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Effect of α-Tocopherol on the Antioxidant Enzymes of Male Wistar Albino Rats Infected with *Trypanosoma brucei brucei*

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ABSTRACT

The research was conducted to determine the effect of α-tocopherol on cardiac tissue antioxidant enzymes of male Wistar albino rats infected with *Trypanosoma brucei brucei*. Fifty-four (54) male Wistar albino rats were randomly divided into six (6) groups of three (3) rats each replicated three (3) times. The rats were marked and kept in stainless wire cages labeled A–F. Groups A, B, and C were blank, negative, and positive controls, respectively. Groups D, E, and F were infected with 1.0×10^6 trypanosomes and treated with 0.5 mg/kg (low dose), 2.5 mg/kg (medium dose), and 5.0 mg/kg (high dose) of vitamin E per body weight per day, respectively. The experiment lasted for 21 days from the day *T. b. brucei* infection was established. A sample of heart tissue homogenate was collected weekly across the groups and subjected to biochemical determination of superoxide dismutase (SOD), catalase, glutathione-s-transferase (GST), glutathione reductase (GR), and glutathione peroxidase (GPx) concentrations. There were significant differences in the effect of α-tocopherol, which was applied during the experiment, on cardiac tissue antioxidant enzymes. After treatment, SOD, catalase, GST, GR, and GPx levels differed significantly ($p < 0.05$) from the negative control. There were significant reductions in the levels of catalase, GST, GR, and GPx and a rise in the SOD level as infections grow. The result, however, showed that α-tocopherol caused a significant elevation in the levels of catalase, GST, GR, GPx, and reduction in the concentration of SOD following treatments with α-tocopherol. In conclusion, there were a significant reduction in the level of SOD and a rise in the concentrations of catalase, GST, GR, and GPx following treatments with α-tocopherol.

Keywords: *Trypanosoma brucei brucei,* α-tocopherol, SOD, catalase, glutathione reductase, glutathione peroxidase

INTRODUCTION

Trypanosomiasis is a debilitating protozoan disease caused by parasites classified in the phylum Sarcomastigophora, the order Kinetoplastida, the family Trypanosomatidae, and the genus *Trypanosoma* (Stevens & Brisse, 2004). Despite decades of research, trypanosomiasis continues to pose important public health and economic problems in many parts of Africa and South America (Sachs, 2010). Two forms of human trypanosomiasis are endemic in various parts of Africa. Rhodesian sleeping sickness, the acute form caused by *T. b. rhodesiense*, is endemic in East Africa, while the chronic form of the disease caused by *T. b. gambiense* occurs in West and Central Africa. *T. b. brucei,* on the other hand, has no regional epizootic limitations and causes a virulent disease described as nagana in animals alongside sleeping sickness in several parts of sub-Saharan Africa. *T. brucei* infection like other trypanosome infections precipitates increased red blood cell destruction, which results in anemia as well as tissue damage (Akanji, Adeyemi, Oguntoye, & Suleiman, 2009; Ekanem & Yusuf, 2008). The complex changes are presumably responsible for the symptoms of African sleeping sickness. The application of trypanocidal drugs has been the most widely practiced means of controlling trypanosomiasis in domestic livestock since the early 1950s, either as curative or as prophylactic drugs. Control of trypanosomiasis may depend in the nearest future on the use of the existing trypanocides. The challenge, therefore, remains to make optimal use of the drugs until new methods of treatment emerge (Adamu, Barde, Abenga, User, Ibrahim, & Esievo, 2009). The transmission of human African trypanosomiasis is mostly cyclical, which is dependent on the interactions of pathogenic parasites, tsetse flies, and mammalian reservoirs (World Health Organization, 2013). Oxidative stress is a common mediator in the pathogenicity of established cardiovascular risk factors (Edwin, Keyvan, Chai-Chi, Ravi, & Gemma, 2013). Catalase is an enzyme used by cells to metabolize the decomposition of hydrogen peroxide into less reactive gaseous oxygen and water (Chelikani, Fita, & Loewen, 2004).

