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Attenuation of Oxidative Enzymes Induction in Palm Oil Fractions Pre-treated Cadmium Intoxicated Rats

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ABSTRACT

Impairing the toxic effects of cadmium (Cd) by diet induced antioxidant defence systems is an innovative approach to managing cadmium poisoning. The present study investigated the ability of crude palm oil (*Elaeis guinensis*) and its fractions to prevent the induction of aldehyde and sulphite oxidative enzymes in acute cadmium intoxicated male rats. The study comprised of six groups, which had group A as control, B as cadium group and C-F as test groups. Group A received No Cadmium and no Palm oil fraction, Group B received 20mgCd/Kg body weight, Groups C-F received 5ml/Kg body weight of appropriate palm oil fraction namely crude palm oil (CPO), Silica Gel Extract (SGE), Bleached Palm oil and unsaponifiable extract for 28 days prior to a single dose of cadmium in form of cadmium chloride on day 29 (20mgCd/Kg body weight). Rats were sacrificed 12h, 24h and 48h post-cadmium administration and the activities of aldehyde oxidase (AO) and sulphite oxidase (SO) were determined. Results obtained indicate a significant rise in AO and SO activities in the liver, kidney, heart, muscle tissues and serum between 12-48 hours following Cd administration compared to control. Administration of palm oil extracts caused significant reduction ($p<0.05$) in AO and SO activities. Results indicate that acute Cd administration induces the expression of oxidative enzyme within 12 hours but pre-treatment of rats with palm oil and its fractions reduces their expression.

Keywords: Attenuate, Induce, Cadmium, Palm oil and Oxidative Enzymes.

Introduction

The use of cadmium for electroplating in most industries makes it one of the elements that come in regular contact with man. In metallurgy, it is used for brazing and soldering of alloys. During oil exploration, it is often disposed primarily along other wastes; its other sources are industrial and agricultural effluents, sewages and sludge.[1,2] When introduced into the environment, it is often absorbed by plants and redistributed through the food chain.[3] Several target organs of cadmium has been identified but it is said to be mainly bound to the metallothionein protein after initial metabolism in the liver and is eventually redistributed through the blood stream to the kidney, testes, lungs, heart, testes, the skeletal architecture and the nervous system were its toxic effects are mostly felt.[4] In the course of these distributions, the cadmium compound induces the generation of several oxidative radicals which in turn shuts or slows down the proper functioning of the entire metabolic system.[5, 6]

Red palm oil is a rich source of vitamins, antioxidants and antitoxin agent.[7,8] The tocotrienol of palm oil is over 60 times higher in antioxidant capacity than ordinary vitamin E.[8] As potent antidote, it is utilized by the south-south people of Nigeria for resuscitating children who accidentally drink kerosene or eat soap.[9] This helps to neutralize the negative effects of the kerosene and soap or in other cases, regurgitate it. Palm oil has been identified to have protective potency against cardiac ischemia and perfusion induced oxidative stress and modulation of serum lipid profile in rat models.[10-12] The antioxidants in palm oil including carotenoids and tocols in silica gel extract of palm oil have the capacity of conferring protection against free radicals arising from ageing, atherosclerosis, cancer, arthritis, and Alzheimer's disease.[13-16] Also supporting the antioxidative protective properties of palm oil, Sutapa and Analava[17] reported that palm oil consumption by humans reverses blockage of the carotid artery and platelet aggregation and so contributes to the prevention of stroke and ischemic heart diseases. Sutapa and Analava, [17] also reported that the tocotrienol fraction of palm oil is able to protect the human brain from oxidative stress and most forms of neuro-degeneration associated with aging. The mechanism by which these palm oil constituents conferred these protections may be due to their ability to scavenge free radicals and being able to induce increased production of neurotransmitters like dopamine (Ref). The study of Achuba and Ogwumu[18] also revealed that palm oil can confer protection against haematotoxicity in rats that were fed diesel contaminated diets.

