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Effect of Administration of Zinc and Selenium on Lipid Peroxidation and Endogenous Antioxidant Enzymes in *Trypanosoma brucei brucei* Infected Albino Rats

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Abstract

The effect of administration of zinc and selenium on lipid peroxidation and endogenous antioxidant enzymes in *Trypanosoma brucei brucei* infected albino rats was investigated. Forty-five (45) healthy rats were divided into nine (9) groups of five (5) rats each. Groups I, II, and III served as control; administered with normal chow and distilled water *ad libitum,* zinc gluconate and selenium and *Trypanosoma brucei brucei* without treatment respectively. Groups IV, V, and VI were the pre-treated infected groups that were administered with daily dose of 50mg per kilogram body weight of zinc, 10mg per kilogram body weight of selenium and combination of zinc and selenium respectively for seven (7) days. Whereas group VII, VIII and IX represented the post-infected treated groups that were administered with daily dose of 50mg per kilogram body weight of zinc, 10mg per kilogram body weight of selenium and combination of zinc and selenium respectively for seven (7) days. The control group of *Trypanosoma brucei brucei* infected without treatment showed a significant ($P<0.05$) increase in the concentration of malondialdehyde (MDA), and decrease in superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) activities when compared with other control groups. Administration of zinc and selenium to the pre and post-infected groups caused significantly ($P<0.05$) lower values in the concentration of malondialdehyde (MDA), with significantly ($P<0.05$) higher activities of superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) in the liver and kidney. Treatment with zinc and selenium may contribute in protection against cellular damage during *Trypanosoma brucei brucei* infection in rats.

Keywords: Zinc, Selenium, MDA, SOD, CAT, GPx, *Trypanosoma brucei brucei*

Introduction

Zinc (Zn) is a micronutrient and a very abundant trace element in the body with diverse roles in biological, clinical and global public health (Dalla Rosa *et al*., 2012; Hambidge *et al*., 2010; Zhou *et al*., 2007). It is an important trace element for all forms of life, and acts as important component of biological antioxidant systems (Debjit *et al*., 2010; Sahin and Kucuk, 2003). A lot of enzymes use zinc in one form or the order to achieve their biological function, and as such, are involved in numerous aspect of cellular metabolism. Due to its interaction with numerous enzymes as a co-factor, they are necessary for growth, optimum performance and modulation of immune system (Zago and Oteiza, 2001). In addition, Zinc is important as they perform roles in the structure and function of biological membranes (Bettger and O‟Dell, 1993), it has been shown to have an antioxidant potential and also exert critical physiological role in regulating the structure and function of cells (Powell, 2000; Sidhu *et al*., 2004). Zinc is specifically needed for the complete formation and function of the antioxidant enzyme; copper-zinc superoxide dismutase (CuZnSOD) and various metallothioneins (Disilvestro, 2000). It performs a vital role in the antioxidative defense of cells (Bonnefont-Rousselot, 2004).

Selenium (Se) is an important component of antioxidant enzymes such as glutathione peroxidase (GPx), thioredoxin reductase (TrxR) and iodothyronine deiodinases (IDD). It is also a natural antioxidant (Tapiero *et al*., 2003) and immunostimulant (Beck *et al*., 2003; Broome *et al*., 2004; Kiremidjian-Schumacher *et al*., 1994). Recently, research on selenium has increased greatly due to its vital function in antioxidant seleno-proteins for protection against oxidative stress initiated by excess reactive oxygen species (ROS) and reactive nitrogen species (RNS) (Rayman, 2012). Its activity helps to maintain membrane integrity, protects prostacyclin production and decreases the likelihood of propagation of further oxidative damage to biomolecules such as lipids, lipoproteins and DNA with the associated increased risk of didtysease conditions such as atherosclerosis and cancer (Néve, 1996).