The serum of camels infected by trypanosomiasis showed a significant fall in superoxide dismutase (SOD) concentration (El-Deeb & Elmoslemany, 2015). The reduced SOD level might be a result of its depletion during oxidative stress caused by *T. evansi.* Glutathione reductase (GR) catalyzes the reduction of glutathione disulfide to glutathione (GSH), thereby regulating the cellular integrity by reducing the oxidative damage caused by reactive oxygen species (Deponte, 2013). Glutathione peroxidase (GPx) is an enzyme that can maintain and restore cellular redox equilibrium and protects the cellular organism from oxidative damage. The physiological role of GPx is to decrease free hydroperoxides to their corresponding alcohols and to change free hydrogen peroxide to water by a reduction reaction (Kannan, Selvaraj, Nirmala, & Vasanthi, 2011).

Vitamin E is a group of fat-soluble compounds with distinctive antioxidant activities (Traber, 2006). It occurs in various chemical forms (α-, β-, γ-, and δ-tocopherol and tocotrienol) that have varying levels of biological activity (Traber, 2006). α-Tocopherol is the most active chemical form that can meet human needs. Vitamin E is a widely known chain-breaking antioxidant hindering lipid peroxidation (Lesenefsky et al., 2001). It is a hydrophobic antioxidant found in lipoprotein membranes and can maintain primary and secondary immunity. Numerous studies have reported an inverse relationship between antioxidant intake and cardiovascular risk factors (Jane & Leopold, 2015). Vitamin E, mostly α-

tocopherol, can ameliorate the modifiable indexes by modulating free radical production (Kaya, 2009).

Various studies have found that vitamin E functions in immune responses (Moriguchi & Muraga, 2000). Satoru and Mayumi (2003) observed that vitamin E deficiency caused a decrease in the differentiation of immature T cells in the thymus of hypertensive rats. Howard, Anna, McNeil, and McNeil (2011) reported the role of vitamin E in maintaining proper skeletal muscle homeostasis. Vitamin E might also hinder the formation of nitric food carcinogenic nitrosamines in the stomach and protect against cancer development by enhancing immunity (Pekmezci, 2011). A study also found that daily supplementation of vitamin E can enhance the immune response to a specific antigen. The mechanisms involved with protection against infectious agents were increased macrophage activity, antibody production, and higher natural killer activity (Han et al., 2000).

Deficiency of vitamin E can also result in neuromuscular problems such as spinocerebellar ataxia and myopathies, dysarthria, an absence of deep tendon reflexes, and the loss of both vibratory sensations. Vitamin E deficiency can also lead to anemia due to the oxidative damage to the red blood cells, retinopathy (Rathore, Suthar, Pareek, & Gupta, 2011), and the impairment of the immune responses. Vitamin E deficiency causes changes in leucocytes' levels and exacerbates the myocarditis and sympathetic denervation of ventricular hearts of *T. cruzi*–infected rats (Carvalho et al., 2006).

The works of Ammouche, Rouaki, Bitam, and Bellal (2002) and Kiron, Puangkaew, Ishizaka, Satoh, and Watanabe (2004) also reported that dietary vitamin E supplement increases the activities of antioxidant enzymes. However, vitamin E significantly reduced serum malondialdehyde (MDA) concentrations of the supplemented *T. b. brucei*–infected rats (Eze et al., 2016). The findings of Edoga, Ejo, Anukwuorji, Ani, and Izundu (2020) indicated that the mixed doses of vitamins B_{12} and E caused a significant increase in the levels of MDA and H_2O_2 and a fall in the concentrations of NO, GSH, and GPx. Trypanocidal drugs are complex, expensive, lethal, and highly resistant. Thus, vitamin E can fill the gap because of its accessibility and affordability. The objective of the study was to determine the ameliorative effect of α-tocopherol on the antioxidant enzymes of male Wistar albino rats infected with *T. b. brucei.*

MATERIAL AND METHODS

Animal Model and Experimental Protocol

Fifty-four (54) male albino Wistar rats (*Rattus norvegicus*) aged 3 months and weighing between 180 and 220 g were procured, housed, and allowed to acclimatize for two weeks at the Pharmacy Animal House, Madonna University, Elele. The research was approved by the Ethics Board of the Faculty of Biological Sciences, University of Nigeria, Nsukka, Enugu State, Nigeria. The rats were grouped into six (6) cages labeled A–F comprising three (3) rats that were replicated three (3) times from each group. The animals were kept under normal room temperature with ad libitum access to feed and water. The cages were cleaned daily to prevent infection of the animals and to minimize extraneous variables. The groups (A–F) were as thus: Group A (blank control) was neither infected with trypanosomes nor treated with vitamins; Group B (negative control) was infected with 1.0×10^6 counts/mL but not treated; Group C (positive control) was infected

