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Hepatocurative Property of Zinc and Selenium in *Trypanosoma brucei brucei* Infected Albino Rats

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Abstract

The effect of administration of zinc and selenium on some biochemical parameters 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 results obtained for serum alkaline phosphatase (ALP), alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities in the infected untreated group were significantly ($P<0.05$) higher when compared to the pre and post-infected treated groups. The result of the liver function parameters showed that the infected untreated control group showed a significantly ($P<0.05$) lower values of TP, ALB and higher values of TB and IB when compared to the normal and combined Zn + Se control groups. All the pre and post infected treated groups shows a significant ($P<0.05$) increase in TP, ALB and decrease in TB, IB when compared with the infected untreated *Trypanosoma brucei brucei* control group.

Keywords: Liver, Zinc, Selenium, Biochemical Parameters, *Trypanosoma brucei brucei*

Introduction

The Liver, the largest gland is a vital organ. It has a pivotal role in regulation of physiological processes. It is involved in several vital functions such as metabolism, secretion and storage. Furthermore, detoxification of a variety of drugs and xenobiotics occurs in liver. The bile secreted by the liver has, among other things, an important role in digestion. Liver diseases are among the most serious ailment. They may be classified as acute or chronic hepatitis (inflammatory liver diseases), hepatosis (non-inflammatory diseases) and cirrhosis (degenerative disorder resulting in fibrosis of the liver). Liver diseases are mainly caused by infections, excess consumption of alcohol, autoimmune/disorder and toxic chemicals (certain antibiotics, chemotherapeutics, peroxidised oil, aflatoxin, carbon-tetrachloride, chlorinated hydrocarbons, etc.). In recent years, researchers have used scientific methods to evaluate the effects of medicinal plants used in traditional medicine for the treatment of liver ailments (Azzam *et al.,* 2007, Luk *et al.,* 2007, Ball and Cowdley 2005, Mayer *et al.,* 2005). In many cases, the mechanisms and modes of action of these plants as well as their therapeutic effectiveness have been confirmed in clinical studies and several hundred plants have been examined.

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).

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).

Materials

Chemicals/Reagents

All assay kits for alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline Phosphatase (ALP), total protein, albumin and bilirubin were purchased from Randox laboratories Ltd® (Northern Ireland, UK), Ardmore, Co. Antrum UK.

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

Collection and Preparation of Sera Samples and Biochemical Analysis

At the end of the experiment, chloroform-inhalation anesthesia was performed on all experimental animals. The anesthetized animals were bled by cardiac

puncture. The blood samples were collected into anticoagulant free tubes, centrifuged at a speed of 3000 r/m for 15 minutes and the resultant serum harvested into plain sample bottles for biochemical analysis. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline Phosphatase (ALP), total protein (TP), albumin (ALB), total Bilirubin (TB), direct bilirubin (DB) and indirect bilirubin (IB) concentrations were determined using Randox kits.

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

Effects on Liver Marker Enzymes

Table 1 presents the effects of pre and post infection administration of zinc and selenium on liver marker enzymes in normal and *Trypanosoma brucei brucei* infected rats. The result shows that there were no significant ($P>0.05$) difference in serum levels of AST, ALT and ALP in animals administered Zn + Se without infection compared to the normal control. There was a significantly ($P<0.05$) higher values in the serum activities of AST, ALT and ALP in *Trypanosoma brucei brucei* infected untreated control when compared to the normal and Zn + Se control groups. The pre and post infected treated groups showed significantly ($P<0.05$) lower values in serum activities of AST, ALT and ALP compared to the infected untreated control group (Table 1).

Table 1: Effect of Pre and Post Infection Administration of Zinc and Selenium on Liver Marker Enzymes in *Trypanosoma brucei brucei* Infected Wistar Albino Rats.

Group	AST (U/I)	ALT (U/I)	ALP (U/I)
NC	33.50 ± 4.85^{a}	33.30 ± 1.75^{a}	120.17 ± 3.67^{a}
N+ Zn +Se	35.00 ± 3.58^{a}	34.67 ± 2.16^{a}	123.83 ± 4.14^{a}
TC	51.33 ± 3.01^{d}	52.83 ± 4.11^{d}	258.67 ± 12.43^{e}
PRE +Zn	35.5 ± 0.83^{ab}	36.17 ± 0.79^{ab}	204.83 ± 1.91^{c}
PRE + Se	44.50 ± 4.22^{c}	35.12 ± 2.89^{ab}	217.33 ± 6.19^{cd}
PRE + Zn+Se	38.00 ± 3.85^{b}	31.50 ± 1.83^{a}	168.50 ± 4.42^{b}
POST + Zn	41.20 ± 5.58^{bc}	45.33 ± 2.47^{c}	213.00 ± 2.29^{cd}
POST + Se	42.16 ± 6.01^{bc}	46.33 ± 3.03^{c}	231.17 ± 3.56^{d}
POST + Zn+Se	36.83 ± 2.53^{ab}	40.83 ± 1.76^{b}	200.50 ± 2.32^{c}

