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Protective Effect of Vitamin E on Potassium Dichromate-induced Haematoxicity and Oxidative Stress in African Catfish (*Clarias gariepinus*)

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Authors' contributions

This work was carried out in collaboration between both authors. Author OIA designed and supervised the study; corrected and processed the manuscript while author SFB carried out the experiment and wrote the first draft of the manuscript. Both authors read and approved the final manuscript.

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ABSTRACT

Effect of potassium dichromate was studied experimentally in African Catfish (*Clarias gariepinus*) with and without vitamin E in induced haematoxicity and oxidative stress. Blood samples were collected for haematology and plasma biochemical parameters while gills, liver and kidney samples were collected for evaluation of markers of oxidative stress. Exposure to potassium dichromate led to a significant decrease in PCV, RBC, haemoglobin concentration, MCV, MCH and total WBC when compared with the unexposed control and those fed with vitamin E-supplemented feed. Nephrotoxicity was also observed as evidenced by increases in plasma creatinine levels. Exposure to potassium dichromate also led to increased oxidative stress as the concentrations of antioxidant endogenous enzymes - GPx, GST and GSH were depleted while potentiating lipid peroxidation and hydroxyl radical generation. The changes in the haematological, biochemical and antioxidant parameters were restored in the fish fed with vitamin E-supplemented feed. The study showed exposure to potassium dichromate to be toxic to African catfish, causing anaemia and kidney damage with free radical generation and depletion of GSH and other antioxidant defence system but the toxic effect can be minimised along with weight gain in fish supplemented by vitamin – E in aquaculture.

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1. INTRODUCTION

Chromium is a ubiquitous metal in the environment found in soil, rocks and living organisms, automobile exhaust and in tobacco products [1]. Hexavalent chromium is used in a variety of industries, ranging from electroplating, dyeing, metal refining, fungicide, wood preservatives, ceramic glass, pigment production and leather tanneries [2,3]. The improper disposal of effluents from these industries has led to the undue presence of chromium in the environment ultimately leading to environmental pollution [4].

Based on the foregoing, this study was conducted to assess the effect of sub-lethal concentrations of potassium dichromate on growth, haematology, plasma biochemistry and markers of oxidative stress in African catfish *(Clarias gariepinus)* and protective effects of vitamin E supplementation in feed.

2. MATERIALS AND METHODS

2.1 Drugs and Chemicals

Analytical grades of copper sulphate (Sigma-Aldrich) and Vitamin E were procured from local agents in Ibadan, Nigeria.

2.2 Experimental Animals

Sixty (60) apparently healthy unsexed juvenile African catfish (*C. gariepinus*), with average weight of 120 g procured from the University of Ibadan Aquaculture Laboratory were used for this study. From our observation, the Aquaculture Unit was devoid of any industrial effluent or any other sources of pollution that could affect the biochemical responses of the control fish. The fish were inspected for general fitness and were allowed to acclimatize for 3 weeks in plastic bowls of 80 litres (80 L) capacity during which time they were fed twice daily. All experimental protocols were in compliance with University of Ibadan ethics committee on research in animals as well as internationally accepted guidelines for laboratory animal use and care. The protocol was approved the University of Ibadan Animal Care Use, Research and ethics Committee with ethical clearance number UI-ACUREC/19/0089.

2.3 Determination of Physico-chemical Parameters of the Water

The physico–chemical parameters of the water consisting of the temperature, dissolved oxygen (D.O), hydrogen ion concentration (pH) and conductivity were determined before the commencement of the study using Sension M156+ Portable multi meter.

2.4 Experimental Feed

Two types of feed were used for this experiment; a pure commercial feed and a vitamin E-supplemented feed. The commercial pelletized feed (Ecofloat) contains 38% crude protein. The vitamin E-supplemented feed was prepared by mixing the commercial feed with vitamin E (240 mg/kg of feed) and then re-pelletized using a pelletizing machine.

2.5 Experimental Protocol

After three weeks of acclimatization during which the fish were all fed with the same feed, the fish were weighed and randomly classified into 6 groups (A-F) at the commencement of the treatment. Group A served as the control was fed with normal pelletized fish feed only, group B was fed with Vitamin E-supplemented feed only, group C was exposed to potassium dichromate (30 mg/L) according to Ekeh et al., [5] with slight modification, plus normal pelletized fish feed, group D was exposed to potassium dichromate (5 mg/L) [5] plus normal pelletized fish feed, group E was administered with potassium dichromate (30 mg/L) plus Vitamin E-supplemented feed (240 mg/kg diet) while group F was given potassium dichromate (5mg/L) plus Vitamin E-supplemented feed (240mg/kg diet) for fourteen (14) days.

