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Haemotological Indices Of Oryctolagus cuniculus (New- Zealand Rabbit) Experimentally Infected With Trypanosoma brucei brucei Treated With Diminazene Diaceturate (Sequzene) In Maiduguri, Bornu State, Nigeria

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Abstract: *This study was conducted to determine the haematological indices of experimentally inoculated Oryctolagus cuniculus (New-Zealand Rabbit) with Trypanosoma brucei brucei at a concentration of 1.5 x 10^6 then treated with Diminazine diaceturate (Sequzene) for twenty one (21) days. A total of thirty rabbits of both sexes were divided into six groups (A-F) of five (5) animals each. Group A were infected with T. brucei brucei only but untreated, while group B were uninfected , untreated control. Group C was infected with T. brucei brucei and treated with Sequzene at 3.5 mg/kg, while group D was infected and treated with the drug at 7.0 mg/kg body weight. Group E were uninfected with T. b. brucei and treated with Sequzene at 3.5mg/kg and group F was uninfected and treated at 7.0 mg/kg of the same drug. Physical symptoms were monitored daily and blood samples taken at seven days intervals then analysed for haematological parameters according to standard laboratory techniques. Physical signs observed include dullness, weakness, anorexia, weight loss, increased respiration, starry hairs and corneal opacity in the infected groups. The major haematological changes observed were anaemia characterized by a significant decline ($p<0.05$) in mean packed cell volume (PCV). Other changes observed were significant decline in haemoglobin (Hb) concentration, red blood cell (RBC) and white blood cell (WBC) counts at $p<0.05$ respectively. Rabbits in group A showed nervous disorder (convulsion) at the point of death. As the disease progressed in all infected groups, neutropenia, lymphopenia and monocytosis increased leading to death. However, the haematological parameters showed signs of restoration in groups treated with Sequzene.*

Keywords: *Haematological, Oryctolagus cuniculus (New-Zealand Rabbit), Trypanosoma brucei brucei, Diminazene diaceturate (Sequzene), Anaemia.*

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I. Introduction

African trypanosomosis in humans and livestock has devastated the livestock industry and jeopardized the health of millions in tropical Africa where the disease is endemic, [1]. The disease has left the health of most afflicted societies precarious and their economies impoverished. In order to develop a better understanding of the disease process, as well as control it in the study of induced laboratory animal models of the disease , several studies to generate a large body of information as part of biomedical research has been undertaken widely. The pathology and pathogenesis of the disease has been thoroughly reviewed by several workers [2, 3, 4]. It was generally accepted that the major disease promoting factor in trypanosomosis is anaemia [5, 6]. Anaemia in trypanosomosis haemolytic in nature, [7] and the aetiology of the haematological changes are complex and multi factorial, [8] which may include the destruction of mature erythrocytes, leucocytes and thrombocytes by macrophages in the bone marrow, spleen, liver and haemolymph nodes [9]. Although anaemia occurs in most trypanosome infection of animals, the severity of haematological changes has been reported to be greater in *Trypanosoma vivax* and *Trypanosoma conglense* infection which are strictly intravascular parasites than *Trypanosoma brucei*, which is a tissue invading parasite [10, 11, 12, 13, 14]. Several erythrocyte morphological abnormalities including anisocytosis, poikilocytosis, polychromasia, punctate basophilia and macrocytosis have been described in various combinations of *Trypanosoma brucei* and *T.congolense* infections [15,3]. Leucopenia has been reported in *Trypanosoma vivax* of cattle [12] and *T.brucei* of red fronted gazelle (*Gazellaru fifrons*) [13].

So far, little has been achieved in terms of understanding the variation in haematological changes of the disease in various laboratory animal species especially as it relates to *T.brucei brucei* and the effects of diminazene diaceturate (sequzene) on experimentally induced hosts such as rabbits.

II. Objectives Of The Study

The study was designed to determine the effect of *diminazene diaceturate* on the parasitological and heamatological parameters of infected animals.

III. Materials And Methods

Sources of New Zealand Rabbits,*Trypanosoma brucei brucei* and drug used

i. New Zealand rabbit

A total of thirty (30) healthy New Zealand rabbits of both sexes were purchased from commercial farmers within Maiduguri. The rabbits on arrival were kept in the animal clinic, University of Maiduguri, Nigeria.