with 1.0×10^6 counts/mL and treated with 0.2 mg/kg diminazene aceturate; Group D (low dose) was infected with 1.0×10^6 counts/mL and treated with 0.5 mg/kg body weight of vitamin E per day; Group E (medium dose) was infected with 1.0×10^6 trypanosomes and treated with 2.5 mg/kg body weight of vitamin E per day; Group F (high dose) was infected with 1.0×10^6 counts/mL and treated with 5.0 mg/kg body weight of vitamin E. The experiment lasted for 21 days after *T. b. brucei* infection was established. A sample of cardiac tissue homogenate was collected weekly from the three (3) rats across the groups and taken to Divine Chemicals and Analytical Laboratory, Nsukka, for the biochemical determination of cardiac tissue SOD, catalase, GST, GR, and GPx.

Procurement and Inoculation of Trypanosomes

T. b. brucei was obtained from an experimentally infected rat previously inoculated with the parasite from the faculty of Veterinary Medicine, University of Nigeria, Nsukka. Each experimental rat was administered 0.1 mL of infected blood in 0.3-mL normal saline containing 1×10^6 counts/mL using the rapid matching method (Herbert & Lumsden, 1976) to determine the level of parasitemia. Inoculation was done intraperitoneally.

Determination of Parasitemia

Wet blood preparations were covered with a coverslip on a slide and examined under ×40 and oil immersion using a microscope at ×100 magnification. The identification of parasites was done using morphological description (Van-Wyk & Mayhew, 2013).

Formulation and Administration of Vitamin E

α-Tocopherol was procured at Science Line, New Parts, Onitsha, Anambra State, Nigeria, in a powder bottle. The working concentrations were weighed at the Department of Biochemistry, Madonna University, Elele, from the result of an acute oral toxicity (LD_{50}) test of vitamin B_{12} as thus:

Mild dose: 0.5 mg/kg body weight per day
Medium dose: 2.5 mg/kg body weight per day
High dose: 5.0 mg/kg body weight per day

The working concentrations were dissolved in 2% ethanol as a vehicle since vitamin E is not soluble in water and administered via intubation.

Standard Drug

Diminazene aceturate was procured from the Faculty of Veterinary Medicine Clinic, University of Nigeria, Nsukka, Nigeria, in 2.36-g granules. The working dosage was 0.2 mL/kg. The administration was intravenous.

Preparation of Cardiac Tissues Homogenate

The heart was weighed and homogenized with a Potter-Elvehjem tissue homogenizer in a potassium phosphate buffer (10 Mm, pH 7.4). The crude tissue homogenate was centrifuged at 10,000 rpm, for 15 min in a cold centrifuge, and the resultant supernatant was used for the different estimations of proteins.

Determination of SOD

The method illustrated by McCord and Fridovich (1969) was used for the determination of SOD.

Catalase Activity

Catalase activity was determined in erythrocyte lysate using Aebi's (1984) method.

GST

The method described by Jocelyn (1972) was used to determine GST concentration.

GR Assay

The method illustrated by Kakkar, Das, and Viswanathan (1984) was followed in the determination of GR.

GPx

The procedure according to Wood (1970) was employed in the estimation of GPx concentration.

Statistical Analysis

Data resulting from the experiments were subjected to a two-way analysis of variance using the SPSS software (version 21), and the difference between means was separated using Duncan's multiple range tests. The test for significance was set at 0.05 probability level.

RESULTS

Cardiac Tissue SOD Concentration

The results of mean SOD level (U/mg) at short and long durations were compared in various groups. A similar pattern occurred at various points during the experiment. On the 14th day after infection (Week 2), the result indicated a significant increase in the negative control (4.972 ± 0.039) when compared to the blank control (3.526 ± 0.162) ($p < 0.05$) (Table 1). There were no significant differences in the mean cardiac tissue SOD between the blank and positive controls at Weeks 1, 2, and 3 after infection ($p > 0.05$).

There was a significant difference in the negative control in mean SOD concentration during the study ($p < 0.05$). This showed that the mean cardiac tissue SOD level increased as the parasitemia increased. A comparison of mean SOD levels in vitamin E–treated groups showed there was a significant reduction in the level of SOD when compared to the negative control at Weeks 1, 2, and 3 after infection and treatment.

