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Study on the infectivity of *Trypanosoma evansi* isolate in Savannah Brown bucks

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Abstract

The infectivity of an isolate of *Trypanosoma evansi* was investigated using five infected and four uninfected control Savannah brown bucks. The goats were each infected intravenously via the jugular vein with approximately 3.0 x 106 *T. evansi* parasites. Following the infection, clinical signs observed, parasitaemia, haematological parameters were measured for 59 days. Body weights were determined once in a week for the duration of the experiment. All infected bucks developed clinical trypanosomosis. The prepatent period varied between 7 and 11 days while the rectal temperatures were significantly higher ($p<0.05$), the mean values of haematological parameters dropped significantly ($p<0.05$) compared to the pre-infection levels. Clinical signs observed in this experiment included dullness, emaciation, recumbency and anaemia. It is suggested that the isolate was pathogenic and infective for Savannah brown bucks.

Keywords: Infectivity, Savannah Brown bucks, *T. evansi*

Introduction

Trypanosomosis caused by *Trypanosoma evansi* (surra) has been documented all over the world for more than 100 years (Indrakamhang, 1998). The disease is important in camels and other domestic animals (Hoare, 1956). It is mechanically transmitted by different vectors including biting flies (*Tabanidae*, *Stomoxys*, etc.) and vampire bats (*Desmodus rotundus*) (Hoare, 1972).

In Nigeria, and indeed Africa, camels are increasingly gaining economic importance particularly in the northern part of the country, where they are being used as a source of meat (Falope, 1991) and in transportation. Animals, especially sheep and goats herded together with camels are likely to be infected with *T. evansi* (Ngerenwa *et al.*, 1993). For many years trypanosomosis research in Nigeria has been focused on tsetse transmitted infections with little attention to mechanically transmitted. *T. evansi* because of the central role played by tsetse flies in the transmission of the disease (Audu *et al.* 1999). *T. evansi* is known to be pathogenic to different animal species in different parts of the world (Mahmoud & Gray, 1980).

With increase in consumption of camel meat and as means of transportation in Nigeria, the risk of other livestock species getting infected with *T. evansi* are becoming more likely, especially sheep and goats that are usually herded together with camels. It is hoped that a good understanding of the parasite behaviour in animals herded together with camels would provide a framework for control measures to protect camels and other susceptible hosts. This present study was therefore designed to study the infectivity of an isolate of *T. evansi* in Savannah brown bucks.

Materials and Methods

Isolation of trypanosomes

The *T. evansi* isolate used in this study was obtained from the blood of a naturally infected camel slaughtered at the Kano abattoir, in northern Nigeria. The blood samples were obtained in sterilized Bijou bottles containing ethylene diamine tetra-acetic acid (EDTA), and transported on ice to the Department of Veterinary Parasitology and Entomology, Ahmadu Bello University, Zaria. The blood samples were immediately examined using wet smear, thin smear and haematocrit centrifugation technique (Murray *et al.* 1977). Ten albino rats were inoculated with the infected camel blood in order to harvest the trypanosomes in sufficient numbers for subsequent infection of the experimental goats.

Experimental Animals and Procedures

Nine Savannah brown male goats aged between 12 and 18 months were obtained locally and conditioned for a period of six weeks. They were screened against the common haemoparasites and helminth infections prior to the experimental infection. The animals were kept in fly-proof pens. The animals were fed grass-hay, concentrates consisting of cottonseed mixed with grain offal, salt licks and water *ad libitum*. The parameters monitored during the period of pre- and post- *T. evansi* infection were daily rectal temperature (°C) using manual

thermometer, body weights taken once a week using a scale (Hana BR-9011), packed cell volume (PCV), haemoglobin concentration, leukocyte count as described by Schalm*et al.* (1975). Total protein was measured using hand refractometer. At the end of six weeks of conditioning, the goats were randomly divided into two groups. Four goats constituted the non- *T. evansi* infected control group and the remaining five goats formed the *T. evansi* infected group. When the *T. evansi* infected rats developed parasitaemia of 3+ (i.e. 20 trypanosomes per field (X40) for three consecutive days, they were bled and the pooled blood in EDTA was subsequently diluted with phosphate buffered saline glucose (PSG) prior to inoculation into the experimental goats. Each of the goats in the infected group was intravenously inoculated via jugular vein using 1ml of the infected rat blood containing approximately 3 x 10^6trypanosomes as estimated using the rapid matching method of Herbert & Lumsden (1976). The goats were daily observed for abnormal behaviour. The inoculated goats were allowed to go through the full course of the infection. Post mortem examinations were conducted on infected animals that were *inextremis* or have died of the infection.

Data Analysis

Data obtained from the infected and control groups during the present study were summarized as means (± SD). The collected data were compared statistically using student t-test as described by Gomez & Gomez (1984).

Results

In this study all goats belonging to the infected group developed parasitaemia. The prepatent period varied between 7 and 11 days post infection.