These rabbits were routinely screened for blood and intestinal parasites using standard criteria by [16], as well as ectoparasites according to [17]. The rabbits were fed on pelleted commercial feeds (Vital Feeds Ltd, Jos , Nigeria) and fresh green vegetables purchased locally. Water was provided *ad libitum*. The rabbits were allowed for

14 days to acclimatize to their new environment before commencement of the research. The experiment was carried out in accordance with international guidelines for the use of animals for biomedical research and welfare [18].

ii. Trypanosoma brucei brucei

The *T.brucei brucei*. (Federe strain) used was obtained from the Nigeria Institute for Trypanosomosis Research (NITOR) in Jos, Nigeria. The trypanosomes were first isolated from a natural infection in 2006 from *N' dama* and *Muturu* cattle. They were identified as *T. brucei brucei* based on the morphology and negative blood inhibition infectivity test (BIIT) and stabilized by four passages in rats before storage in liquid nitrogen.

iii .Source of drug (diminazene diaceturate).

Diminazene diaceturate was obtained from the manufacturer (Ferberwarke, Germany) and dissolved in 15ml of distilled water and administered intraperitonealy at 3.5 mg/kg and 7.0 mg/kg to the rabbits.

***In vivo* EXPERIMENTAL DESIGN.**

The 30 rabbits were weighed using Cammry scale model P2117, China and randomly separated into six (6) groups (A, B, C, D, E, and F).

The groups were infected and treated as follows;

Group A. infected and treated control (negative control)

Group B. uninfected and untreated control (normal)

Group C. infected and treated with a single standard dose of diminazene diaceturate (sequzene) administered intravenously at 3.5 mg/kg by day 10 and terminated on day 21.

Group D. infected and treated with single standard dose of diminazene diaceturate (sequzene) at 7.0 mg/kg by day10

Group E. uninfected and treated with a single standard dose of diminzene diaceturate (sequzene) at 3.5 mg/kg by day10

Group F. uninfected and treated with a single dose of diminazene diaceturate (sequzene) at 7.0 mg/kg by day 10.

This experiment was approved by the ethical committee for the use of animals for biomedical research, of the faculty of veterinary medicine, University of Maiduguri.

COLLECTION OF BLOOD SAMPLES FOR THE DETERMINATION OF HAEMATOLOGICAL PARAMETERS.

Blood samples from rabbits were obtained via the ear vein and collected in vacutainers containing ethylenediamine tetra acetate (EDTA) as anticoagulant every seven (7) days for a period of three weeks (21 days) according to standard methods by[19] and [20].

DETERMINATION OF PACKED CELL VOLUME (PCV%)

The micro haematocrit method was used to estimate the PCV as described by [20]. Five (5) to ten (10) milliliters of blood was thoroughly mixed with 1-2 milliliters of EDTA. The capillary tubes were filled to about three quarters with the blood. One end of each capillary tube was sealed with plasticine by holding the tube vertically, pushing it down firmly into the plasticine and twisting at the same time. The tubes were then loaded symmetrically in the micro hacmatocrit centrifuge (Hawsley,England) with the sealed end outermost. The capillary tubes were centrifuged at 1200 rpm for 5minutes and the PCV read using the hacmatocrit reader then expressed in percentage.

DETERMIANTION OF MEAN RED BLOOD CELLS COUNT (X 10^6 mm^3)

5ml of blood was mixed with the red blood count diluting fluid known as hayems solution and drawn up to O.5 mark of the Neubauer counting chamber [20]. It is further diluted with the hayems solution and filled to 101 mark by slow but continuous exertion and allowed to stand for three (3) minutes. The RBCS were counted using x10 eyepiece and x40 objective lenses of the microscope.The counting chamber had 5 tertiary squares each containing sixteen (16) smaller squares. All RBC' s found were counted per square of the chamber. The number of red cells was calculated as follows:-

The number of RBC/mm^3=number of cells counted x correction for volume x correction for dilution.

DETRMINATION OF MEAN WHITE BLOOD CELLS COUNT (X $1O^3mm^3$).

