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Short Communication

PATHOGENICITY OF *TRYPANOSOMA BRUCEI* IN AFRICAN GIANT RATS (Cricetomys gambianus, Water House)

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SUMMARY

The course of trypanosomosis was investigated over a period of two weeks in six African giant rats (*Cricetomys gambianus)* experimentally infected with *Trypanosoma brucei.* Six other rats served as uninfected control. The rats were each infected intraperitoneally with 0.2mls of blood containing approximately $2.0x10^5$ *T. brucei* parasites. The prepatent period varied between 3 and 8 days post infection. The course of the disease was acute (6-8 days post infection). The mean rectal temperature of the infected group was not significantly different (P>0.05) from the control group, while the mean values of packed cell volume of the infected rats declined significantly (P<0.05) compared to the pre-infection and control values. The findings in this study are suggestive of susceptibility of the African giant rats to *T. brucei.*

Keywords: *Trypanosoma brucei*; giant rat pathogenicity; aracmia

INTRODUCTİON

Trypanosomosis due to *Trypanosoma brucei* is one of the most important tsetse-borne diseases which impede increased productivity of livestock in African (Ikede, 1972) and causes pathological damage in cattle, sheep and goats (Isoun and Anosa, 1974). Pathological studies on ruminant trypanosomosis have been subject of intensive research (Saror, 1980; Sekoni 1992; Ikede, 1979; Losos and Ikede 1972). However, detailed pathology of the infections and behaviour of the trypanosomes in the tissues of wild animals are not too well known. One of the few reports was on the susceptibility of the African giant rats to *Trypanosoma evansi* (Lariviere, 1961). A study of the behaviour of *T. brucei* in African giant rats is important considering efforts being made at domesticating giant rats to augment meat protein especially in the Western part of Nigeria (Ajayi, 1975). Giant rats are very successful terrestrial mammals with high reproduction and breeding potentials. In view of the mechanical transmission of trypanosomes, the productivity of the rats might be impeded.

The present study was to investigate the pathogenicity *T. brucei* isolate in African giant rats.

MATERİALS AND METHODS

Animals and experimental infection

Twelve intact adult male and female African giant rats (*Cricetomys gambianus*) were captured from the wild in Samaru Zaria either by physically digging them out of their burrows or wooden and iron traps were used. They were subsequently kept in cages and housed in a fly proof unit in the Department of Biological Sciences, Ahmadu Bello University, Zaria and conditioned to captivity for 4 weeks before the experiment commenced. They were

maintained on potatoes, vegetables and groundnut cake. Water was supplied *ad libitum*. A drop of blood was collected from the tail of each rat and examined for haemoparasites using thin smear preparations which indicated that the rats were free of haemoparasites. Six giant rats were non-infected control group 1, while 6 others were the experimentally infected group II. The rats were tagged for the purpose of identification.

Trypanosomes

Trypanosoma brucei used in this study was originally isolated from the blood of infected Swine in Zaria, Kaduna State, Nigeria. The parasite was obtained in the laboratory by serial passages in Swiss albino mice until required. The blood samples were obtained in sterilized bijou bottles containing EDTA. The blood was diluted 1 in 2mls of phosphate buffered saline glucose prior to inoculating the rats. Each of the infected rats received approximately $2.0x10^5$ parasites as estimated using the improved Neubauer haemocytometer method (Petana, 1963). The parameters determined during the pre- and post- infection were the packed cell volume (PCV) using the Haematocrit Centrifuge Technique (HCT) (Woo, 1969), serum protein using the hand refractometer . These parameters were determined at 2 days intervals. Rectal temperature was daily taken using the clinical thermometer. The inoculated rats were allowed to go through full course of the infection without treatment.

Parasitaemia was scored as +,++,+++ and massive (representing 1-5, 6-10,11-20 and more than 20 trypanosomes per microscopic field) at X 100 magnification. The rats were observed daily for any abnormal behaviour. All rats that died as a result of the infection were immediately followed by postmortem examination. Similarly, postmortem examination was conducted on one randomly selected control rat. Gross pathological examinations of various organs were carried out on the carcasses

Data analysis

The data obtained were summarized as means ± standard deviation. The means were compared statistically using the Student's t-test. (Wishart and Sanders, 1958).

