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


**Department of Veterinary Medicine, University of Nigeria, Nsukka, Nigeria**

**Department of Veterinary Medicine, University of Abuja, Abuja, Nigeria**

**Department of Animal Health and Production, University of Nigeria, Nsukka, Nigeria**

**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.

**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.

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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...
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 trypanotolerance 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 hampered by numerous limitations such as toxicity, prohibitive cost and development of resistance by the parasites (Clarkson et al., 1984; Anene et al., 2001). Also, trypanosomiasis 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 antienthythrocyte antibodies), hemolysis, non-specific reticuloendothelial system activation, direct traumatic effect of trypanosomes, microangiopathy associated with disseminated intravascular coagulation, splenic phagocytosis and splenic pooling, erythropagocytosis, 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 trypanosomiasis 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 the all 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 (Broomé 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 follows; groups A, B, C and D were infected with 0.5 ml of saline diluted trypanosome infected rat blood containing about 1 × 10⁶ 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 Eppendorf tube. The erythrocytic profile and parasitaemia were determined on day 0 and every 7th day, while deaths were recorded as it occurred. The blood samples were analyzed for the following parameters: packed cell volume (PCV) and hemoglobin (Hb) concentration.
PCV was determined using the microhematocrit method while hemoglobin concentration was determined spectrophotometrically by the cyanometemoglobin method (Jain 1986) using SP6-500UV spectrophotometer (PYE UNICAM, England). Parasitemia was estimated using the matching method of Herbert and Lumsden, 1976.

2.7. Determination of serum protein, albumin and globulin

Following centrifugation, the supernantant (serum) was separated into separate tubes. The serum was used for the determination of total protein, albumin and globulin levels in each group. Total serum protein concentration was determined by the biuret method as described by Weichselbaum (1946) using the standard Randox® diagnostic kit (Randox Laboratories LED, U.K.). The albumin fraction was determined spectrophotometrically using SP6-500UV spectrophotometer (PYE UNICAM, England). Globulin fraction of the serum was calculated by subtracting the albumin fraction from the total serum protein.

2.8. Survivability

The rats in different groups were monitored and time of death of each rat recorded. The survival rate was recorded as the number of rats alive over the number in the group.

2.9. Statistical analysis

Data generated were presented as means with standard deviation. The data were subjected to analysis of variance (ANOVA). Means were considered significant at *P* < 0.05 and the means separated using Duncan’s multiple range test.

3. Result

The selenium supplementation at 4 and 8 ppm led to increase in mean PCV (Table 1) of 4 ppm, 8 ppm groups on day 7 of selenium supplementation (SS) when compared with the infected not supplemented groups though not statistically significant. Following infection with Trypanosoma brucei on day 14 of selenium supplementation, the PCV of 16 ppm and infected not supplemented groups were significantly (*P* < 0.05) lower than other groups on days 28 and 35 of SS. On days 42 and 49 of SS, the PCV of 4 ppm supplemented groups was significantly (*P* > 0.05) lower than other groups in total protein and albumin from day 28 of SS. The 16 ppm supplemented group was significantly (*P* < 0.05) lower in total protein and albumin values on days 28 and 35 of SS when compared with other groups. The infection did not produce any significant (*P* > 0.05) changes on globulin values (Table 5). The supplementation did not significantly (*P* > 0.05) increase the pre-putent period of the supplemented groups. However, the supplementation led to a sig-

<table>
<thead>
<tr>
<th>Day</th>
<th>PS 4 ppm</th>
<th>8 ppm</th>
<th>16 ppm</th>
<th>D infected not supplemented</th>
<th>E not infected not supplemented</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>38.2 ± 4.96 37.4 ± 2.60</td>
<td>36.0 ± 3.24</td>
<td>35.6 ± 2.70</td>
<td>37.4 ± 2.70</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>39.0 ± 2.91 38.4 ± 3.43</td>
<td>38.2 ± 3.11</td>
<td>34.4 ± 3.84</td>
<td>36.8 ± 2.16</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>38.6 ± 2.07 38.4 ± 2.96 38.6 ± 4.39</td>
<td>35.2 ± 2.30</td>
<td>36.9 ± 2.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>33.8 ± 3.89 32.4 ± 3.20 30.6 ± 3.20</td>
<td>33.6 ± 0.89</td>
<td>32.4 ± 3.91</td>
<td></td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>34.0 ± 1.43 33.2 ± 2.63 27.8 ± 2.73</td>
<td>23.0 ± 1.58</td>
<td>34.0 ± 2.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>32.0 ± 3.54 32.8 ± 2.63 22.0 ± 4.94</td>
<td>19.4 ± 1.39</td>
<td>36.0 ± 7.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>42</td>
<td>28.0 ± 1.01 26.2 ± 4.83 17.9 ± 2.98</td>
<td>15.2 ± 7.62</td>
<td>35.4 ± 8.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>49</td>
<td>23.4 ± 2.58 23.9 ± 2.03 15.8 ± 1.98 16.9 ± 2.07</td>
<td>36.6 ± 1.51</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 1: Mean packed cell volume (%) of rats infected with T. brucei and fed feed containing different levels of selenium.

