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Ameliorative effect of methanol extract of *Telfairia occidentalis* Hook. and *Amaranthus hybridus* Linn. against cadmium induced oxidative stress in rats

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ABSTRACT

Oxidative stress caused by cadmium in living systems leading to various clinical interpretations has been well documented. The study investigated if methanol extract of *Telfairia occidentalis* and *Amaranthus hybridus* could ameliorate the toxicity caused by cadmium. Administration of cadmium lead to an increase in lipid peroxidation marker MDA 0.42±018, 1.66±1.13 and reduced the concentration of enzymatic antioxidants SOD 10.22±2.13, 10.92±0.54, Catalase 22.82±14.74, 19.90±1.16 and GSH 18.02±0.49, 12.89±085 in liver and kidney of the rats. Result for liver function assay showed a significant increase in AST 24.00±3.31, ALT 60.40±10.85 and ALP 78.05±15.75 in the serum of rats administered cadmium without treatment. Total protein concentration was significantly higher in the vegetable treated group when compared to the group administered cadmium without treatment 3.72±041. Kidney function assay, creatinine and urea were significantly high in the rat administered cadmium without treatment when compared to the rats that received the vegetables. Phytochemical screening showed the presence of flavonoids, phenols, alkaloid in high concentration in the vegetables. *In vitro* antioxidant reflected high radical scavenging activity. From the result of this study, the vegetable showed robust ameliorative effect on cadmium induced toxicity which can be labelled to the rich antioxidant content of the vegetables and also the ability of the vegetable to promote phase 2 bio-transformation of xenobiotic.

INTRODUCTION

Cadmium is a heavy metal present in the environment and causes serious environmental and occupational hazard to human (Tarasub *et al.*, 2008). Cadmium causes lipid peroxidation by stimulating the production of superoxide anions and inhibits antioxidants such as superoxide dismutase and glutathione peroxidase, causing accumulation of free radicals that damage the cells and produce chronic disease (Amara *at al.*, 2011).

Cadmium affects cell proliferation, differentiation, apoptosis and other cellular activities (Yuan *et al.*, 2013). Cadmium is readily available in industrialized regions of the world. Studies by Ejiofor *et al.* (2016) showed accumulation of cadmium in water bodies around oil refinery and cement regions in Southern Nigeria. In previous literature, cadmium toxicity is associated with several clinical complications, renal dysfunction, bone diseases, hepatic dysfunction, with renal tubular damage is probably the most common adverse effect. Studies have shown that it toxicity is dose dependent, route of exposure and time (Jarup *at al.*, 2000). The liver is also a point of cadmium toxicity, leading to apoptosis and necrosis (Lopez *et al.*, 2003). The International Agency for Research on Cancer has classified cadmium a potent human carcinogen.

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Practitioners of traditional medicine believe that the constituents of plants are distinctive as they contain active compounds that are crucial in enhancing the well-being of their patients. The ignited interest in the pharmaceutical importance of plants has led to the discovery and adoption of plant extracts which were commonly used in traditional medicine, as alternative source of remedy (Ekaiko *et al.,* 2015). Dietary antioxidants are useful because of their protective roles against oxidative stress involved in the pathogenesis of diseases such as anaemia, diabetics, cardiovascular diseases and most prevalent cancer.

Telfairia occidentalis is a tropical vine grown in Nigeria as a leaf vegetable. The plant is commonly known as fluted gourd, fluted pumpkin, and ugu (Akoroda, 1990). The *Telfairia occidentalis* has been traditionally used in Nigerians as a cure for fatigue and diabetes (Alada, 2000). The high rich iron content of the leaves is employed in boosting blood of lactating and pregnant women (Okoi and Mgbeogu, 1983). Flour produced from the seeds can be used for high-protein breads (Emebiri and Nwufo, 1990). The vegetable *T. occidentalis* can also be prepared for herbal medicine, it is used to treat sudden attack of convulsion, malaria, and anaemia; it also plays a vital and protective role in cardiovascular diseases (Badifu, 1993). Two important antioxidant compounds that have been isolated and elucidated from *T. oidentallius* include; Kaempferol-3-O-rutinoside and Kaempferol. *Amaranthus hybridus* is a species of flowering plant in the amaranth genus. It is commonly known as careless weed, dioecious amaranth, it is edible and highly nutritious (Culpepper, 2006). Suffo *et al*. (2016) reported that *A. hybridus* is rich in phytol, squalene and 1- Eicosanol. In Nigeria, these vegetables have been prepared collectively as a component of food.