Table 1. Effect of α-Tocopherol on Cardiac Tissue Superoxide Dismutase Level (U/mg)

Groups	Week 1	Week 2	Week 3
A	$3.444 \pm 0.095^{a,1}$	$3.526 \pm 0.162^{a,1}$	$3.460 \pm 0.167^{a,1}$
B	$3.216 \pm 0.136^{b,1}$	$4.972 \pm 0.039^{b,2}$	$5.002 \pm 0.096^{b,2}$
C	$3.485 \pm 0.057^{a,1}$	$3.657 \pm 0.071^{a,1}$	$3.635 \pm 0.090^{a,1}$
D	$3.943 \pm 0.042^{c,1}$	$3.965 \pm 0.070^{c,1}$	$3.915 \pm 0.003^{c,1}$
E	$3.912 \pm 0.008^{c,1}$	$3.940 \pm 0.032^{c,1}$	$3.906 \pm 0.002^{c,1}$
F	$3.947 \pm 0.024^{c,1}$	$4.259 \pm 0.326^{d,2}$	$3.878 \pm 0.007^{c,1}$

Note. Mean values (mean ± SEM) with the same letters and numbers (superscript) per column and per row, respectively, denote no significant differences ($p > 0.05$).

Cardiac Tissue Catalase Concentration

The results of mean catalase level (U/mg) at short and long durations were compared in various groups. A similar pattern occurred at various points during the experiment. On the seventh day after infection (Week 1), the result indicated a significant decrease in the negative control (42.252 ± 0.064) when compared to the blank control (49.423 ± 0.190) ($p < 0.05$) (Table 2). There were no significant differences in the mean cardiac tissue catalase between the blank and positive controls at Weeks 1, 2, and 3 after infection ($p > 0.05$).

There was a significant difference in the negative control in mean catalase concentration during the study ($p < 0.05$).

This showed that the mean cardiac tissue catalase level decreased as the parasitemia increased. A comparison of mean catalase levels in vitamin E–treated groups showed there was a significant rise in the level of catalase when compared to the negative control at Weeks 1, 2, and 3 after infection and treatment.

Table 2. Effect of α-Tocopherol on Cardiac Tissue Catalase Level (U/mg)

Groups	Week 1	Week 2	Week 3
A	$49.423 \pm 0.190^{a,1}$	$50.155 \pm 0.481^{a,1}$	$51.412 \pm 1.237^{a,1}$
B	$42.252 \pm 0.064^{b,1}$	$40.510 \pm 0.340^{b,2}$	$38.345 \pm 0.191^{b,3}$
C	$48.131 \pm 0.017^{c,1}$	$49.435 \pm 0.156^{c,1}$	$49.527 \pm 0.507^{c,1}$
D	$45.493 \pm 0.117^{d,1}$	$44.611 \pm 0.116^{d,1}$	$43.650 \pm 0.239^{d,1}$
E	$45.688 \pm 0.059^{d,1}$	$44.712 \pm 0.117^{d,1}$	$44.681 \pm 0.058^{d,1}$
F	$46.381 \pm 0.116^{d,1}$	$44.825 \pm 0.055^{d,1}$	$44.751 \pm 0.116^{d,1}$

Note. Mean values (mean ± SEM) with the same letters and numbers (superscript) per column and per row, respectively, denote no significant differences ($p > 0.05$).

Cardiac Tissue GST Concentration

The result of mean GST level (U/mg) was compared in various groups. A similar pattern occurred at various points during the study. On the 14th day after infection (Week 2), the result indicated a significant drop in the values of the negative control (21.125 ± 0.083) when compared to the blank control (25.022 ± 0.176) ($p < 0.05$) (Table 3). There were no significant differences in the mean cardiac tissue GST between theblank and positive controls at Weeks 1, 2, and 3 after infection ($p > 0.05$).

There was a significant difference in the negative control in mean GST concentration during the study ($p < 0.05$). This showed that the mean cardiac tissue GST level dropped as the parasitemia increased. A comparison of mean GST levels in vitamin E–treated groups showed there was a significant increase in the level of GST when compared to the negative control at Weeks 1, 2, and 3 after infection and treatment.