With these pieces of evidence, and the knowledge of the high level of antioxidants in crude palm oil and palm oil extracts it has been adjudged as an effective natural product for the protection against cadmium poisoning hence this study investigated the ability of palm oil and its extracts to confer protection against the induction of aldehyde and sulphite oxidase enzymes in acute cadmium intoxication.

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Materials and Methods

Chemicals and Reagent
The cadmium salt (CdCl) used as a source of cadmium was supplied by Sigma Aldrich Co. All other chemicals used which were of analytical grade were supplied by (Sigma Aldrich Co, May & Baker, Dagenham, England, and British Drug House Chemicals, Poole, England).

Palm Fruits Collection
Palm fruit purchase was from a local oil palm plantation in Obiaruku, Delta State, Nigeria. *Elaeis guinensis* Tenera was used for this research. Identification was carried out at the Department of Botany and registered with the voucher number ID/2017/16807/*Tenerra* Spp.

Extraction of Palm Oil
The ripen palm fruits of relative weight 10 kg were boiled for four hours. Having been cooked, extraction from the pulp was done by pounding in a wooden mortar and transferred into 10 L bowl of water with concurrent stirring, thoroughly. Removal of the oil palm fruit fibers was achieved through the use of a handmade basket as sieve and the collected filtrate transferred into a cooking pot of about 20 liter capacity and boiled under a controlled temperature of 150^0C for five hours. At the end of the boiling period, the heavily heated mixture was allowed to stand for 30 minutes and 2L of cold water sprinkled at the surface using a sprinkler. After cooling, the palm oil already set on top was collected into a fresh containers and heated for ten minutes to remove any trace of water.

Fractionation of Crude Palm Oil
The details of the methods for the fractionation of the three palm oil fractions (unsaponifiable extract, silica gel extract and bleached extract) used in this study have been previously described by Osei, [18] Twumasi *et al*[19] and Ichipi-Ifukor *et al*.[20] These methods are Unsaponifiable Extract (UPE), of which its preparation was done based on the methods of Meloan as modified by Twumasi, *et al*[19]. Silica Gel Extracts (SGE) based on the method of Ahmad *et al*.[21] and Bleached Extract carried out based on the method described by Patterson.[22]

Ethical consideration
Study approval was obtained from the Faculty of Science ethical committee Delta State University Abraka with approval number ETH/15/16/PG223056 and all procedures involving animals conformed to the guidelines for animal researches stated by Animal Research Ethics in 2009.

Experimental Design
The protective ability of crude palm oil (CPO) and the various palm oil fractions pre-treatment against AO and SO activities induction were investigated by carrying out assay for these enzymes at various time intervals following acute cadmium exposure. The dose of the palm oil and palm oil extracts used were based on an established lethal dose of undiluted palm oil greater than 5 g/kg body weight[23] and available data in literature on the time dependent toxicological effects of cadmium intoxication in rats.[24]
A total of seventy-two male rats with an average weight of 180-200g were obtained from the animal house of Emma Maria Research Laboratory and Consultancy Abraka Nigeria. These rats were allowed to acclimatize for two weeks and eventually distributed randomly into six groups of twelve rats each. The rats in group A served as the control and were neither treated with palm oil nor administered cadmium. The rats in group B were not treated with palm oil but were exposed to a single dose of 20 mg/kg body weight of cadmium chloride on the 29th day of the experiment. Rats in Groups C-F were treated with 5ml/Kg body weight of crude palm oil and the various fractions of palm oil for a period of 28 days as follows. (Group C= Crude palm oil (CPO); Group D = Silica Gel Extract (SGE); Group E= Unsaponifiable Extract (UPE); Group F = Bleached Extract (BE). On the 29th day of the experiment, these rats were exposed to a single dose of 20mgKg^{-1} body weight of cadmium chloride orally by gavage and four animals from each group were sacrificed at intervals of 12hrs, 24hrs and 48 hrs.