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

Effect on Liver Function Parameters

The effect of pre and post infection administration of zinc and selenium on liver function parameters in the serum of normal and *Trypanosoma brucei brucei* infected wistar albino rats is presented in Table 2. The result shows that the infected untreated control group showed a significantly ($P<0.05$) lower values of TP, ALB and higher values of TB and IB when compared to the normal and combined Zn + Se control groups. All the pre and post infected treated groups shows a significant ($P<0.05$) increase in TP, ALB and decrease in TB, IB when compared with the infected untreated *T. brucei brucei* control group. The control groups, pre and post infected treated groups showed no significant ($P>0.05$) difference in the DB (Table 2).

Table 2: Effects of Pre and Post Infection Administration of Zinc and Selenium on Liver Function Parameters in *Trypanosoma brucei brucei* Infected Wistar Albino Rats.

Group	TP (g/dl)	ALB (g/dl)	TB (mg/dl)	IB (mg/dl)	DB (mg/dl)
NC	44.17±1.47^{b}	42.67±1.75^{e}	10.88±1.12^{a}	4.36±1.33^{a}	6.52±0.48^{a}
N+ Zn +Se	42.33±1.50^{b}	40.00±2.28^{e}	10.13±1.39^{a}	3.03±1.27^{a}	7.10±0.42^{a}
TC	37.83±3.54^{a}	31.83±2.48^{a}	14.15±1.29^{c}	7.07±1.30^{b}	7.08±0.60^{a}
PRE + Zn	40.17±0.52^{ab}	37.50±1.64^{cd}	11.62±1.28^{ab}	4.72±1.02^{a}	6.90±0.85^{a}
PRE + Se	39.67±2.92^{ab}	36.50±2.26^{b}	11.85±1.02^{ab}	4.85±1.43^{a}	7.00±0.84^{a}
PRE+Zn+Se	44.47±2.56^{b}	39.83±0.47^{d}	11.01±0.65^{a}	4.18±0.41^{a}	6.83±0.45^{a}
POST + Zn	42.67±1.75^{b}	34.67±2.26^{b}	12.88±1.07^{b}	5.25±1.48^{a}	7.63±0.38^{a}
POST + Se	38.83±4.00^{ab}	32.00±1.67^{ab}	12.20±1.84^{ab}	5.18±0.95^{a}	7.02±0.58^{a}
POST+Zn+Se	43.03±3.67^{b}	36.83±2.32^{c}	11.78±0.98^{ab}	5.78±1.31^{a}	6.00±0.31^{a}

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. *Trypanosoma brucei* infection, like other trypanosome infections may precipitate increased biochemical changes in the host in response to invading parasites and these changes in part could be responsible for infection-induced tissue damage.

Hepatic injury is often associated with alterations in the serum and liver levels of Aspartate aminotransferase (AST), Alanine aminotransferase (ALT) and Alkaline phospatase (ALP) (Arhoghro et al., 2014). The increase in serum alkaline phosphatase activity may indicate hepatic damage probably by the altered cell membrane permeability leading to the leakage of the enzymes from the tissues to the serum (Appidi et al., 2009; Akanji and Yakubu, 2000). Alanine and aspartate aminotransferase are considered to be sensitive indicators of hepatocellular damage and within limit can provide a quantitative evaluation of the degree of damage to the liver (Al-Habori et al., 2002). Hepatic damage can affect the metabolic processes in the body due to the role of liver in general metabolism. Hepatic cells appear to participate in a variety of enzymatic metabolic activities. Enzymes are necessary for normal cellular metabolism including that of the liver (Rajamanickam and Muthuswamy, 2008). Infection with *T. brucei brucei* damaged the hepatic cells leading to a significant increase in serum levels of AST, ALT, and ALP respectively (Table 1). The significantly ($P<0.05$) higher levels of serum AST, ALT, and ALP activities were observed in infected untreated rats compared to the pre and post-infected treated and control groups. These results are in agreement with previous studies where ALT was elevated in Trypanosoma evansi infected (Sazmand et al., 2011) and T. brucei brucei infected (Yusuf et al., 2012) animals. Several other studies have also reported elevated serum AST, ALT, and ALP (Abd El-Baky and Salem, 2011; Allam et al., 2011; Oluwatosin et al., 2013; Umar et al., 2007). Since these enzymes are the major liver marker enzymes; the elevation of these enzymes is usually an indication of liver damage, haemolytic conditions or partly to cellular damage caused by lysis or destruction of the trypanosomes (Yusuf et al., 2012). The significant decrease in the activities of the liver marker enzymes in all the pre and post-infected treated rats could be attributed to the reduction in hepatic granuloma size