Table 3. Effect of α-Tocopherol on Cardiac Tissue GST Level (U/mg)

Groups	Week 1	Week 2	Week 3
A	$25.058 \pm 0.053^{a,1}$	$25.022 \pm 0.176^{a,1}$	$25.057 \pm 0.152^{a,1}$
B	$21.563 \pm 0.272^{b,1}$	$21.125 \pm 0.083^{b,1}$	$21.125 \pm 0.083^{b,1}$
C	$24.253 \pm 0.263^{c,1}$	$24.317 \pm 0.278^{c,1}$	$24.712 \pm 0.311^{c,2}$
D	$22.835 \pm 0.058^{d,1}$	$22.995 \pm 0.114^{d,1}$	$22.861 \pm 0.232^{d,1}$
E	$22.886 \pm 0.116^{d,1}$	$22.903 \pm 0.060^{d,1}$	$22.721 \pm 0.116^{d,1}$
F	$23.016 \pm 0.002^{d,1}$	$22.896 \pm 0.115^{d,1}$	$22.723 \pm 0.117^{d,1}$

Note. Mean values (mean ± SEM) with the same letters and numbers (superscript) per column and per row, respectively, denote no significant differences ($p > 0.05$).

Cardiac Tissue GR Concentration

The results of mean GR level (U/mg) at short and long durations were compared in various groups. A similar pattern occurred at various points during the experiment. On the seventh day after infection (Week 1), the result indicated a significant reduction in the negative control (15.117 ± 0.111) when compared to the blank control (19.914 ± 0.037) ($p < 0.05$) (Table 4). There were no significant differences in the mean cardiac tissue GR between the blank and positive controls at Weeks 1, 2, and 3 after infection ($p > 0.05$).

There was a significant difference in the negative control in mean GR concentration during the study ($p < 0.05$). This showed that the mean cardiac tissue GR level decreased as the parasitemia increased. A comparison of mean GR levels in vitamin E–treated groups showed there was a significant rise in the level of GR when compared to the negative control at Weeks 1, 2, and 3 after infection and treatment.

Table 4. Effect of α-Tocopherol on Cardiac Tissue Glutathione Reductase Level (U/mg)

Groups	Week 1	Week 2	Week 3
A	$19.914 \pm 0.037^{a,1}$	$20.186 \pm 0.315^{a,1}$	$20.101 \pm 0.320^{a,1}$
B	$15.117 \pm 0.111^{b,1}$	$15.069 \pm 0.076^{b,1}$	$14.693 \pm 0.244^{b,2}$
C	$19.062 \pm 0.176^{c,1}$	$19.217 \pm 0.135^{c,1}$	$19.707 \pm 0.125^{c,2}$
D	$17.106 \pm 0.058^{d,1}$	$17.384 \pm 0.034^{d,1}$	$17.612 \pm 0.117^{d,1}$
E	$17.263 \pm 0.059^{d,1}$	$17.518 \pm 0.002^{d,1}$	$17.638 \pm 0.059^{d,1}$
F	$17.318 \pm 0.172^{d,1}$	$17.710 \pm 0.116^{d,1}$	$17.889 \pm 0.002^{d,1}$

Note. Mean values (mean ± SEM) with the same letters and numbers (superscript) per column and per row, respectively, denote no significant differences ($p > 0.05$).

Cardiac Tissue GPx Concentration

The result of mean GPx level (U/mg) was compared in various experimental groups. A similar pattern occurred at various points during the experiment. On the seventh day after infection (Week 1), the result indicated a significant reduction in the negative control (20.414 ± 0.333) when compared to the blank control (24.381 ± 0.116) ($p < 0.05$) (Table 5). There were no significant differences in the mean cardiac tissue GPx between the blank and positive controls at Weeks 1, 2, and 3 after infection ($p > 0.05$).

There was a significant difference in the negative control in mean GPx concentration during the study ($p < 0.05$). This showed that the mean cardiac tissue GPx level decreased as the parasitemia increased. A comparison of mean GPx levels in vitamin E–treated groups showed there was a significant rise in the level of GPx when compared to the negative control at Weeks 1, 2, and 3 after infection and treatment.

Table 5. Effect of α-Tocopherol on Cardiac Tissue GPx Level (U/mg)

Group	Week 1	Week 2	Week 3
A	$24.381 \pm 0.116^{a,1}$	$24.585 \pm 0.264^{a,1}$	$24.585 \pm 0.168^{a,1}$
B	$20.414 \pm 0.333^{b,1}$	$20.619 \pm 0.170^{b,1}$	$19.879 \pm 0.074^{b,2}$
C	$23.056 \pm 0.099^{c,1}$	$23.116 \pm 0.036^{c,1}$	$23.518 \pm 0.060^{c,1}$
D	$22.826 \pm 0.071^{c,1}$	$22.825 \pm 0.072^{c,1}$	$22.842 \pm 0.096^{c,1}$
E	$22.966 \pm 0.053^{c,1}$	$22.807 \pm 0.030^{c,1}$	$22.826 \pm 0.083^{c,1}$
F	$22.823 \pm 0.242^{c,1}$	$22.714 \pm 1.196^{c,1}$	$23.179 \pm 0.006^{c,1}$

Note. Mean values (mean ± SEM) with the same letters and numbers (superscript) per column and per row, respectively, denote no significant differences ($p > 0.05$).