Neaubauer haemocytometer counting chamber [19] was used for the WBC count. 10 ml of blood was thoroughly mixed and diluted with Turk's diluting fluid then aspirated to the $0.5mm^3$ mark of the haemocytometer. Further aspiration was done to the $11mm^3$ mark. The content was shaken by slow and continuous eversion for 3minutes. Four (4) drops of the mixture was expelled on to a piece of gauze to charge the counting chamber which is covered with a glass on top of the ruled areas, the mixture was allowed to sit for one minute before the WBC was counted. All WBC' s were counted using low power magnification of the microscope in the four square milliliters of the haemocytometer. The number of WBC' s per $10^3/mm^3$ was calculated as follows:-

Number of WBC x $10^3/mm^3$ = white cells counted x correction for volume x correction for dilution.

DETERMINATION OF THE HAEMOGLOBIN CONCENTRATION (HBC) (g/dl)

The haemoglobin concentration was determined using Sahli' s method [19].Twenty cubic milliliters of blood was drawn up to the $20mm^3$Sahli' spippete which was cleaned and dried before the exercise. 2mls of 0.1N Hcl was added to the $20mm^3$ blood gently and thoroughly mixed. Distilled water was added drop by drop while at the same time stirring with a glass rod. The distilled water was continuously added until the colour matched that of the standard mixture. The haemoglobin value was calculated and expressed in grams per deciliter.

DETERMINATION OF DIFFERENTIAL LEUCOCYTE COUNT.

The differential leucocyte count (lymphocytes(L) , monocytes (M) and neutrophils (N))
were determined by counting and differentiating 100/WBC and the values were converted to absolute numbers according to [19].

Statistical Analysis

Data generated from the study were expressed as mean$\pm$ standard deviations (S.D) using 2 way analysis of variance (ANOVA) at $p< 0.05$ was considered significant [22].

IV. Results

Haematological indices of *Oryctolagus cuniculus* (New-zealand Rabbit) infected with *Trypanosoma brucei brucei* and treated with *Diaminazene diaciturate Sequzene*)

The mean PCV, RBC, HBC, WBC, NC, ML and MN of the New-ZeaLand Rabbits experimentally infected with *T. brucei brucei* and treated with Sequzene is presented in Tables 1-7 respectively below. In group A, table 1, (infected but untreated control), the pre-infection (P.I) PCV of 30.25 $\pm$ 2.63 was obtained, the rabbits experienced a continuous but significant decline at ($p<0.05$) from day 7(P.I) to 16.00 $\pm$ 0.81 by day 14 post infection when all rabbits died of the infection.

In group B (uninfected/treated control), group E (uninfected and treated with 3.5mg/kg) of sequzeneand group F (uninfected and treated with sequzene at 7.0mg/kg) their pre-infection value of 32.00$\pm$2.94, 33.50 $\pm$ 2.38 and 33.50 $\pm$ 3.00 remain fairly constant at ($p>0.05$) throughout the study.

In group C (infected/treated pre-infected with 3.5mg/kg of sequzene and group D (infected/treated post infection with sequzene at 7.0mg/kg), the pre- infected values of 34.00 $\pm$ 0.81 and 32.75 $\pm$ 3.20 experienced a sharp and significant decline at ($p<0.05$) from day 7 (P.I) to 37.25 $\pm$ 1.25 and 34.50 $\pm$ 0.57 by day 21 post infection when all the rabbits in both groups died of the infection.

Table1: Effects of *Diminazene diaceturate* on mean packed cell volume (%) of New- Zealand rabbits experimentally infected with *Trypanosoma brucei* with their controls.

Groups (n=5)	PPTb	day of treatment		day of post-infection
	7	10	14	21
A	30.25+2.63[a]	24.25+2.87[b]	27.00 + 1.41[c]	27.00 + 1.41[c]
B	32.00+ 2.94[a]	31.25+1.50[a]	32.75+0.95[a]	32.75+0.50[a]
C	34.00+0.89[a]	24.00+0.89[b]	33.75+1.35[a]	34.50+0.57[a]
D	32.75+3.20[a]	24.00+0.89[b]	33.75+1.35[a]	34.50+0.57[a]
E	33.50+2.38[a]	32.75+2.27[a]	34.25+0.95[a]	34.75+0.50[a]
F	32.00+2.94[a]	32.35+1.70[a]	39.75+0.95[a]	33.00+2.30[a]

Numbers with different superscripts in rows and in columns differed significant at ($p<0.05$)
Key:
A: infected/untreated control
B: uninfected/untreated control
C: infected/treated with 3.5 mg/kg of sequzene
D: infected/treated with 7.0mg/kg of sequzene
E: uninfected/treated with 3.5mg/kg of sequzene
F: uninfected/ treated with 7.omg/kg of sequzene
PP Tb = Pre-patent period for *Trypansoma brucei brucei*.

