COMBATING THE NEGATIVE EFFECTS OF Trypanosoma Bruclei-bruce (Federe strain) INFECTION IN WISTAR RATS USING SELECTED ANTI-OXIDANTS AND TRYPAMIDUM

Abstract: Combating the negative effects of Trypanosoma bruclei-bruce (Federe strain) infection in Wistar rats using antioxidants and Trypamidium was investigated. Thirty Wistar rats with average weights of 170-200±20 g were randomly divided into six groups. Group A uninfected, group B, C, D, E and F were inoculated with 1 × 104 of T. brucei brucei. Group A rats were not infected and not treated, Group B rats were infected but not treated, Group C rats were infected and treated with 1 mg of Trypamidium, Group D rats were infected and treated with 4.7mg of vitamin B-complex, Group E rats were infected and treated with 10mg of vitamin K, while Group F rats were infected and treated with 4.7mg of vitamin B-complex tablet and 10mg of vitamin K tablet combined together respectively, the treatment were given after three days post infection daily, orally except Group C which were administered intramuscularly. Body weight of the animals observed decrease significantly (P < 0.05) in group B at 9-day post infection (9DPI) and (12DPI), and in groups D, E and F at 12DPI compared to group A not infected and not treated. Packed cell volume of the rats decreased significantly (P < 0.05) in all the groups infected at 6DPI and 12DPI compared to group A, but increased towards the end of the experiment. The weight of the liver and spleen in all the groups infected increased significantly (P < 0.05) when compared to group A, but heart and lung remained unchanged. In conclusion oral administration of the antioxidants prevented the decrease in body weight, packed cell volume and organ weight associated with Trypanosoma bruclei-bruce infection in Wistar rats.

Keywords: Trypamidium; weight; Trypanosoma bruclei brucei; Wistar rats

Introduction
African trypanosomiasis is a debilitating disease that has been ravaging both man and animal for ages. The disease is localized in some regions of sub-Saharan Africa between latitude 14° N and 29° S with expression in 37 countries with about 60 million people and 48 million cattle at risk of the infection (World Health Organization, 2005).
disease is caused by a protozoan parasite of the genus *Trypanosoma* spp. The species of interest are *Trypanosoma brucei gambiense*, and *Trypanosoma brucei rhodesiense* which predominantly affect humans in Western-Central and Eastern-Southern Africa, respectively causing 'sleeping sickness'; while *T. congolense, T. vivax, T. evansi* and *T. brucei brucei* are the major parasites that cause disease called 'nagana or sammore' in livestock (Grebaut *et al.*, 2009). The disease is classified as human African trypanosomiasis (HAT) and animal African trypanosomiasis (AAT) and both are transmitted by the vector called *Glossina* spp. (tsetse fly) which is commonly found in rainforest, savannah and woodland ecological zones (Nagamune *et al.*, 2004). Nagana is transmitted via blood sucking tsetse and has clinical sign such as cachetism, undulating fever, lymphadenopathy, anaemia, rough hair coat and in some cases alopecia of infected skin regions (Nagamune *et al.*, 2004). It has been documented that anatomicopathologically, disease is produced as a result of cellular damage of tissues and organs such as the blood, liver, kidney and the lymph nodes through the excess production of free radicals, like the peroxides, superoxides and the low level glutathione (GSH), thereby decreasing the plasma level of ascorbic acid (Umar *et al.*, 2000). Although, it has been reported that a good dieting for infected and uninfected animals will cause both to grow at the same rate (Holmes *et al.*, 2000). Continuous increase in lipid peroxidation, generation of free radicals and reduced systemic antioxidants as a result of untreated infection leads to systemic oxidative stress and these results into the clinical manifestation of the trypanosomiasis (Eze *et al.*, 2008). In Africa, several strains of *T. brucei brucei* have been reported such as the Basa, Mkar/84/NITR/6, TREU 667, Federe 2001 strain and so on. But the Federe 2001 strain has been acclaimed to be highly virulent particularly in the northern region of Nigeria causing increased livestock mortality (Wurochekke and Anyanwu, 2012). The control of trypanosomiasis has been particularly difficult as trypanocidal drugs have not been satisfactory in their actions against the parasites due to the following reasons: resistance development by the parasites, toxicity of drugs to the host, high cost of the existing drugs and slow of discovery of new drugs (Kennedy *et al.*, 2002). However, past research results has revealed that the destruction of homeostasis as a result of oxidative stress may be corrected if the body system is supplemented with natural antioxidants, such as vitamin C (Pietta, 2000). Other research efforts have documented that oxidative stress was prevented in the aqueous compartment and lipid bilayer of cell membranes by antioxidants such as vitamins A and C which are lipid soluble and water-soluble respectively (Packer *et al.*, 2001). Therefore, this study was carried out to investigate the effect of Trypamidium, vitamins B-complex and vitamin K on the pack cell volume (PCV), live body weight and organs weight in Wistar rats infected with *T. brucei brucei* (Federe strain).
Materials and Methods
Experimental Site
The study was conducted at the Nigerian Institute for Trypanosomiasis and Onchocerciasis Research (NITR) Kaduna, Nigeria, which is situated within latitude 10° 30' 00" N and longitude 7° 25' 50" E.