Sample Collection and Preparation
Animals were sacrificed at the time intervals indicated, blood samples were collected using hypodermic syringe and needle by cardiac puncture. The tissues (liver, kidney, heart, brain and muscle) were excised, weighed and transferred immediately into labelled containers while the blood samples were transferred into labelled plain tubes. The serum was then collected by centrifugation of the clotted blood at 3000xg and stored in the refrigerator at 4^0C. The tissues already collected were homogenized using pre-chilled mortar and pestle in cold normal saline solution. The homogenates were also centrifuged at 5000xg for 10 minutes and the supernatants collected and stored in the refrigerator at 4^0C.

Assay for Aldehyde and Sulphite Oxidases
Assay for the activities of sulphite oxidase was done by the method of Macleod, et al.,[25] while aldehyde oxidase was assayed by the method of Johns (1967) modified by Omarov.[26] These were based on the ability of SO to catalyse the oxidation of sulphite to sulphate and that if AO to catalyse the conversion of benzaldehyde to benzoate using 2,6–dichlorophenol (DCIP) as the electron acceptor respectively.

Statistical Analysis
Data was analysed using the computer software statistical package for social science version 21 (SPSS 21). The simple analysis of variance (ANOVA) was used while multiple comparisons across groups were done using Tukey HSD analysis and significance level set at $p<0.05$.

Results and Discussion

Oxidative enzymes (AO and SO) have been identified as cytosolic enzymes which are absolutely necessary in the breakdown of a number of aldehydes, nitrogenous heterocyclic compounds and endogenous sulphites. They are rich in FAD, molybdenum and iron-sulfur centres and participates in the reduction of sulfoxides, N-oxides, and aromatic nitro compounds and 1,2-benzisoxazole derivatives.[26] The result presented in Table 1 shows the activity of Sulphite Oxidase (SO) in tissue and serum after 12 h, 24 h and 48 h of acute cadmium administration in rats pre-treated with palm oil and palm oil extracts. The activity of SO in the organs (liver, kidney, and heart) of cadmium treated rats (group B) showed a significant ($P \leq 0.05$) increase after 12 h, 24 h and 48 h periods of exposure compared to control (group A). Also, SO activities in these organs of Cd exposed rats pre-treated with varying palm oil fractions (C-F) indicated significant increase relative to control but was reduced compared to the cadmium only group (group B). Likewise, Exposure to cadmium (group B) significantly increased AO activity in the serum and tissues of rats at the end of the duration of exposure except in brain, muscle and serum after 12 h relative to control (Table 2). Administration of palm oil and palm oil extracts (groups C-F) decreased the activity of AO in serum and tissue relative to rats treated with Cd only (group B). Also, administration of palm oil and palm oil extracts (groups C-F) decreased the activity of AO in serum and tissue relative to rats treated with Cd only (group B). No significant change was observed in the brain, muscle and serum AO activity across all experimental groups irrespective of the duration except in group B after 24 h and 48 h time interval.
Based on the earlier stated roles of AO and SO in xenobiotic metabolism, there is no doubt that the observed increase of tissue and serum AO and SO after cadmium administration may be traceable to the activation of synthesis of these enzymes in order to clear or minimize the tissue levels of aldehydes, sulfoxides, N-oxides and aromatic oxides. Cadmium induced rise in brain AO and SO activities of *Clarias gariepienus* has been previously reported by Ichipi-Ifukor *et al*.[27] Likewise, Asagba[28] revealed that following Cd intoxication, there is increased depletion of tissue proteins and generation of endogenous heterocyclic compounds which over time increases the inhibition of the AO and SO activities.
Relative to the palm oil extracts and crude palm oil, it does appear that oxidative enzyme induction occurs as a function of the extent to which the palm oil extracts were able to limit the occurrence of lipid peroxidation occasioned by the free rise of oxidative radicals.[29]

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Table 1: Effect of Palm Oil and Palm Oil Extracts on Activities of Tissue and Serum Sulphite Oxidase (SO) of Rats Administered Cadmium. (Unitsg^{-1} tissue) or (Unitsml^{-1}) in serum.