There were variations in the intensity of parasitaemia between and within each of the infected group. There was no correlation between parasitaemia and rise in rectal temperatures (Fig.1). The parasites were detected even during the afebrile period.

Four of the infected goats died of the infection, while the remaining one was able to survive the infection.

The *T. evansi* infected goats developed moderate levels of anaemia as evident by gradual fall in PCV and haemoglobin concentrations, which also coincided with fluctuating parasitaemia (Fig. 2).

The weight of the infected goats was also found to have decreased with infection.

Post mortem examination of the goats that died as a result of infection showed pale carcasses with atrophy of body fats, congestion of liver, lungs and kidney, enlarged lymph nodes and catarrhal enteritis.

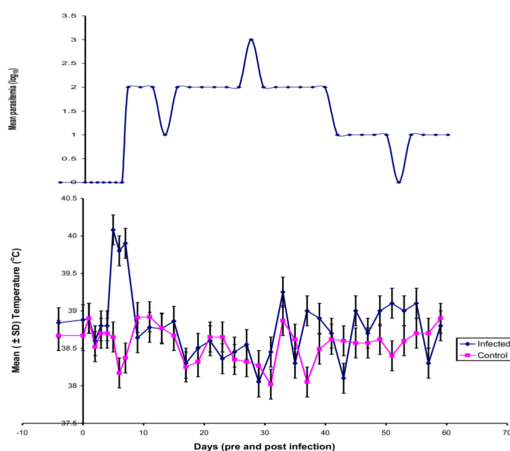

Figure 1: Mean parasitaemia in relation to rectal temperature in Savannah brown Bucks

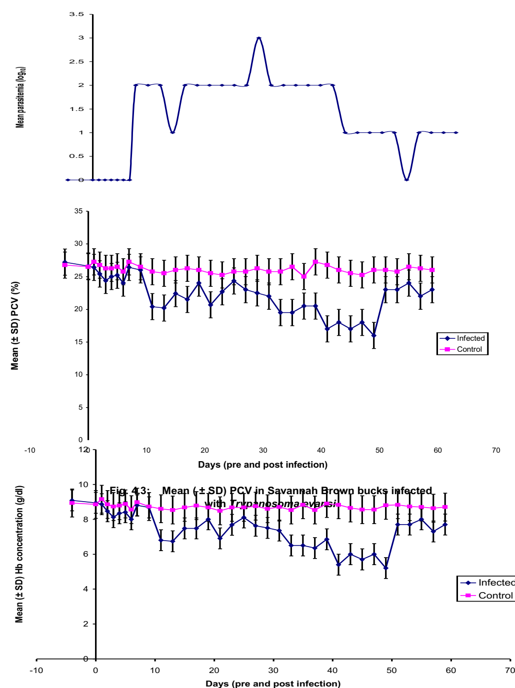

Figure 2: Mean PCV, Haemoglobin concentrations in relation to parasitaemia in Savannah brown Bucks

Discussion

The prepatent period following inoculation of the goats with *T. evansi* ranged between 7 and 11 days. This is in line with the reports of Verma & Gautam (1978) for *T. evansi* infected buffaloes and cow calves and that of Stephen (1986) for *T. evansi* infected goats. However, it appeared longer than what was reported by Audu *et al.*, (1999) for *T. evansi* infected Yankasa sheep. The intensity of parasitaemia ranged between 1+ (10^3) and 3+ ($5x10^4$) with the highest levels of parasitaemia recorded during the early phase of the infection. This pattern was also reported by Anosa & Isoun (1980) for *T. vivax* infected small ruminants.

The *T. evansi* infected goats developed moderate levels of fall in PCV and haemoglobin concentration. This coincided with fluctuating parasitaemia, which suggested that living trypanosomes were responsible for the progressive development of anaemia as previously observed by Ogbadoyi *et al.* (1999).

The mechanism for development of anaemia in the present study was not investigated. However, it could be due to bone marrow defects, immunological reactions, erythrophagocytosis, haemorrhagic syndrome, severe haemolysis (Murray & Dexter, 1988) and perioxidative injury to erythrocytes (Igbokwe *et al.*, 1994).

The observed pyrexia in the present study indicates metabolic disorder due to the presence of circulating trypanosomes which could play a significant role in haemolysis and consequently fall in PCV as earlier reported by Audu *et al.* (1999). The fall in total plasma protein concentrations in infected bucks in this study is in agreement with the reports of Losos& Ikede (1972) and Audu *et al.* (1999) for *T. evansi* infection in different domestic animals. The low protein levels may be adduced to loss of blood associated with anaemia, it could also be as a result of increased protein breakdown or urea loss and haemodilution.

The loss of weight observed in the infected bucks was earlier reported by Sackey (1998) for T. vivax infected bucks and Audu *et al.* (1999) for *T. evansi* infected Yankasa sheep, indicating the wasting nature of the disease.

The results of this study have conclusively shown that *T. evansi* isolate used was infective to the Savannah brown bucks.

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