Experimental Animals (Rats)
Thirty (30) adult (10 weeks of age) Wistar rats of average weight between 170 and 200 g were purchased from the rat's colony of the Nigerian Institute for Trypanosomiasis Research (NITR).

Grouping of the Animals (Rats)
The rats were randomly divided into six groups, group A negative control, group B positive control, while C, D, E and F were the groups tested. Each group contained five rats kept in well ventilated plastic cages in the laboratory, dewormed with albendazole through their drinking water and acclimatized to laboratory condition for two weeks before commencement of the experiment. The animals were fed with a pelleted basal diet obtained from a commercial feed outlet (Vital Feeds Plc., Kaduna, Nigeria) and water was given (ad-libitum).

Source of the Parasites
The parasite Trypanosoma brucei brucei (Federe strain) was obtained from the cryopreserved stabilates kept in vector and parasitology Department of Nigerian Institute for Trypanosomiasis Research (NITR), Kaduna, Nigeria.

Inoculation of the Parasites into Donor Rat
The T. brucei brucei was inoculated into a clean rat free of worms through deworming with albendazole which served as donor rat. Infected blood from a donor rat at peak parasitaemia (4 days post infection (DPI) was collected by means of tail picking and diluted with cold physiological saline (Normal saline 0.9% w/v). The number of parasites in the diluted blood were determined through the method described by Herbert and Lumsden (1976) and a volume of blood diluted with Normal saline containing approximately $1 \times 10^7$ cells of trypanosomes was injected intraperitoneally into each rat in the group B, group C, group D, group E and group F using syringe and needle, while group A remained uninfected which served as control (Ajakaiye et al., 2013).
Preparation of the antioxidants and the trypanidium for treatment
The antioxidants (vitamin B-complex and vitamin K) and the Trypanidium used were obtained from a commercial outlet in Kaduna, Nigeria. Four point seven milligram (4.7mg) of vitamin B complex tablet containing vitamins B1, 0.5mg, B2, 0.1mg, B6, 1mg and Nicotinamide 3mg was first dissolved in 10 ml of distilled water and given as drinking water in the morning. The same procedure above was followed in preparing ten milligram (10 mg) of vitamin K tablet which contained menadione sodium bisulphate. While one milligram (1mg) of trypanidium was dissolved and reconstituted in distilled water according to manufacturer’s instruction and was injected intramuscularly from the onset of parasitaemia. The vitamin B-complex is a product of Nigerian Company (Franoson Mannyon int’l Co Ltd, Anambra state Nigeria) and the vitamin K is a product of Indian Company (Mancare Pharm. Pvt Ltd, India). While the Trypanidium is product of the France Company (Merial, Lyon, France).

