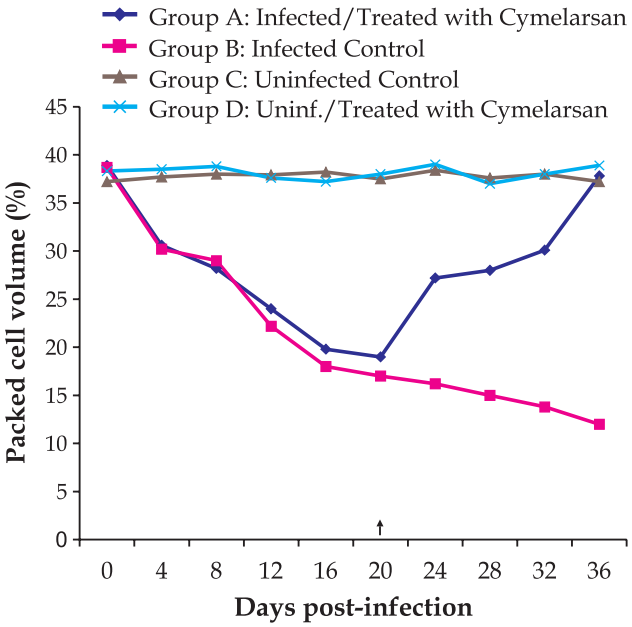

Fig 2. Mean packed cell volume (%) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

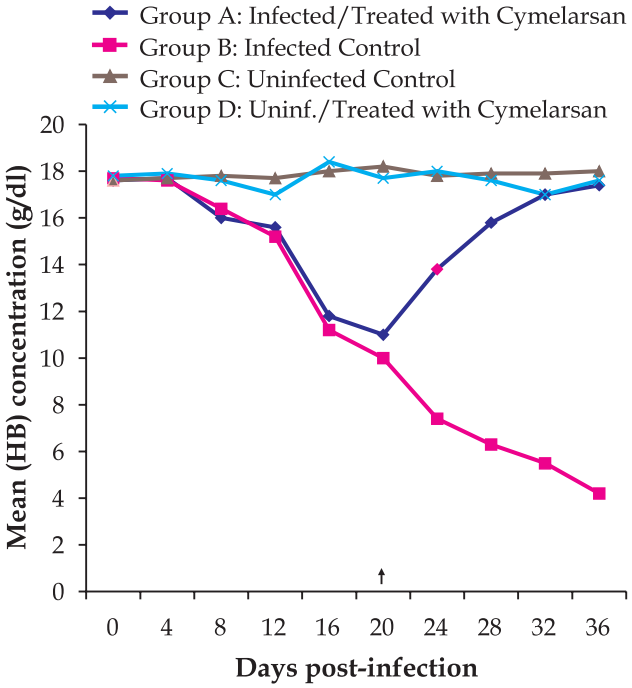

Fig 4. Mean haemoglobin concentration (g/dl) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

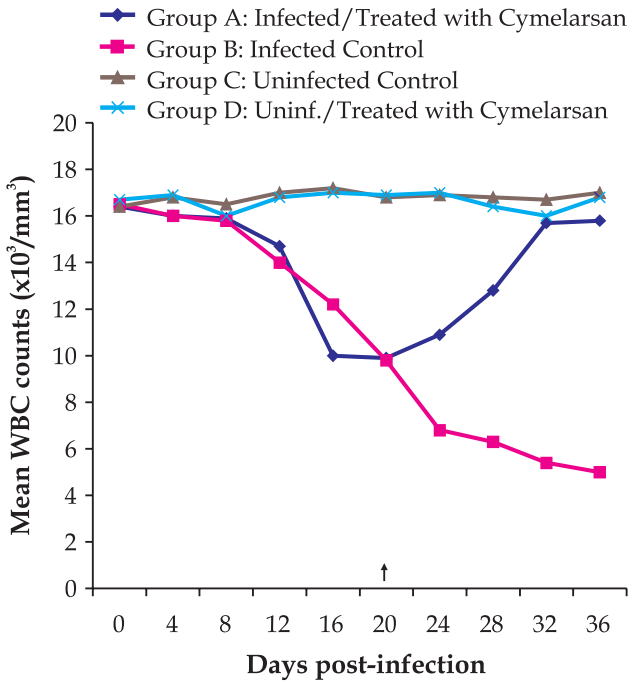

Fig 5. Mean white blood cell counts (x10^3/mm^3) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