Tissues	Experimental Groups					
			12h			
	A	B	C	D	E	F
LIVER	1.57 ± 0.61^{a}	5.40 ± 0.77^{b}	2.02 ± 0.23^{ac}	3.45 ± 0.77^{d}	2.32 ± 0.37^{ad}	1.67 ± 0.53^{a}
%Change		(244)	(29)	(120)	(48)	(6)
KIDNEY	0.95 ± 0.12^{a}	4.82 ± 1.04^{b}	2.82 ± 0.66^{c}	1.13 ± 0.37^{a}	1.69 ± 0.15^{ac}	1.90 ± 0.25^{ac}
%Change		(407)	(197)	(19)	(78)	(100)
HEART	1.35 ± 0.66^{a}	5.51 ± 0.92^{b}	2.00 ± 0.73^{a}	1.36 ± 0.55^{a}	1.81 ± 0.84^{a}	1.23 ± 0.34^{a}
%Change		(308)	(48)	(7)	(34)	(-9)
BRAIN	1.14 ± 0.33^{a}	1.69 ± 0.21^{a}	1.58 ± 0.32^{a}	1.56 ± 0.19^{a}	1.55 ± 0.32^{a}	1.56 ± 0.46^{a}
%Change		(48)	(39)	(37)	(36)	(42)
MUSCLE	0.79 ± 0.15^{a}	1.74 ± 0.24^{b}	1.24 ± 0.38^{a}	1.59 ± 0.14^{b}	1.60 ± 0.19^{b}	1.04 ± 0.08^{a}
%Change		(120)	(57)	(101)	(103)	(32)
SERUM	1.15 ± 0.39^{a}	2.66 ± 0.26^{b}	1.22 ± 0.29^{a}	1.28 ± 0.21^{a}	1.99 ± 0.23^{c}	1.44 ± 0.17^{a}
%Change		(131)	(6)	(11)	(42)	(25)
24h						
LIVER	1.60 ± 0.59^{a}	6.18 ± 1.14^{b}	2.47 ± 0.25^{a}	4.61 ± 0.67^{c}	2.83 ± 0.38^{a}	5.07 ± 0.92^{bc}
%Change		(286)	(54)	(118)	(77)	(217)
KIDNEY	1.85 ± 0.47^{a}	5.21 ± 1.21^{b}	2.59 ± 0.33^{ac}	3.63 ± 0.39^{c}	2.43 ± 0.42^{ac}	3.18 ± 0.91^{c}
%Change		(182)	(40)	(96)	(31)	(72)
HEART	1.39 ± 0.58^{a}	5.73 ± 0.75^{b}	2.58 ± 0.65^{a}	1.76 ± 0.46^{a}	2.31 ± 0.43^{a}	1.41 ± 0.31^{a}
%Change		(312)	(86)	(27)	(66)	(1)
BRAIN	1.53 ± 0.32^{a}	1.93 ± 0.22^{a}	1.70 ± 0.28^{a}	1.64 ± 0.23^{a}	1.63 ± 0.27^{a}	1.95 ± 0.72^{a}
%Change		(21)	(11)	(7)	(7)	(27)
MUSCLE	0.68 ± 0.16^{a}	2.22 ± 0.31^{b}	1.43 ± 0.16^{c}	1.62 ± 0.11^{bc}	1.87 ± 0.60^{bc}	1.14 ± 0.07^{ac}
%Change		(226)	(110)	(138)	(175)	(68)
SERUM	1.22 ± 0.35^{a}	3.64 ± 0.80^{b}	1.55 ± 0.06^{a}	1.88 ± 0.66^{b}	2.99 ± 0.32^{b}	2.65 ± 0.57^{b}
%Change		(198)	(27)	(54)	(145)	(117)
			48h			
LIVER	1.57 ± 0.35^{a}	6.65 ± 0.60^{b}	2.70 ± 0.12^{c}	2.92 ± 0.41^{c}	5.41 ± 0.70^{d}	4.70 ± 0.58^{d}
%Change		(324)	(72)	(86)	(244)	(199)
KIDNEY	1.94 ± 0.65^{a}	5.75 ± 0.89^{b}	2.72 ± 0.27^{ac}	2.64 ± 0.19^{a}	3.80 ± 0.16^{c}	3.68 ± 0.48^{ac}
%Change		(196)	(40)	(36)	(96)	(90)
HEART	1.53 ± 0.43^{a}	6.08 ± 0.57^{b}	2.73 ± 0.52^{c}	1.91 ± 0.44^{ad}	2.49 ± 0.21^{cd}	1.63 ± 0.17^{ad}
%Change		(297)	(78)	(25)	(63)	(7)
BRAIN	1.50 ± 0.19^{a}	2.23 ± 0.49^{a}	1.88 ± 0.20^{a}	1.76 ± 0.29^{a}	1.80 ± 0.16^{a}	2.12 ± 0.72^{a}
%Change		(47)	(25)	(17)	(20)	(41)
MUSCLE	0.68 ± 0.29^{a}	2.61 ± 0.29^{b}	1.62 ± 0.19^{c}	1.83 ± 0.13^{c}	1.91 ± 0.44^{c}	1.36 ± 0.32^{c}
%Change		(417)	(138)	(169)	(181)	(100)
SERUM	1.43 ± 0.17^{a}	4.11 ± 1.18^{b}	1.72 ± 0.23^{ac}	2.12 ± 0.37^{ac}	2.88 ± 0.41^{bc}	2.58 ± 1.07^{bc}
%Change		(187)	(20)	(48)	(101)	(80)