Considering the use of these vegetables in folklore medicine and for nutritional purposes, the study investigated of methanol extract of the vegetables could be useful in ameliorating toxicity induced by cadmium in rats.

MATERIALS AND METHODS

Plant material

The vegetables were collected from a Farm in Arochukwu Area of Abia State, Nigeria. The plants were identified by Dr. Garuba Omosun of the Department of Plant Science and Biotechnology, Michael Okpara University of Agriculture Umudike, as *Telfairia occidentalis* and *Amaranthus hybridus* and were issued specimen number MOUAU/COLNAS/ PSB/15/215 and MOUAU/COLNAS/PSB/ 15/216 respectively. The plant materials were obtained in the month of October, 2016.

Plant extraction

The leaves of *Telferia occidentalis* and *Amaranthus hybridus* were washed with running water to remove sand and debris. The leaves were chopped into small pieces, air-dried for five days under shade at room temperature and pulverized using an electronic blender. The dried and pulverized leaves were extracted by cold maceration method for 72 hours at room temperature using methanol (Sigma- Aldrich) in a winchetser bottle (Sukhev, 2008). The mixture was filtered with Whatman No. 1 filter paper. The filtrate was concentrated using vacuum rotary evaporator at 40^{o}c to give residue. The extract was stored in refrigerator at 4^{o}c.

Phytochemical test

Concentration of flavonoids, tannins, cyanide, alkaloids and phenols in the extracts were determined by the method described by Harborne (Sukhdev, 2008) and Edeoga *et al*. (2005)

In vitro antioxidant

In *vitro* antioxidant activity was achieved using *2, 2-Diphenyl-1-picrylhydrazyl* (DPPH) photometric assay as described by Mensor *et al*. (2001). The ferric reducing antioxidant power was carried out as described by Benzie and Strain, (1999).

Experimental animals

Wistar albino rats were used for this study. The animals were obtained from the animal house of the Department of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike. They were kept in aluminium cages in a well-ventilated room, fed with standard pelleted grower feed (Vital Feed, Nigeria) with access to clean drinking water. They were kept at normal environmental temperature. The animals were maintained in accordance with the recommendation of the Guide for the Care and Use of Laboratory Animals (NIH, 2002). The animals were allowed one week for acclimatization. Ethical approval was obtained from the Ethical Committee of the Department of Biochemistry, College of Natural Sciences, Michael Okpara University of Agriculture, Umudike, and was given ethical approval number BCH/ETH/16/035.

Toxicity testing (LD_{50})

The acute toxicity of cadmium chloride ($Cdcl_2$) was determined by single oral administration of $Cdcl_2$ ranging from 150mg/kg to 300mg/kg body weight. The animals were observed for signs of toxicity for 72hours after administration. LD_{50} values calculated showed 212mg/kg B.W of $Cdcl_2$ as toxic dose.

Experimental design and animal grouping

Twenty-five animals were randomly distributed into five groups of five animals per group. The weight of the animals was checked during distribution to ensure a weight difference of +/- 5 inter and intra cages.

Group A served as the control group and received distilled water only.

Group B received 50mg/kg body weight of cadmium chloride once every two days and received 300mg/kg of *Telferia occidentalis* extract daily

Group C received 50mg/kg body weight of cadmium chloride once every two days and received 300mg/kg of *Amaranthus hybridus* extract daily

Group D received 50mg/kg body weight of cadmium chloride once every two days and received 300mg/kg of *Telferia occidentalis* and *Amaranthus hybridus* combined in equal grams daily.

Group E received 50mg/kg body weight of cadmium chloride once every two days.

Experimental period lasted for twenty-one days, after which blood samples were collected through retro-orbital venous plexus. Blood was spun at 895xg for 10mins to obtain serum. The animals were later euthanized and the liver and kidney were dissected out immediately for preparation of liver and kidney homogenate used in antioxidant assay.