2.6 Evaluation of Growth Parameters

All fish were weighed at the commencement and the end of the experiment (14 days), to calculate weight gain (WG) and feed conversion ratio (FCR). The amount of feed consumed was recorded throughout the period of the experiment to determine the feed intake (FI). Growth performance and feed utilization were assessed in terms of weight gain (WG) and feed conversion ratio (FCR) using the following formulae:

$$WG\ (g) = W_2 - W_1$$

$$FCR = \frac{FI}{WG}$$

Where, W_2= final body weight, W_1= initial body weight, W = body weight, FI = feed intake, WG = weight gain

2.7 Determination of Haematological Parameters

After 14 days, blood samples were collected from the caudal vein of each fish into heparinized tubes. From the samples collected, the Packed Cell Volume (PCV) was determined by microhaematocrit method, Red Blood Cells (RBC) and White Blood Cells (WBC) by haemocytometer method using the improved Neubauer slide [6]. Haemoglobin concentration (Hb) was determined by cyanmethaemoglobin method while the Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH) and Mean Corpuscular Haemoglobin Concentration (MCHC) were calculated from the PCV, RBC and Hb values according to (Jain, 1986), whereas erythrocyte osmotic fragility was determined according to the method described by Azeez et al. [7].

2.8 Determination of Plasma Biochemical Parameters

Blood samples were centrifuged at 4000 rev/min for 10 min to obtain the plasma. From the plasma, urea and creatinine were determined spectrophotometrically according to the methods of Coloumbe and Farreau as described by Kuribayashi et al. [8] and Taussky by Onuegbu et al. [9], respectively. Total protein and albumin in plasma samples were determined by the methods described by Bradford [10] and Doumas et al. [11] while plasma globulin was calculated as the difference between total protein and albumin. Alkaline phosphatase activity was determined by the method of Bessey [12] while activities of ALT and AST were determined by the method of Reitman and Frankel as described by Mallhi et al. [13]. The serum concentrations of total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) and triglyceride (TG) were determined by enzymatic colorimetric method using the following Roche kits according the manufacturer's protocol.

2.9 Determination of Markers of Oxidative Stress

Lipid peroxidation was quantified as malondialdehyde (MDA) according to the method described by Farombi et al. [14] reduced glutathione (GSH) concentrations by the method of Jollow et al. [15] while Glutathione-S-transferase (GST) activity was determined according to the method described by Farombi et al. [16]. Hydrogen peroxide generation was evaluated according to the method of Woff [17]. Protein concentration was determined by Biuret method as described by Omobowale et al. [18] Glutathione peroxidase activity was measured according to the method described by Rotruck et al. [19].

2.10 Statistical Analysis

All values are expressed as mean ± S.D. "One-way Analysis of Variance" (ANOVA) with Tukey's post-hoc test was performed to compare the data between groups using GraphPad Prism version 7.0 with probability value of $P < 0.05$ considered statistically significant.

3. RESULTS

3.1 The Physico-chemical Parameters of the Water Used

From the results in Table 1, the physico-chemical parameters of the water used were within the accepted international EPA standard. For example, the pH of the water was neutral at 7.33 ± 0.84, which corresponds to the average acceptable international EPA standard. The dissolved oxygen was also high enough for survival of the fish at 5.71 ± 0.88, which was higher than the acceptable international Environmental Protection Agency (EPA) standard.

3.2 Growth Performance and Feed Conversion

Fig. 1 shows the growth performance and feed utilization parameters of the African catfish exposed to copper sulphate. At 14 days, the fish in all the groups increased in weight along with group B that was fed with vitamin E-supplemented feed having the highest weight gain whereas group C that was exposed to 30mg/L of potassium dichromate had the lowest weight gain. In a pattern similar to the weight

gain, the feed conversion ratio was also highest in group B and lowest in group C.

The weight of liver and kidney compared to the body weight as shown in Table 2 revealed that the percentage weight of liver to body ratio of fish in group C that were exposed to potassium dichromate (30 mg/L) alone relative to body ratio was significantly higher than groups of B and F at $p<0.05$ while there was no significant difference in the percentage heart-body weight ratio across the groups.

3.3 Haematological Parameters

As shown in Table 3, the packed cell volume (PCV) of the group exposed to 30mg/L of potassium dichromate was significantly reduced ($p<0.05$) when compared with the control group (group A) and group F that was co-treated with vitamin E, while the haemoglobin concentration of group D that was exposed to potassium dichromate (5 mg/L) was significantly lower than that of the control group at $p<0.05$. The red blood cell count of group D was also significantly lower ($p<0.05$) than those obtained in groups E or F. The mean corpuscular volume of group D was significantly higher ($p<0.05$) than the values in groups B, E or F. The mean corpuscular haemoglobin of group D was significantly higher ($p<0.05$) than groups E or F.

The total WBC count of the fish in groups C and D that were exposed to 30mg/L and 5mg/L respectively were significantly lower than those of groups A, B and F. The lymphocyte count of group C that was exposed to potassium dichromate (30 mg/L) alone was significantly lower ($P<0.05$) than those of groups B or F. The neutrophil counts of groups B, E and F were significantly higher ($P<0.05$) than those of groups C or D.