Treatment of the Infected Groups
Group A rats were not infected and not treated, Group B rats were infected but not treated, Group C rats were infected and treated with 1 mg of Trypanidium, Group D rats were infected and treated with 4.7 mg of vitamin B-complex, Group E rats were infected and treated with 10 mg of vitamin K, while Group F rats was infected and treated with 4.7 mg of vitamin B-complex tablet and 10 mg of vitamin K tablet combined together respectively, the treatment were given daily after three days post infection through the method adopted by Ajakaiye et al. (2013). Regardless of the quantity each rat drunken in the group, but anticipating that they drank approximately equal amount.

Blood Samples and Organs Collection
Blood samples were collected from the tail of each rat in the groups daily using heparinised tubes (Capillary tubes) to check the packed cell volume (PCV) using microhaematocrit centrifuge technique (HCT) through the method described by Hettich laboratory technology (2016) in which the capillary tubes were arranged in a rotor and spun at maximum speed of 12,000 revolution per minute, then reading were taken using microhaematocrit reader, and parasitaemia levels which was estimated by wet mount method described by Herbert and Lumsden (1976) in which blood from the tail of the rats were placed on a clean slide and covered with cover slip and directly observed microscopically using rapid matching method at ×400 magnification and involved the counting of the number of parasites per field in pure blood. On 28 days post infection (DPI), the rats were sacrificed by humane decapitation prior anaesthesia with sterile cotton impregnated chloroform and organs were removed aseptically from all the groups and kept in 10% buffered formalin adopted by Ajakaiye et al. (2013).
Measurement of live Body Weight
The live body weight of the rats in all groups were taken twice per week throughout the experimental period with the aid of a standard weighing balance with a maximum calibration of 5kg as described by Ajakaiye et al. (2013), in which each rat is placed gently on the weighing balance, allowed to stand for some period and then the reading were taken.

Measurement of Organ Body Weight
Organ weight of the rats in all groups were measured using Smart Weight Digital Pocket Scale (SWS 100) New York USA, Made in China. Gently, each individual organ were placed on the weighing balance and were allowed for a period of time to display the value of each organ obtained were recorded immediately as described by Ajakaiye et al. (2013).

Statistical Analysis
All the data obtained from this experiment are presented as mean ± standard deviation (SD). Data were analyzed by the one-way analysis of variance (ANOVA) and the difference between experimental groups were compared using Duncan (1955) post-hoc test and mean values that differs at p < 0.05 were considered significant.

Results
The results of live body weight recorded in all the groups within the period of this study are shown in Table 1. Significant (P < 0.05) decrease in body weight was observed in group B at 9 days post infection (9DPI) (160.0 ± 6.3) and 12DPI (1440.0 ± 6.8), and in groups D (156.0 ± 8.1), E (156.0 ± 9.3) and F (168.0 ± 8.6) at 12DPI when compared to group A. Reduction in body weight was highly significant (P < 0.05) in group B as observed at 12DPI (1440.0 ± 6.8). However, in the case of all the groups, there was no significant (P > 0.05) difference observed at 3DPI and 6DPI when compared to the initial values of body the body weight (pre-infection).

The results of Packed Cell Volume (PCV) of all the Wistar rats groups obtained in this experiment are presented in Table 2. Packed Cell Volume reduced significantly (P < 0.05) in all the groups infected at 6DPI and 12DPI compared to uninfected group. The level of significance observed is more pronounced in group B which is infected untreated at 6DPI (43.0 ± 1.1) and 12DPI (43.0 ± 0.9), as well as in groups D (44.4 ± 0.7) and E (44.4 ± 0.8) at 12DPI when compared to the group infected and treated with the Trypamidium. But towards tail end of the experiment the group C treated with
Trypanosoma brucei-brucei (Federer strain) Infection in Wistar Rats Using Selected Anti-oxidants and Trypamidium

Trypamidium and the group F treated with Vitamin B-complex and Vitamin K combined together, increased significantly (P < 0.05) at 12DPI (48.8 ± 1.6) and (47.4 ± 1.1) which is higher than the groups treated with single antioxidant respectively. However there is no statistically significant (P < 0.05) difference observed in all the groups of Wistar rats at 3DPI and 6DPI when compared to the initial values of PCV recorded.