Trypanosoma brucei belongs to the order Kinetoplastida and is considered part of the earliest diverging eukaryotic lineages (Simpson *et al*., 2006). Therefore, they are regarded as a “model organism” for studying other alternative mechanisms by which eukaryotes are able to perform basic functions. During their life cycle, trypanosomes encounter the vastly different environments of the mammalian bloodstream and various tissues within the tsetse vector. They respond to these by dramatic morphological and metabolic changes, including adaptation of their lipid and energy metabolism (Hannaert *et al*., 2003).

There are three sub-species of the *Trypanosoma brucei*, namely; *Trypanosoma brucei brucei, Trypanosoma brucei rhodesiense* and *Trypanosoma brucei gambiense* (Hoare, 1972). *Trypanosoma brucei brucei* belongs to the sub-genus Trypanozoon, which is responsible for causing Nagana in cattle. Horses, dogs, cats, camels and pigs are very susceptible to *Trypanosoma brucei brucei* infection. Infection of cattle, sheep, goats and sometimes pigs results in mild or chronic infection (Mulligan, 1970). Moulton and Sollod, (1976) showed evidence of this organisms wide-spread in East and West sub-Saharan Africa causing serious disease and high death rate in cattle, sheep, and goats.

Trypanosomiasis is one of the oldest and neglected tropical diseases (WHO, 2008) causing much trouble to man, mainly in sub-Saharan Africa and parts of South America. Tse tse fly, belonging to the genus *Glossina* is responsible for transmitting the disease (Trypanosomiasis), endemic in 36 sub-Saharan African countries posing a serious setback to improved and profitable livestock production and mixed crop-livestock farming development in the African continent (Adamu *et al*., 2008; Stevens and Brisse, 2004; Swallow, 2002). The disease is called sleeping sickness in humans, Nagana in cattle and Surra in Camels (Welburn *et al*., 2006).

Materials and Methods

Materials

Chemicals/Reagents

All chemicals used were of analytical grade. Hydrogen Peroxide, Sodium chloride, Sodium hydroxide, Sodium carbonate, Sodium bicarbonate, Acetic acid, Potassium dichromate, Potassium phosphate, Potassium iodide, Picric acid, Hydrochloric acid, Dinitrophenyl hydrazine, Magnesium chloride, Copper sulphate, Sodium citrate, Dipotassium phosphate, Trichoroacetic acid, Adrenalin, Thiobarbituric acid were all purchased from Sigma-Aldrich Company limited®. Zinc gluconate (Good‟N Natural, Bohemia USA) and Selenium (Sigma-Aldrich®).

Experimental Animals

A total of fifty four (54) healthy albino rats of both sexes, weighing between 200 – 250g, were used for the experiment. The rats were purchased from the Department of Pharmacology animal house, Ahmadu Bello University Zaria, Nigeria. The animals were kept in well-aerated laboratory cages and allowed to adjust to the laboratory environment for a period of two weeks before commencement of the experiment. They

were fed with standard feed (Vital Feeds, Jos, Nigeria) and water was provided *ad libitum.*

Trypanosome Isolates

Strain of *Trypanosoma brucei brucei* was obtained from the stabilates that was cryopreserved in a Vector in Parasitology Studies Department, Nigerian Institute for Trypanosomiasis Research (NITR), Kaduna, Nigeria.

Methods

Induction of Parasitaemia

The parasite was maintained by serial passage in a donor rat. The infected blood from the donor rat was collected and diluted with phosphate buffered saline (PBS). The number of parasites in the diluted blood was determined (Herbert and Lumsden, 1976) and 0.1mL of blood containing approximately 1 x 103 trypanosomes was inoculated intraperitoneally into each rat in the infected groups.

