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Original Research Article

Neurological Consequences of Cadmium Exposure and *Telfairia occidentalis* Extract Intervention in Wistar Rats

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Abstract: This study investigated the neurologic effect of *Telfairia occidentalis* on cadmium chloride-induced toxicity in male rats. Twenty-five male rats were grouped as follows: Group A: control, Group B-E: $CdCl_2$ 0.5ml; $CdCl_2$+ *T. occidentalis* 0.3ml; $CdCl_2$+*T. occidentalis* 0.5ml and $CdCl_2$+ *T. occidentalis* 0.7ml, respectively. Gas chromatography coupled to flame ionization detector, Ultraviolet visible ray spectrophotometry and total antioxidant capacity of *T. occidentalis* was done. Oxidative stress markers, muscle function markers and plasma electrolyte were assayed. The gait parameters of the animals were observed using gait test apparatus. This study lasted for 4 weeks. Blood and brain samples were collected. The phytochemical results revealed alkaloids, flavonones, terpenes, phenols, anthrocyanins and epicatechin. The biochemical results were in comparison with control and group B. The results were statistically significant at 95% confidence interval. MDA decreased in groups C, D and E, PC decreased in groups C, D and E and F_2ISO_P decreased in group C, D and E. An increase in SOD, CAT, GPx and GSH in groups C, D and E. A decrease in plasma creatine kinase level and an increase in AChE levels of groups C, D and E was revealed. There was no change in level. SL, SDL and BOS values increased in groups C, D and E. This study revealed that *T. occidentalis* has the tendency to ameliorate neurotoxicity and anemia induced by cadmium chloride.

Keywords: Neuroscience, Neurotoxicity, Heavy Metals, Gait, Brain.

INTRODUCTION

Telfairia occidentalis (fluted pumpkin) commonly called *ugu* is a vegetable which belongs to the family Cucurbitaceae [1]. *T. occidentalis* (family Cucurbitacea) is a herbal plant found along the fringes of the closed forest in Africa and particularly cultivated from Sierra Leone to Southern Nigeria among other areas [2]. It is a crop of commercial importance grown in West Africa with Nigeria, Ghana and Sierra Leone, being the major producers [2, 3]. It is called *ugu* in Igbo land, *iroko* in Yoruba land and *umeke i*n Edo. Fluted pumpkin is cultivated majorly for its leaves and is eaten as potherbs. A potherb being, any plant, used to add flavour in cooking and the seeds can be eaten whole by boiling [4]. In Nigeria, the herbal preparation of this plant has been employed in the treatment of sudden attack of convulsion, malaria and anaemia [5]. Despite its widespread usage as food and medication, information on the biological activity of the plant is very scanty but from the available, the medicinal potential of the leaf of *T. occidentalis* is no longer in doubt. It is now obvious that the plant has been proven to possess beneficial antioxidant, antidiabetic, hepatoprotective, haematological, antiplasmodial, antimicrobial, testiculoprotective, anticancer, anti-inflammatory, anxiolytic and sedative properties. Fluted pumpkin has been found to be medicinally useful, apart from its nutritional, agricultural and industrial importance [6]. *T. occidentalis* is used ethnopharmacologically for the treatment of anemia [6-8]. The aim of the study is to investigate the possible antioxidant role of *T. occidentalis* in cadmium chloride-induced toxicity in the brain of male rats.

MATERIALS AND METHODS

Ethical Approval

This study was approved by the Research Ethics Committee of Madonna University, Elele, with

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reference; MAU/DRC/HD/E/PHYS/2024/0211. Every member of the research team obeyed standard protocols regarding the use of experimental animals in conducting biomedical research.

Procurement of *Telfaira Occidentalis*

Sample Collection and Sample Treatment

The sample of *T. occidentalis* used in this study was collected from a farm site at Elele in Rivers state, Nigeria. Prior to analysis, the leaves were washed with tap water then rinsed with distilled water. The residual moisture was evaporated at room temperature thereafter the leaves were sun dried at about 35^0C until properly dried. The dried leaves were then ground in porcelain mortar, sieved through 2 mm mesh sieve and stored in polythene bag. The powdered sample was used for both proximate and mineral analysis. Moisture content was determined using fresh leaves.

Procurement of Cadmium Chloride

The cadmium chloride was purchased from Sigma-aldrich limited Germany with EC number 233-296-7.

Phytochemical Screening

The Phytochemical Screening of different constituents of *T. occidentalis* was done by Gas Chromatography Coupled to flame ionization detector (GCFID). Using Standard Procedure [9], Ultraviolet-Visible spectrophotometry (UV-VIS) was done using standard procedure by Wanyika *et al.*, (2011) and Total Antioxidant Capacity (TAC) using already established standard procedures [10].

Concentration and Dosage of Treatments

Concentration of *T. Occidentalis* Administered

10000 mg of *T.occidentalis* was dissolved in 250ml of water 400mg/ml of *T.occidentalis* was administered to the rats 0.3ml and 0.7ml were administered to the rats as low and high dose respectively.