Table 3: showed the results of visceral organs weight of experimental animals across the various treatment groups. The organ-body weight of the liver and spleen as observed in all the groups infected increased significantly (P < 0.05) when compared to uninfected untreated group (Liver 7.28±0.11 and Spleen 0.81±0.02), but the increase is significant (P < 0.05) in group B. (Liver 11.5±0.50 and Spleen 2.84±0.32) compared to the groups infected and treated. However, there was no significant (P > 0.05) increase observed in the organ-body weight of kidney in all the groups (i.e. infected not treated group, infected treated groups and uninfected untreated group). Statistically significant (P < 0.05) difference in the organ-body weight of lung was observed only in groups B (2.56 ± 0.62) and E (2.56 ± 0.73) compared to rest of the groups. Significant (P < 0.05) difference in the organ-body weight of heart was observed only in groups B (1.44 ± 0.36), D (1.04 ± 0.14) and E (1.04 ± 0.14) when compared to groups A (0.78 ± 0.03), C (0.81 ± 0.10) and F (0.87 ± 0.12) respectively.

Table 1: Live Body Weight (g) of Experimental Wistar Rats (animals) across the various Treatment Groups at Different Time

<table>
<thead>
<tr>
<th>Groups</th>
<th>Uninfected untreated</th>
<th>Infected untreated</th>
<th>Trypamidium treated</th>
<th>Vitamin B treated</th>
<th>Vitamin K treated</th>
<th>Vitamin B+K treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>174.0 ± 2.5a</td>
<td>174.0 ± 4.0a</td>
<td>172.0 ± 2.0a</td>
<td>176.0 ± 2.0a</td>
<td>174.0±2.4a</td>
<td>173.7 ± 1.0a</td>
</tr>
<tr>
<td>3DPI</td>
<td>176.0 ± 5.1a</td>
<td>176.0 ± 5.1a</td>
<td>176.0 ± 5.1a</td>
<td>180.0 ± 7.1a</td>
<td>176.0±5.1a</td>
<td>180.0 ± 7.1a</td>
</tr>
<tr>
<td>6DPI</td>
<td>184.0 ± 3.7a</td>
<td>180.0 ± 3.2a</td>
<td>180.0 ± 7.1a</td>
<td>178.0 ± 5.8a</td>
<td>182.0±3.7a</td>
<td>182.0 ± 5.8a</td>
</tr>
<tr>
<td>9DPI</td>
<td>188.0 ± 3.7a</td>
<td>160.0 ± 6.3b</td>
<td>174.0 ± 4.0a</td>
<td>174.0 ± 5.1a</td>
<td>172.0±5.8a</td>
<td>172.0 ± 8.6a</td>
</tr>
<tr>
<td>12DPI</td>
<td>190.0 ± 6.3a</td>
<td>144.0 ± 6.8b</td>
<td>182.0 ± 8.0a</td>
<td>156.0 ± 8.1b</td>
<td>156.0±9.3b</td>
<td>168.0±8.6ab</td>
</tr>
</tbody>
</table>

Results are given as mean ± SD. In each row, values with different superscripts have statistically significant difference (p < 0.05).
Table 2: Packed Cell Volume (PCV) of Experimental Wistar Rats (animals) across the various Treatment Groups at Different time