and fibrosis as well as absence of necrotic hepatic tissue in the pre and post-infected treated rats. Apparently it appears that the membrane damage seems to be the prime culprit for the marked increase in the serum marker enzymes, AST, ALT, and ALP. The pre-treated infected rats were better at reducing hepatic damage than the post-infected treated rats.

Serum albumin and total protein are some of the markers of liver dysfunction while albumin transports bilirubin and other substances in blood (Vasudevan and Sreekumari, 2007). Serum levels of total protein and albumin were reduced significantly ($P<0.05$) in the infected untreated as compared to normal control groups (Table 2). However administration of zinc and selenium to the pre and post-infected rats resulted in elevation of total protein and albumin levels when compared to the infected untreated rats. The increased synthesis of protein occurs at the expense of muscle protein catabolism and loss in body weight. The gradual decrease in the mean values of serum total proteins, observed in the infected untreated rats during this study, agrees with previous findings (Biryomumaisho et al., 2003; Katunguka-Rwakishaya, 1996), but contradicts observations made in sheep infected with *T. brucei* by Taiwo et al., (2003), who observed no change in levels of total plasma proteins from pre-infected values at the initial stage of the infection, but in the later stage the levels increased significantly above pre-infection levels. Albumin is synthesized in the liver; therefore decrease in albumin concentration may be attributed to the damage in the liver where there could be less synthesis of albumin. The result obtained here agrees with that of Ogunsanmi et al., (1994) who studied the serum biochemical changes in West African dwarf sheep experimentally infected with *T. brucei*. They found that the serum albumin values were markedly decreased. The findings suggest that there might be a hepatic and/or renal malfunction. Similar observations were noticed by Arora and Pathak, (1995) and Yusuf *et al.*, (2012). The cause of the decrease in albumin is difficult to elucidate. Albumin is a negative acute phase protein during trypanosomiasis (Karori *et al*., 2008). Its decrease could result from reduced synthesis in the liver as part of the acute phase response, loss through the kidney and intestine or increased utilization by the trypanosomes as a nutrient, since they require it for optimal survival (Coopens *et al*., 1987).

Bilirubin is the main bile pigment that is formed from the breakdown of heme in the red blood cells. In addition, it is transported to the liver where it is secreted by the liver into the bile. As such interference with the normal liver functions affects its rate of conjugation or excretion. Thus a high level of bilirubin is used as an index for liver function and bile excretion status (Usha *et al.*, 2008). The present results showed significantly ($P<0.05$) higher level of total bilirubin in *T. brucei* infected untreated rats. The high level of total bilirubin in infected untreated rats in this experiment supports earlier observations in several trypanosome-infected animals (Adeyemi *et al.*, 2012; Boniface *et al.*, 2011; Ezeokonkwo *et al*., 2012). The bilirubin formed from breakdown of red blood cells in the reticulo endothelial cells are transported in plasma bound to albumin (Vasudevan, and Sreekumari, 2007), so the increase in bilirubin is suggestive of haemolytic anaemia which may be due to the activity of proliferating parasites. It could also be associated to the inability of the liver to conjugate bilirubin (Adeyemi *et al*., 2012). The liver detoxifies harmful substances, secretes bile into the intestine, synthesizes and stores up important material, hence, it is common in clinical practice to screen for liver disease, monitor the progression of a known disease and monitor the effect of potentially hepatotoxic drugs (Kapoor, 2011).

Conclusion

The findings of this study have shown potential efficacy of Zinc and Selenium in ameliorating some negative effects of Trypanosomiasis on the liver. Understanding the role of these micronutrients in the pathogenesis of trypanosomiasis may help in designing nutritional support and control programs as a strategy to combating the effect of the disease in areas where it is endemic.