DISCUSSION AND CONCLUSION

Discussion

The present experiment showed a higher level of SOD in the heart of *T. b. brucei*–infected rats from Days 7–21 after infection when compared to the blank control. This aligns with the work of Ogunsanmi and Taiwo (2007), who reported hiked SOD activity in the serum and kidney of *T. brucei*–infected rats. Atalay and Laaksonen (2002) also observed that, under the condition of oxidative stress, the activity of antioxidant SOD increases. The result of the present experiment is consistent with the observation of Eze et al. (2016), who reported that vitamin E supplementation enhances SOD activities in *T. b. brucei*–infected rats.

The result of the present study has shown that *T. b. brucei* caused a significant reduction in the level of antioxidant catalase in the heart tissues of Wistar albino rats. This observation is consistent with the work of Matheus et al. (2016), who reported a significant reduction of enzymatic activities of catalase in the heart of rats infected with *T. evansi*. They attributed these decreased enzymatic activities to cardiac injury. The levels of catalase enzyme in the groups infected and treated with vitamin E significantly increased the trypanosome-induced catalase reduction. This aligns with the observation of Eze et al. (2016), who reported that vitamin E supplementation significantly increases catalase activities of *T. b. brucei*–infected rats.

The results of the present experiment showed that *T. b. brucei* infection of the experimental rats caused a significant decrease in the cardiac tissue GST concentration. The observations agreed with the observations of Matheus et al. (2016), who reported that cardiac tissue GST activity of *T. evansi* rats significantly decreased after infection. However, the results of the present study contradict the observation of Anschau et al. (2013), who reported no difference in GST activity in the heart of rats infected with *T. evansi*. The levels of GST in the heart tissues of albino rats infected with *T. b. brucei* and treated with α-tocopherol increased the trypanosome-induced GST reduction even though it could not get to the level of the blank or positive control value. The present experiment is in line with observations of many studies on vitamin E. Harald, Kathrin, Dietmar, and Johanna (2014) reported an inverse relationship between vitamin E and cardiovascular risk factors. The works of Atalay and Laaksonen (2002) and Matheus et al. (2016) reported that dietary vitamin E supplement increases the activities of antioxidant enzymes.

The study showed that the proliferation of trypanosomiasis caused a reduction in the levels of GR in the experimental group. In the groups infected and treated with α-tocopherol, the GR significantly increased. The observed elevation of GR in the treated groups is indicative of antioxidant and trypanocidal properties of vitamin E. This is consistent with the result of Verhagen, Buijse, Jansen, and Bueno-de-Mesquita (2006), who reported that antioxidant vitamins can prevent the damaging effects of reactive oxygen species. The result of the experiment showed that the proliferating parasites caused GPx reduction in the experimental group. This is in line with the observation of Anschau et al. (2013), who reported a fall in GPx activities in the blood of *T. evansi*–infected rats.

However, the present observation conflicts with the findings of Omer, Mousa, and Al-Wabel (2007), who reported the elevated GPx activity in the whole blood of infected rats following trypanosomiasis. In the groups infected and treated with vitamin E, the trypanosome-induced reduced GPx returned close to the level of the positive control group. The present experiment is in line with observations of many studies on vitamin E. Jane and Leopold (2015) described vitamin E as the most potent and efficient scavenger of lipid peroxyl radicals. Harald, Kathrin, Dietmar, and Johanna (2014) reported an inverse relationship between vitamin E and cardiovascular risk factors.

CONCLUSION

The findings of the present study indicated that the pathogenesis of *T. b. brucei* caused a significant rise in the concentration of SOD and a profound reduction in the concentrations of catalase, GST, GR, and GPx. However, the administration of α-tocopherol caused a dose-dependent reduction in the concentrations of SOD and increased cardiac tissue catalase, GST, GR, and GPx. This research has affirmed the ameliorative capacity of vitamin E in managing *T. b. brucei*–related diseases.

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