RESULTS

All the infected rats developed parasitaemia between days 3 and 8 post-infection (PI) with a mean prepatent period of 4.23± 0.18 days. The parasitaemia in the infected rats increased progressively, and fluctuated between days 5 and 8 post-infection. One of the infected rats died by day 6 post-infection, 3 died day 7 PI and 2 died by day 8 PI. Anaemia was a consistent feature of the disease as the mean PCV of the infected rats declined significantly ($P<0.05$) from 43.3± 1.12% on day 2 to 15.0± 0.02 on day 7 (Fig 1). The pre-infection mean serum protein in the control and infected rats remained fairly constant within the normal range. However, a non-significant ($P>0.05$) decrease occurred in the infected group between days 3 and 8 post-infection.

The mean live body weight of the control rat dropped slightly from the pre-infection value of 1.08± 0.16kg to 0.96 and increased through out the duration of the experiment. The mean live body weight of the infected rats decreased from the pre-infection mean values of 1.12± 0.7kg to 0.88± 0.01 on day 4 and later up to the point the rats died. The infected rats lost an average of 21.4% body

weight by the end of the experiment. Observed clinical signs were rough hair coat, weakness and loss of weight. Postmortem examination of giant rats that died as a result of the infection showed anaemia as indicated by paleness of the tissues, congested lungs with evidences of splenomegaly, enlarged adrenal glands, hepatomegaly, congested blood vessels and haemorrahage.