Table 1. The physico-chemical parameters of the water used

Parameters	Values	EPA standard
Temperature (°C)	27.34±2.67	25
pH	7.33±0.84	≥6 ≤9
Dissolved oxygen (mg/L)	5.71±0.88	≥5
Conductivity (µS/cm)	215.53±13.01	200-1000

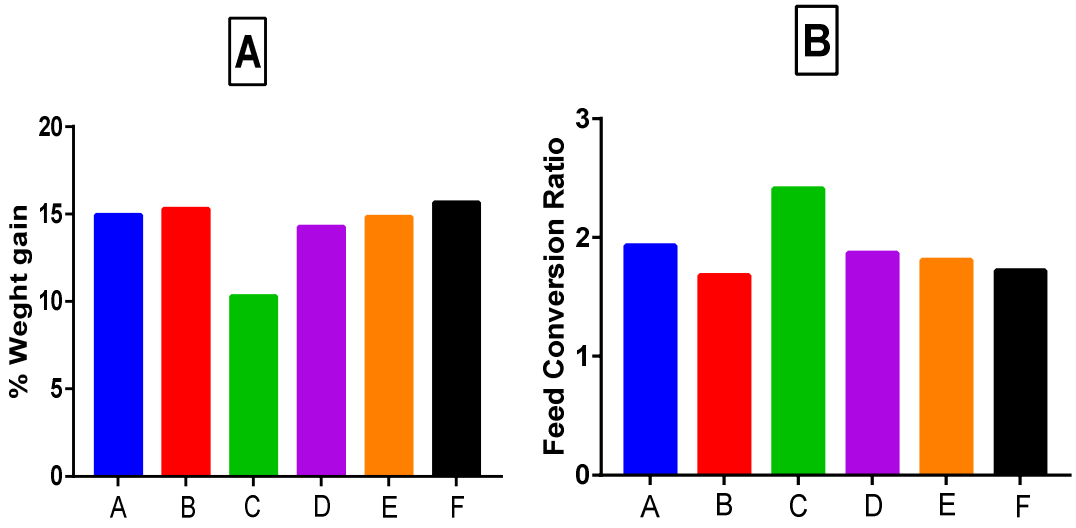

Fig. 1. Effect of potassium dichromate toxicity on percentage weight gain (A) and feed conversion ratio (B) in African catfish (*Clarias gariepinus*) as modulated by vitamin E

A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone, D = Potassium dichromate (5mg/L) alone, E = Potassium dichromate (30mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5mg/L) alone + Vitamin E-supplemented feed, n = number of fish

Table 2. Effect of potassium dichromate toxicity on organ-body weight ratio in african catfish (*Claria gariepinus*) as modulated by vitamin E

Parameters	A	B	C	D	E	F
BW (g)	185.70±20.81	163.10±28.02	169.60±11.75	161.00±28.88	169.10±39.70	125.1±7.16
LW (g)	3.27±0.38	1.79±0.23	4.00±0.61	3.01±0.80	2.72±1.37	1.49±0.10
L-B weight (%)	1.79±0.38	1.10±0.05[a]	2.36±0.35[ab]	1.86±0.31	1.55±0.55	1.20±0.12[b]
HW (g)	0.16±0.05	0.15±0.04	0.20±0.05	0.16±0.01	0.22±0.08	0.10±0.02
H-B weight (%)	0.08±0.02	0.09±0.01	0.12±0.04	0.10±0.02	0.14±0.08	0.08±0.01

Data are presented as mean ± SD. Where BW = bodyweight (g), LW= liver weight (g), L-B = percentage liver/body ratio (%), HW = heart weight (g), H-B = percentage heart/body ratio. Values with the same superscript alphabets along the same row are significantly different at P<0.05
A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone,
D = Potassium dichromate (5mg/L) alone, E = Potassium dichromate (30mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5mg/L) alone + Vitamin E-supplemented feed, n = number of fish

Table 3. Effect of potassium dichromate on erythrocyte parameters of African catfish (*Clarias gariepinus*) as modulated by vitamin E

Parameters	A	B	C	D	E	F
PCV (%)	47.20±8.08[a]	43.40±3.85	37.33±4.88[ab]	38.40±3.13	42.00±5.00	43.21±2.30[b]
Hb (g/dl)	10.80±1.66[a]	10.38±1.50	8.26±1.32	7.48±1.65[a]	9.50±0.76	8.60±1.86
Rbc (x10^6/µL)	2.41±0.32	2.55±0.30	2.09±0.39	1.47±0.23[ab]	2.86±1.25[a]	2.67±0.49[b]
MCV	199.10±45.21	171.40±20.40[a]	192.10±28.69	264.80±32.68[abc]	170.70±79.98[b]	154.9±26.78[c]
MCH	45.60±10.21	41.46±9.72	40.09±6.29	50.51±3.73[ab]	38.88±18.00[a]	32.88±8.7.7[b]
MCHC	23.64±6.1	24.04±3.77	21.01±2.74	19.40±3.49	22.72±1.23	21.23±4.31

