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Effects of dietary selenium supplementation on parasitemia, anemia and serum proteins of *Trypanosoma brucei brucei* infected rats

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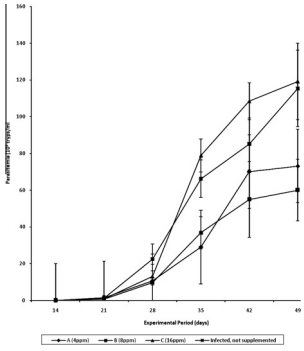
HIGHLIGHTS

- Supplementation was able to, reduced anemia.
- Protect the protein, albumin and globulin.
- Increase the pre-patent period.
- Reduction in the parasitemia levels.
- Increased survival intervals.

GRAPHICAL ABSTRACT



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ABSTRACT

Trypanosomosis has been associated with immunosuppression, anemia and oxidative damage while selenium possesses both immunostimulatory and antioxidative effects. This study was designed to assess the effect of dietary selenium supplementation on parasitemia, anemia, survival pattern and serum protein profiles of trypanosome-infected rats. Twenty five rats, divided into five groups (A–E) of 5 each, were treated as follows: 4, 8 and 16 ppm (ppm) of selenium in their feed, respectively throughout the experimental period and were infected with *Trypanosoma brucei brucei* on day 14 post supplementation, infected not supplemented and the negative control. Supplementation at 4 and 8 ppm increased the packed cell volume (PCV) and hemoglobin (Hb) concentration on day 7 of supplementation (PS) when compared with the unsupplemented groups. Following infection on day 14 PS, the PCV, Hb of 16 ppm and infected not supplemented groups were significantly ($P < 0.05$) lower than other groups on days 28 and 35 PS. Supplementation did not lead to significant ($P > 0.05$) changes on the total protein, albumin and globulin by day 14 PS. Infection, however, caused significant ($P > 0.05$) decrease in the total protein and albumin from day 28. The supplementation did not significantly ($P > 0.05$) increase the pre-patent period but caused a significant reduction in the parasitemia levels and increased survival intervals. Dietary selenium supplementation, from the results, may show promise in the management of African trypanosomosis as the supplementation was able to: reduce anemia and parasitemia and increase survival intervals of trypanosome infected rats.

1. Introduction

Trypanosomosis is a debilitating as well as fatal tropical disease of livestock and man. It currently causes annual losses of about USD 1.5 billion and, over the long run, has had the effect of limiting

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Africa's agricultural income to about USD 4.5 billion a year, below its potential level (FAO, 2000). In addition, it is estimated that about USD 30 million per year is spent on prophylaxis and treatment. Thus, African livestock keepers are faced with serious challenges of controlling or reducing the impact of the disease. Controlling the disease has been directed towards vector control, chemotherapy and chemoprophylaxis and use of trypanotolerence breeds. Trypanocidal drugs remain the principal method of animal trypanosomiasis control in most African countries (Anene et al., 2001). However, the therapeutic and prophylactic use of trypanocides is hampared by numerous limitations such as toxicity, prohibitive cost and development of resistance by the parasites (Clarkson et al., 1984; Anene et al., 2001). Also, trypanosomosis has been associated with immunosuppression (Godwin et al., 1972; Murray et al., 1974; Osma et al., 1992), and induction of lipid peroxidation in the host (Eze et al., 2008).

Because of their presence in the blood, these invading parasites produce numerous changes in the cellular and biochemical constituents of blood (Igbokwe and Mohammed, 1992; Taiwo et al.,2003). Anemia, a common feature of trypanosome infections, is a complex process and remains unclear (Anosa and Kaneko, 1983). Increased red blood cell destruction, extravascular and intravascular hemolysis by immune system (trypanosome antigen/antibody complex and antierythrocyte antibodies), hemolysins, nonspecific reticuloendothelial system activation, direct traumatic effect of trypanosomes, microangiopathy associated with disseminated intravascular coagulation, splenic phagocytosis and splenic pooling, erythrophagocytosis, increased plasma volume, non compensatory and/or decreased erythropoiesis and anemia of chronic disorders are proposed as causes of anemia in acute and chronic African trypanosomosis (Amole et al., 1982; Jenkins and Facer 1985; Katugunka-Rwakishaya et al., 1995). The rate of development and recovery from the anemia is an indication of how resistant an animal is to the disease. The degree of anemia in trypanosomosis has been positively correlated with the onset and level of *T. brucei* parasitemia. Also, increase in parasitemia corresponds with rise in rectal temperature, rapid weight loss, packed cell volume decline and decrease in total plasma protein in all the infected animals. Susceptibility to trypanosomiasis depends on malnutrition, overwork, intercurrent infection, pregnancy, parturition lactation, stress and degree of parasitemia (Katugunka-Rwakishaya et al., 1995).