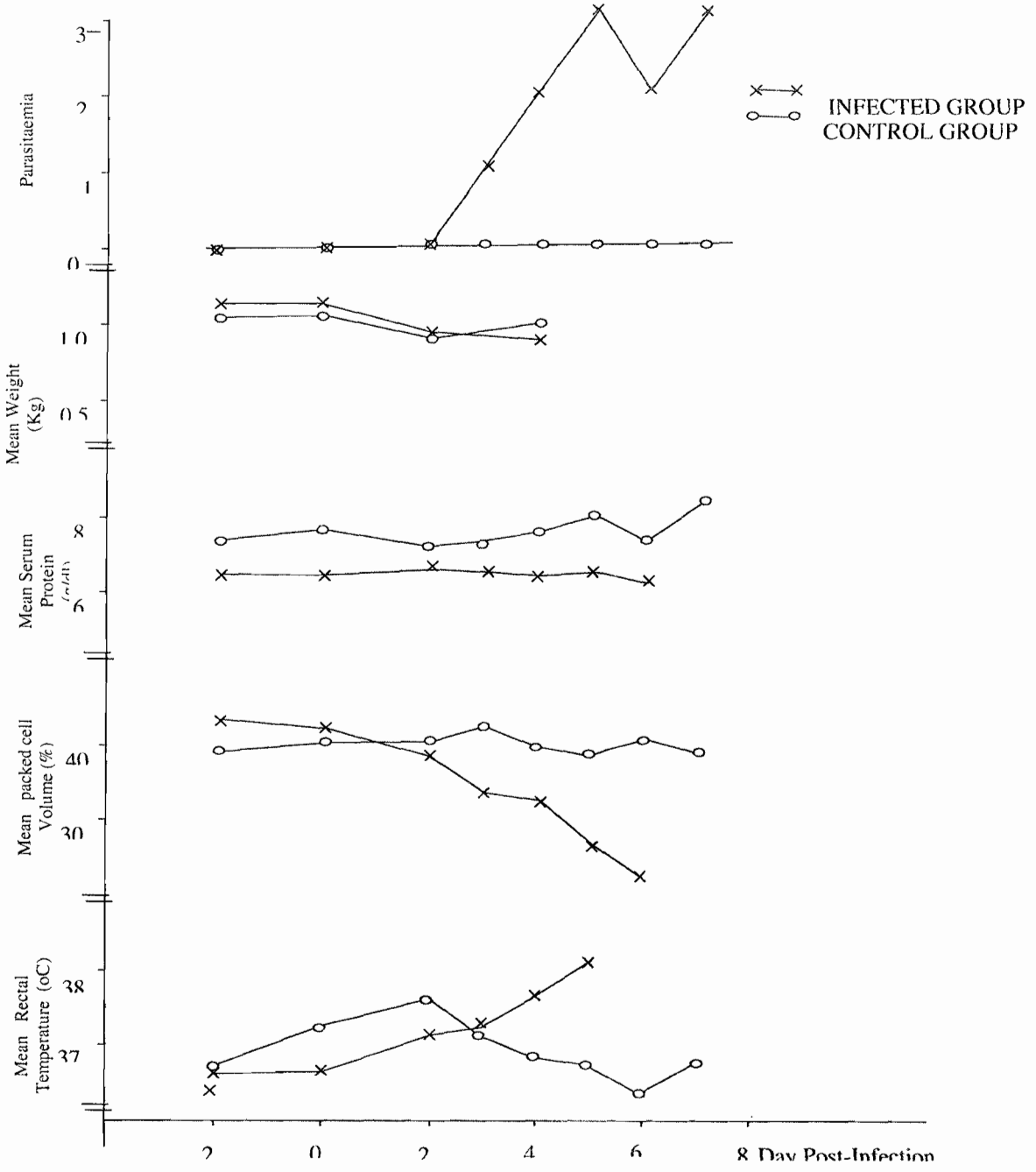

Fig. 1 Mean Rectal Temperature, Packed Cell Volume. Total Serum Protein, Body Weight and Parasitaemia of infected and non-infected Giant Rats

DISCUSSION

In this study, it is evident that *T. brucei* is pathogenic for African giant rats. The degree of pathogenicity is similar in all the infected rats. The giant rats developed acute trypansomosis and died within 8 days PI. Postmortem examinations revealed that in the entire infected giant rat. *T. brucei* evoked similar pathological lesions in the spleens, liver and kidneys. This might indicate that *T.brucei* caused irreparable damages in the rats as reported for albino rats (Egbe *et al.,* 2003) and domestic animals (Waisa *et al.,* 2003a). It would appear that the grade of parasitaemia affected the ultimate course of the disease in the rats by accelerating the progress to death. It was also observed in the infected rats that, temperature peaks corresponded with parasitoemic rises suggesting that the elevated temperature was induced by an antibody-antigen reaction (Edwards *et al;* 1956, Audu *et al* 1999).

The post infection anaemia was characterized by decreased in the pack cell volume which coincided with the patency of the infection. This might suggest that the presence of live trypanosomes is necessary for the development of anaemia (Omotainse and Anosa, 1992; Egbe *et al*; 2003, Waisa *et al*; 2003b). The actual mechanism for the development of anaemia in the present study was not investigated. However, hypothesis on this include the role of specific immunoglobulin against trypanosomes to form complexes with antigens and complement on the surface of red blood cells leading to their sequestration and destruction in the reticulo endothelial system as reported for rabbits infected with *T. brucei* (Dodd *et al.,* 1978). Other possible factors include the role of 'non-specific haemolytic factors' which might facilitate the destruction of red blood cells by macrophages (ILRAD, 1984.)

The serum protein concentration of infected rats was slightly lower than the control group. This might suggest an increase in protein break down or urea loss (Ikede and Losos, 1972) or under utilization of the feed provided due to the presence of the parasites (Ngeranwa *et al.*, 1992).
The fall in the body weights of the infected rats is an indication of the wasting nature of trypanosomosis (Sackey, 1998).

Occurrence of haemorrhages in the organs of infected rats might be responsible for the appearance of cell congestion in the spleen, liver and lungs. These changes which occurred within a few days post infection might be of immense significance in the light of anaemia associated with trypanosomes (Karram *et al*; 1991), and could be of special importance in the immunology and therapy of trypanosomosis (Godwin, 1990).

The findings in the present study have shown that the *T. brucei* used in this study is infective for the giant rat and could have very important economic implications to the livestock industry in Nigeria. Giant rats are becoming more and more popular for their meat particularly in Western Nigeria where its domesticaltion is being encouraged. The *T. brucei* used in this study originally came from the blood of Swine, as such possibility of infection becoming important in giant rats should be considered in the effort at domesticating the rats.

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