Concentration of Cadmium Chloride

1000 mg of Cadmium was dissolved in 500ml of water, the concentration is 2mg/ml 0.5ml was administered to the rats.

Study Animals

Procurement

30 male Wistar rats (*Rattus norvegicus*) weighing 80g to 120g aged 2 to 3 weeks were Purchased and Housed in Research animal house, Department of Human Physiology, Madonna University, Elele, Rivers State, Nigeria. The animals were allowed to acclimatize for a period of and 12 days exposed to 12/12 hours' light/dark cycle. Animals had access to feed and water ad libitum. The animals were kept in line with laid down principles for animal care as prescribed in Helsinki`s 1964 declaration. The animal ethics committee of Madonna University, Nigeria approved our study protocol kindly.

The animals were randomly assigned differently into 5 groups of 5 rats each. After 12 days of acclimatization, administration of Cadmium Chloride and *T. occidentalis* commenced.

Exclusion Criteria

The following qualities disqualified a sample in this study;

- ✓ Female wistar rat
- ✓ Above or below 4 to 6 weeks of age
- ✓ Body weight Above or below 80 to 120g
- ✓ Physically deformed
- ✓ Already tested

Animal Welfare

They were housed in clean cages with a room temperature of 23 to 25^0C after acclimatization for 10 days. They were allowed to roam freely within their cages. The feeding depended on the Study design. No animal was Subjected to pain suffering or restraint.

Study Design

Table 1: Study Design

Groups	Administration
1	Water + Feed *Ad libitum*
2	$CdCl_2$ (0.5ml)
3	$CdCl_2$ + *T. occidentalis* 0.3ml
4	$CdCl_2$ + *T. occidentalis* 0.5ml
5	$CdCl_2$ + *T. occidentalis* 0.7ml

N=5

Study Duration: After 12 days of acclimatization, this study lasted for about 3 weeks and half (24 days).

Gait Test

The gait test was performed in a well-lit and ventilated room using a Gait Test Apparatus (GTA). This apparatus consists of a closed chamber and an open runway. Both hind paws of the rats were placed in blue-black ink. The rats were placed on a plain white paper and allowed to walk freely towards the closed chamber. All animals were assayed and the apparatus was cleaned with methylated spirit before and after each assay.

Gait Function Parameters include:

Step length (SL): The distance between the point of initial contact of one paw and the point of initial contact of the opposite paw in one step.

Stride Length (SDL): The distance between successive points of initial contact of the same paw.

Base of Support (BOS): The farthest distance between opposite paws usually in equal plane.

Number of Steps (NOS): The amount of step taken to reach the inner chamber.

Sample Collection

The animals were anaesthetized with chloroform before an incision was made in the cranial region, the skull was opened and the brain was carefully removed and Placed on Ice before Homogenization.

Laboratory Analysis

Brain Tissue Homogenization

All animals were anaesthetized with chloroform (0.5 ml i.p.) and perfused with normal Saline (0.9% NaCl) followed by 4% Paraformaldehyde (PFA) in phosphate buffer (PB; 0.1 M pH 7.4). The brains were removed from the skull and placed in normal saline for biochemical analysis.

Biochemical Analysis

Oxidative stress markers and Muscle function markers were assayed for. Oxidative stress markers used include δ-amino levulinic acid (D-ALA), Glutathione Peroxidase (GPx), Superoxide dismutase (SOD), Malondialdehyde (MDA), Catalase (CAT), Protein Carbonyl (PC), Glutathione Reductase (GR) and Reduced Glutathione (GSH). The Muscle Function Markers used include Creatine Kinase (CK) and Acetylcholinesterase (AChE).

Assay for Glutathione Reductase (GR)

Glutathione reductase together with its co-factor, NADPH, catalyzes the reduction of oxidized glutathione (glutathione disulfide, GSSG) to glutathione wherein the oxidation of NADPH to NADP+ is monitored as a decrease in absorbance at 340 nm. This rate of decrease in A340 is directly proportional to the glutathione reductase activity in the sample because the enzyme is present at rate limiting concentrations. The unit is U/g. The reaction mixture consisted of 1.65 ml phosphate buffer: (0.1 mol; pH 7.6), 0.1ml ethylene diamine tetra acetic acid (EDTA) (0.5 mmol), 0.05 ml oxidized glutathione (1 mmol), 0.1 ml nicotinamide adenine dinucleotide phosphate (NADPH) (0.1 mmol) and 0.1 ml of homogenate in a total volume of 2 ml. Enzyme activity were quantitated at 25°C by measuring disappearance of NADPH at 340 nm and was calculated as nmol NADPH oxidized/ min/mg protein using a molar extinction coefficient of 6.22×10^3 $M^{-1}cm^{-1}$.