Experimental Design

A total of forty-five (45) rats were used. The rats were randomized into nine (9) groups consisting of five (5) rats per group. Groups I, II and III served as control while group IV, V and VI served as pre-treated infected rats that were administered with daily dose of 50mg per kilogram body weight of zinc gluconate (Ambali *et al*., 2011), 10mg per kilogram body weight of selenium (Rayman, 2012) and combination of zinc gluconate and selenium respectively for seven (7) days. Groups VII, VIII and IX served as post-infected treated groups that were administered with daily dose of 50mg per kilogram body weight of zinc gluconate (Ambali *et al*., 2011), 10mg per kilogram body weight of selenium (Rayman, 2012) and combination of zinc gluconate and selenium immediately after parasite was sighted in the blood for seven (7) days.

Grouping and Treatment

Control groups:

Group I (NC): Normal rats fed with normal chow and distilled water *ad libitum*
Group II (N+ Zn +Se): Normal rats treated with zinc gluconate + selenium
Group III (TC): *Trypanosoma brucei brucei* infected untreated rats

Pre-infected treated groups:

Group IV (Pre +Zn): *Trypanosoma brucei brucei* infected rats + zinc gluconate
Group V (Pre + Se): *Trypanosoma brucei brucei* infected rats + selenium
Group VI (Pre + Zn +Se): *Trypanosoma brucei brucei* infected rats + zinc gluconate + selenium

Post-infected treated groups:

Group VII (Post + Zn): *Trypanosoma brucei brucei* infected rats + zinc gluconate
Group VIII (Post + Se): *Trypanosoma brucei brucei* infected rats + selenium
Group IX (Post + Zn +Se): *Trypanosoma brucei brucei* infected rats + zinc gluconate + selenium

The samples were reconstituted in distilled water, and administered intraperitoneally, daily for 7 days. Blood samples were collected from the tail vein of rats daily to monitor the development of parasitaemia.

Determination of Parasitaemia

Blood sample was collected from the tail vein of rats, pre-sterilized with methylated spirit every day to monitor the development of parasitaemia. The number of parasites was determined microscopically at x 40 magnification using the “Rapid Matching” method described by Herbert and Lumsden (1976) to estimate the number of parasite per field. The method involves microscopic counting of parasites per field in blood appropriately diluted with buffered phosphate saline (pH 7.2). Logarithm values of these counts were obtained by matching with the table of Herbert and Lumsden (1976) and converted to antilog to provide absolute number of trypanosomes per ml of blood.

Collection and Preparation of Samples for analysis

At the end of the experiment, the liver and kidney were excised, trimmed of connective tissues, rinsed with saline to eliminate any form of blood contamination, dried by blotting with filter paper, and kept in the freezer. The liver and kidney were crushed; 50mM potassium phosphate buffer (pH 7.4) was used for homogenization. A certain weight of the organ was taken and buffer was added to a certain total volume, and then homogenised. It was centrifuged at 4000rpm for 15min. Then, the supernatant was collected using Pasteur pipette. Lipid peroxidation of low density lipoprotein (LDL) as evidenced by the level of malondialdehyde (MDA) concentration was assessed by Thiobarbituric acid reactive substances (TBARS) formation described by Ohkawa *et al.,* 1979. Superoxide dismutase (SOD) activity was determined by method of Fridovich (1989), Catalase (CAT) activity was determined using the method described by Sinha (1972) and Glutathione peroxidase (GPx) activity by method described by Ellman (1959).

Data Analysis

Results were expressed as mean ± standard deviation (SD). The data obtained were analyzed using analysis of variance (ANOVA) (SPSS program, version 20 SPSS Inc., Chicago, IL, USA for windows Computer software Package). The difference between the experimental groups were compared using the Duncan Multiple Range Test. Values of P less than 0.05 (P<0.05) were taken as significant.