<table>
<thead>
<tr>
<th>Groups</th>
<th>Uninfected untreated</th>
<th>Infected untreated</th>
<th>Trypanidium treated</th>
<th>Vitamin B treated</th>
<th>Vitamin K treated</th>
<th>Vitamin B + K treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial</td>
<td>56.4 ± 4.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>51.0 ± 3.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>53.0 ± 4.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>48.2 ± 1.5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>50.0 ± 6.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>48.8 ± 1.4&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>3DPI</td>
<td>55.0 ± 3.7&lt;sup&gt;a&lt;/sup&gt;</td>
<td>49.0 ± 1.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>50.0 ± 2.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>48.2 ± 1.5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>49.4 ± 2.9&lt;sup&gt;a&lt;/sup&gt;</td>
<td>47.8 ± 1.3&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>6DPI</td>
<td>57.0 ± 3.8&lt;sup&gt;a&lt;/sup&gt;</td>
<td>46.0 ± 1.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>50.6 ± 1.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>49.0 ± 2.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>50.6 ± 2.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>49.2 ± 2.1&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>9DPI</td>
<td>50.6 ± 1.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>43.0 ± 1.1&lt;sup&gt;b&lt;/sup&gt;</td>
<td>48.8 ± 1.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>48.2 ± 1.6&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>48.6 ± 1.8&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>48.6 ± 1.3&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>12DPI</td>
<td>52.0 ± 3.0&lt;sup&gt;a&lt;/sup&gt;</td>
<td>43.0 ± 0.9&lt;sup&gt;b&lt;/sup&gt;</td>
<td>48.8 ± 1.6&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>44.4 ± 0.7&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>44.4 ± 0.8&lt;sup&gt;b&lt;/sup&gt;</td>
<td>47.4 ± 1.1&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Results are given as mean ± SD. In each row, values with different superscripts have statistically significant difference (P< 0.05).

Table 3: Mass (g) of Visceral Organs of Experimental Wistar Rats (animals) across the various Treatment Groups

<table>
<thead>
<tr>
<th>Groups</th>
<th>Uninfected untreated</th>
<th>Infected untreated</th>
<th>Trypanidium treated</th>
<th>Vitamin B treated</th>
<th>Vitamin K treated</th>
<th>Vitamin B + K treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Liver</td>
<td>7.28 ±0.11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>11.5±0.50&lt;sup&gt;d&lt;/sup&gt;</td>
<td>9.26 ±0.99&lt;sup&gt;b&lt;/sup&gt;</td>
<td>10.5 ±0.29&lt;sup&gt;c&lt;/sup&gt;</td>
<td>10.1 ±0.65&lt;sup&gt;c&lt;/sup&gt;</td>
<td>9.89 ±0.71&lt;sup&gt;bNC&lt;/sup&gt;</td>
</tr>
<tr>
<td>Lung</td>
<td>1.39 ±0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.56±0.62&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.88 ±0.26&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.80±0.13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.56±0.73&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.66±0.13&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Kidney</td>
<td>0.67 ±0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.90±0.07&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.86 ±0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.97 ±0.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.88 ±0.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.00 ±0.50&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Spleen</td>
<td>0.81 ±0.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.84±0.32&lt;sup&gt;d&lt;/sup&gt;</td>
<td>1.56 ±0.29&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.07 ±0.36&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.07 ±0.36&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.75±0.45&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Heart</td>
<td>0.78 ±0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.44±0.36&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.81 ±0.10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.04±0.14&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.04 ±0.14&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.87 ±0.12&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

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Combating the Negative Effects of Trypanosoma brucei-brucei (Federe strain) Infection in Wistar Rats Using Selected Anti-oxidants and Trypamidium

Discussion

Trypanosomiasis was imposed on the Wistar rats when they were injected with trypanosome and the parasite Trypanosoma brucei brucei produces a very severe damage in the infected rats. The high significant decrease in live body weight observed in infected untreated group at 9 days post infection (9DPI) and 12 days post infection (9DPI) and the groups infected and treated with Antioxidant at 12DPI is an indication of the severity of the disease infection. This phenomenon may possibly be as a result of parasite-induced anorexia. Our observation in this study agrees with the findings of Ajakaiye et al. (2013) which reported that the apparent weight loss was due to increased parasitaemia. However, weight loss recorded in the group of animals treated with vitamins B, K, combination of B+K, and trypamidium was not significant (P > 0.05) when compared to uninfected untreated group. This could probably be as a result of antioxidants and the trypamidium exerted their potential in preventing the loss of live body weight. This agrees with reports that said the potential use of antioxidant in combined form is possibly more efficient and crucial than single antioxidant nutrients (German and Traber, 2001; Umar et al., 2007).