Assay for Reduced Glutathione (GSH)

The reduced form of glutathione comprised in most instances the bulk of cellular non-protein sulfhydryl groups. This method is therefore based upon the development of a relatively stable yellow colour when 5,5 dithiobis-2-nitrobenzoic acid (Ellman's reagent) is added to sulfhydryl- compounds. The chromorphoric product resulting from the reaction of Ellman's reagent with the reduced GSH is measured at 412nm. The unit is U/g. 0.2ml of the sample was added to 1.8ml of d/w and 3ml of the precipitating solution and mixed. Allow to stand for 5 min and centrifuged at 4000rpm for 10 mins. 1ml of filtrate was added to 4ml of 0.1m phosphate buffer. 0.5ml of DTNB was finally added. Read and record the absorbance at 412nm using a prepared blank to zero the spectrophotometer.

Assay for Creatine Kinase (CK-MM)

Creatine kinase (CK-MM), present in the sample, catalyses the transfer of a high energy phosphate group from creatine phosphate to ADP. The ATP produced in this reaction is subsequently used to phosphorylate glucose to produce glucose-6-phosphate (G-6-P) in the presence of hexokinase. G-6-P is then oxidized by glucose-6-phosphate dehydrogenase (G-6-PDH) with the concomitant reduction of nicotinamide adenine dinucleotide phosphate (NADP) to nicotinamide adenine dinucleotide phosphate reduced (NADPH). The rate of formation of NADPH is monitored at 340 nm and is proportional to the activity of CK in the sample. The unit is (U/L). 110 µL water and (10 µL Calibrator + 100 µL water) were transferred into separate wells of a clear bottom 96-well plate. 10 µL of sample was transferred into separate wells.100 µL Reconstituted Reagent was added and the plate was tapped to mix. It was then incubated at room temperature or 37°C. CK was fully activated within 20 min by glutathione provided in the Substrate Solution. OD340nm was read at 20 min and again at 40 min.

Assay for Acetylcholinesterase (AChE)

It is based on the reaction between thiols and chromogenic 5,5'- dithiobis-2-nitrobenzoic acid (DTNB) as it measures the formation of the yellow ion of 5-thio-2-nitrobenzoic acid (TNB). The unit is (U/L). The reaction mixture (3 ml) in quartz cuvette having 1cm path length contained 0.50mM of ATI, 0.5 mM of DTNB and 50 mM phosphate buffer (pH 7.4). The change in optical density was measured at 412 nm for 3 min at each interval of 30 sec. The AChE activity was calculated using extinction coefficient $13.6x10^3 M^{-1} cm^{-1}$.

Electrolyte Assay

Calcium is measured using a Potentiometer. This method determines the potential difference that develops between the inner and outer phases of an ion selective electrode. The electrode is made of a selectively permeable material to calcium ion. The potential is measured by comparing it to the potential of reference electrode. Since the reference electrode has a constant potential, the voltage difference between the two electrodes is attributed to the concentration of ionized calcium in the sample[10]. The unit is mg/dl. Determination of Sodium Ion (Na^+) and Serum Potassium (K^+) Concentration were measured using Centronic GmbH kit via turbidimetric determination method. The unit of Na^+ is mmol/L and for K^+ meq/L.

Statistical Analysis

Data was expressed as mean ± SEM and ($P<0.05$) was considered statistically significant. Data collected from this study was analyzed using One-Way analysis of variance (ANOVA) and Post Hoc analysis with the aid of IBM®SPSS Version 21.0.

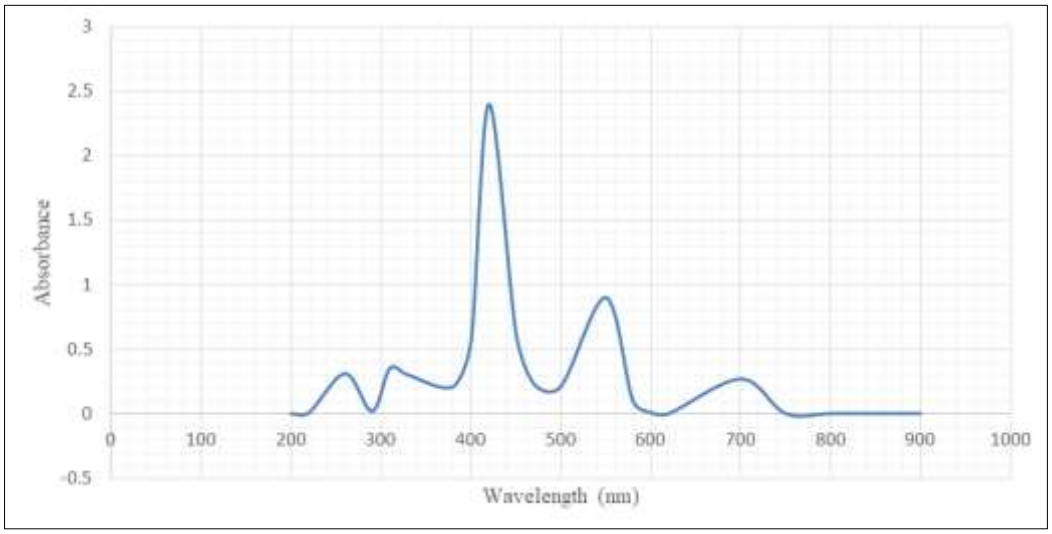
Results: The results of this study are presented as follows;