Results

Effect on Liver Lipid Peroxidation

Table 1 shows lipid peroxidation level of malondialdehyde (MDA) in the liver of normal and trypanosomiasis infected rats administered with zinc and selenium for seven (7) days. Rats that were infected with *Trypanosoma brucei brucei* untreated shows significantly (P<0.05) higher concentration of MDA compared to other control groups. Animals that were treated; pre and post infection showed a significantly (P<0.05) higher concentration of MDA when compared to the normal feed and water and combined Zn + Se control groups. However, there was an exception in the pre-infected treated and post-infected treated groups administered with Zn +Se. The MDA concentration of all the pre and post infected treated showed a significantly (P<0.05) lower value when compared to the infected untreated *Trypanosoma brucei brucei* control group.

Effect on Liver Antioxidants

The result shows that normal rats that were administered combined Zn + Se without infection had no significant (P>0.05) difference in liver antioxidant enzymes (SOD, CAT, and GPx) activities compared to the normal feed and water control group animals (Table 1). There was a significantly (P<0.05) lower activities of liver antioxidant enzymes (SOD, CAT, and GPx) in infected untreated group when compared to the normal feed and water and combined Zn +Se control groups. The pre and post infected treated rats showed a significantly (P<0.05) lower activity in SOD when compared with the normal feed and water control group animals. All the pre and post *Trypanosoma brucei brucei* infected treated rat showed a significantly (P<0.05) higher activities of liver antioxidant enzymes (SOD, CAT, and GPx) when compared to the infected untreated *Trypanosoma brucei brucei* group.

Table 1: Effect of Administration of Zinc and Selenium on Lipid Peroxidation and Endogenous Antioxidant Enzymes in *Trypanosoma brucei brucei* Infected Wistar Albino Rats in the Liver.

Group	MDA (mmol/g of tissue)	SOD (mmol/min/g of tissue)	CAT (moles of H_2O_2/min/g of tissue)	GPx (mU/mL)
NC	2.35±0.33^{a}	3.18±0.19^{e}	52.00±1.26^{b}	51.00±2.23^{bc}
N+ Zn +Se	2.36±0.18^{a}	3.13±0.16^{e}	53.17±2.14^{b}	49.83±1.94^{bc}
TC	2.95 ±0.26^{c}	2.28±0.46^{a}	43.33±1.63^{a}	42.00±4.38^{a}
PRE +Zn	2 .65±0.28^{b}	2.92±0.15^{d}	53.00±1.03^{bc}	49.33±1.86^{bc}
PRE + Se	2.67±0.29^{b}	2.87±0.20^{bc}	53.00±1.74^{bc}	48.59±2.59^{bc}
PRE+Zn+Se	2.43±0.38^{a}	3.00±0.05^{cd}	55.67±2.80^{c}	51.50±1.23^{c}
POST + Zn	2.70±0.33^{b}	2.77±0.18^{c}	53.83±3.06^{bc}	49.67±2.73^{bc}
POST + Se	2.77±0.23^{b}	2.53±0.31^{b}	53.50±3.08^{bc}	47.17±3.06^{b}
POST+Zn+Se	2.44±0.32^{a}	2.90±0.21^{bcd}	52.17±2.79^{bc}	49.00±1.53^{bc}

Values are means ± SD of five replicate determinations. Values with different superscript down the column are significantly different (P<0.05).

Effect on the Kidney Lipid Peroxidation

Trypanosoma brucei brucei infected untreated rats showed a significantly (P<0.05) higher concentration of MDA when compared to the normal feed and water and combined Zn +Se control groups (Table 2). Rats in the pre and post infected treated groups showed a significantly (P<0.05) high concentration of MDA when compared to the normal feed and water and combined Zn +Se control groups. The pre and post infected treated groups showed a significantly (P<0.05) lower concentration of MDA compared to the infected untreated *Trypanosoma brucei brucei* control group.

Effect on the Kidney Antioxidants

The result shows that there was a significantly (P<0.05) lower activities of kidney antioxidant enzymes (SOD, CAT and GPx) in the infected untreated group when compared to the normal feed and water and combined Zn + Se control groups (Table 2). The treated pre and post infected groups showed a significant (P<0.05) increase in SOD, CAT and GPx activities when compared to *Trypanosoma brucei brucei* infected untreated control group.