References

Abd El-Baky, A.A. and Salem S.I. (2011). Clinicopathological and cytological studies on naturally infected camels and experimentally infected rats with *Trypanosoma evansi.* World Applied Science Journal, 14(1):42–50

Adamu, S., Ige, A.A., Jatau, I.D., Neils, J.S., Useh, N.M., Bisalla, M., Ibrahim, N.D.G., Nok, A.J. and Esievo, K.A.N. (2008). Changes in the serum profiles of lipids and cholesterol in sheep experimental model of acute African trypanosomosis. *African Journal of Biotechnology*, 7: 2090-2098.

Adeyemi O.S, Akanji M.A. and Ekanem J.T. (2012). Ethanolic extract of Psidium guajava influences protein and bilirubin levels in *Trypanosoma brucei brucei* infected rats. Journal of Biological Science. 12(2):111–116.

Akanji, M. A. and Yakubu, M. T. (2000). α-Tocopherol protects against metabisulphite induced tissue damage in rats. Nigerian Journal of Biochemistry and Molecular Biology. 15(2), 179-183.

Al-Habori, M, Al-Aghbari, A, Al-Mamary, M. and Baker, M. (2002). Toxicological evaluation of *Catha edulis* leaves: a long term feeding experiment in animals. Journal of Ethnopharmacology, 83(3): 209-217

Allam, L., Ogwu, D., Agbede, R.I.S. and Sackey, A.K.B. (2011). Haematological and serum biochemical changes in gilts experimentally infected with *Trypanosoma brucei*. Veterinarski Arhiv. 81(5):597–609.

Ambali, S.F., Abubakar, Kawu, M.U., Uchendu, C., Shittu, M. and Salami, S.O. (2011). Biochemical alterations induced by subchronic chlorpyrifos exposure in Wistar rats: ameliorative effect on zinc. *The Journal of American Science*, 7(9): 73-81

Appidi, J.R, Yakubu, M.T, Grierson, D.S and Afolayan A.J. (2009). Toxicological evaluation of aqueous extracts of *Hermannia incana* Cav. Leaves in male Wistar rats. African Journal of Biotechnology, 8(10), 2016-2020.

Arhoghro, E.M., IKeh, C. and Prohp, T.P. (2014). *Cymbopogon citratus* aqueous extract alleviates cisplatin-induced hepatic oxidative stress and toxicity in albino rats. International Journal of Current Microbiology and Applied Sciences, 3(4): 586-604.

Arora, J.K and Pathak, M.L. (1995). Clinico-haematological and biochemical damages associated with *Trypanosoma evansi* in dogs. Indian Animal Health.

Azzam H.S, Goertz C, Fritts M, Jonas WB (2007). Natural products and chronic hepatitis C virus. Liver Int.; 27:17–25.

Ball K.R, and Kowdley K.V (2005). A review of *Silybum marianum* (milk thistle) as a treatment for alcoholic liver disease. Journal of Clinical Gastroenterol.; 39:520–8.

Beck, M. A., Levander, O. A. and Handy, J. (2003). Selenium deficiency and viral infection. *Journal of Nutrition*, 133, 1463S-1467S.

Bettger, W.J. and B.L. O'Dell (1993). Physiological roles of zinc in plasma membrane of mammalian cells. *Journal of Nutrition and Biochemistry*, 4: 194-207.

Biryomumaisho, S., Katunguka-Rwakishaya, E. and Rubaire-Akiiki, C. M. (2003). Serum biochemical changes in experimental *Trypanosoma congolencse* and *Tryapanosoma brucei* infection in small east African goats. Veterinary archive, 73, 167-180.

Boniface, A., Augustine, I., Ikechukwu, I. and Paschal, U. (2011). Prevalence and haemato-biochemical parameters of trypanosome-infected pigs at Nsukka, Nigeria. Comparative Clinical Pathology. 20(1):15–18.

Bonnefont-Rousselot, D. (2004). The role of antioxidant micronutrientents in the prevention of diabetic complications. *Treat Endocrinology*, 3(1): 41-52.

Broome, C. S., Mcardle, F., Kyle, J. A. M., Andrews, F., Lowe, N. M., Hart, C, A., Arthur, J. R. and Jackson, M. J. (2004). An increase in selenium intake improves immune function and poliovirus handling in adults with marginal selenium status. *American Journal of Clinical Nutrition*, 80, 154-162.