Values are presented as mean ± SD while values with the same superscript alphabets along the same row are significantly different at P<0.05
A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone,
D = Potassium dichromate (5mg/L) alone, E = Potassium dichromate (30mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5mg/L) alone + Vitamin E-supplemented feed, n = number of fish

Table 4. Effect of potassium dichromate on leucocyte parameters of African catfish (*Clarias gariepinus*) as modulated by vitamin E

Parameters	A	B	C	D	E	F
TWBC	1.20±0.07^{abc}	1.23±0.10^{def}	0.85±0.09^{adg}	0.92±0.12^{beh}	1.02±0.03^{cf}	1.10±0.09^{gh}
Lymph. (/µl) (%)	657.00±84.16 (79.40±5.18)	782.80±40.31^{a} (78.60±6.54)	603.50±54.38^{ab} (77.80±6.54)	743.80±110.60 (82.40±4.51)	629.80±36.13 (62.80±6.54)	761.50±116.20^{b} (61.60±3.51)
Neut. (/µl) (%)	109.20±81.10 (10.80±5.98)	175.80±83.25^{ab} (15.60±6.73)	52.60±27.52^{ace} (7.20±4.38)	41.90±41.06^{bdf} (4.00±3.39)	192.10±34.53^{ef} (18.80±5.89)	201.10±43.91^{cd} (18.60±3.36)
Mono. (/µl) (%)	91.50±12.48^{ab} (9.80±2.86)	63.30±25.72^{cde} (5.80±2.78)	117.60±39.73^{fg} (15.00±4.00)	135.20±14.69^{chi} (13.60±3.05)	199.90±35.96^{adfh} (18.80±1.64)	218.90±47.18^{begi} (20.20±3.27)

Values are presented as mean ± SD while values with the same superscript alphabets along the same row are significantly different at P<0.05
A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone, D = Potassium dichromate (5mg/L) alone, E = Potassium dichromate (30mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5mg/L) alone + Vitamin E-supplemented feed, n = number of fish

Table 5. Effects of potassium dichromate toxicity on plasma biochemical parameters in the African catfish (*Clarias gariepinus*) as modulated by vitamin E

Parameters	A	B	C	D	E	F
AST (IU/L)	14.00±2.00	14.33±3.79	14.67±2.52	11.67±1.16	14.33±1.53	14.33±4.04
ALT (IU/L)	10.33±1.53	11.33±2.89	12.00±2.00	8.00±2.00	12.00±1.00	10.67±2.08
ALP(IU/L)	42.67±7.64	40.67±9.07	44.33±6.11	33.33±4.51	42.00±4.00	36.33±6.51
Urea (mg/dl)	26.67±3.06	27.67±3.51	30.67±2.52	33.33±5.86	23.00±3.61	27.00±4.00
Creatinine (mg/dl)	0.57±0.12	0.60±0.10	0.73±0.06	0.77±0.06^{a}	0.50±0.10^{a}	0.60±0.10
Plasma proteins (g/dl)	6.67±0.38	6.87±0.25	7.23±0.21	7.17±0.25	6.60±0.36	6.80±0.35
Albumin (g/dl)	3.73±0.32	3.90±0.27	4.20±0.10	4.00±0.17	3.70±0.27	3.80±0.46
Globulin (g/dl)	2.93±0.06	2.97±0.12	3.03±0.12	3.17±0.15	2.90±0.20	3.03±0.12
Total cholesterol (mg/dl)	164.70±5.51	155.00±7.94	180.70±31.56	146.70±14.05	164.00±7.55	154.00±6.25
Triglycerides(mg/dl)	70.00±12.53	57.33±4.73	75.00±23.64	59.67±14.05	66.67±18.15	67.67±2.08
HDL (mg/dl)	44.67±2.89	40.33±3.51	49.33±9.87	33.33±9.29	45.67±3.51	40.67±2.52
LDL (mg/dl)	135.00±9.64	120.70±10.50	147.00±26.15	119.00±11.53	135.30±10.41	119.30±5.03

Values are presented as mean ± SD while values with the same superscript alphabets along the same row are significantly different at P<0.05
A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone,
D = Potassium dichromate (5mg/L) alone, E = Potassium dichromate (30mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5mg/L) alone + Vitamin E-supplemented feed, n = number of fish

The monocyte count of groups E and F that were co-treated with potassium dichromate and vitamin E was significantly higher (P<0.05) than that of all other groups.

Potassium dichromate exposure also affected the erythrocyte osmotic resistance in hypotonic solution as seen in Fig. 2. At 0.5% NaCl concentration, the erythrocyte osmotic fragility of the fish in group B that were fed with vitamin E-supplemented feed was significantly lower ($p<0.05$) than groups A, D or E while that of group F that was exposed to potassium dichromate (5 mg/L) plus vitamin E-supplemented feed was significantly lower ($p<0.05$) than groups D or E.