Table 2: Effect of Administration of Zinc and Selenium on Lipid Peroxidation and Endogenous Antioxidant Enzymes in *Trypanosoma brucei brucei* Infected Wistar Albino Rats in the Kidney.

Group	MDA (mmol/g of tissue)	SOD (mmol/min/g of tissue)	CAT (moles of H_2O_2/min/g of tissue)	GPx (mU/mL)
NC	2.40±0.13^{a}	3.37±0.30^{d}	48.50±1.33^{b}	46.17±1.17^{b}
N+ Zn +Se	2.30±0.14^{a}	3.22±0.21^{d}	49.00±1.22^{b}	46.67±2.80^{b}
TC	2.78±0.23^{d}	2.63±0.27^{a}	34.67±3.93^{a}	39.50±1.38^{a}
PRE + Zn	2.58±0.17^{bc}	3.25±0.24^{b}	47.83±1.14^{b}	47.17±1.94^{b}
PRE + Se	2.63±0.25^{bc}	2.95±0.24^{ab}	47.17±3.06^{b}	49.83±1.60^{c}
PRE+Zn+Se	2.45±0.22^{b}	3.98±0.27^{b}	48.60±0.22^{b}	47.00±2.37^{b}
POST + Zn	2.68±0.19^{bc}	2.80±0.28^{ab}	46.00±3.22^{b}	46.15±3.11^{b}
POST + Se	2.70±0.23^{c}	2.75±0.19^{ab}	45.25±1.82^{b}	47.12±2.81^{b}
POST+Zn+Se	2.46±0.24^{b}	3.91±0.32^{c}	47.08±2.00^{b}	46.08±3.28^{b}

Values are means ± SD of five replicate determinations. Values with different superscript down the column are significantly different (P<0.05).

Discussion

Trypanosomiasis was inflicted on the rats when they were injected with trypanosomes and therefore, provides animal models of experimentally infected trypanosomiasis. The parasite *Trypanosoma brucei brucei* produces a very severe acute stress in the infected rats. Oxidative stress describes a higher pro-oxidant load compared to the antioxidant defense system in the body, leading to a potential damage (Sies *et al*., 2005). During oxidative stress, there is a general reduction of the free radical defense mechanism in the body, which includes changes in the activities of antioxidant enzymes, essential polyunsaturated fatty acids, compromise of vitamins and minerals elements, thereby exposing the body's own antioxidant defense system and making them vulnerable to damage by reactive oxygen species (ROS) (Omoregie and Osagie, 2011). Malondialdehyde (MDA) is the major oxidation product of the peroxidation of polyunsaturated fatty acids, and MDA formed is measured quantitatively by the reaction with thiobarbituric acid reactive substances (TBARS), thereby an elevated MDA concentration, reflects as an important index to lipid peroxidation.

Free radical scavengers and chain terminator enzymes such as superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) system protect the biological system against the damaging effects of oxidants and activated species (Kurata *et al*., 1993).

Catalase is an enzymatic antioxidant that can be found virtually in all animal tissues including the red blood cell and liver. It degrades hydrogen peroxide (H_2O_2) and helps protect the tissues from highly reactive hydroxyl radicals. SOD, another antioxidant enzyme, removes superoxide radical by converting it to H_2O_2 (Krishnaraju *et al*., 2009). Glutathione protects the cellular system against the harmful negative effects of lipid peroxidation. In this present study, the results shown in (Table 1 and 2) shows that concentrations of MDA were significantly ($P<0.05$) higher in liver and kidney tissue homogenates of infected untreated rats compared to the pre and post-infected treated and normal feed and water control groups. The depression in the concentration of MDA in liver and kidney tissue homogenates following administration of zinc and selenium confirm their antioxidant properties (DiSilvestro, 2000). The micronutrients may have also prevented or delayed the on-set of oxidative stress which arises as a result of imbalance between radical-generating and radical-scavenging activity. Ogunsanmi and Taiwo (2001) have shown in earlier study that oxidative stress plays an important etiologic role in the pathogenesis of trypanosomiasis. In the same vein, it has also been demonstrated that infections with *Trypanosoma brucei brucei* group of parasites may change the host's antioxidant defense against free radicals (Igbokwe *et al*., 1996; Omer *et al*., 2007).

