

Short Communication

Effect of corticosteroid administration on the infectivity of *Trypanosoma brucei* (8/18) in the Nigerian domestic chickens (*Gallus Gallus Domesticus*)

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Abstract

This study was aimed at determining how the infectivity of *trypanosoma brucei* could be affected by steroid administration to the local chickens. It has been shown that Nigerian domestic chickens were susceptible to *Trypanosoma brucei* (8/18). In the course of this study, the *Trypanosoma brucei* (8/18) was found to have lost its infectivity to the local chickens. Even steroid administration at both low (0.2mg/ml) and high (1.0mg/ml) dosage and prolonged administration of the steroid could not change this occurrence. The study thus concluded that serial passage of *T. brucei* (8/18) over a time will make it lose infectivity to Nigerian domestic chickens (*Gallus gallus domesticus*).

Keywords: *T. brucei*, *Trypanosomiasis*, *Gallus gallus*, Dexamethazone

Introduction

Trypanosomiasis has been intensively studied in recent times with the aim of finding a cure as well as immunoprophylactic approach to the control of the disease. Efforts are also being made to understand sufficiently the details of the biology of trypanosomes, the causative agent of the disease (Losos and Ikede, 1972; Jennings, 1976; Tabel, 1978; Kalu and Agu, 1984; Kalu *et al.*, 1991; Ahmed and Agbede, 1993; Daniel *et al.*, 1994; Anosa *et al.*, 1995; Kalejaye *et al.*, 1995; Onyia, 1997).

Another approach to the study of the disease is its study in abnormal hosts. A number of workers (Desowitz, 1963; Goedblood, 1972; Josuha, 1979) have carried out extensive studies on trypanosomiasis in domestic chickens. Domestic chickens (*Gallus gallus domesticus*) infected with

a stock of *Trypanosoma brucei* exhibited a chronic infection that was spontaneously terminated by self cure. The local chickens were then found to be immunized to challenge with derivatives of the same trypanosome stock.

It was suggested that the ability of the birds to cure the infection and retain immunity might be due to the particular efficient nature of their immune system. These results encouraged the view that it may be possible to design effective trypanosome vaccines for use in animals (Joshua, 1983).

A study involving inoculation of local Nigerian chickens with *T. brucei* (8/18) and *T. vivax* (Y58) showed that these chickens did not develop a clinical disease but rather they all continued to feed and increase in body weight (Dina and

Arowolo, 1988). This study also revealed that chickens infected with both trypanosomes did not show parasitaemia for 4 weeks post inoculation.

Dexamethasone, a glucocorticoid, inhibits the release of inflammatory mediators from macrophages and eosinophils but do not inhibit the release of granules from mast cells. Glucocorticoids also decrease synthesis of prostaglandins, leukotrienes, and platelet-activating factor, which play important roles in the pathophysiology of respiratory tract diseases. Glucocorticoids are also known to have immunosuppressive effects, hence are generally avoided in infectious respiratory diseases (Dowling, 1998). Glucocorticoids also suppress both inflammatory and immunological responses and thereby attenuate associated tissue destruction and fibroplasia. Inhibition of a number of lymphocyte functions form part of the basis for immunosuppression (Bryette, 1998).

It was thought that using dexamethasone (corticosteroid) to suppress the cell-mediated immunity in the domestic chickens; one will be able to evaluate the infectivity of *T. brucei brucei* (8/18) in local chickens.

Materials and Methods

Experimental Animals

20 local chickens aged 8 – 10 weeks were brought from 2 different locations in Ibadan and were acclimatized for 4 weeks at the Experimental Animal House of the Faculty of Veterinary Medicine, University of Ibadan. During this period the animals were given antibiotic and anthelmintic (Pyrantel pamoate {Canex[®]- Pfizer Agricare Pty Ltd., Australia} at 5mg/kg body weight) treatment for 3 – 5 days. Feed and water were also given *ad libitum*.

20 albino mice were also purchased at the Animal House, faculty of Veterinary Medicine University of Ibadan for xeno-diagnosis of trypanosomosis. Infected blood containing

Trypanosoma brucei brucei was collected from infected mice.

Trypanosoma brucei brucei 8/18

A strain of *Trypanosoma brucei* (8/18) was obtained from the experimental animal house of the Faculty of Veterinary Medicine, University of Ibadan where it has been passaged and maintained in laboratory mice and rats. The strain was first isolated from a pig at Nsukka, Nigeria in 1962 and brought to the university of Ibadan in 1990.

Drugs

10 ampoules of 4 mg/ml of dexamethazone (Cortisol[®]) were used in the course of this study. They were given intramuscularly (i/m). Pyrantel pamoate {Canex[®]-Pfizer Agricare Pty Ltd., Australia, an anthelmintic and neomycin (antibiotic) were given separately to the local chickens during acclimatization. Each administration lasted 5 days.

Experimental design

4 experiments were carried out in the course of this study.

Experiment 1

12 local chickens were divided into 3 groups (A, B, C) of 4 birds each. 0.2 mg/ml of dexamethazone was administered for 10 days to the animals in group B intramuscularly (i/m). each chicken in groups A and B was infected with about 2.5×10^4 trypanosomes intraperitoneally i/p on day 6 of the experiment. Group C served as uninfected control.