Table 2: Mean Hb (g/dl) concentration rats infected with T. brucei and fed feed containing different levels of selenium.

<table>
<thead>
<tr>
<th>Day</th>
<th>PS 4 ppm</th>
<th>8 ppm</th>
<th>16 ppm</th>
<th>D infected not supplemented</th>
<th>E not infected not supplemented</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>10.74 ± 0.97 11.94 ± 0.71</td>
<td>11.32 ± 2.02</td>
<td>10.48 ± 1.09</td>
<td>10.82 ± 0.81</td>
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</tr>
<tr>
<td>7</td>
<td>11.25 ± 2.99 10.46 ± 3.06</td>
<td>11.50 ± 1.98</td>
<td>9.22 ± 2.51</td>
<td>10.72 ± 2.79</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>12.02 ± 1.54 13.32 ± 1.93 12.94 ± 1.05</td>
<td>10.82 ± 1.70</td>
<td>11.32 ± 0.70²</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>11.74 ± 0.11 13.50 ± 0.84</td>
<td>11.98 ± 1.23</td>
<td>11.20 ± 1.63</td>
<td>9.78 ± 1.84</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>11.66 ± 1.31 12.66 ± 1.58</td>
<td>8.76 ± 2.42</td>
<td>7.52 ± 2.13</td>
<td>10.90 ± 2.67²</td>
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</tr>
<tr>
<td>35</td>
<td>10.01 ± 4.11 9.88 ± 4.48 8.70 ± 3.8</td>
<td>6.62 ± 4.28</td>
<td>9.68 ± 1.94</td>
<td></td>
<td></td>
</tr>
<tr>
<td>42</td>
<td>8.55 ± 2.12 7.84 ± 4.42 6.23 ± 3.97</td>
<td>5.82 ± 5.58</td>
<td>11.08 ± 0.94b</td>
<td></td>
<td></td>
</tr>
<tr>
<td>49</td>
<td>7.44 ± 3.14 5.52 ± 0.98 4.09 ± 2.10</td>
<td>3.62 ± 3.62</td>
<td>9.96 ± 1.21b</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Mean total protein (g/dl) concentration of rats infected with T. brucei and fed feed containing different levels of selenium.

<table>
<thead>
<tr>
<th>Day</th>
<th>PS 4 ppm</th>
<th>8 ppm</th>
<th>16 ppm</th>
<th>D infected not supplemented</th>
<th>E not infected not supplemented</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>7.24 ± 1.00 6.66 ± 0.50</td>
<td>5.60 ± 1.50</td>
<td>6.84 ± 1.24</td>
<td>6.94 ± 0.70</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>7.14 ± 0.86 6.58 ± 1.86</td>
<td>8.74 ± 1.87</td>
<td>7.91 ± 1.87</td>
<td>7.50 ± 1.30</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>8.60 ± 1.34 8.06 ± 1.10 7.10 ± 1.04</td>
<td>8.72 ± 0.89</td>
<td>8.22 ± 1.37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>7.18 ± 0.54 6.40 ± 0.40</td>
<td>6.30 ± 0.50</td>
<td>6.60 ± 0.65</td>
<td>8.33 ± 0.86</td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>5.72 ± 0.64 5.56 ± 0.27 3.92 ± 1.00</td>
<td>3.36 ± 1.17</td>
<td>7.04 ± 1.38²</td>
<td></td>
<td></td>
</tr>
<tr>
<td>35</td>
<td>4.59 ± 2.63 3.86 ± 0.42 2.78 ± 1.74</td>
<td>2.47 ± 0.18</td>
<td>7.80 ± 0.79²</td>
<td></td>
<td></td>
</tr>
<tr>
<td>42</td>
<td>3.32 ± 1.31 3.55 ± 1.29 2.32 ± 1.89</td>
<td>3.20 ± 1.87</td>
<td>7.28 ± 0.79</td>
<td></td>
<td></td>
</tr>
<tr>
<td>49</td>
<td>1.96 ± 2.68 2.33 ± 1.09</td>
<td>2.22 ± 1.09</td>
<td>1.86 ± 1.92</td>
<td>6.67 ± 0.60b</td>
<td></td>
</tr>
</tbody>
</table>

Table 4: Mean serum albumin (g/dl) concentration of rats infected with T. brucei and fed feed containing different levels of selenium.