Table 2: GC-FID Phytochemical screening of *T. occidentalis*

Component	Concentration	Unit
Tannins	5.1110	ug/ml
Phytate	7.2717	ug/ml
Saponins	5.1020	ug/ml
Terpenes	12.4424	ug/ml
Anthocyanin	8.6706	ug/ml
Cardiac glycosides	7.2312	ug/ml
Resveratol	0.1242	ug/ml
Procyanidin	4.8427	ug/ml
Phenol	12.8186	Ppm
Epicatechin	10.5146	ug/ml
Flavonones	10.7127*	Ppm
Steroids	1.4011	Ppm
Alkaloids	13.0167	ug/ml
Flavone	6.1040	Ppm
Lectin	2.6121	ug\ml
Anthraquinone	10.1051	ug/ml
Balsam	4.0243	ug/ml
Oxalate`	8.3614	ug/ml
Catechin	7.1004	ug/ml

Table 3: UV-VIS wavelengths and absorbance of *T. occidentalis*

Wavelength (nm)	*Telfaira occidentalis*
200	0.001
220	0.010
260	0.311
290	0.020
310	0.354
330	0.301
380	0.210
400	0.561
420	2.400
450	0.624
500	0.212
550	0.900
580	0.100
600	0.010
620	0.001
700	0.270
750	0.001
800	0.002
850	0.001
870	0.001
900	0.001



The result from the UV-VIS Analysis show peaks at 260nm, 310nm, 330nm, 400nm, 420nm 450nm and 550nm with absorbance of 0.311, 0.354, 0.301, 0.561, 2.4, 0.624 and 0.9 respectively. The spectra for phenolic compounds (tannins) and flavonoids typically lie in the range of 230-290 nm [11]. The peak 420 was also detected in this study; a previous study highlighted that peaks occurring at 400–450nm indicate the presence of carotenoids [12]. The peak 420 was also identified, and this is characteristic for tannins due their occurrence at 350–500nm [11, 12]. The peaks 420, and 550 were characteristic for terpenoids, known to occur at 400–550nm [13-15]. The peak 610 was identified as chlorophyll, and this is due to its occurrence at 600–700nm [16-18]. In previous studies, absorption bands that occur at 234–676nm are characteristic for alkaloids, flavonoids, and phenolic compounds [13-18]. In another study peaks at 208-881 nm reveals that the absorption bands are due to the presence of flavonoids, phenol and its derivatives [18-20].

Table 4: Total antioxidant Capacity of *T. occidentalis*

Conc. of T. *occidentalis*	Abs. of *T. occidentalis*
20mg/ml	0.96
40mg/ml	0.112
60mg/ml	0.172

80mg/ml	0.227
100mg/ml	0.280

Table 5: Total antioxidant capacity of Ascorbic acid standard

Concentration of Ascorbic Acid Standard	Absorbance
20mg/ml	0.301
40mg/ml	0.373

At 20mg/ml and 40mg/ml the absorbance of *T. occidentalis* was 0.96 and 0.112 and that of Ascorbic Acid (A well-known antioxidant which was used as a standard), 0.301 and 0.373 respectively. Consequently, an increased concentration of *A. sativum* (100mg/ml) showed an absorbance of 0.280 indicating a positive correlation in the absorbance of *T. occidentalis* and Ascorbic acid. This indicates that *T. occidentalis* has a similar antioxidant activity to ascorbic acid.

Table 5: Organic content screening of *T. occidentalis*

Content	Composition (%)
Moisture	43.27
Ash content	7.62
Crude fiber	15.11
Lipid content	2.28
Crude protein	11.44
Carbohydrate	10.33

Table 6: Mineral content screening of *T. occidentalis*

Mineral	Composition (Mg/kg)
Na	96.10
Ca	87.32
K	57.12
Mn	0.61
Mg	1.34
Cu	0.002
Zn	0.010

Table 7: Changes in brain stress markers in response to *T. occidentalis*

Groups	SOD	CAT	GPx
Control	$2.12\pm0.06^{\wedge}$	$1.38\pm0.01^{\wedge}$	$2.86\pm0.23^{\wedge}$
$CdCl_2$ 0.5ml	$0.94\pm0.14^{*}$	$0.07\pm0.01^{*}$	$0.19\pm0.03^{*}$
$CdCl_2$+To 0.3ml	$2.24\pm0.14^{\wedge}$	$1.62\pm0.09^{\wedge}$	$3.72\pm0.13^{*\wedge}$
$CdCl_2$+To 0.5ml	$4.06\pm0.31^{*\wedge}$	$2.86\pm0.11^{*\wedge}$	$4.49\pm0.07^{*\wedge}$
$CdCl_2$+To 0.7ml	$5.96\pm0.21^{*\wedge}$	$4.28\pm0.12^{*\wedge}$	$5.61\pm0.27^{*\wedge}$
Total	15.32	10.21	16.87
Average	3.06	2.04	3.374