Coopens, I., Opperdoes, F.R., Courtoy, P.J. and Baubhuin, P. (1987). Receptor- mediated endocytosis in the bloodstream form of *Trypanosoma brucei*. Journal of Protozoology. 34:465-473.

Dalla Rosa, L., Aleksandro, S.D., Camila B.O., Isabela, B., Erika, B., Fellipe, D., Jeandre, A.J., Kaio, C.S.T., Luis, C.M., Marta, R.L. and Silvia, G.M. (2012). *Trypanosoma evansi*: Effects of Zinc and Copper in experimentally infected Rats. *Experimental Parasitology* 131: 358-362

Debjit, B., Chiranjib, K.P. and Sampath, K. (2010). A potential medicinal importance of zinc in human health and chronic disease. *A Review*. *International Journal of Pharmacy Biomedical Science*, 1(1), 5-11.

DiSilvestro, R.A. (2000). Zinc in relation to diabetes and oxidative disease. *Journal of Nutrition*, 130:1509s-1511s.

Ezeokonkwo, R.C., Ezeh, I.O., Onunkwo, J.I., Onyenwe, I.W., Iheagwam, C.N. and Agu, W.E. (2012). Comparative serum biochemical changes in mongrel dogs following single and mixed infections of *Trypanosoma congolense* and *Trypanosoma brucei brucei.* Veterinary Parasitology, 190(1–2):56–61.

Hambidge, K.M., Miller, V.V., Westcott, J.E., Sheng, X. and Krebs, N.F. (2010). Zinc bioavailability and homeostasis. *Journal of Clinical Nutrition*, 91: 1478s-1483s.

Hannaert, V., Bringaud, F., Opperdoes, F.R. and Michels, P.A. (2003). Evolution of energy metabolism and its compartmentation in Kinetoplastida. *Kinetoplastid Biology Disorder*, 2:11.

Herbert, W.J. and Lumsden, W.H.R. (1976). *Trypanosoma brucei*: A rapid "matching" method for estimating the host‟s parasitemia. *Experimental Parasitology*, 40:427-431.

Hoare, C.A. (1972). *The Trypanosomes of mammals: a zoological monograph*. Blackwell Scientific Publications, Oxford UK, pp. 749.

Kapoor S.H. (2011). A new liver function test. Journal of Clinical Diagnostic Research, 5(1):155–156.

Karori, S., Ngure R.M, Wachira F.N, Wanyoko J.K and Mwangi J.N. (2008). Different types of tea products attenuate inflammation induced in *Trypanosoma brucei brucei* infected mice. Parasitology International. In press (Parint-D-00179).

Katunguka-Rwakishaya, E. (1996): The prevalence of trypanosomosis in small ruminants and pigs in a sleeping sickness endemic area of Buikwe country Mukono district, Uganda. Revue d'elevage et de medecine veterinaire des pays tropicaux, 49(1), 5658.

Kiremidjian-Schumacher, L., Roy, M., Wishe, H. I., Cohen, M. W. and Stotzky, G. (1994). Supplementation with selenium and human immune cell functions. II. Effect on cytotoxic lymphocytes and natural killer cells Biology. *Trace Element Research*, 41, 115-127.

Luk J.M, Wang X, Liu P, Wong K.F, Chan K.L, Tong Y, et al. (2007). Traditional Chinese herbal medicines for treatment of liver fibrosis and cancer: From laboratory discovery to clinical evaluation. Liver Int.; 27:879–90.

Mayer K.E, Myers R.P, Lee S.S (. 2005). Silymarin treatment of viral hepatitis: A systematic review. Journal of Viral Hepatitis; 12:559–6

Moulton, J.E. and Sollod, A.E. (1976).Clinical, serological and pathological changes in calves with experimentally induced *Trypanosoma brucei* infection. *American Journal of Veterinary Research*, 37: 791.

Mulligan, H.W. (1970). *The African trypanosomiasis*. George Allen and Unwin Ltd, UK. pp 950.

Néve, J. (1996). Selenium as a risk factor for cardiovascular diseases. *Journal of Cardiovascular Risk*, 3: 42–47.

Ogunsanmi, A. O., Akpavie, S. O and Anosa, V. O. (1994). Serum biochemical changes in West African dwarf sheep experimentally infected with *Trypanosoma brucei* Revue d'elevage et de medecine veterinaire des pays tropicaux, 47, 195-200.