Biochemical estimation

Liver enzyme parameters

Aspartate aminotransferase (AST) and alanine transaminase (ALT) were done by the method described by Reitman and Frankel, (1957). Alkaline phosphatase (ALP) was described by the method described by Klein *et al*. (1960), using Randox test kits (Randox Laboratories, UK). Serum total protein was assayed using standard diagnostic kit (Randox Laboratories, UK).

Liver and kidney tissue preparation

Phosphate buffer (9ml) was added to one gram of liver and kidney tissue in well labelled organ bottles. The mixture was homogenized and centrifuged at 1000 x g for 5mins. The supernatant was separated from the pellet and labelled the aliquot. This was used for antioxidant assay.

***In vivo* antioxidant**

Estimation of catalase activity

Catalase activity in liver and kidney homogenate was determined using the modified method described by Atawodi, (2011).

Estimation of SOD activity

Superoxide dimutase (SOD) activity in liver and kidney homogenate was determined by the method described by Sun *et al*. (1988)

Determination of lipid peroxidation

The level of thiobarbituric acid reactive substance (TBARs) and malondialdehyde (MDA) production in liver and kidney homogenate was determined by the method described by Draper and Hadley, (1960).

Determination of reduced gluthathione

GSH activity in liver and kidney homogenate was determined using method described by Owen and Belcher, (1965)

Kidney function parameter

Creatinine and urea concentration was determined using Randox commercial kits from Randox Laboratories, UK.

Statistical analysis

Data obtained were statistically analysed using one-way analysis of variance (ANOVA) followed by Duncan post hoc tests on SPSS (Ver. 22) and mean was separated by least significant difference of the different groups. Significant difference was accepted at the level of $P<0.01$.

DISCUSSION

Phytochemical screening of the methanol extract of the vegetables showed the presence of phenols, flavonoids, alkaloids, cyanide and tannins. Phenol concentration was highest in the vegetables and concentration of tannins were the lowest. *Amaranthus hybridus* however had the highest concentration of all phytochemicals screened for as presented in Table 1. Phytochemicals present in plants have been well documented to have medicinal and therapeutic activities (Himani *et al.*, 2014).

The percentage (%) inhibition of DPPH radicals showed that the extract inhibited DPPH in a dose dependent manner as shown in Table 2. From the obtained result, IC_{50} value for the extract will be slightly above 800µg/mL. Also, *Amaranthus hybridus* extract showed more activity than *Telferia occidentalis*. Result obtained for FRAP assay also showed that the extract possesses reducing power and this was in a dose dependent manner, although *Amaranthus hybridus* was more potent than *Telferia occidentalis* as shown in Table 3. This result justifies the result of values obtained from the phytochemical screening of the vegetables. As earlier reported, *Amaranthus hybridus* showed greater concentration of the phytochemicals reported in this study. The ability of the plant extract to reduce Fe^{3+} to Fe^{2+} would lie in the antioxidant activity of the plant which is a function of it phenolic, flavonoids and tannin content.

Result for LD_{50} for cadmium which was administered to Rats as presented in Table 4 showed a value of 212mg/kg B.W when calculated. Signs of toxicity were paralysis, stretching, weakness, dizziness, urination, chocking followed by death within 48hrs of administration. LD_{50} value for cadmium obtained from our study is in strong agreement with the LD_{50} of cadmium reported by Kotsonis and Klaasen, (1997) as 225mg/kg of cadmium administered as cadmium chloride in water for Rats.

Cadmium, a potent toxic and heavy metal is constantly present in our environment. It concentration is known to increase in industrialised regions (Ejiofor *et al.*, 2016). It exposure occurs through inhalation or contamination of the food chain (Abernethy *et al.*, 2010). It main target point of toxicity is liver and kidney after absorption. Cadmium induces it deleterious effects through generation of oxidative stress (Matovi´c *et al.*, 2011) and also affect liver functioning and metabolism. Valko *et al*. (2005) earlier reported that cadmium toxicity induces oxidative stress, promotes lipid peroxidation actively and depletes glutathione.

Result for antioxidant in liver and kidney homogenate (Table 5 and 7) showed that cadmium toxicity promoted the generation of free radicals and progressed to the generation of lipid peroxidation in liver and kidney organs.