3.4 Plasma Biochemical Parameters

The effects of potassium dichromate toxicity on plasma biochemical parameters in African catfish are presented in Table 5. Fish in group D that were exposed to potassium dichromate (5 mg/L) alone had a significantly higher ($p<0.05$) plasma creatinine levels than those in group E that were exposed to potassium dichromate (30 mg/L) plus vitamin E-supplemented feed. Marginal increases were observed in AST, ALT and ALP levels in the fish exposed to sub-lethal dose of potassium dichromate (30 mg/L), but they were not statistically significant. There was no significant difference in plasma protein levels and lipid profiles across the groups

3.5 Markers of Oxidative Stress

The effects of potassium dichromate toxicity on markers of oxidative stress in the gills (Fig. 3), liver (Fig. 4) and kidneys (Fig. 5) of African catfish and the modulatory effect of vitamin E are shown below. In the gills (Fig. 3), the GPx and GST activities were reduced while lipid peroxidation was potentiated in those groups exposed to different concentrations of potassium dichromate alone. These abnormalities were restored in the groups fed with vitamin E-supplemented feed. As shown in Fig. 4, the GPx, GSH and GST activities as well as the total protein concentration in the liver was reduced in the groups exposed to potassium dichromate (30mg/L and 5mg/L) alone when compared with the control group. These alterations were brought back to normal in the groups fed with vitamin E-supplemented feed. Fig. 5 shows that the GPx and GSH activities in the kidney were reduced in the fish exposed to 30mg/L and 5mg/L of potassium dichromate alone while hydrogen peroxide generation was increased when compared with the control groups. These abnormalities were restored in the groups fed with vitamin E-supplemented feed.

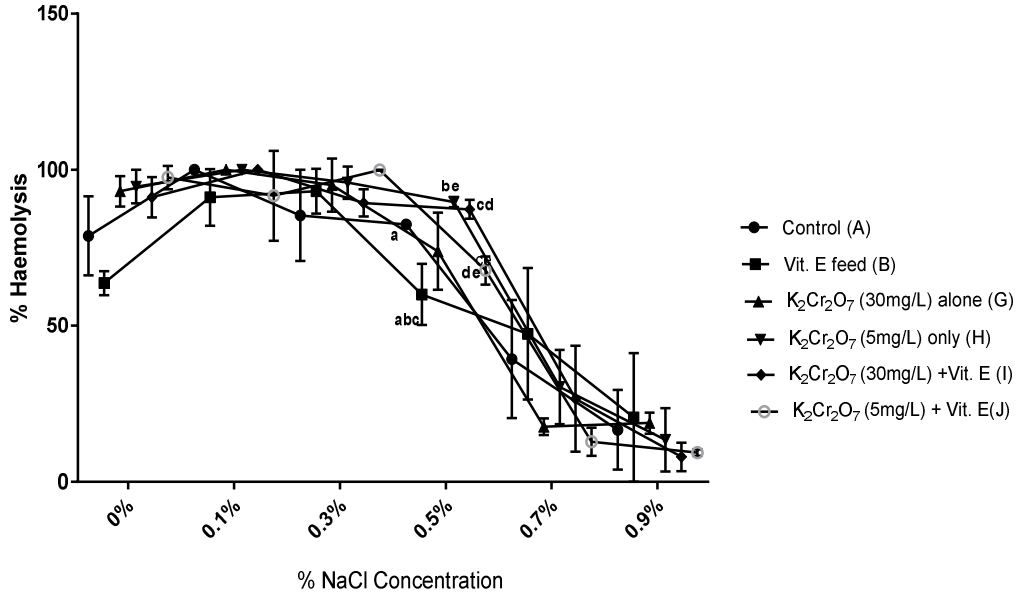

Fig. 2. Erythrocytes osmotic fragility of African catfish (*Clarias gariepinus*) exposed to potassium dichromate toxicity and concurrent treatment with vitamin E. Values are expressed as mean ± SD and n is 5 for each group