The results of packed cell volume (PCV) of all the groups of Wistar rats obtained in this experiment could be proved to be important in livestock health and production status by restoring the level of PCV almost back to the normal after treatment. This statement is further supported by the studies of Ekanem et al. (2006) who established and documented that, the measurement of anemia gives a reliable indication of the disease status and productive performance of trypanosome infected animals. Anaemia following trypanosome infection is typically diagnosed by the presence of a low PCV of erythrocytes and mechanisms of anaemia are thought to arise due to symptoms from the infection, induced by the innate immune response, leading to haemolysis (Noyes et al., 2009). Anaemia is recognized as the most important clinical manifestation of animal trypanosomiasis while PCV is used as an indicator in evaluating the level of anemia. In this experiment the packed cell volume observed decreased significantly (P < 0.05) in group B when compared to the values of group A and the treated groups. The low PCV observed in the infected groups especially group B could be as a result of acute hemolysis due to growing infection. This agrees with studies reported by Ogunsanmi and Taiwo, (2001); Umar et al. (2007); Ekanem et al. (2008); Saleh et al. (2009); Sulaiman and Adeyemi, (2010) who revealed that acute anemia in trypanosomiasis reflects the intensity and duration of parasitemia. Unlike the untreated group, the groups treated with vitamins B-complex, vitamin K and Trypamidium showed significant (P < 0.05) percentage increase in the PCV, its means that the antioxidants and the trypanocide have appreciated the effect of the trypanosome
infection on the PCV values. Previous studies have shown that infection with trypanosomes resulted in increased susceptibility of red blood cell membrane to oxidative damage probably as a result of depletion of vitamins on the surface of the red blood cell (Taiwo et al., 2003).

The enlargement or increase in size of the organs (Heart, Liver, Lung and Spleen) otherwise known as cardiomegaly, Hepatomegaly and Splenomegaly respectively as observed in the result, is presumably due to membrane damage caused by the large amount of free radicals and other oxidative species being generated and the concomitant reduction in systemic antioxidant reserves. This statement is in conformity with the findings of Ajakaiye et al. (2014b) who reported Hepatomegaly and Splenomegaly in trypanosomiasis. The increase in size of liver and spleen is caused by the activation of the immune system during trypanosome infection because spleen is involved in producing antibodies that fight infection, as a result of this intense activity the organ enlarged (Ajakaiye et al. 2014b), while the kidney serves many important functions, which includes; Filtering out wastes to be excreted in the urine and stimulating red blood cell production via the release of the hormone erythropoietin, for this reason, it tends to increase in size so as to meet the needs of the infected animals (Ajakaiye et al., 2014a). The prevention of organs damage by vitamins B-complex and K as well as Trypamidium is predicated on their activity, this was contained in a similar study conducted by Ajakaiye et al. (2014a) using vitamins C and E. but this statement is not in agreement with the findings of this study because no significant (P > 0.05) increase in size was observed in the values of kidney in all the experimental groups of the Wistar rats. It was earlier reported that infection of trypanosome causes anemia, this mean that there is no enough red blood cell to carry adequate oxygen to the tissues (Ajakaiye et al., 2013). If the anemia becomes chronic, it will lead to rapid or irregular heartbeat, in this case, the heart must pump more blood to make up for the lack of oxygen in the blood. For this reason, the heart tends to enlarge above normal, since the animal is untreated (Ajakaiye et al., 2014a).

Conclusion
This study shows that administration of vitamin B Complex, vitamin K and Trypamidium had prevented the depletion of live body weight and suppressesanaemia by restoring the level of PCV to the normal as well as parasitaemia. In addition the trypamidium cleared the parasites from the blood of the group of Wistar rats treated with the trypanosome. Thus, understanding the function of these antioxidants and the trypamidium in the pathogenesis of trypanosomiasis related to oxidative stress
possibly will help in designing nutritional support and control programs as a policy to combating the effect of the sleeping sickness.

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