Table2: Effects of *Diminazene diaceturate* on red blood cell counts($x10^6mm^3$) of New- Zealand rabbits experimentally infected with *Trypanosoma brucei brucei* with their controls.

Groups (n=5)	PPTb	day of treatment		day of post-infection
	7	10	14	21
A	7.69+0.10[a]	4.79+0.67[b]	2.75 + 0.39[c]	2.18+1.10[d]
B	7.48+ 0.53[a]	7.19±0.43[a]	7.33±0.35[a]	7.47±0.41[a]
C	7.69±0.46[a]	4.93±0.15[b]	6.47±1.17[a]	7.36±0.40[a]
D	7.46±0.36[a]	4.50±0.45[b]	6.76±0.76[a]	7.32±0.36[a]
E	7.44±0.30[a]	7.19±0.48[a]	7.09±0.04[a]	7.03±0.16[a]
F	7.36±0.43[a]	7.32±0.36[a]	7.40±0.52[a]	7.09±0.14[a]

Numbers with different superscripts in rows and in the columns differed significantly at ($p<0.05$).
Key:
A: infected/untreated control
B: uninfected/untreated control
C: infected/treated with 3.5mg/kg of s*equzene*
D: infected/treated with 7.0mg/kg of*sequzene*
E: uninfected/treated with 3.5mg/kg of *sequzene*
F: uninfected/treated with 7.0mg/kg of*sequzene*
PP Tb = Pre-Patent period for *Trypanosomabruceibrucei*

Table 3: Effects of Diminazene diaceturate on mean haemoglobin concentration (g/dl) of New- Zealand rabbits experimentally infected with *Trypanosoma brucei brucei* with their controls.

Groups (n=5)	PPTb	day of treatment		day of post-infection
	7	10	14	21
A	13.97±1.19[a]	9.95±1.69[b]	7.32 ± 0.67[c]	4.70±0.47[d]
B	14.05± 1.25[a]	14.22±0.93[a]	14.87±0.29[a]	14.02±0.55[a]
C	14.75±0.69[a]	9.20±1.64[b]	13.25±0.78[a]	14.15±0.26[a]
D	14.27±0.91[a]	9.80±1.33[b]	13.57±1.87[a]	13.77±0.90[a]
E	14.32±0.69[a]	14.42±0.78[a]	14.07±0.53[a]	14.22±1.73[a]
F	14.55±0.75[a]	14.17±0.25[a]	14.00±0.40[a]	14.55±0.58[a]

Numbers with different superscripts in rows and in columns differed significantly at ($p<0.05$).
Key:
A: infected/untreated control
B: uninfected/untreated control
C: infected/treated with 3.5 mg/kg of sequzene
D: infected/treated with 7.0mg/kg of sequzene
E: uninfected/treated with 3.5mg/kg of sequzene
F: uninfected treated with 7.omg/kg of sequzene

PP Tb = Pre-patent period for Trypanosoma brucei brucei.

Table 4: Effects of diminazene diaceturate on mean white blood cell count ($x10^3/mmm^3$) New- zealand rabbits experimentally infected with *Trypanosoma brucei brucei* with their control.

Groups (n=5)	PPTb	day of treatment		day of post-infection
	7	10	14	21
A	9.52 ± 0.41^a	7.42 ± 0.43^b	5.69 ± 0.68^c	5.69 ± 0.68^c
B	9.85 ± 0.92^a	9.79 ± 0.26^a	9.75 ± 0.64^a	10.62 ± 1.37^a
C	9.83 ± 0.63^a	6.94 ± 0.40^b	9.25 ± 0.64^a	9.75 ± 0.95^a
D	9.52 ± 0.41^a	6.87 ± 0.62^b	10.39 ± 0.74^a	9.62 ± 0.75^a
E	9.70 ± 0.47^a	9.65 ± 0.94^a	10.75 ± 0.64^a	9.87 ± 0.62^a
F	9.82 ± 0.20^a	9.82 ± 0.20^a	9.84 ± 0.17^a	9.99 ± 0.06^a

Numbers with different superscripts in rows and in the column differed significantly ($p<0.05$)
Key:
A: infected/untreated control
B: uninfected/untreated control
C: infected/treated with 3.5mg/kg of sequzene
D: infected/treated with 7.0mg/kg of sequzene
E: uninfected/treated with 3.5mg/kg of sequzene
F: uninfected/treated with 7.0mg/kg of sequzene
PP Tb = Pre-Patent period for *Trypanosoma brucei brucei*

Table 5: Effects of diminazene diaceturate n mean absolute neutrophil count (%) of new Zealand rabbits experimentally infected with *Trypanosoma brucei brucei* with their controls.

