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## Studies on experimental infection of pigs with *Trypanosoma brucei*

Short communication

W. E. AGU, Z. T. BAJEH

*Trypanosoma simiae* causes very virulent and fatal infection in pigs (Unsworth, 1952). The other trypanosome species, *T. brucei* and *T. congolense*, are thought to cause only mild infections in the pig (Hoare, 1936). Recently, however, an outbreak of fatal *T. brucei* infection of pigs in Mkar, Benue State, Nigeria has been reported (Agu and Bajeh, in press). The present report describes clinical and haematological studies carried out on piglets experimentally infected with a *T. brucei* stock (MKAR/84/NITR/6) isolated from the outbreak. The organism was identified as *T. brucei* based on rat inoculation, morphology, negative BIIT result and biologically by tsetse transmission with the parasites invading salivary glands of *Glossina palpalis palpalis* and developing metacyclic forms.

Eight large-white piglets aged between 4 to 5 months were used for the study. They were housed in concrete pens and fed on concentrates and vegetables. Six of the piglets (Nos. 1, 2, 3, 4, 6, 7) were each inoculated with approximately  $3 \times 10^6$  parasites contained in 2 ml of infected rats' blood, by the intramuscular route. Two piglets (Nos. 5, 8) were left as uninfected controls. The body temperature of all the piglets was taken daily, and their peripheral blood was also examined for the appearance of trypanosomes by wet film examination. They were also clinically examined daily. White (WBC) and red blood-cell (RBC) counts as well as packed cell volume (PCV) were estimated weekly on blood taken from the anterior vena cava of control and infected animals, using EDTA as the anticoagulant. Thin films were also made weekly and were stained with Giemsa for differential WBC count.

Trypanosomes appeared in the peripheral blood of the infected piglets 3 to 4 days post inoculation and then produced undulating parasitaemia for several weeks. Intermittent pyrexia accompanied the peaks of parasitaemia with the temperature reaching 104.8°F at times. Clinically the infected piglets became progressively emaciated. They were off-feed particularly at heights of parasitaemia. Some of them showed nervous signs like the incoordination of hind limbs. At the terminal stages, they were recumbent and exhibited paddling of the limbs. There was profuse subcutaneous haemorrhage in the flank, abdomen and the ears. 5 out of 6 infected piglets died of the infection within 70 to 138 days after patent infection while one piglet (No. 7) had a self-cure of the infection.

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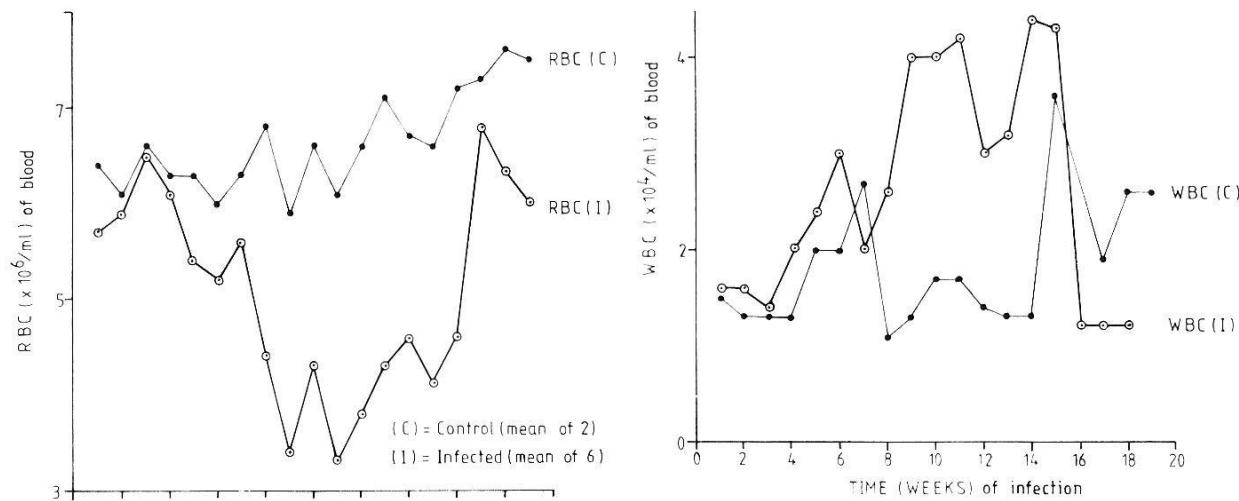


Fig. 1. Effect of *T. b. brucei* on RBC and WBC of pigs.

The changes in weekly values of RBC, WBC and PCV in both the control and infected piglets are given in Fig. 1. There was a drop in both PCV and RBC as from 4th and 5th week post infection, respectively. With the exception of the piglet that underwent self-cure, the values of both parameters remained low throughout the rest of the course of infection. Statistically significant changes in WBC counts, both total and differential, were not observed as measured by Student's "t" test.

The present study has shown that the *T. brucei* stock isolated from a natural outbreak of trypanosomiasis in pigs is pathogenic to this host species. Piglets experimentally infected with this trypanosome stock developed anaemia as demonstrated by the reduction in PCV and RBC, severe clinical signs and death in 5 out of 6 infected animals. The factors responsible for the pathogenicity of this stock to pigs are not known. The terminal symptoms shown by some of the infected piglets indicated involvement of the brain. Such nervous signs as swaying-gait was noted in *T. brucei* infections in pigs (Stephen, 1966). The histopathology of the brain of piglets infected with the same stock of *T. brucei* as in the present study, showed extensive interstitial and perivascular accumulation of mononuclear cells (Agu and Bajeh, in press). Thus *T. brucei* could be more pathogenic in pigs than hitherto believed.

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