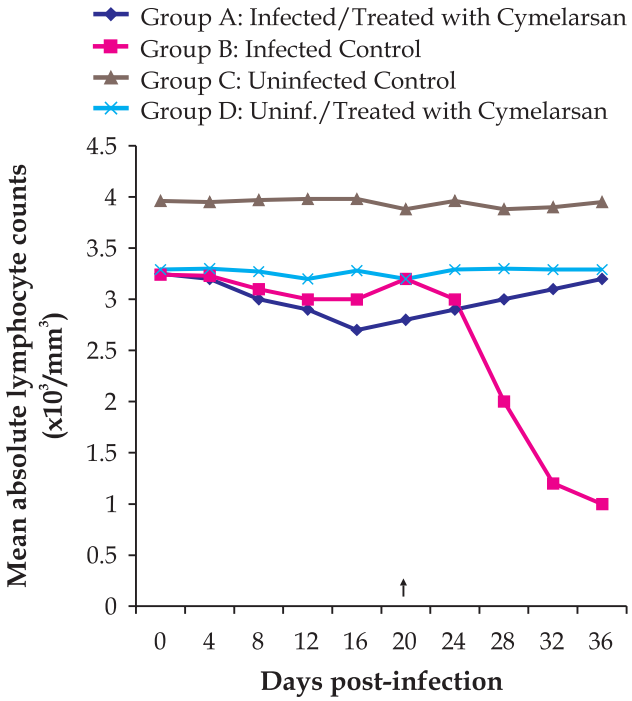

Fig 6. Mean absolute lymphocyte counts (x10^3/mm^3) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

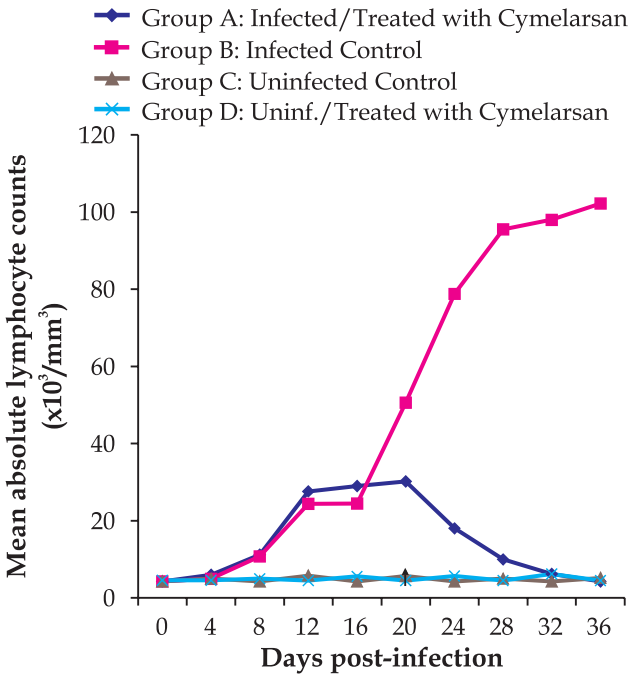

Fig 7. Mean absolute monocyte counts (x10^3/mm^3) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

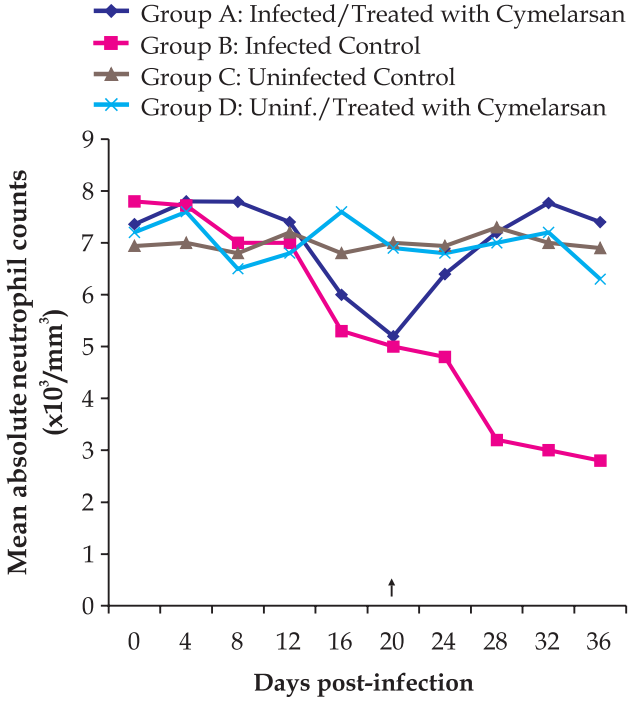

Fig 8. Mean absolute neutrophil counts (x10^3/mm^3) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

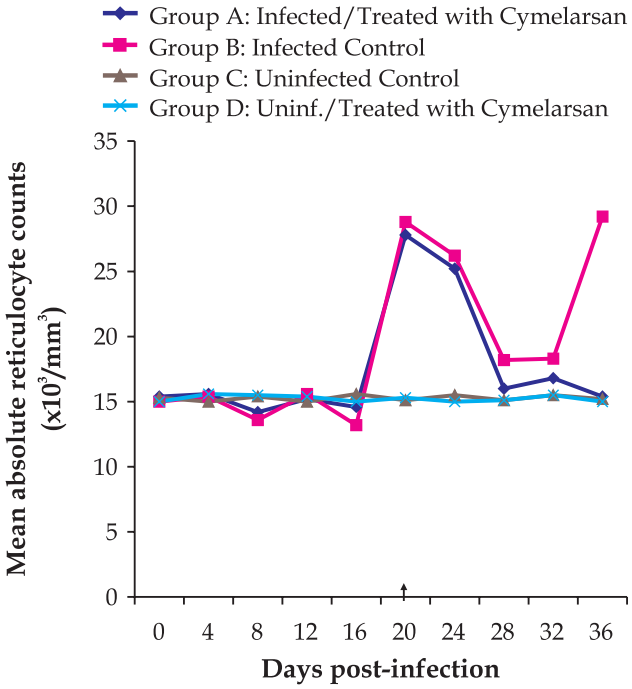

Fig 9. Mean absolute reticulocyte counts ($x10^3/mm^3$) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