Key: *($P\leq0.05$)-Significantly different compared to control group, $^{\wedge}$($P<0.05$)-Significantly different compared to $CdCl_2$ group. To-*T. occidentalis*

Results showed a significant increase in SOD levels in group C ($CdCl_2$+To 0.3ml), D ($CdCl_2$+To 0.5ml) and E ($CdCl_2$+To 0.7ml) when compared to the control and $CdCl_2$ group. A significant decrease in SOD levels in Group B ($CdCl_2$ 0.3ml) when compared to control was also shown in the result.

Results showed an insignificant increase in CAT levels in group C ($CdCl_2$+To 0.3ml) when compared to control, but it, together with D ($CdCl_2$+To 0.5ml) and E ($CdCl_2$+To 0.7ml), showed significant increase when compared to the $CdCl_2$ group. Group Dand E showed significant increase when compared to control. A significant decrease in CAT levels in Group B ($CdCl_2$ 0.3ml) when compared to control was also shown in the result.

Results showed a significant increase in GPx levels in group C ($CdCl_2$+To 0.3ml), D ($CdCl_2$+To 0.5ml) and E ($CdCl_2$+To 0.7ml) when compared to the control and $CdCl_2$ group. A significant decrease in GPx levels in Group B ($CdCl_2$ 0.3ml) when compared to control was also shown in the result.

Table 8: Percentage change in SOD, CAT and GPx levels in cerebral cortex in response to *T. occidentalis*

Groups	SOD(U/g)	%c*	CAT(U/g)	%c*	GPx(U/g)	%c*
Control	2.12±0.06^	-	1.38±0.01^	-	2.86±0.23^	_
$CdCl_2$ 0.5ml	0.94±0.14*	-55.66	0.07±0.01*	-94.93	0.19±0.03*	-267
$CdCl_2$+To 0.3ml	2.24±0.14^	5.66	1.62±0.09^	17.39	3.72±0.13*^	30.06
$CdCl_2$+To 0.5ml	4.06±0.31*^	91.51	2.86±0.11*^	107.25	4.49±0.07*^	57.00
$CdCl_2$+To 0.7ml	5.96±0.21*^	181.10	4.28±0.12*^	210.15	5.61±0.27*^	116.21
Total	15.32		10.21		16.87	
Average	3.06		2.04		3.374	

Key: SOD- Superoxide dismutase, CAT- Catalase, (%c)- Percentage Change, asterisk (*) indicates statistical significance, ^(P<0.05)-Significantly different compared to $CdCl_2$ group

There was significant increase (P≤0.05) in SOD levels in group C with percentage change (5.66), group D with percentage change (91.51) and group E ($CdCl_2$+AS 1.5ml) with percentage change (181.10) compared to control. There was a significant decrease (P≤0.05) in CAT levels in group B ($CdCl_2$ 0.3ml) with percentage change (-55.66) compared to control.

There was significant increase (P≤0.05) in CAT levels in group C with percentage change (17.39), group D with percentage change (107.25) and group E with percentage change (210.15) compared to control. There was a significant decrease (P≤0.05) in CAT levels in group B ($CdCl_2$ 0.3ml) with percentage change (-94.93), compared to control.

There was significant increase (P≤0.05) in GPx levels in group C with percentage change (30.06), group D with percentage change (57.00) and group E with percentage change (116,21) compared to control. There was a significant decrease (P≤0.05) in GPx levels in group B ($CdCl_2$ 0.3ml) with percentage change (-267), compared to control.

Table 9: Changes in F_2IsoP and GSH levels in cerebral cortex in response to *T. occidentalis*

Groups	GSH(U/g)	%c*	F_2IsoP(U/g)	%c*
Control	4.3±0.08	-	1.6±0.08	_
$CdCl_2$ 0.5ml	0.38±0.05*	-91.16	8.2±0.24*	412.5
$CdCl_2$+To 0.3ml	4.5±0.15	4.65	0.7±0.11	-56.3
$CdCl_2$+To 0.5ml	4.98±0.10	15.81	0.35±0.04	-78.1
$CdCl_2$+To 0.7ml	6.24±0.18*^	45.12	0.08±0.04*^	-95
Total	20.4		10.93	
Average	4.08		2.186	

Key: SOD- Superoxide dismutase, CAT- Catalase, (%c)- Percentage Change, asterisk (*) indicates statistical significance, ^(P<0.05)-Significantly different compared to $CdCl_2$ group

Results showed an insignificant increase in GSH levels in group C ($CdCl_2$+TO 0.3ml) when compared to control, but it, together with D ($CdCl_2$+To 0.5ml) and E ($CdCl_2$+TO0.7ml), showed significant increase when compared to the $CdCl_2$ group. Group D and E showed significant increase when compared to control. A significant decrease in GSH There was significant increase in GSH levels in group D with percentage change (15.81) and group E with percentage change (45.12).