Also, pathological and biochemical changes are associated with trypanosome infection. This may be due to the presence of trypanosomes in the blood of the host, thus, producing numerous changes in the cellular and biochemical constituents of blood (Igbokwe and Mohammed, 1992; Taiwo et al., 2003). Hypoproteinemia, hypoalbuminemia and elevated serum alanine aminotransferase activity have been reported in trypanosome infections (Kalu et al., 1989; Adah et al., 1992). The onset of anemia, and the extent to which the packed cell volume fall, correlates closely with the appearance, level and duration of parasitemia (Luckins and Gray, 1978).

Selenium (Se), a trace element essential to man and animals plays a role as an antioxidant, providing protection against free radical damage and oxidative stress (Jelicks et al., 2011) and as an immunostimulant (Broome et al., 2004; Eze et al., 2011). Selenium deficiency in animal and humans is characterized by pathological changes including growth retardation, skin lesions and hair loss, visual defects, reproductive disorders, pancreas atrophy, liver necrosis and dystrophy of the skeletal muscle and of the heart muscle and increased immature erythroid cellular elements (Bartholomew et al., 1998). Selenium supplementation has been shown to increase antibody titre to sheep red blood cells in *Trypanosoma brucei* infected rats (Eze et al., 2011), tissue selenium concentration (Kim and Mahan, 2001) and potent antioxidant (Jelicks et al., 2011). Selenium supplementations have shown to ameliorate *Trypanosoma cruzi* (Chagas disease) in murine models (Davis et al., 1998; Gomez et al., 2002; Rivera et al., 2002; de Souza et al., 2003; de Souza et al., 2010; Jelicks et al., 2011).

The aim of this study was, therefore, to determine the effect of dietary selenium supplementation on parasitemia, anemia and serum proteins of *Trypanosoma brucei brucei* infected rats.

2. Materials and methods

2.1. Experimental animals

Twenty five (25) adult male outbred albino rats, weighing between 278–302 g, were used for the study. The rats were acquired from The Laboratory Animal Unit of the Department of Veterinary Pathology and Microbiology, University of Nigeria, Nsukka. The rats were housed in a fly-proof house and given feed and water *ad libitum*. A period of 10 days was allowed for acclimatization of the rats.

2.2. Trypanosome

Trypanosoma brucei brucei (Federe strain) used for this work was obtained from National Institute for Trypanosomosis Research (NITR), Vom, Nigeria. The strain was isolated from N'dama cattle from Federe village in Plateau State, Nigeria and has been maintained in liquid nitrogen at the NITR, Vom. The strain was passaged in rats from where the experimental animals were infected.

2.3. Selenium

Selenium as sodium selenite was manufactured by Biorganics Nigeria Limited, Ikeja-Lagos, Nigeria.

2.4. Experimental design

The twenty-five rats were randomly divided into five groups (A, B, C, D and E) of 5 rats each and each group received treatment as follows; groups A, B and C received 4, 8 and 16 ppm (ppm) selenium supplementation in their feed from day 0 till termination of the experiment. The selenium content of their feed was assayed and made up to 4, 8 and 16 ppm of the feed respectively. On day 14 on the selenium supplementation (PS), each rat in groups A, B, C and D were infected with 0.5 ml of saline diluted trypanosome infected rat blood containing about 1×10^6 trypanosomes intraperitoneally. The erythrocytic profile and parasitaemia were determined on day 0 and every 7th day, while deaths were recorded as it occurred. Animal studies were in compliance to the ethical procedure of the Animal Use and Care Committee, Faculty of Veterinary Medicine, University of Nigeria, Nsukka which corresponds with NIH guidelines (NIH 1996).

2.5. Collection of blood sample from rats

About 0.5 ml of blood was collected from each rat through the retro bulbar plexus of the medial canthus of rats using hematocrit tube. About 0.2 ml of the blood was put into bijou bottle with ethylenediamine tetra-acetic acid (EDTA) (BDH, England) for PCV and Hb concentration while the remainder was put in Ependof tube and allowed to clot, and later was centrifuged at 906*g* for 10 min to separate the serum for determination of serum protein levels.

2.6. Determination of PCV, Hb and parasitaemia

The blood samples were analyzed for the following parameters: packed cell volume (PCV) and hemoglobin (Hb) concentration. The