Table 1: Table showing concentration of some phytochemicals (g/100g) in the plant extract.

	Flavonoids (g/100g)	Tanins (g/100g)	Cyanide (g/100g)	Alkaloids (g/100g)	Phenols (g/100g)
Tel. occ.	1.68±0.00	0.06±0.00	0.47±0.01	3.25±0.01	0.86±0.00
Ama. hyb.	1.84±0.01[a]	0.15±0.02[a]	0.55±0.02[a]	3.79±0.04[a]	1.63±0.14[a]

[a] significantly (P<0.05) higher in the same column
values are expressed as mean±S.D of triplicate determination
key- *Tel. occ.* (*Telferia occidentalis) Ama. Hyb* (*Amaranthus hybridus)*

Table 2: Table showing result for *in vitro* antioxidant (DPPH Assay) of plant extract.

	Tel. occ.	*Ama. hyb.*	Ascorbate
25µg/mL	12.48±0.38*	17.52±0.77*	95.51±0.32
50µg/mL	22.31±0.35*	24.11±3.43*	95.67±0.45
100µg/mL	28.87±0.10*	31.10±0.49*	95.74±0.31
200µg/mL	30.43±0.90*	34.60±0.56*	95.17±0.27
400µg/mL	37.32±0.53*	38.00±0.79*	94.96±0.23
800µg/mL	41.36±0.45*	43.26±0.25*	95.74±0.31

* significantly (P<0.05) lower than ascorbate in the same column
key- *Tel. occ.* (*Telferia occidentalis) Ama. Hyb* (*Amaranthus hybridus)*

Table 3: Table showing result for *in vitro* antioxidant (FRAP) of plant extract.

	Tel. occ.	*Ama. hyb.*	Ascorbate (125µg/mL)
25µg/mL	0.29±0.00*	0.31±0.07*	
50µg/mL	0.31±0.00*	0.31±0.00*	
100µg/mL	0.33.±0.00*	0.32±0.01*	2.00±0.00
200µg/mL	0.36±0.00*	0.38±0.04*	
400µg/mL	0.38±0.01*	0.41±0.01*	
800µg/mL	0.42±0.01*	0.46±0.01*	

* significantly *(P<0.05)* lower than ascorbate in the same column
key- *Tel. occ.* (*Telferia occidentalis) Ama. Hyb* (*Amaranthus hybridus)*

Table 4: Table showing acute oral toxicity of $Cdcl_2$.

Acute oral toxicity test after administration of $Cdcl_2$			
S/No.	**Dose (mg/kg)**	**Mortality**	**Toxicity sign**
1	150	0/3	Paralysis, weakeness, urination, choking,
2	300	1/3	same as above, followed by death.

At 150mg/kg B.W dose, paralysis, weakness, frequent urination and chocking were signs of toxicity observed. However, at 300mg/kg B.W, death was recorded.

Table 5: Showing result of antioxidant activity in liver homogenate of test and control animals.

Group	MDA (µMole/mg protein)	SOD (Unit/g protein)	CATALASE (µMole/mg protein)	GSH (µMole/mg protein)
A	0.07±0.00	22.08±2.02[b]	49.84±3.29	20.82±0.47
B	0.20±0.17	19.48±1.57[b]	41.14±14.07	21.50±0.38
C	0.16±0.19	20.50±2.18[b]	57.00±2.90	21.97±1.68
D	0.72±0.05[a]	13.58±1.75	42.36±9.91	22.40±1.11
E	0.42±0.18[a]	10.22±2.13	22.82±14.74[c]	18.02±0.49[c]

([a])- Significantly *(P<0.01)* higher when compared to other groups in the same column.
([b])- Significantly *(P<0.01)* higher when compared to group D and E in the same column.
([c])- Significantly *(P<0.01)* lower when compared to other groups in the same column.
The table above showed that GSH and Catalase concentration was significantly (p<0.01) low in group E compared to group A-D. For SOD, group A-C was significantly higher compared to D and E. For MDA, group D and E were significantly low when compared to group A-C.

Table 6: Showing result of liver function assay of test and control animals.