From day 6 – 14 post infection of the birds, blood was collected from the wing veins of all the birds and examined for parasitaemia using x40 objective lens. At day six post infection of the chickens, blood collected from the infected chicken was inoculated intraperitoneally into mice. The blood of the mice was examined for parasitaemia, 3 – 6 day post inoculation with chicken blood.

At day 13 post inoculation of the chickens, blood samples were also collected and administered

intraperitoneally to the mice. Subsequently, parasitaemia was examined in the mice 3 – 6 days post inoculation with chicken blood.

Experiment II

The 12 local chicken used in experiment I were used for this experiment after two weeks of the first experiment. They were divided into 3 groups (A,B,C) of 4 birds each. 0.4 mg/ml of dexamethazone was given to chickens in group B intramuscularly for 7 days. Thereafter chickens in groups A and B received approximately 5.0×10^4 trypanosomes intravenously (i/v) through the wing vein. Group C animals however served as uninfected control. From day 6 to 14 post infection of the chickens, blood samples were collected from the wing vein of all birds and examined for parasitaemia. Also at day six, post inoculation of the birds, the blood samples collected from all the chickens were administered intraperitoneally to mice. The blood of the mice was examined 3 – 6 days post inoculation for parasitaemia using x 40 objective lens.

Blood was collected from the infected chickens at day 11 post infection and inoculated into mice. The inoculated mice were thereafter screened for parasitaemia 3 – 6 days post inoculation.

Experiment III

This procedure was similar to that of the previous experiment except that 1 mg/ml of dexamethasone was administered into group B animals for 10 days. Also approximately 5.5×10^5 were inoculated into chickens in groups A and B with group C serving as uninfected control.

It needs be stressed that simple wet smear was performed while enumerating trypanosomes in the course of this study. This involved obtaining blood from the wing veins of all the chickens and placed a drop on a microscope slide and then examined for parasitaemia using X 40 objective lens.

Experiment IV

8 chickens were used in this experiment and the birds were each injected with approximately 1×10^6 trypanosomes intravenously (i/v) through the jugular vein. The infected birds were screened for parasitaemia 7 – 20 days post infection blood from the infected birds were inoculated into mice which was later at 3 – 6 days post inoculation screened for parasitaemia.

Results

- The results of this study showed that the blood samples of all chickens and laboratory mice revealed no parasitaemia. Even when the dosage of dexamethazone was increased and the duration of administration prolonged, no parasitaemia was revealed. Infact, the concentration of the trypanosomes was increased with each experiment yet no parasitaemia was observed both in the chickens and the mice.

Discussion

The results of this study showed that the local chickens were not susceptible to trypanosomal infection. It is therefore consistent with the findings of Goedblood (1972) who reported that infection of chickens with trypanosomes through the intra-peritoneal route showed no infectivity. In fact xeno-diagnosis using laboratory mice revealed no parasitaemia confirming lack of infectivity.

The result of this study is however, a contrast of earlier work carried out in local chickens using the strain of *T. brucei brucei* (8/18). Dina and Arowolo (1988) showed that chickens inoculated with *T. brucei* or *T. vivax* did not develop a clinical disease but rather they all continued to feed and increase in weight. Though these chickens infected with *T. brucei* or *T. vivax* showed no parasitaemia under the microscope for four weeks post inoculation, all laboratory mice infected with the blood of *T. brucei* infected chickens died after intensive parasitaemia. It thus means that the birds were susceptible to *T. brucei*.

The fact that these chickens did not show clinical manifestations of trypanosomosis means they must possess special mechanisms for controlling the disease since infected blood of the chickens inoculated into laboratory mice produced intense parasitaemia in mice. Joshua (1983) suggested that the immune response of the chickens (notably the Bursa ad Fubricus) was responsible for this. In fact Oyewale (1987) reported that lymphocytes levels of local Nigerian chickens were quite high when compared to commercial stock tested in the same environment suggesting a cell-mediated immunity. This is thus proposing a similar hypothesis for the mechanism of resistance to infection exhibited by N'dama cattle when compared to the Boran cattle.

Dexamethazone like other glucocorticoids are used in hormonal therapy for neoplasia in veterinary medicine because their direct antitumor effects are related to their lympholytic properties, inhibit mitosis, RNA synthesis, and protein synthesis in sensitive lymphocytes (Kochevar, 1998). In this study, chickens that were administered with dexamethasone prior to trypanosome inoculation neither showed any parasitaemia nor clinical manifestation of trypanosomosis implying that *T. brucei* 8/18 could not initiate infection in the chickens even after the immune status of the chickens were compromised by the administration of the dexamethasone. The same stock of trypanosomes had caused sub-clinical infection in Nigerian domestic chickens (Dina and Arowolo, 1988). This species of trypanosomes perhaps have lost infectivity in the chickens following continuous passage in laboratory animals.

It should be noted that *trypanosome brucei brucei* 8/18 was first isolated from a pig in 1962 and have since been maintained by passage in rats, sheep, cryo-preservation and laboratory mice. Incidentally, this stock of *T. brucei* 8/18 was used to show sub-clinical infection in Nigerian local chicken to trypanosomosis. The result of these

experiments suggests that serial passage of *T. brucei* 8/18 over a long period of times makes them loose infectivity to Nigerian domestic chickens. This therefore supports the work of Desowitz (1963) that a loss of ability to infect the anthropoid host, and also to undergo cyclical developments in the same are features of cyclical transmitted forms of trypanosomes that have been passaged continuously by mechanical means.

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