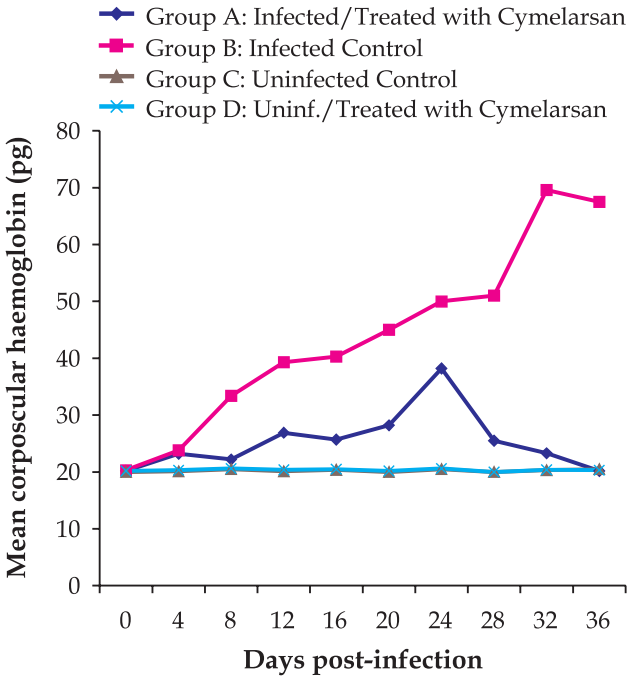

Fig 11. Mean corpuscular haemoglobin (pg) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and theircontrols

Key: Day of treatment (arrowed)

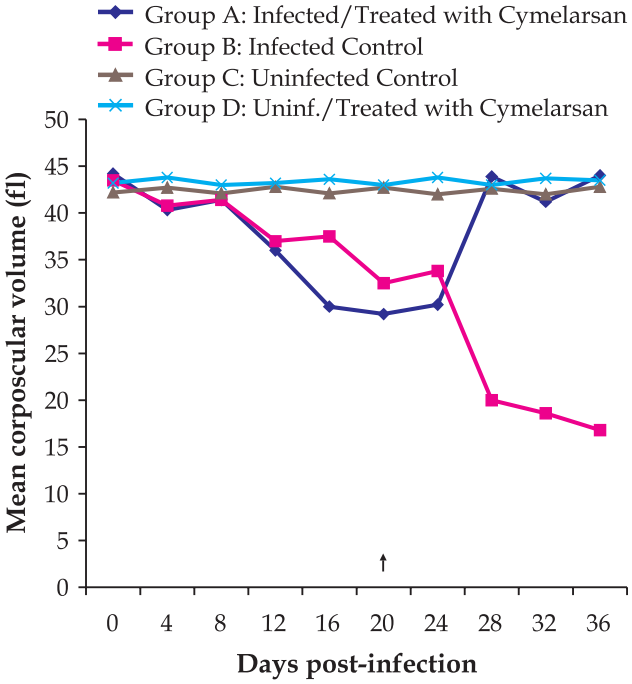

Fig 10. Mean corpuscular volume (fl) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

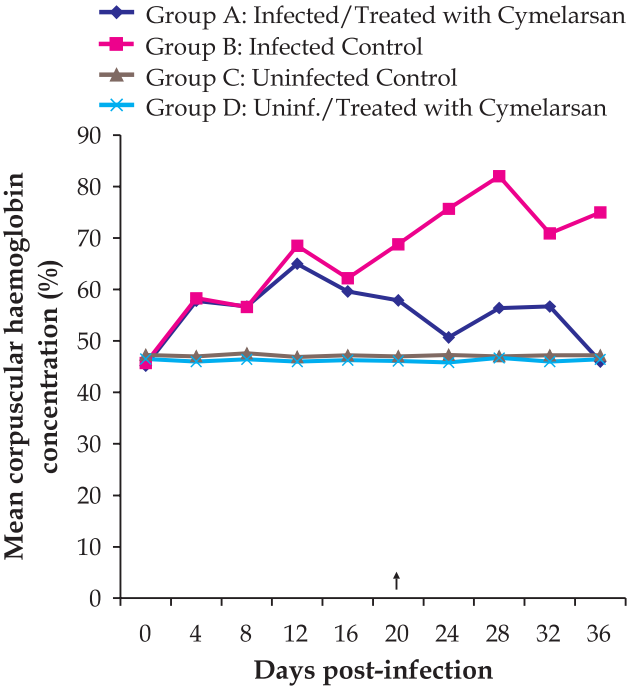

Fig 12. Mean corpuscular haemoglobin concentration (%) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