Group	AST (IU/L)	ALT (IU/L)	ALP (IU/L)	TP (IU/L)
A	15.40±5.94	20.20±18.75	19.35±4.65	6.07±0.74
B	19.40±4.78	45.20±14.87	37.58±5.66[c]	6.65±0.56
C	16.80±4.76	44.40±17.64	30.38±8.87	7.05±0.12
D	17.20±6.57	45.94±13.80	21.13±3.25[b]	7.21±0.63[e]
E	24.00±3.31	60.40±10.85[a]	78.05±15.75[a]	3.72±0.41[d]

([a])- Significantly *(P<0.01)* higher when compared to other groups in the same column.
([b])- Significantly *(P<0.01)* lower when compared to group B and E in the same column.
([c])- Significantly *(P<0.01)* higher when compared to group A and D in the same column.
([d])- Significantly *(P<0.01)* lower when compared to other groups in the same column.
([e])- Significantly *(P<0.01)* higher when compared to group A and E in the same column.
Result for ALT and ALP was significantly higher in group E when compared to group A-D. Result for TP was significantly low in group E when compared to A-D.

Table 7: Showing result of antioxidant activity in kidney homogenate of test and control animals.

Group	MDA (µMole/mg protein)	SOD (Unit/g protein)	CATALASE (µMole/mg protein)	GSH (µMole/mg protein)
A	0.65±0.05	18.17±0.79[d]	32.19±0.74[b]	17.80±1.50
B	0.80±0.10	16.63±0.60	28.87±1.23	16.55±1.70
C	0.58±0.26	15.91±1.19	28.87±2.21	16.21±0.62
D	0.69±0.14	16.51±1.15	30.85±1.00	17.04±0.77
E	1.66±1.13[a]	10.92±0.54[c]	19.90±1.16[c]	12.89±0.85[c]

([a])- Significantly *(P<0.01)* higher when compared to other groups in the same column.
([b])- Significantly *(P<0.01)* higher when compared to group B, C and E in the same column.
([c])- Significantly *(P<0.01)* lower when compared to other groups in the same column.
([d])- Significantly *(P<0.01)* higher when compared to group C and E in the same column.
The table above showed that GSH, SOD and Catalase concentration was significantly (p<0.01) low in group E compared to group A-D. For MDA, group E was significantly high when compared to group A-D.

Table 8: Table showing concentration of creatinine and uric acid in test and control animals.

	Creatinine (mg/dl)	Urea (mg/dl)
A	0.65±0.30	30.39±2.21
B	0.72±0.25	34.64±3.70
C	0.58±0.38	31.16±0.80
D	0.61±0.25	41.64±4.19[b]
E	1.58±0.36[a]	79.25±4.65[a]

([a])- Significantly *(P<0.01)* higher when compared to other groups in the same column.
([b])- Significantly *(P<0.01)* higher when compared to groups A and C in the same column.
Urea and Creatinine concentration was significantly high in group E when compared to group A-D.

This resulted in decrease of antioxidant enzymes, catalase (CAT), superoxide dismutase (SOD) and glutathione (GSH). GSH concentration was significant *(P<0.01)* lower in group E when compared to other group (A-D) in both liver and kidney homogenates. Transport of cadmium to the liver and kidney requires albumin as a potent carrier. Once in the liver, cadmium binds to GSH and is transported to the bile where it can be excreted. The significant *(P<0.01)* increase in the groups (B-D) administered the vegetables when compared to the group administered cadmium alone without treatment indicated the vegetables promoted the synthesis of GSH and promoted it activities.

Result for SOD and CAT showed that cadmium treatment decreased the concentration of these enzymes in liver and kidney tissues. This is an indication of oxidative stress generated by cadmium in the tissues. Shagirtha *et al*. (2011) observed cadmium causes oxidative stress, leading to an increase in oxidative stress biomarker, depletion of antioxidant enzymes and also disturbances in membranes structures. SOD and CAT must work collectively to achieve their purpose (Bakirel *et al.*, 2008). SOD dis-mutation of superoxide anion into hydrogen peroxide and oxygen. CAT breaks down hydrogen peroxide, a potent precursor for generation of free radicals to water and oxygen (Kaushik and Aryadeep, 2014) protecting biological system from oxidative damage. Wijeratne, (2005) reported that hydrogen peroxide can lead to membrane and DNA damage. Administration of the vegetables increased the concentration of these antioxidant enzymes in both liver and kidney homogenates and also upregulated their activities.