Groups (n=5)	PPTb	day of treatment		day of post-infection
	7	10	14	21
A	37.25 ± 2.21^a	28.75 ± 3.40^b	22.75 ± 4.50^c	22.75 ± 4.50^c
B	38.00 ± 2.16^a	37.75 ± 1.50^a	37.50 ± 1.00^a	37.50 ± 1.00^a
C	38.00 ± 2.16^a	27.25 ± 0.95^b	34.75 ± 4.50^a	37.25 ± 2.06^a
D	38.75 ± 0.95^a	28.25 ± 1.70^b	35.75 ± 3.09^a	38.25 ± 1.70^a
E	38.75 ± 1.70^a	38.75 ± 1.50^a	38.50 ± 1.73^a	39.25 ± 0.95^a
F	37.50 ± 1.73^a	37.75 ± 1.50^a	38.25 ± 0.95^a	37.75 ± 1.70^a

Numbers with different superscripts in rows and in the columns differed significantly at ($p<0.05$).
Key:
A: infected/untreated control
B: uninfected/untreated control
C: infected/treated with 3.5mg/kg of sequzene
D: infected/treated with 7.0mg/kg of sequzene
E: uninfected/treated with 3.5mg/kg of sequzene
F: uninfected/treated with 7.0mg/kg ofsequzene
PP Tb = Pre-Patent period for *Trypanosoma brucei brucei*

Table 6: Effects of diminazene diaceturate on mean absolute lymphocytes(%) of New- Zealand experimentally infected with Trypanosoma brucei brucei with their controls.

Groups (n=5)	PPTb	day of treatment		day of post-infection
	7	10	14	21
A	64.25 ± 3.30^a	53.50 ± 1.29^b	42.00 ± 0.81^c	33.50 ± 1.29^d
B	62.00 ± 4.24^a	64.00 ± 4.08^a	63.75 ± 4.34^a	65.00 ± 3.55^a
C	66.50 ± 1.29^a	52.00 ± 0.81^b	60.50 ± 0.57^a	65.25 ± 1.70^a
D	66.50 ± 1.29^a	50.75 ± 0.95^b	59.25 ± 1.70^a	63.25 ± 3.50^a
E	66.75 ± 1.70^a	63.50 ± 3.69^a	60.75 ± 4.19^a	63.00 ± 2.82^a
F	65.75 ± 4.57^a	64.75 ± 4.42^a	64.50 ± 3.10^a	65.00 ± 3.55^a

Numbers with different superscripts in rows and in the column differed significantly at ($p<0.05$)
Key:
A: infected/untreated control
B: uninfected/untreated control
C: infected/treated with 3.5mg/kg of sequzene
D: infected/treated with 7.0mg/kg of sequzene
E: uninfected/treated with 3.5mg/kg of sequzene

F: uninfected/treated with 7.0mg/kg of sequzene
PP Tb = Pre-Patent period for *Trypanosomabruceibrucei*

Table 7: Effects of diminazene diaceturate on mean absolute monocytes (%) of New- Zealand rabbits experimentally infected with *Trypanosoma brucei brucei* with their controls.