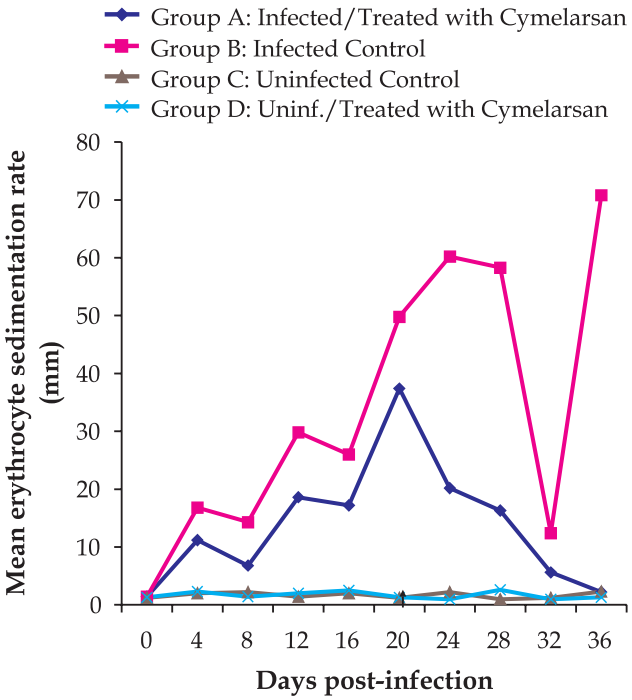

Fig 13. Mean erythrocyte sedimentation rate (mm) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

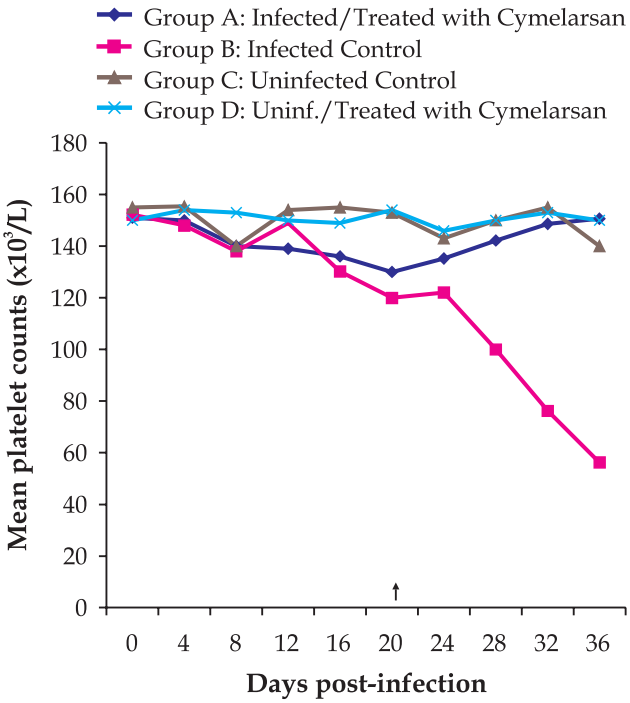

Fig 14. Mean platelet counts (x10^3/L) of one humped camels (*Camelus dromedarius*) experimentally infected with *Trypanosoma evansi* and treated with melarsenoxyde cysteamine hydrochloride (Cymelarsan®) and their controls

Key: Day of treatment (arrowed)

Statistical analysis

The data obtained from the study were summarised as means ± standard deviation and the differences between the means determined at 5% level of significance using the analysis of variance ANOVA (Graph Pad Instat 2000).

Results

The mean parasite count of the camels infected with *T. evansi* and treated with Cymelarsan® and their controls are presented in Fig 1. In groups A and B infected with the parasite, a uniform pre-patent period of 4 days was observed. In group A, the parasitaemia reached a peak count of 210.2 ± 1.81 by day 20 post-infection (p.i.). Following treatment with Cymelarsan®, the parasitaemia declined and was eliminated by day 32 (p.i.) or by day 12 post-treatment (p.t.). All the camels in this group survived. In group B (Infected Control), parasitaemia appreciated significantly ($p<0.05$) to a peak count of 400.2 ± 2.50 by day 36 (p.i.). One camel died in this group by day 28 (p.i.) while the remaining 4 died by day 36 (p.i.). In group C (uninfected control) and group D (uninfected but treated with Cymelarsan®) no deaths were recorded (Table 1).

The packed cell volume (%) of the camels and their controls are presented in Fig 2. In group A, the pre-infection PCV of 38.9 ± 0.78 declined significantly ($p<0.05$) from day 4 (p.i.) to 19.0 ± 0.54 by day 20 (p.i.). Following treatment with Cymelarsan®, its pre-infection value was attained by day 36 (p.i.) or by day 16 (p.t.). In group B, the pre-infection PCV of 38.7 ± 0.78 declined significantly ($p<0.05$) to 12.0 ± 0.43 by day 36 (p.i.). Meanwhile, the pre-infection PCV of Group C and D remained fairly constant ($p>0.05$) throughout the study.