Oluwatosin, K.S, Habibat U and Usman Y.U. (2013). Effect of methanolic leaf extract of *Thymus vulgaris* on some biomarker enzymes in *Trypanosoma brucei* infected rats. International Journal of Pharmaceutical and Biomedical Research, 4 (2), 83-87.

Powell, S.R., (2000). The antioxidant properties of zinc. *Journal of Nutrition*, 130: 1447-1454.

Rajamanickam, V and Muthuswamy, N. (2008). Effect of heavy metals induced toxicity on metabolic biomarkers in common carp (*Cyprinus carpio* L.). Maejo International Journal of Science and Technology, 2(01), 192-200.

Rayman, M.P. (2012). Selenium and human health. *A Review*. *Lancet.* 379:1256-68.

Sahin, K. and Kucuk, O. (2003). Zinc supplementation alleviates heat stress in laying Japanese quail. *Journal of Nutrition*, 133: 2808-2811.

Sazmand, A., Aria, R., Mohammad, N., Hosein, H and Seyedhossein, H. (2011). Serobiochemical alterations in subclinically affected dromedary camels with *Trypanosoma evansi* in Iran. Pakistan Veterinary Journal, 31(3):223–226.

Sidhu, P., Garg, M.L. and Dhawan, D.K. (2004). Protective effects of zinc on oxidative stress enzymes in liver of protein deficient rats. *Nutrición Hospitalaria*, 19(6): 341-347.

Simpson, A.G., Stevens, J.R. and Lukes, J. (2006). The evolution and diversity of kinetoplastid flagellates. *Trends in Parasitology*, 22:168–74.

Stevens, J.R and Brisse, S. (2004). Systematics of trypanosomes of medical and veterinary importance. In: Maudlin, I., Holmes, P.H. and Miles, M.A., (Ed). *The trypanosomiasis.* CABI Publishing, Wallingford, pp. 1-23.

Swallow, B.M. (2002). Impacts of Trypanosomiasis on African agriculture. PAAT Technical and Scientific Series 2. Food and Agriculture Organisation (FAO), Rome. pp. 52.

Taiwo, V.O., Olaniyi, M.O and Ogunsanmi, A.O. (2003). Comparative plasma biochemical changes and susceptibility of erythrocytes to in vitro peroxidation during experimental *T. congolense* and *T. brucei* infections in sheep. Israel Journal of Veterinary Medicine, 58, 1-10.

Umar, I.A., Ogenyi, E., Okodaso, D., Kimeng, E., Stancheva, G.I., Omage, J.J., Isah, S. and Ibrahim, M.A. (2007). Amelioration of anaemia and organ damage by combined intraperitoneal administration of vitamins A and C to *Trypanosoma brucei brucei* - infected rats. African Journal of Biotechnology, 6:2083-2086.

Usha, K., Mary, K.G. and Hemalatha, P. (2008). Hepatoprotective effect of *Hygrophila spinosa* and *Cassia occidentalis* on carbon tetrachloride induced liver damage in experimental rats. Indian Journal of Clinical Biochemistry, 22(2): 132-135.

Vasudevan, D.M. and Sreekumari, S. (2007). Textbook of Biochemistry for Medical Students. 5th Ed. Jaypee Brothers, Medical Publishers Ltd, New Delhi, 348-349.

Welburn, S.C., Coleman, P.G., Maudlin, I., Fevre, E.M., Odiit, M. and Eisler, M.C. (2006). Crisis, what crisis? Control of Rhodesian sleeping sickness. *Trends in Parasitology*, 22(3): 123-128.

World Health Organization. (2008). Control of Neglected Tropical Diseases (NTD). On-line [http://www.int/neglected-diseases/faq/en/index4.html]

Yusuf, A.B., Umar I.A. and Nok A.J. (2012). Effects of methanol extract of Vernonia amygdalina leaf on survival and some biochemical parameters in acute *Trypanosoma brucei brucei* infection. African Journal of Biochemistry Research, 6(12):150–158.

Zago, M. and Oteiza, P.I. (2001). The antioxidant properties of zinc interactions with iron and antioxidants. *Free Radical Biology and Medicine*, 31: 266-274.

Zhou, Z., Kang, X., Jiang, Y., Song, Z., Feng, W., Mcclain, C.J. and Kang, Y.J. (2007). Preservation of hepatocyte nuclear factor-4á is associated with zinc protection against TNF-á hepatotoxicity in mice. *Experimental Biology and Medicine*, 232: 622-28, 32.

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