Groups (n=5)	PPTb	day of treatment		day of post-infection
	7	10	14	21
A	6.50 ± 1.29^{a}	12.75 ± 0.95^{b}	17.75 ± 2.21^{c}	23.75 ± 0.95^{d}
B	6.50 ± 1.29^{a}	6.00 ± 0.81^{a}	6.50 ± 1.29^{a}	6.50 ± 1.29^{a}
C	7.00 ± 0.81^{a}	13.25 ± 0.95^{b}	8.00 ± 0.81^{a}	6.75 ± 0.95^{a}
D	7.50 ± 0.57^{a}	14.00 ± 0.81^{b}	7.50 ± 0.57^{a}	7.00 ± 0.81^{a}
E	6.50 ± 1.29^{a}	6.75 ± 0.95^{a}	6.25 ± 0.95^{a}	6.00 ± 0.81^{a}
F	6.50 ± 0.57^{a}	6.50 ± 0.57^{a}	6.75 ± 0.95^{a}	6.50 ± 0.57^{a}

Numbers with different superscripts in rows and in the columns differed significantly at ($p<0.05$).
Key:
A: infected/untreated control
B: uninfected/untreated control
C: infected/treated with 3.5mg/kg of sequzene
D: infected/treated with 7.0mg/kg of sequzene
E: uninfected/treated with 3.5mg/kg of sequzene
F: uninfected/treated with 7.0mg/kg of sequzene
PP Tb = Pre-Patent period for *Trypanosomabruceibrucei*

V. Discussion

The study shows that the infected groups (A, C and D) presented physical signs characterized by pyrexia, weakness, anorexia, emaciation, increased respiration, alopecia and corneal opacity. However, following treatment in group C (infected treated with sequzene at 3.5mg/kg) and group D (infected treated with sequzene at 7.0mg/kg), these signs were reduced or even reversed suggesting an attempt by the administered drug to restore cellular functions to its pre-infection status. These reversals can be attributed to the efficacy of diminazene diaceturate in the treatment of Trypanosomosis [23 and 14].

In this study, standard doses of the inocula were administered and uniform pre-patent periods were encountered, which shows that the initial parasite replication rate were similar irrespective of the host susceptibility. Similar observation have been reported in *T.brucei* infection in dogs [24] and red fronted gazelles (G. rufifrons) [13].All infected rabbits became pale looking , a sign of anaemia at 7 days of post infection in conformity with the observations made at the pre-patent period of 5-10 days [16]. Similarly, the pre-patent period of 7days observed among the rabbits were in consonance with the reports of [23] and [26]. However haemolytic anaemia and pre-patent periods in most cases often depends on the species of Trypanosomes involved and the route of inoculation of the parasite [25]. A significant decline in red blood cell count (RBC), packed cell volume (PCV) and haemoglobin concentration (Hb) parameters were observed in the infected groups (A, C and D). This was indicative of anaemia which started at the onset of the infection, from day7 (P.I). This is in agreement with several reports that anaemia in trypanosomosis often started during the 1^{st} wave of infection which is haemolytic in nature [23; 27 and 4]. However the haecmolytic nature of the anaemia in most cases will depend on the species of trypanosomes involved [4].The expanded and active mononuclear phagocytic system (MPS) has been a major player in haemolytic anaemia in trypanosomosis through erythrophagocytosis which developed soon after infection and continued thereafter, in the various phases of the disease [4]. The presence of MPS might have been associated with increased demand on the system to remove dead red blood cells, tissue cells, trypanosome antigen-antibody complexes and to participate in immune response [24]

The infected rabbits also exhibited leucopenia which was indicative of immunosuppression, similar to observations in *T.evansi* infected in camels by [6].

The lower counts of WBC, lymphocyotes and neutrophilis observed in this work were in agreement with investigations in rodents infected with trypanosomosis by (1). Lymphopaenia encountered was probably associated with the increased demand on the system for lymphocyte which is a common requirement in both immune and inflammatory response in trypanosomosis [27]. Meanwhile, the significant neutropenia encountered among the infected rabbits might be associated with splenic sequestration of leucocytes which was often suppressed by the raising of neutrophil numbers [24]. The infected rabbits however, showed significant monocytosis, this has been reported as a common feature of tissue macrophages likely due to increased demand on the system to remove dead red cells, antigen/antibody complexes and to participate in immune responses since macrophage are formed from blood monocytes . The increased need for macrophages may have been responsible for the consistent monocytosis encountered in this study.

VI. Conclusion

From the study, it could be concluded that *O. cuniculus* is susceptible to *T.brucei brucei* infection which might serve as carriers of infection to other domestic animals reared by the farmers .The trypanosomes had deleterious infects on haematological parameters of the rabbits . A single dose of Diaminazene diaciturate at 3.5 mg/kg and 7.0 mg/kg was promising in ameliorating the damaging effects of *T.brucei brucei* on RBC, WBC and Hb of New-Zealand rabbits.

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