Lipid peroxidation biomarker, malondialdehyde (MDA) increased significantly *(P<0.01)* in group E when compared to other groups (A-D). As earlier reported by Shagirtha *et al*. (2011) cadmium promotes generation of reactive oxygen species (ROS) which are free radicals. These ROS reacts strongly with membrane lipids and leads to lipid peroxidation (LPO) and cell death (Wijeratne, 2005). LPO affects membrane fluidity, damages membrane structures and proteins, deactivating membrane receptors (Arulselvan and Subramanian, 2007). Administration of the vegetables, controlled positively the oxidative stress induced by cadmium, by preventing lipid peroxidation. The antioxidant ability of the vegetable can be attributed to it rich phenolic contents. Dietary source of polyphenol has been shown to possess scavenging ability for superoxide anion in biological system (Emmanuel *et al.*, 2015).

Result for liver enzymes in blood serum showed that cadmium induced hepatic damage, and administration of the vegetables was able to either ameliorate the adverse damage induced by cadmium or prevented the damage, indicating that the vegetables possessed hepato-protective ability. As earlier mentioned, cadmium bind with albumin to and successfully enters the liver, where it destroys amino acids in the liver. Once in the liver, metallothionein a protein is made active which binds cadmium and transports it to the kidney for excretion (Bernard, 2008). Cadmium affects liver proteins negatively and also reduces the concentration of these proteins.

Result for ALT and ALP presented in Table 6 was significantly *(P<0.01)* higher in the group E when compared to other groups (A-D). During liver damage, the concentration of liver enzymes increases in liver due to increase in membrane permeability and necrosis. These enzymes leak from the damaged cells and find their way in blood, and when assayed for, their blood concentration is bound to increase above normal.

Administration of the vegetables, protected and promoted normal functioning of the liver under cadmium toxicity. An increase in ALP in group E confirms the hepatotoxic effect of cadmium. In conditions of liver damage, ALP is released into blood stream. This is an indication that cadmium might induce liver carcinogenesis.

Result for total protein (TP) in this study, showed that cadmium administration reduced significantly *(P<0.01)* the concentration of total protein in group E. however, the vegetables administered singly or combined, increased the concentration of TP. The binding of cadmium and albumin can significantly affect the concentration of total protein in blood. Also, cadmium binds strongly to amino acids once in the liver. This binding may also be responsible for the low concentration of total protein in group E. Decline in total protein concentration is a strong indication of liver disease and furthermore, can lead to immunodeficiency. From this study, it could be suggested that the vegetables promoted the synthesis of GSH, which also binds strongly to cadmium and transports it to the bile for excretion, thereby preventing cadmium from binding to amino acids in the liver.

Result for kidney function parameters (Table 8) showed that the concentration of urea and creatinine were significantly (*P<0.01)* higher in the group E when compared to other groups (A-D). Creatinine in the liver where it is synthesized is a breakdown product of creatine phosphate in muscle and serve as biomarker for kidney injury reporting kidney and nephron damage. Creatinine is removed mainly through the kidney, and it concentration in biological systems can be altered by various muscles sizes or decreased muscular activity (Ndukaku *et al.*, 2015).

The increase in creatinine and urea concentration in group E indicates that cadmium is nephrotoxic. Cadmium in the kidney is bound to metallothionein and the toxic form of cadmium Cd^{2+} in the kidney reacts with proteins and cellular components. Cadmium toxicity in kidney affects tubular reabsorption capacity and also glomerular filtration.

This leads to a high loss of proteins in urine. Result from this study, clearly showed that the vegetables administered singly or combined improved kidney function as shown by the concentration of urea and creatinine.

From this study, it is clear that cadmium induced oxidative stress in liver and kidney tissue. However, the vegetables play a protective role in ameliorating the harmful effects of cadmium on the kidney and liver tissue. Phytochemical studies indicated that the vegetables are rich in phenolic compounds, which may be responsible for the obtained result of the study. Also, fractionation and identification of bio active compound of the vegetables will be useful in promoting the aim of this study.

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