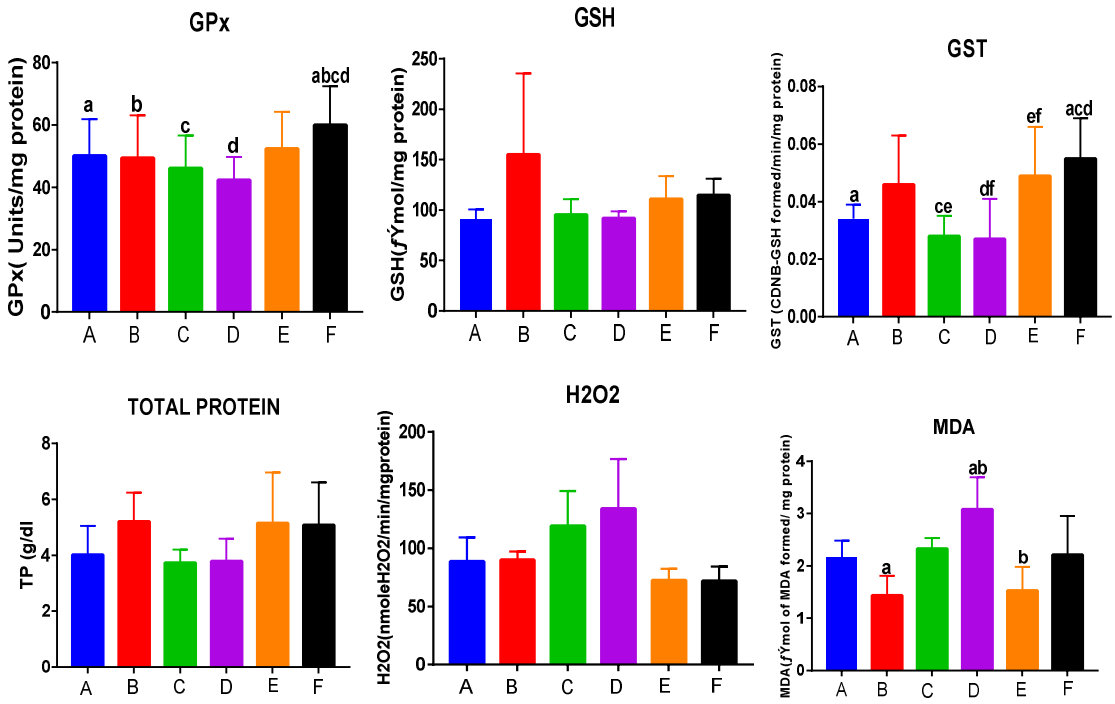

Fig. 3. Effect of potassium dichromate toxicity on markers of oxidative stress in the gills of African catfish as modulated by vitamin E

A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone, D = Potassium dichromate (5mg/L) alone, E = Potassium dichromate (30mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5mg/L) alone + Vitamin E-supplemented feed.

4. DISCUSSION

The deleterious effect of potassium dichromate in this study was evidenced by the reduction in growth performance parameters in the groups of fish exposed to potassium dichromate only, as the body weight gain and feed efficiency were lower than those exposed to the toxicant plus vitamin E-supplemented feed. The observed reduction in body weight of potassium dichromate treated groups was probably due to high-energy demand and reduced feed consumption. Exposure to toxicants such as pesticide could result in reduced body weight due to reduced feed consumption as reported by Nieves-Puigdoller et al. [20]. In the present study, the growth parameters improved significantly in vitamin E-supplemented groups compared to control group. This was also in agreement with the work of Hossein et al. [21] who recorded an increase in growth parameters in vitamin E-supplemented capsian brown trout fish.

Potassium dichromate increased the relative liver weight when compared with the group B that was fed with vitamin E-supplemented feed where the relative liver weight was decreased. The elevation in the relative liver weight was thought to be due to reduced body weight caused by potassium dichromate while the absolute weight of the liver remained unchanged. Vitamin E is a biological antioxidant that could contribute to improved growth because of its ability to neutralize free radicals and reduce lipid peroxidation in both plasma and muscles [22,23]. According to Buckley et al. [24], apart from microbial spoilage, lipid oxidation is the primary process by which quality loss of muscle foods occurs.

Exposure of African catfish to potassium dichromate resulted in macrocytic normochromic anaemia, which was evidenced by a significant decrease in the PCV, RBC and haemoglobin values; and a significant increase in MCV. The reduction in the RBCs count after exposure to pollutants may be attributed to the inhibition of erythropoiesis or due to an increased rate of erythrocyte destruction in the hematopoietic organs [25]. Reduction of haemoglobin