The mean RBC counts of the camels and their controls are presented in Fig 3. In Group A, the pre-infection value of 8.80 ± 0.37 declined significantly ($p<0.05$) to 3.9 ± 0.25 by day 20 (p.i.). Following treatment with Cymelarsan®, it appreciated significantly ($p<0.05$) to its pre-infection value by day 36 (p.i.) or by day 16 (p.t.). In Group B, the pre-infection value declined significantly ($p<0.05$) to 2.4 ± 0.19 by day 36 (p.i.). There were no significant ($p>0.05$) changes in the values of the camels in Groups C and D. The mean haemoglobin concentration of the camels and their controls are presented in Fig 4. In Group A, the mean pre-infection value of 17.6 ± 0.52 declined significantly ($p<0.05$) to 11.0 ± 0.41 by day

20 (p.i.). Following treatment with Cymelarsan®, it appreciated significantly ($p<0.05$) to its pre-infection value by day 36 (p.i.) or by day 16 (p.t.). However, in group B, the pre-infection value of 17.7 ± 0.52 declined significantly ($p<0.05$) to 4.2 ± 0.26 by day 36 (p.i.). In groups C and D the values remained fairly constant (p>005).

The mean WBC counts of the camels and their controls are presented in Fig 5. In group A, the pre-infection value of 16.4 ± 0.51 declined significantly ($p<0.05$) to 9.9 ± 0.39 by day 20 (p.i.). Following treatment with Cymelarsan®, its pre-infection value was attained by day 36 (p.i.) or by day 16 (p.t.). However, in group B, the pre-infection value of 16.5 ± 0.51 declined significantly ($p<0.05$) to 5.0 ± 0.51 by day 36 (p.i.). Meanwhile, the values for group C and D remained fairly constant throughout the study (p>005). The mean absolute lymphocyte counts of the camels and their controls are presented in Fig 6. In group A, the pre-infection value of 3.26 ± 0.40 declined significantly ($p<0.05$) to 2.7 ± 0.21 by day 20 (p.i.). Following treatment with Cymelarsan®, it declined significantly ($p<0.05$) to its pre-infection value by day 36 (p.i.) or by day 16 (p.t.). In group B, the pre-infection value of 3.26 ± 0.40 declined significantly ($p<0.05$) to 1.2 ± 0.14 by day 36 (p.i.). Meanwhile, in groups C and D the values remained fairly constant throughout the study ($p>0.05$). The mean absolute monocyte counts of the camels and their controls are presented in Fig 7. In group A, the pre-infection value of 4.26 ± 0.26 appreciated significantly ($p<0.05$) to 30.2 ± 0.69 by day 20 (p.i.). Following treatment with Cymelarsan®, its pre-infection value was attained by day 36 (p.i.) or by day 16 (p.t.). In group B, the pre-infection value of 4.27 ± 0.26 appreciated significantly ($p<0.05$) to 102.2 ± 2.63 by day 36 (p.i.). Meanwhile, the monocyte values of camels in groups C and D remained fairly constant ($p>0.05$). The mean absolute neutrophil counts of the camels and their controls are presented in Fig 8. In group A, the pre-infection value of 7.36 ± 0.80 declined significantly ($p<0.05$) to 5.2 ± 0.29 by day 20 (p.i.). Following treatment with Cymelarsan® , its pre-infection value was attained by day 36 (p.i.). In group B, the pre-infection value of 7.80 ± 0.17 declined significantly ($p<0.05$) to 2.8 ± 0.21 by day 36 (p.i.) while there were no significant variation ($p>0.05$) in the values for groups C and D. The mean absolute reticulocyte counts of the camels and their controls are presented in Fig 9. In group A, the pr-infection value of 15.4 ± 0.49 appreciated significantly ($p<0.05$) to 27.8 ± 0.66 by day 20 (p.i.). Following treatment with Cymelarsan®, its pre-infection value was attained by day 36 (p.i.) or by day 16 (p.t.). In group B, the pre-infection value of 15.0 ± 0.48 appreciated significantly ($p<0.05$) to 29.2 ± 0.68 by day 36 (p.i.) while the values for groups C and D remained fairly constant ($p>0.05$).