PS: Different superscripts in a row indicates significant difference between the means at the level of probability: *P* < 0.05; PS means post supplementation.

Other groups. The infection with trypanosomes on day 14 of SS led to a decrease in the Hb concentration with 16 ppm group being significantly (*P* < 0.05) lower than other groups on day 28. Uninfected, not supplemented group maintained significantly (*P* < 0.05) higher Hb than other groups on days 42 and 49 of SS. The supplementation with selenium did not lead to significant (*P* > 0.05) increase in the total protein, albumin and globulin (Tables 3–5) by day 14 of SS. The infection with trypanosomes on day 14 of SS led to significant (*P* > 0.05) decrease in the total protein and albumin from day 28 of SS. The 16 ppm supplemented group was significantly (*P* < 0.05) lower in total protein and albumin values on days 28 and 35 of SS when compared with other groups. The infection did not produce any significant (*P* > 0.05) changes on globulin values (Table 5). The supplementation did not significantly (*P* > 0.05) increase the pre-putent period of the supplemented groups. However, the supplementation led to a sig-
significant reduction in the parasitaemia levels (Fig. 1) of 4 ppm and 8 ppm groups on day 28 and 35 of SS when compared with the control. However, 16 ppm group had significantly ($P < 0.05$) higher parasitemia than 4 ppm, 8 ppm and infected not supplemented groups on days 35 and 42 of SS.

The survival patterns of the mice groups showed that the first death was recorded in infected not supplemented group on day 32 of SS. However, by day 56, all mice in infected not supplemented group (100%) have died as well as 3/5 (60%), 4/5 (80%) and 2/5 (40%) rats survived from 4 ppm, 8 ppm and 16 ppm groups, respectively.

### 4. Discussion

The supplementation with selenium at 4, 8 and 16 ppm of rats’ diet improved the packed cell volume and hemoglobin concentration of the supplemented groups prior to infection on day 14 PS. This could be attributed to the immunostimulatory and anti-oxidative properties of selenium (Sidhu et al., 1993; Miller et al., 2001; McKeever and Britton, 2004). According to McKeever and Britton (2004), selenium is a powerful destroyer of free radicals and is also involved in antioxidant defenses as a coenzyme in glutathione peroxidase. Selenium exerts its protective effect against oxidative damage by decreasing the amount of free radicals and increasing the synthesis of glutathione peroxidase, which catalyzes the breakdown of toxic hydrogen peroxide and lipid hydroperoxides (Sardessai, 2003). Generally, antioxidants help in maintenance and stabilization of lipid membrane from peroxidative damage by inhibition and destruction of endogenous peroxides (Koller and Exon, 1986; Umar et al., 1999).

The infection with trypanosomes on day 14 post supplementation however led to, progressive decrease in PCV and Hb

### Table 5
Mean serum globulin (g/dl) concentration of rats infected with *T. brucei* and fed feed containing different levels of selenium.

<table>
<thead>
<tr>
<th>Day PS</th>
<th>A 4 ppm</th>
<th>B 8 ppm</th>
<th>C 16 ppm</th>
<th>D infected not supplemented</th>
<th>E not infected not supplemented</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1.60 ± 0.74</td>
<td>3.02 ± 0.98</td>
<td>2.56 ± 1.34</td>
<td>1.26 ± 1.18</td>
<td>1.66 ± 0.41</td>
</tr>
<tr>
<td>7</td>
<td>1.74 ± 0.33</td>
<td>1.40 ± 0.84</td>
<td>2.20 ± 0.29</td>
<td>2.23 ± 1.74</td>
<td>1.30 ± 0.90</td>
</tr>
<tr>
<td>14</td>
<td>1.46 ± 1.14</td>
<td>1.96 ± 1.06</td>
<td>1.18 ± 0.58</td>
<td>2.02 ± 0.77</td>
<td>1.78 ± 1.20</td>
</tr>
<tr>
<td>21</td>
<td>2.31 ± 0.31</td>
<td>1.96 ± 0.56</td>
<td>2.08 ± 0.67</td>
<td>2.00 ± 0.65</td>
<td>2.14 ± 0.78</td>
</tr>
<tr>
<td>28</td>
<td>1.20 ± 0.63</td>
<td>1.24 ± 1.40</td>
<td>1.18 ± 1.70</td>
<td>1.94 ± 1.14</td>
<td>1.92 ± 0.52</td>
</tr>
<tr>
<td>35</td>
<td>1.20 ± 1.41</td>
<td>1.54 ± 1.03</td>
<td>1.32 ± 0.71</td>
<td>1.57 ± 2.37</td>
<td>1.90 ± 0.74</td>
</tr>
<tr>
<td>42</td>
<td>1.84 ± 2.66</td>
<td>1.91 ± 1.81</td>
<td>1.00 ± 1.02</td>
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<td>2.72 ± 0.45</td>
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<td>49</td>
<td>0.54 ± 0.77</td>
<td>1.10 ± 0.98</td>
<td>0.96 ± 0.97</td>
<td>0.98 ± 1.02</td>
<td>1.26 ± 1.04</td>
</tr>
</tbody>
</table>