There was significant decrease in GSH levels in group B (-91.16) compared to control levels in Group B ($CdCl_2$ 0.3ml) when compared to control was also shown in the result.

There was significant decrease in F_2IsoP levels in group C with percentage change (-56.3), group D with percentage change (-78.1) and group E with percentage change (-95). There was significant increase in group B (412.5) compared to control.

Table 10: MDA and PC levels in the cerebral cortex in response to *T. occidentalis*

Groups	MDA (mmol/g)	PC (mmol/g)
Control	1.14±0.03^	3.63±0.16^
$CdCl_2$ 0.5ml	6.25±0.10*	3.17±0.32*
$CdCl_2$+To 0.3ml	1.12±0.05^	3.24±0.05^
$CdCl_2$+To 0.5ml	0.73±0.06*^	1.67±0.38*^
$CdCl_2$+To 0.7ml	0.33±0.08*^	0.14±0.03*^
Total	9.57	11.85
Average	1.914	2.37

Key: MDA-Malondialdehyde, PC-Protein carbonyl, *(P≤0.05)-Significantly different compared to control group, ^(P<0.05)-Significantly different compared to $CdCl_2$ group.

Results showed a significant decrease in MDA levels in group C ($CdCl_2$+To 0.3ml), D ($CdCl_2$+To 0.5ml) and E ($CdCl_2$+To 0.7ml) when compared to the control and $CdCl_2$ group. A significant increase in MDA levels in Group B ($CdCl_2$ 0.3ml) when compared to control was also shown in the result. Results showed an insignificant decrease in PC levels in group C ($CdCl_2$+To 0.3ml) when compared to control, but it, together with D ($CdCl_2$+To 0.5ml) and E ($CdCl_2$+To 0.7ml), showed significant decrease when compared to the $CdCl_2$ group. Group D and E showed significant increase when compared to control. A significant decrease in PC levels in Group B, group D and group E when compared to control was also shown in the result. There was insignificant decrease (P≤0.05) in MDA levels in group C with percentage change (-1.75) compared to control. There was a significant decrease in MDA levels in group D with percentage change (-35.96) and group E (-71.05) compared to control. There was significant increase in group B with percentage change (448.2) compared to control. There was significant decrease in PC levels in group B with percentage change (-12.67), group C with percentage change (-10.74), group D with percentage change (-54) and group E with percentage change (-96.14) compared to control.

Table 11: CK and AchE levels in the blood plasma in response to *T. occidentalis*

Groups	CK-MM(U/L)	AchE(U/L)
Control	10.77±0.25	8.63±0.46
$CdCl_2$ 0.5ml	20.95±2.09	3.92±0.95
$CdCl_2$+To 0.3ml	9.77±0.65	9.21±0.34
$CdCl_2$+To 0.5ml	7.61±0.70	13.32±0.65
$CdCl_2$+To 0.7ml	5.85±0.46	14.26±0.58
Total	54.95	49.34
Average	10.99	9.87

Key: CK-MM= Creatine Kinase-Muscle, AChE= Acetylcholinesterase, *(P≤0.05) = Significantly different compared to control group

Results showed a significant decrease (P≤0.05) in CK-MM levels in group C ($CdCl_2$+TO 0.3ml), D ($CdCl_2$+TO 0.5ml) and F ($CdCl_2$+TO 0.7ml) when when compared to the control and $CdCl_2$ group. A significant increase (P≤0.05) in CK-MM levels in Group B when compared to control, was also shown in the result. Results showed a significant increase (P≤0.05) in AChE levels in group C ($CdCl_2$+TO 0.3ml), D ($CdCl_2$+TO 0.5ml) and E ($CdCl_2$+TO 0.7ml) when compared to the control and $CdCl_2$ group. A significant decrease (P≤0.05) in AChE levels in Group B ($CdCl_2$ 0.3ml) when compared to control was also shown in the result.

Table 12: Plasma Electrolyte (Ca^{2+} and Na^{+}) levels

Groups	Ca^{2+}(mg/dL)	Na^{+} (mmol/L)
Control	2.52±0.14	0.50±0.01
$CdCl_2$ 0.5ml	3.48±2.13	0.47±0.02
$CdCl_2$+TO 0.3ml	2.52±0.21	0.46±0.01
$CdCl_2$+TO 0.5ml	2.70±0.26	0.84±0.02
$CdCl_2$+TO 0.7ml	3.10±0.24	1.43±0.9
Total	14.32	3.70
Average	2.864	0.74

Key: *(P≤0.05)-Significantly different compared to control group, ^(P≤0.05)-Significantly different compared to $CdCl_2$ group. Data represented as Mean ±SEM. P≤0.05 was considered significant.