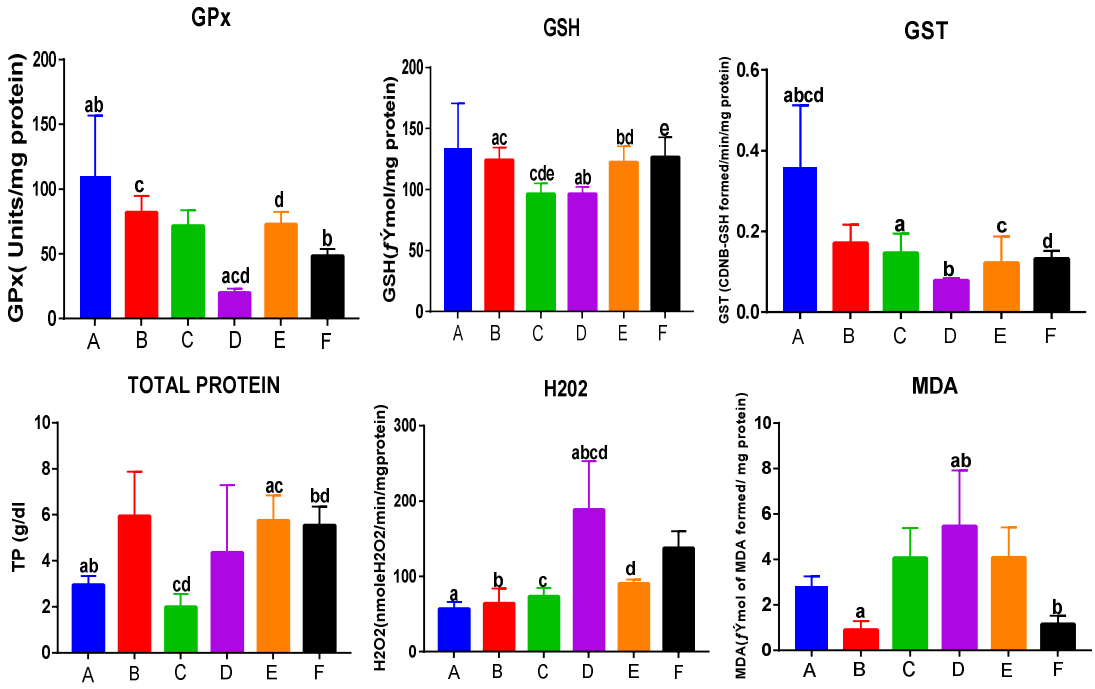

Fig. 4. Effect of potassium dichromate toxicity on markers of oxidative stress in the liver of African catfish as modulated by vitamin E

A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone, D = Potassium dichromate (5 mg/L) alone, E = Potassium dichromate (30 mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5 mg/L) alone + Vitamin E-supplemented feed

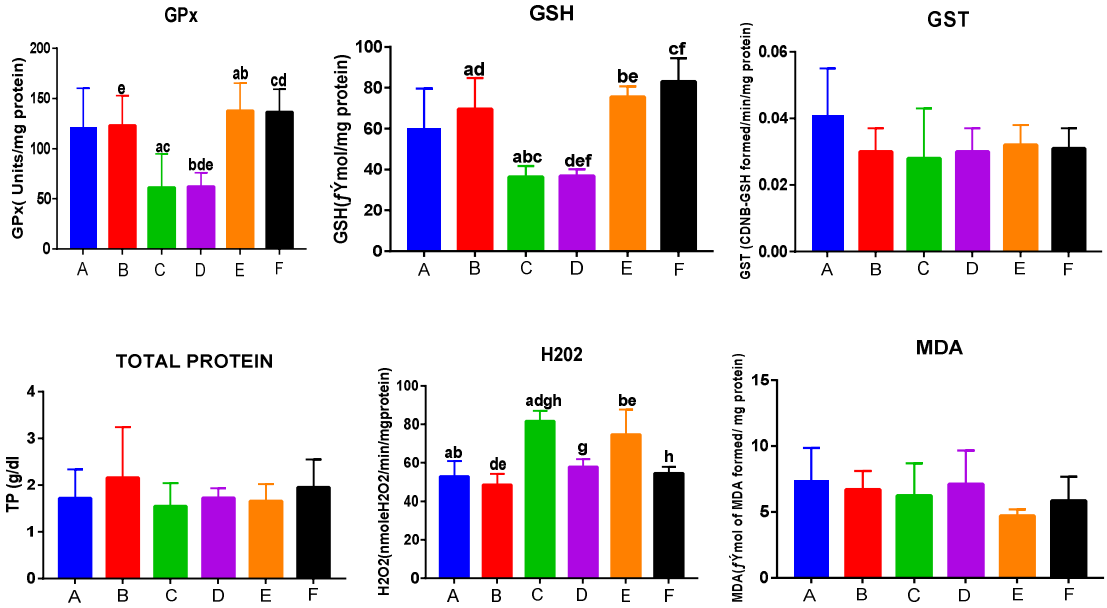

Fig. 5. Effect of potassium dichromate toxicity on markers of oxidative stress in the kidney of African catfish as modulated by vitamin E

A = Control, B = Vitamin E-supplemented feed, C = Potassium dichromate (30mg/L) alone, D = Potassium dichromate (5 mg/L) alone, E = Potassium dichromate (30 mg/L) alone + Vitamin E-supplemented feed, F = Potassium dichromate (5 mg/L) alone + Vitamin E-supplemented feed

concentration affects its oxygen binding capacity [26] and also manifests anaemic condition in fish, which may be due to stress related haemolysis [27]. This reduction in the haemoglobin values in the blood of exposed fish is usually caused by the effect of heavy metals on blood, as well as decrease in the oxygen carrying capacity, which also imply anaemia or validate the toxic effects of potassium dichromate on *Clarias gariepinus* [28,29]. Co-treatment with antioxidant (vitamin E) increased red cell parameters and restored the parameters the level similar to those obtained in the untreated control. This might be due to the antioxidant property of vitamin E favouring integrity and fluidity of membranes and enhancing cellular respiration, which in turn, increases oxygen carrying capacity of haemoglobin.

Erythrocyte osmotic fragility is a measure of erythrocyte strength and its ability to withstand varying osmotic gradients and it has been reported to be increased in situations of low oxygen tension, red blood cell membrane abnormality and during oxidative stress [30]. The result of this study showed a increase in erythrocyte osmotic fragility after exposure to potassium dichromate (5mg/L) at 0.5% NaCl when compared to group F that was co-treated with vitamin E. The reduced erythrocyte osmotic fragility observed in group F was probably due to the protective effect of vitamin E on erythrocyte osmotic fragility [31].

