Sokoto Journal of Veterinary Sciences
(ISSN 1595-093X)
Neils *et al*. /*Sokoto Journal of Veterinary Sciences* (2010). **8**(1):39-41.

The response of serum manganese in *Trypanosoma congolense* infection in Yankasa sheep

JS Neils*[1], AKB Sackey[2], US Abdullahi[2] & KAN Esievo[3]

[1]*Department of Animal Production, Adamawa State University, Mubi. Nigeria.*
[2]*Department of Veterinary Surgery and Medicine*
[3]*Department of Veterinary Pathology and Microbiology*
Ahmadu Bello University, Zaria. Nigeria.

*Correspondence: Tel.: 08038971347, neilsjoel@gmail.com;

Abstract
Twenty two (22) Yankassa sheep obtained from a tsetse free area were acclimatized for a period of 4 weeks and divided into three groups (A, B and C) of eight, eight and six sheep, respectively, based on their mean packed cell volume (PCV) values. Sheep in groups A and B were experimentally infected with 2ml of blood from donor sheep containing 10^5 *T. congolense* organisms via the jugular vein, while group C animals were left as uninfected control. On the day of peak parasitaemia levels (day 28), in both groups, animals in group A were treated with diminazene aceturate (Berenil®, Hoechst AG, Frankfurt, Germany) at dose rate of 3.5mg/kg, through deep intra muscular injection while group B animals were not treated. Serum samples were collected weekly from 6 animals in both groups beginning weeks before infection and lasted 8 weeks post-infection. Atomic Absorption Spectrometry (Unicam Solaar 32) technique was used to determine the concentrations of manganese (Mn) in both pre- and post-infection sera. The mean serum concentration of Mn in groups A and B initially increased and then fluctuated between weeks 2 and 5 post-infection. There was significant difference serum concentrations of Mn ($P<0.001$) between the infected and the uninfected but not significant ($P>0.05$) between the infected groups. The initial increase in concentration may have been protective and from desialylated red blood cells that have been lysed by the trypanosomes. Variations in concentration of Mn likely assisted in the haemolysis in *T. congolense* infection in Yankasa sheep.

Keywords: Manganese; serum; stress; trypanosomosis; Yankasa sheep.

Introduction
Trypanosomosis on the African continent has now become a matter of 'living with the problem' however, the major control method is still by using chemotherapy (Taylor & Authié, 2004). While searching for the diverse behaviours of trypanosomes, the aspect of the effects of trypanosome infection on microminerals has not been fully investigated. Among microminerals of interest, manganese has been found to be part of many enzymes for instance pyruvate carboxylase and arginase in the body and it is also involved in oxidative phosphorylation and mitochondrial superoxide dismutase (Anon. 2010b) as well as assisting in carbohydrate and lipid metabolisms (Anon. 2010a). Mn also functions in oxidative cellular respiration and the formation of sialic acid (Underwood, 1977; Georgievskii *et al*., 1989; Awolaja *et al*., 2005; Anon. 2010b).
Despite the importance of manganese in the daily activity of animal cell, it has the lowest concentration in the body of the animal (Anon. 2010b). There are no much data/information on the effect of trypanosome infection on the serum level of manganese in animals. The effect of *T. congolense* infection on the serum concentration of manganese was therefore investigated in this study.

Materials and Methods
Experimental animals twenty two Yankasa breed of sheep were obtained from an apparently tsetse free area in northern Nigeria where the breed is predominantly found. The sheep were divided into three groups, A and B, which had eight animals each, and C, which had six animals. Animals in groups A and B were each infected with 2 ml of donor blood containing 10^5 parasites (*T. congolense,* Karu strain) administered via jugular venipuncture, while group C animals served as uninfected control. All animals in group A were treated at first peak of parasitaemia with Berenil® (Hoechst AG, Frankfurt, Germany) at dose rate of 3.5 mg/kg body weight intramuscularly while group B animals were allowed to run the full course of the disease.

Sample collection
Blood: Pre-infection blood samples for manganese assay were obtained through jugular venipuncture from each animal once a week and twice in a week when

parasitaemia was observed. After attainment of the peak of parasitaemia, samples were taken once a week. Blood from ear pricking was used for daily monitoring of parasitaemia.

Serum: Six ml of blood was collected in a clean test tube and kept at room temperature for the serum to be expressed. The clotted blood was centrifuged at 2000 g for 15 minutes and serum then transferred in a clean dry plastic vial and stored as described by Cheesbrough (1991).

Analysis of samples:
The stored sera were used to determine manganese concentration using the Atomic Absorption Spectrometry (UNICAM SOLAAR 32) technique (Petrucci & Wismer, 2005).

Statistical analysis:
SAS V8 (2004) statistical package was used.

Results

The mean serum concentration of manganese varied in group A. There was an increase from 0.26ppm to 0.35 ppm by week 1 post-infection. Thereafter, the mean concentration of Mn decreased to 0.10 ppm by week 3. The mean concentration of Mn increased to 0.30 ppm by week 6 before dropping to 0.23ppm by week 8 when the experiment was terminated (Fig1.0). There was significant difference ($P<0.05$) between the mean values in group A and the ones in group C.