There was no significant change in the Ca^{2+} levels in all groups when compared to control and $CdCl_2$ groups. There was also no significant change in the Na^{+} levels in all groups when compared to control, $CdCl_2$ group.

Table: 13: Gait test Analysis showing Step Length, Stride Length and Base of Support.

Groups	SL(cm)	SDL(cm)	BOS(cm)
Control	4.32±0.97	6.88±0.12	4.44±0.18
$CdCl_2$ 0.5ml	3.24±0.81	4.04±1.02	2.72±0.71
$CdCl_2$+TO 0.3ml	5.10±0.23	7.8±1.20	4.68±0.20
$CdCl_2$+TO 0.5ml	5.04±0.34	7.1±0.27	5.00±0.10
$CdCl_2$+TO 0.7ml	5.32±0.25	8.38±0.23	5.40±0.19
Total	23.02	34.2	22.24

Average	4.604	6.84	4.45

DISCUSSION

Alkaloids are nitrogen-containing natural products found in bacteria, fungi, animals, and plants with complex and diverse structures [21]. They are commonly isolated from plants; however, they have also been found in animals, insects, marine invertebrates, and some microorganisms [22, 23]. Currently, the Dictionary of Alkaloids reports more than 40,000 compounds [24]. Flavonones, also known as dihydroflavones, are an important class of flavonoids widely found in citrus fruits. For instance, flavanone glycosides, like naringin, naringenin, and naringenin 7-O-neohesperidoside, can be found in grapefruits, hesperidin, hesperetin, and hesperetin 7-O-srutinoside in oranges, mandarins, limes, and lemons, and eriocitrin, eriodictyol, and eriodictyol 7-O-rutinoside in lemons [21]. Many natural flavanones are also linked to sugars, usually in the form of 7-O-glycosides, but several examples present prenyl side chains [21, 22]. Flavonones are associated with a number of health benefits because of their free radical scavenging properties. Plant phenolic compounds combine metabolites that contain one or more phenolic residues and have different numbers of oxygroups and substituents [22-24]. they play an important role in the metabolism of plant cells, and are also involved in chemical interactions between organisms due to allelopathic, antibacterial and antifungicidal activity, regulate the functions of the rhizosphere and participate in the formation of humus [23]. Anthocyanins are a class of water-soluble flavonoids widely present in fruits and vegetables. Cyanidin, delphinidin, malvidin, peonidin, petunidin, and pelargonidin are the six common anthocyanidins. Following consumption, anthocyanin, absorption occurs along the gastrointestinal tract, the distal lower bowel being the place where most of the absorption and metabolism occurs. In the intestine, anthocyanins first undergo extensive microbial catabolism followed by absorption and human phase II metabolism. This produces hybrid microbial–human metabolites which are absorbed and subsequently increase the bioavailability of anthocyanins [24, 25]. Epicatechins are phytochemicals that aid in decrease of blood pressure (Corti *et al*., 2009). There is a large body of literature proposing that one of the main beneficial effects of (−)-epicatechin is via its ability to directly or indirectly scavenge ROS by chemically reacting with ROS or by modulating pathways that regulate ROS scavenging compounds and enzymes, respectively. The antioxidant activity of *A. cepa* has been studied by various researchers [10, 11]. Its similar antioxidant activity with the ascorbic acid standard is an indicator of the high antioxidant activity of *T.occidentalis*. The activity of GR and GPx is also inhibited by cadmium administration [26]. These findings can be explained by direct interaction of cadmium and functional groups of enzymes such as cadmium binding to -SH groups, or metal cofactors replacement with cadmium from the enzyme active sites, whereas the decrease in GPx activity could be due to the competition between GPx and metallothioneins for S-amino acids [26-28]. Antioxidant catalysts, such as SOD enzymes, help to regulate and "deactivate" ROS, turning them into less harmful molecules such as oxygen (O_2) and hydrogen peroxide (H_2O_2) (Sheng, *et al*., 2014). Results from this study showed a significant increase in SOD, CAT, GPx and GR levels in treatment groups when compared to the control indicating the antioxidant role of T.occidentalis. From previous studies, *T.occidentalis* had a reductive effect on cadmium-induced toxicity as manifested by the increase activities of the antioxidant enzymes, SOD and GPx, post treatment in groups. The increase and restoration of the enzyme activities following the treatment could be attributed to the presence of phenolic compounds and flavonoids. The plant has higher free than the bound polyphenols and the free soluble polyphenols had better antioxidant activity than the bound one [29, 30]. The leaf has the ability to reduce iron from Fe^{+3} to Fe^{+2} state [31]. Telfairia occidentalis leaf is very rich in vitamin C, flavonoids and phenolics. The antioxidant property of *T. occidentalis* has been attributed to its high level of polyphenols, particularly flavonoids [32]. GSH play an important role in the detoxification and metabolism of many xenobiotic compounds. The increase in GSH levels in groups treated with *T.occidentalis* may be due to the activation of the nuclear factor erythroid-2-related factor 2 (Nrf2). The Nuclear factor erythroid-2-related factor 2 (Nrf2) regulates the expression of genes encoding for GCL and other cysteine-metabolizing enzymes. The induction of glutamate-cysteine ligase (GCL) protects cells from oxidative damage by increasing GSH [33]. Oxidative injury and lipid peroxidation can be monitored by a measure of MDA level. MDA can be enzymatically metabolized by some enzymes, such as cyclooxygenases, or react with DNA. It is considered a good marker of oxidative stress, being able to cause damage and form adducts which can become mutagenic, and with irreparable alterations [34]. In this present study, there was an increase in MDA level in the cadmium treated rats (group B) compared to the control. This study confirmed that MDA (resulting from the oxidative breakdown of polyunsaturated fatty acids) was reduced significantly after *T.occidentalis* administration. *T.occidentalis* has been shown to reduce MDA in plasma, red blood cells, and this *T.occidentalis* also decreases oxidation reaction and increases antioxidant enzymes [35, 36]. Protein carbonyl content is actually the most general indicator and by far the most commonly used marker of protein oxidation [13-14], and accumulation of protein carbonyls has been observed in several human diseases including Alzheimer's disease (AD), diabetes mellitus, inflammatory bowel disease (IBD), and arthritis [37, 38]. The reduction in protein carbonyl level in groups administered *T.occidentalis* indicates its possible antioxidant property. CK-MM is