Exposure to potassium dichromate resulted in decreased number of white blood cells (leukopenia). According to Shrivastava et al. [32], leukopenia may result from generalized injury caused to the hematopoietic stem cells in the bone marrow and other erythropoietic organs as a result of heavy metal exposure. This was in line with the work of Adjroud [33] who observed decrease in the number of white blood cells after potassium dichromate exposure in both male and female rats. This was corrected in the fish fed with vitamin E-supplemented feed where the leucocyte values were elevated. This activity of vitamin E may be associated with its antioxidant effect as it plays a vital role in scavenging free radicals [34] and maintenance of fish immunity [35].

Serum biochemical indices including urea and creatinine can be utilized as sensitive and suitable biomarkers in aquatic ecotoxicology, as they provide a primary alarm for potentially dangerous variations in polluting aquatic organisms [36]. The present study showed a significant increase in creatinine level in the fish exposed to potassium dichromate indicating a possibility of kidney damage. This was corrected in the fish co-treated with vitamin E as the creatinine level was reduced even better than the control level.

The present study also revealed significant changes in the activities of markers of oxidative stress in the gill, liver and kidney tissues. Potassium dichromate significantly increased GPx, GSH and GST activities in the liver, kidney and gills. The reduction in the activities of these antioxidants could be due to their exhaustion as a result of the increased production of free radicals. The observed decreased levels of GSH was also in line with that observed by Rasool et al. [37] upon exposure of male mice to potassium dichromate. The reduced GST activity was in conformation with the work of Arojojoye et al. [38] who observed a lowered GST activity in African catfish exposed to heavy metals. Supplementation with vitamin E showed a significant improvement in the concentrations of these antioxidants in the gill, liver and kidney of the exposed fish.

Malondialdehyde (MDA) is produced by lipid peroxidation and considered as an indicator of oxidative stress, which results from the free radicals damage to membrane complements of cells [39]. Bioaccumulation of heavy metals led to increased malondialdehyde deposits, products of lipid peroxidation in both gill and hepatic tissues (Figs. 3 and 4) of fish exposed to potassium dichromate. Alongside, increases were observed in hydrogen peroxide generation in the liver and kidney. Elevated malondialdehyde contents agree with previous works, which showed such as a response to oxidative stress from heavy metals [40,41].

Chromium and its compounds are common environmental pollutants found in soil, rocks, in automobile exhaust and in tobacco products such as traditional and electronic cigarettes and hookahs [1]. According to Wilbur et al. [42], it has been estimated that 66% of current or former hazardous waste sites on the National Priorities List also contain Chromium. Chromium compounds are used in a variety of industries, ranging from electroplating, dyeing, metal refining, fungicide, wood preservatives, ceramic glass, pigment production and leather tanneries [2,3] from where they find their ways into effluents ultimately leading to environmental

pollution [4] Most of the heavy metals cause their toxicities by induction of oxidative stress leading to a disruption of redox signalling and control and/or molecular damages [43,44]. These molecular damages by reactive oxygen species if not detoxified can oxidize proteins, lipids and nucleic acids, often leading to cellular damage or even cell death [45,46].

Vitamin E serves as a chain-breaking antioxidant that prevents the propagation of free radical reactions [47]. One of the roles of Vitamin E as an antioxidant is that it mops up free radicals so as to terminate lipid peroxidation, which can initiate damage to unstable intracellular components including nucleic acids and enzymes, thereby resulting in pathological conditions and indirectly reducing growth [48].

Fish are ideal organisms to monitor aquatic systems because of the position they occupy towards the apex of food pyramids and may therefore, reflect the effects of heavy metals toxicity and environmental pollution on other organisms, including human beings [49]. The advantage of African catfish (*Clarias gariepinus)* as an aquaculture candidate cultured in many parts of the world lies in its ability to withstand adverse environmental conditions, utilize atmospheric oxygen and effectively convert different feedstuff to flesh [50]. Tissue concentrations of heavy metals can be of public health concern to both animals and humans due to the fact that heavy metals can easily accumulate in the tissues of aquatic animals [4].

5. CONCLUSION

Potassium dichromate has proved attractive to fish farmers for use in bacterial and parasites control. This present study showed that potassium dichromate exert a significant influence on the antioxidant system by overproduction of reactive oxygen species while dietary supplementation with vitamin E could modulate and diminish the toxic effects of potassium dichromate on growth and haematological parameters as well as antioxidant defences.

ETHICAL APPROVAL

All experimental protocols were in compliance with University of Ibadan ethics committee on research in animals as well as internationally accepted guidelines for laboratory animal use and care. The protocol was approved by the University of Ibadan Animal Care Use, Research and ethics Committee with ethical clearance number UI-ACUREC/19/0089.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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