The mean corpuscular volume (MCV) of the camels and their controls are presented in Fig 10. In group A, the pre-infection value of 44.2 ± 0.83 declined significantly ($p<0.05$) to 29.2 ± 0.68 by day 20 (p.i.). Following treatment with Cymelarsan®, the value appreciated significantly ($p<0.05$) to its pre-infection value by day 36 (p.i.) or by day16 (p.t.). In group B, the pre-infection value of 43.5 ± 0.82 decreased significantly ($p<0.05$) to 16.8 ± 0.81 by day 36 (p.i.). Similarly, no significant changes ($p>0.05$) were observed for camels in groups C and D. The mean corpuscular haemoglobin (MCH) of the camels and their controls are presented in Fig 11. In group A, the pre-infection value of 20.2 ± 0.56 appreciated significantly ($p<0.05$) to 28.2 ± 0.66 by day 20 (p.i.). Following treatment with Cymelarsan®, its pre-infection value was attained by day 36 (p.i.) or by day 16 (p.t.). In group B, the pre-infection value of 20.3 ± 0.56 appreciated significantly ($p<0.05$) to 67.5 ± 1.03 by day 36 (p.i.). Meanwhile, the values for groups C and D remained fairly constant ($p>0.05$). The mean corpuscular haemoglobin concentration (MCHC) of the camels and their controls are presented in Fig 12. In group A, the pre-infection value of 45.0 ± 0.84 appreciated significantly ($p<0.05$) to 59.6 ± 0.97 by day 16 (p.i.). Following treatment with Cymelarsan®, it declined significantly ($p<0.05$) to its pre-infection value by day 36 (p.i.) or by day 16 (p.t.). In group B, the pre-infection value of 45.7 ± 0.85 appreciated significantly ($p<0.05$) to 75.0 ± 1.08 by day 36 (p.i.) while the values for groups C and D remained fairly constant ($p>0.05$). The mean erythrocyte sedimentation rate (ESR) of the camels and their controls are presented in Fig 13. In group A, the pre-infection value of 1.4 ± 0.13 appreciated significantly ($p<0.05$) to 37.4 ± 0.76 by day 20 (p.i.). Following treatment with Cymelarsan®, its pre-infection value was attained by day 36 (p.i.) or by day 16 (p.t.). In group B, the pre-infection value of 1.4 ± 0.15 appreciated significantly ($p<0.05$) to 70.8 ± 1.05 by day 36 (p.i.) while no significant variations ($p>0.05$) were observed in groups C and D. The mean platelet counts of the camels and their controls are presented in Fig 14. In group A, the mean pre-infection value of 151.0 ± 1.54 declined significantly ($p<0.05$) to 136.2 ± 1.46 by day 36 (p.i.) or by day 16 (p.t.) and attained its pre-infection value Following treatment with

Cymelarsan®. Mean while the pre-infection value of 56.2 ± 0.94 in group B declined significantly ($p<0.05$) to 56.2 ± 0.94 by day 36 (p.i.). The values for group C and D remained fairly constant ($p>0.05$).

Discussion

In this study, fluctuations of parasitaemia were observed among the infected camels. Fluctuations of parasitaemia are known features of trypanosomosis commonly caused by antigenic variation (Nwosu and Ikeme, 1992; Mbaya *et al,* 2009a, b, c, d). The ability of the host to limit the peak and number of each wave of parasitaemia is however, dependant on whether the infection is acute, sub-acute or chronic (Katunguka-Rwakishaya *et al,* 1992). In this study where a standard dose of the inoculums were administered and uniform pre-patent periods were encountered, showed that the initial parasite replication rates were similar irrespective of the host susceptibility. These observations have been reported in *T. brucei* infection of dogs (Nwosu and Ikeme, 1992), red fronted gazelles (*G. rufifrons*) (Mbaya *et al,* 2009d; 2010) and in *T. brucei* gambiense infection in baboons (*Papio anubis*) (Mbaya *et al,* 2009a).

In this study, the infected camels showed a significant decline in red cell (PCV, RBC, Hb) parameters. The decline which was indicative of anaemia started at the onset of parasitaemia, from day 4 (p.i) up to day 20 (p.i.) for camels treated with Cymelarsan® and up to day 36 among the infected controls. This development was the same in all infected camels (Groups A and B) with the difference being only in the severity. This is in agreement with several reports quoting that the anaemia in trypanosomosis often started during the 1st wave of parasitaemia which is haemolytic in nature (Anosa, 1988; Igbokwe, 1994; Mbaya *et al,* 2011, 2012). However, the haemolytic nature of the anaemia in most cases would depend on the species of trypanosomes involved (Mbaya *et al,* 2012). The expanded and active mononuclear phagocytic system (MPS) has been a major player in haemolytic anaemia in trypanosomosis through erythrophagocytosis which develop soon after infection and continued thereafter, in the various phases of the disease (Mbaya *et al,* 2012). The presence of the MPS might have been associated with increased demand on the system to remove dead red blood cells, tissue cells, trypanosomes, antigen-antibody complexes and to participate in immune responses (Nwosu and Ikeme, 1992).

The fact that the red cell parameters (PCV, RBC, Hb) decreased sharply during bouts of parasitaemia but maintained a gradual increase during the periods of low parasitaemia showed an inverse relationship with parasitaemia (Nwosu and Ikeme, 1992; Mbaya *et al,* 2009c). The infected camels also showed decline in red cell indices (MCV) and an increase in MCHC and MCH. An increase in circulating haemoglobin and its concentration during trypanosomosis often follow the development of haemolytic anaemia commonly characterised by a significant increase in MCH and MCHC (Igbokwe, 1994). Therefore, the morphological classification of the anaemia in this case was normocytic normochromic in the acute phase and macrocytic hypochromic in the chronic phase. This is in consonance with the *T. brucei* infection in dogs (Ogunsanmi *et al,* 1994) or in chronic *T. evansi* infection in camels (Jatkar and Purohit, 1971). Similarly, a decline in platelet count was also encountered which was indicative of thrombocytopaenia, while reticulocytosis and increase in erythrocyte sedimentation rate (ESR) was also encountered. The reticulocyte response however, occurred by days 20 and 36 (p.i.). The reticulocytosis encountered among the camels on those days was in response to the haemolytic crises which is commonly accompanied by increased eryhropoesis and reticulocyte response (Igbokwe, 1994; Mbaya *et al,* 2012). The increase in ESR in this study agrees with the *T. brucei* infection of cattle (Antia *et al,* 1995), red fronted gazelles (*G. rufifrons*) (Mbaya *et al,* 2009c, 2012) and in *T. evansi* infection of camels (Raisinghani and Lodha, 1980). Increase in ESR in trypanosomosis has been associated with anaemias which commonly relates to the antigenic status of the animals (Katunguka-Rwakishaya *et al,* 1992).