The serum profile of manganese in group B followed a similar pattern to that of group A. There was decrease in mean concentration from 0.33 ppm to 0.18 ppm by week 4 post-infection. The mean serum concentration then increased to 0.28ppm by week 8 post-infection. However, there was no significant difference ($P>0.05$) between the pre- and post-infection mean values of this group. There was significant difference ($P<0.001$) between the mean values of the infected (groups A and B) and the uninfected control group C.

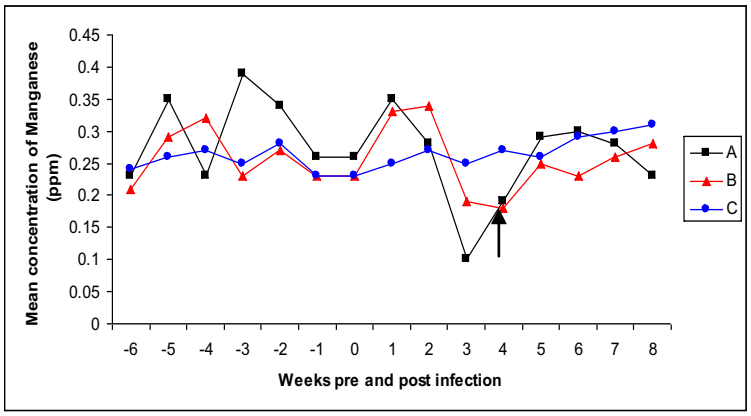

Figure 1: Mean concentration of Manganese in serum of Yankassa sheep infected with *T. congolense.*
Key: A = Infected and treated group; B = Infected untreated group
C = Uninfected control group; = Point of treatment.

Discussion

The observed sudden increase in the concentration of Mn in week 1 post-infection agrees with the observations made by Berger (2002), who explained that this could be the usual initial reaction of microminerals to infections as a protective measure. At times, red blood cell (RBC) destruction could occur without the parasites being observed, which was the case here; desialyilated RBCs might have been easily destroyed and the substantial quantity of Mn found on the membrane of RBCs released into circulation (Underwood, 1977; Awolaja *et al*., 2005). However, the subsequent decline in the mean concentrations of Mn may be due to the fact that Mn was utilized in the production of Mn-SOD for the maintenance of cell membrane integrity and functions (Underwood, 1977). Also, Mn might have been utilized in the production of sialic acid which is known to protect RBC against attack by cells of the mononuclear phagocytic system (Esievo *et al*., 1986).

The second increase was that Mn was released from the storage organs (bone, kidney, liver, pancreas and pituitary glands) and the lysed red blood cells into the plasma. The kidney which is very sensitive to hypoxia could release Mn with the slightest decrease in O_2 tension (Anon. 2010c). The increase in RBC destruction during peak parasitaemia is a contributory factor to the variations in concentration of Mn.

In conclusion, variations in concentration of Mn assisted the haemolysis of red blood cells in *T. congolense* infection in Yankasa sheep.

Acknowledgement

The staff of the Instrumentation unit, National Research Institute of Chemical Technology (NARICT) Zaria and Adamawa State University, Mubi are hereby acknowledged.

References

Anonymous (2010a). Manganese Minerals Information – Facts, benefits, deficiency disease of Manganese. http://www.online-vitamins.org/minerals/manganese.htm accessed 11-03-2010.

Anonymous (2010b). Draft Toxicological profile for Manganese http://www.atsdr.cdc.gov/tox profiles/tp151-c3.pdf

Anonymous (2010c). Salt and trace minerals for Livestock, poultry and other animals: Manganese for animals. http://www.seaagri.com/docs/salt_and_trace_elements_in_animal_nutrition.pdf accessed 11-03-2010.

Awolaja OA, Antia RE & Oyejide A (2005). Trace element levels in plasma/serum and erythrocytes of Keteku and White Fulani cattle. *Tropical Animal Health and Production,* **29**: 2-6.

Berger LL (2002). Copper Toxicity in Sheep. Treasure Valley Sheep Producers. www.tvsp.org/copper.html accessed 11-12-10.

Cheesbrough M (1991). Medical Laboratory Manual for Tropical Countries. Vol. 1, 2nd, Edition. Cambridge University Press, Cambridge. Pp 481-530.

Esievo KAN, Sarror DI, Kolo MN & Eduvie IO (1986). Erythrocyte surface Sialic acid in N'dama and Zebu cattle. *Journal of Comparative Pathology*, **96**:95-99.

Georgievskii VI, Annenkov BN & Samonkhin VT (1989). *Mineral Nutrition of Animals*. Butterworth, London. Pp 459.

Petrucci RH & Wismer RK (2005). General Chemistry with Quantitative analysis. 2nd edition, Preparing solid sample for analysis- Procedure 35-36. Oxford University Press. Pp 884.

Taylor K & Authié M-L (2004). Pathogenesis of Animal trypanosomiasis. In: *The Trypanosomiases*. (I Maudlin, PH Holmes & MA Miles editors). CABI Publishing. Pp 331-353.

Underwood EJ (1977). *Trace Elements in Human and Animal Nutrition*. 4th Edition, Academic Press. Pp 170-195.