the most sensitive serum marker for muscle damage and can be elevated in any condition that causes muscle injury [10, 11]. It is routinely used in the evaluation of patients with suspected myopathy (e.g., patients presenting with unexplained muscle weakness or myalgia), and the investigations of myositides and suspected rhabdomyolysis [39]. The significant increase in CK-MM levels when compared to control in the group administered $CdCl_2$ 0.3ml may be due to the increased muscle necrosis or myofibril degeneration [40]. *T.occidentalis* maintained the functional integrity of the independent muscle fibers, this was clearly seen in the significant decrease in CK-MM levels group C, D and E. Other studies have proven the therapeutic effects of *T.occidentalis* against muscle myopathy [41, 42]. Acetylcholinesterase (AChE) is an enzyme that degrades (through its hydrolytic activity) the neurotransmitter acetylcholine, producing choline and an acetate group which is mainly found at neuromuscular junctions and cholinergic nervous system, where its activity serves to terminate synaptic transmission [43]. AChE is known to be inactivated by high Cadmium concentrations and show dose dependent inhibition [44]. This explains the significant decrease in the $CdCl_2$ 0.3ml group. AChE levels in group C, D and E were significantly increased compared to control probably due presence on pyridine, a type of alkaloid that acts directly on the nervous system. [45] Calcium initiates and regulates responses of CNS to nerve injury [40, 41]. calcium plays a key role in many physiological processes, including contraction of skeletal, cardiac, and smooth muscles, blood clotting, and transmission of nerve impulses [40-43]. The was an increase in the level of calcium in the group administered $CdCl_2$. This may likely be due to the osteoporotic effect of the heavy metal [40]. Cadmium may replace calcium in the bones because it competes with calcium absorption, thus increasing the plasma level of calcium [46, 47]. *T. occidentalis* groups showed a significant decline in Ca2+ levels likely as a result of its ameliorative effect on osteoporosis except for the group with the highest dose of *T. occidentalis*. It has been shown that *T. occidentalis* has effect on interleukins increase which can protect bone density. Interleukins are naturally occurring proteins that mediate communication between cells and regulate cell growth and differentiation. It is suggested that *T. occidentalis* has a preventive role in bone mass reduction [48]. There was no significant difference in the sodium ion level of all groups when compared with control. Cadmium dependent neurotoxicity has also been linked to neurodegenerative diseases such as Alzheimer's (AD) and Parkinson's diseases (PD) [49], as well as amyotrophic lateral sclerosis and multiple sclerosis, and myalgia [50]. Cadmium exposure has been shown to cause motor activity impairment and behavioural alterations both in adults and in children [48-50]. Step length, stride length and Base of support in all the groups treated with doses of *T. occidentalis* extract significantly reduced when compared to control. *T. occidentalis* and Cadmium chloride co-treatments caused a positive change in gait and kinematic response because at these doses the adverse effect of cadmium chloride on the parameters was prevented, suppressed or opposed.

CONCLUSION

From the findings of this study, the administration of aqueous extract *Telfaira occidentalis* to different treatment groups enhanced the brain antioxidant levels and decreased lipid peroxidation, an effect that was adversely altered by cadmium chloride. *Telfaira occidentalis* also improved the gait of animals treated with $CdCl_2$. Thus, *Telfaira occidentalis* may be an effective natural agent in alternative or complementary medicine for combating neurotoxicity.

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