The thromocytopenia encountered among the camels is associated with coagulation defects due to low platelet numbers which have been reported to be associated with disseminated intravascular coagulation in trypanosomosis (Igbokwe, 1994; Mbaya *et al,* 2012). This has been reported commonly in acute and chronic *T. brucei* infection of sheep (Ogbechie and Oyejide, 1988) but it does not agree with the reports of Antia *et al (*1995), which perhaps is the only report of thrombocytosis in cattle trypanosomosis. The infected camels also exhibited leucopoenia which was indicative of immunosuppression commonly encountered in *T. evansi* infection in camels (Njiru *et al,* 2004). Leucopoenia has been reported as a consistent feature in trypanosomosis (Nwosu and Ikeme, 1992). Similarly, lymphopeania was encountered among the infected camels. This is probably associated with an increased demand on

the system for lymphocytes, which is a common requirement in both immune and inflammatory responses in trypanosomosis (Igbokwe, 1994). Meanwhile, the significant neutropaenia encountered among the infected camels might be associated with splenic sequestration of leucocytes which often suppressed the raising of neutrophil numbers (Nwosu and Ikeme, 1992). The infected camels however, showed significant monocytosis. This has been reported as a common feature in trypanosomosis (Mbaya *et al,* 2009a). Monocytosis in trypanosomosis has been associated with a proliferation of tissue macrophages likely due to an increased demand on the system to remove dead red cells, antigen/ antibody complexes and to participate in immune responses. Since macrophages are formed from blood monocytes, this increased need for macrophages may have been responsible for the consistent monocytosis encountered in this study. In all cases, camels treated with Cymelarsan®, had all haematological parameters modulated to their pre-infection status with 0% mortality which showed that the drug is a good candidate for the treatment of surra in camels. It is therefore concluded that *T. evansi* can cause severe haematological changes in camels which can be reversed following treatment with Cymelarsan®.

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References

Anosa VO (1988). Haematological and biochemical changes in human and animal trypaosomiasis. Revue d'élevage et de Medicine vétérinaire des Pays Tropicaux 41:151-164.

Antia RE, Ohore OG, Ogunsanmi AO, Michael J, Awolaya OA, Ajuwape ATP, Adeneye TO and Oyejide A (1995). Clinicopathological changes associated with trypanosome antigenaemia in Nigerian Cattle. Organization of African Unity/International Scientific Council for Trypanosomiasis Research and Control 18: 136-137.

Aradib IE and Majid A (2006). A simple and rapid method for detection of *Trypansoma evansi* in the dromedary camel using PCR. Kinetopastid Biology and Diseases 5:2-12.

Broom DM and Johnson KG (1993). Stress and Animal Welfare, London, UK. Chapman and Hall.

Cadioli FA, Marques LC, Machedo R.Z, Alessi AC, Aquino LPCT and Barnabas PA (2006). Experimental *Trypanosoma evansi* infection in donkeys: haematological, biochemical and histopathological changes. Arquivo Brasileiro de Medicina Veterináriae Zootecnia 58(5): 123-126.

Coles FW (1986). Veterinary Clinical Pathology, 4th Ed. WB. Saunders Company, London.

Enwezor FNC and Sackey AKB (2005). Camel trypanosoniasis: A review. Veterinarski Arhiv 75(5):439-452.

Graph Pad Instat, 2000 "Version 3.00 for windows, Graph Pad Software, San Diego, CA, USA, www.graphpad.com

Gutierrez G, Justa MC, Corbera JA, Magnus E, Verloo D and Montoya JA (2000). Camel trypanosomiasis in the Canary Islands: assessment of seroprevalence and infection rates using the card agglutination test (CATT/ *Trypanosoma evansi*) and parasite tests. Veterinary Parasitology 90:155-159.

Herbert WJ and Lumsden WHR (1976). *Trypanosoma brucei:* A rapid matching method for estimating host's parasitaemia. Experimental Parasitology (40)43:427-432.

Herrera HM, Alessi AC, Marqes LC, Santana AE, Aquino LPCT, Menezes RF, Mornes MAV and Machado RZ (2002). *Trypanosoma evansi*: experimental infection in the South American coati (*Nasua nasua*): haematological, biochemical and histopathological changes. Acta Tropica 81(3):203-210.

Igbokwe IO (1994). Mechanisms of cellular injury in African trypanosomosis. Veterinary Bulletin 64(7):1684-5315.