Elevated MDA concentrations have been related to the amount of stress and are well correlated with lipid membrane damage and loss of membrane integrity. Increased production of MDA in plasma, tissues and erythrocytes was also reported in murine models and humans infected with *Trypanosoma cruzi* (Malvezi *et al*., 2004). Aleksandro *et al*., (2009) reported that plasma MDA concentrations were significantly higher in animals infected by *Trypanosoma evansi* than in the uninfected group, and in animals with acute (day 21) and chronic (day 49) trypanosomiasis, MDA levels were also found to be proportional to the time of infection.

Intraperitonial administration of zinc and selenium in the pre and post-infected rats resulted in increased activities of GPx in liver and kidney (Table 1 and 2) when compared with the infected untreated rats. The depressed activity of GPx observed in the liver and kidney of infected untreated rats is an indication of an increased utilization due to oxidative stress. This is probably due to their antioxidant defense system which includes GPx was mobilized to fight the presence of the parasites. This is congruent with previous studies that showed a decrease in erythrocytic and hepatic glutathione concentrations in rats infected with *Trypanosoma brucei brucei* (Ameh, 1984). The trypanosome infection causes the oxidation of GSH to GSSH by activated oxygen and is partly responsible for the significantly lower concentration of glutathione peroxidase in the liver and kidney tissue homogenate of infected untreated rats compared to the pre and post-infected treated groups (Igbokwe *et al*., 1996; Ogunsanmi and Taiwo, 2001), also, the high increase in the activity of glutathione peroxidase can also be attributed to its lower concentration (Omer *et al*., 2007) since the reaction catalyzed by this enzyme consumes GSH. Likewise, reports have shown that depletion of endogenous glutathione antioxidant may be a significant factor in the pathogenesis of *Trypanosoma Scongolense* infection but upon administration of exogenous vitamin C to infected animals prevented these disease-induced decreases in glutathione and ascorbic acid (Umar *et al*., 2010).

Catalase (CAT) is a peroxisomal maker enzyme found in blood, bone marrow, mucous membrane, kidney and liver. It functions assumed to be destruction of hydrogen peroxide. The result of this study showed an increase in liver and kidney CAT activities in the the pre and post infection treated groups when compared with the infected untreated group (Table 1 and 2). Oluwatosin *et al*., (2013) observed that the decrease in the activity of catalase could be attributed to its use in mopping up the free radicals overload which is produced during trypanosome infection.

Superoxide dismutase (SOD) is also an endogenous antioxidant enzyme that protects blood cells from oxidative stress and damage. The result of this study showed lower SOD activities in liver and kidney in the infected untreated control compared to the pre and post-infected treated groups. This could be as a result of increased removal of superoxide anions. Omer *et al*., (2007), reported decreased SOD activity in *Trypanosoma evansi* infected rats. However, others reported increased SOD activity (Ogunsanmi and Taiwo, 2001; Yusuf *et al*., 2012). Conditions of oxidative stress increases the activities of antioxidant enzymes such as SOD, CAT and glutathione peroxidise as reported by Ataley *et al*., (2000).

Conclusion

This study shows that administration of zinc and selenium prevented depletion of endogenous antioxidant enzymes (superoxide dismutase, catalase and glutathione peroxidase) in the liver and kidney. Therefore, understanding the role of these micronutrients in the pathogenesis of diseases

associated with oxidative stress may help in designing nutritional support and control programs as a strategy to combating the effect of the diseases.

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