![Fig. 1. Mean parasitemia of *Trypanosoma brucei* infected rat groups given different levels selenium in their diet (10^6 trypanosomes/ml of blood).](image-url)
concentration of the infected groups. Anemia which the decrease indicates is a common feature of trypanosomosis. (Anosa, 1988). However, the anemia in selenium supplemented groups was less severe than the one recorded for the unsupplemented infected rats. This may be due to the relatively lower parasitemia recorded in the supplemented group, since the degree of anemia in trypanosomosis has been positively correlated with the onset and level of parasitemia (Dargie et al., 1979). The decrease in PCV and Hb concentration in the 16 ppm group may be due to the toxicity of selenium. This justifies previous results that supplementation with antioxidants (vitamin C and/or vitamin E) reduced anemia in trypanosomosis (Umar et al., 2000, 2001).

The supplementation with selenium did not lead to significant increase in the total serum protein, albumin and globulin by day 14 on the SS. This agrees with the finding of Kumar and Clark (2001) that selenium supplementation has no significant effect on the levels of serum total protein, albumin and globulin and albumin/globulin ratio. The primary function of selenium in animal systems is as a component of the antioxidant enzymatic cascade responsible for protection of cells from potentially damaging lipid peroxides, oxygen and nitrogen free radicals and immunomodulation. Thus, the slight increase noticed may be due to proper functioning of the animal system and improved immune response.

However, the infection with trypanosomes on day 14 PS led to significant decrease in the total protein and albumin from day 28. This is in agreement with some reports (Anosa, 1988; Katunguka-Rwakishaya et al., 1999; Osaer et al., 2000) but differs with reports that trypanosomosis led to elevated serum protein and albumin (Kalu et al., 1989; Orhue et al., 2005; Ekanem and Yusuf, 2008). The decrease in serum total protein could be attributed to a decrease in serum albumin probably from decreased hepatic biosynthesis, plasma expansion, proteinuria (Bruitin et al., 1987) or hepatocellular damage (Saror, 1980). Also, uptake of albumin-bound fatty acids and lipoproteins (Vickerman and Tetley, 1979) may lead to decrease in plasma albumin concentrations in trypanosome infected animals. The decline in albumin could also be due to initiation of the immune response and synthesis of immunoglobulins (Katunguka-Rwakishaya et al., 1999).

According to Adah et al., 1993, T. brucei induces an elevation of serum transaminases which indicates that the infection causes reduction in liver function including protein synthesis. The supplemented groups maintained higher protein and albumin levels which may suggest the ability of selenium to prevent hepatocellular damage caused by the trypanosome infection. Selenium supplementation led to significant suppression of parasitemia in supplemented groups. Ability of selenium supplementation to reduce T. brucei parasitemia has been reported (Davis et al., 1998; Eze, 2007). Selenium is an antioxidant and immunostimulant (Elango et al., 2006; Ortac et al., 2006) which is necessary for maximum performance of the immune system needed to control parasite proliferation. Deficiency of selenium impairs both circulating (humeral) and cell-mediated immunity. Estimation of parasitemia has become an important tool in understanding the pathogenesis of trypanosomosis because increase in population of the parasites corresponds with rise in rectal temperature, weight loss, decline in packed cell volume and decrease in total plasma protein in all the infected groups. The main mechanism, generally considered to mediate parasitemia control in a mammalian host, is the continuous interaction between antibodies and the parasite surface, covered by variant specific surface glycoproteins (Gjini et al., 2010). The effectiveness of selenium in suppression of parasitemia while it was not able to clear it may be due to the fact that specific immune response to trypanosomes is limited by the ability of the parasite to undergo antigenic variation leading to persistence of the infection (Gjini et al., 2010).

The prolonged survival intervals of rats in the groups supplemented with selenium could be attributed to the beneficial effect of selenium. This is in agreement with result obtained by Davis et al. (1998) in T. cruzi infected mice. The ability of selenium supplementation to prolong the survival intervals could be attributed to its immunostimulatory (Eze et al., 2011) and antioxidant effects (McKeever and Britton, 2004) effects.

From the result, it is evident that selenium supplementation has beneficial effects on the anemia, serum proteins, survival pattern and parasitemia of Trypanosoma brucei brucei infected rats. However, lower doses of 4 and 8 ppm seem to be better than 16 ppm as they improved the parameters studied more in trypanosome infected rats.

References