Jaktar PR and Purohit MS (1971). Pathogenesis of anaemia in *Trypanosoma evansi* infections. I. Haematology. Indian Veterinary Journal 48:239-244.

Joshi PP, Chaudhari VR, Shegokar VR, Power RM, Dani VS, Somalwar AM, Jannin J and Truc P (2006). Treatment and follow-up of the first case of human trypanosomosis caused by *Trypanosoma evansi* in India. Transactions of the Royal Society of Tropical Medicine Hygiene 100:989-991.

Katunguka-Rwakishaya E, Murray M and Holmes PH (1992). The pathophysiology of ovine typanosomiasis: haematological and blood biochemical changes. Veterinary Parasitology 45(1-2):17-32.

Mbaya AW, Nwosu CO and Aliyu MM (2009a). An assessment of the efficacy of DFMO in baboons (*Papio anubis*) infected with *Trypanosoma brucei gambiense*. Global Journal of Veterinary Sciences 15(1):69-78.

Mbaya AW, Aliyu MM and Ibrahim UI (2009b). Clinico-pathology and mechanisms of trypanosomosis in captive and free-living wild animals: A review. Veterinary Research Communication 33:793-809.

Mbaya AW Aliyu MM, Nwosu CO and Egbe-Nwiyi TNC (2009c). The relationship between parasitaemia and anaemia in a concurrent *Trypanosoma brucei* and *Haemonchus contortus* infection in red fronted gazelles (*Gazella rufifrons*). Veterinarski Arhiv 79(5):451-460.

Mbaya AW, Aliyu MM, Nwosu CO, Taiwo VO and Ibrahim UI (2009d). Effects of melarsamine hydrochloride (Cymelarsan®) and diaminazene aceturate (Berenil®) on the pathology of experimental *Trypanosoma brucei* infection in red fronted gazelles (*Gazella rufifrons*). Veterinary Parasitology 163(1-2):140-143.

Mbaya AW, Ibrahim UI, Onyiche T and Sanya L (2010). Toxicity and potential anti-trypanosomal activity of ethanolic extract of *Azadirachta indica* (Maliacea) stem bark: An

in-vivo and in-vitro approach using *Trypanosoma brucei*. Journal of Ethnopharmacology 128:495-500.

Mbaya AW, Nwosu CO and Kumshe HA (2011). Genital lesions in male red fronted gazelles (*Gazella rufifrons*) experimentally infected with Trypanosoma brucei and the effect of melarsamine hydrochloride (Cymelarsan®) and diminazene aceturate in their treatment. Theriogenology 76(4):721-728.

Mbaya AW, Kumshe HA and Nwosu CO (2012). The Mechanisms of Anaemia in Trypanosomosis, A Review. In: Donald, S. Silverberg (Ed). Anaemia, In Tech. Publishers, Croatia.

Nwosu CO and Ikeme MM (1992). Parasitaemia and clinical manifestations in Trypanosoma brucei infected dogs. Revue d'élevage et de medicine vétérinaire des pays tropicaux 45(3-4):273-277.

Njiru ZK, Bett O, Ole-Murphy SM, Githioni JB and Ndung'u JM (2002). Trypanosomosis and helminthosis in camels: comparison of ranch and traditional camel management systems in Kenya. Journal of Camel Practice and Research 55:67-71.

Njiru ZK, Constantine CC, Ndungu JM, Robertson I, Okayes S, Thompsorza RCA and Reida SA (2004). Detection of *Trypanosoma evansi* in camels using PCR and CATT/*Trypanosoma evansi* tests in Kenya. Veterinary Parasitology 124:187-219.

Ogbechie CA and Oyejide A (1988). Thrombocytopenia and coagulation defects in acute and chronic *Trypanosoma brucei* infections. Tropical Veterinarian 6(1-2):77-82.

Ogunsanmi AO, Akpavie SO and Anosa VO (1994). Haematological changes in ewes experimentally infected with *Trypanosoma brucei*. Revue d'élevage et de medicine vétérinaire des pays tropicaux 47(1):55-57.

Parija SC and Bhattacharya S (2005). The tragedy of the tigers (Panthera tigris). Lesson to learn from Nandankan episode. Indian Journal of Medical Microbiology 23: 43-48.

Raisinghani PM and Lodha KR (1980). Prognostic values of some haematological and biochemical parameters of camels affected with surra following the treatment with Antrypol, Antrycide methyl sulphate, Naganol and Berenil. Indian Veterinary Journal 57(6):479-484.

Silva RAMS, Arosemena NAE, Herrera HM, Sahib CA and Ferrera MSJ (1995). Outbreak of trypanosomiais due to *Trypanosom evansi* in horses of Pantela. Mato-Grossense, Brazil. Veterinary Parasitology 60:167-171.

Shalm OW, Jain NC and Carroll EJ (1995). Veterinary Haematology, 3rd Ed. Lea and Fabiger, Philadelphia.

Soulsby EJL (1982). Helminthes, Arthropods and Protozoa Parasites of Domesticated Animals, Baillere Tindal, London.