All values are expressed as Mean ± SD, values sharing different letters of the alphabet as superscript on the same row are significantly different. Values expressed in bracket indicate percentage change relative to control.
Key: A= Control; B= Administered 20mg/Kg Body Weight of $CdCl_2$; C= Crude Palm Oil (CPO) + 20 mg/kg $CdCl_2$; D = Silica Gel Extract (SGE) + 20 mg/kg $CdCl_2$; E= Bleached Extract (BE) + 20 mg/kg $CdCl_2$; F= Unsaponifiable Extract (UPE) + 20 mg/kg $CdCl_2$

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Table 2: Effect of Palm Oil and Palm Oil Extracts on Activities of Tissue and Serum Aldehyde Oxidase (AO) of Rats Administered Cadmium. (Unitsg^{-1}tissue) or (Unitsml^{-1}) in serum.

Tissues	Experimental Groups					
	A*	B	C	D	E	F
LIVER	3.04 ± 0.94^{a}	14.43 ± 1.35^{b}	7.11 ± 2.38^{c}	8.07 ± 1.93^{c}	5.15 ± 1.06^{ac}	3.44 ± 1.11^{a}
%Change		(375)	(134)	(165)	(69)	(13)
KIDNEY	2.27 ± 0.65^{a}	9.80 ± 1.10^{b}	6.94 ± 2.17^{bc}	4.53 ± 0.94^{ac}	6.45 ± 0.69^{bc}	4.73 ± 2.20^{ac}
%Change		(332)	(206)	(100)	(184)	(108)
HEART	2.12 ± 0.68^{a}	8.13 ± 1.62^{b}	5.09 ± 1.46^{c}	2.71 ± 0.87^{a}	4.92 ± 1.10^{c}	1.87 ± 0.57^{a}
%Change		(283)	(140)	(28)	(132)	(-12)
BRAIN	2.66 ± 0.21^{a}	2.61 ± 0.42^{a}	2.41 ± 0.35^{a}	2.13 ± 0.60^{a}	2.24 ± 0.36^{a}	2.28 ± 0.09^{a}
%Change		(-2)	(-9)	(-20)	(-16)	(-14)
MUSCLE	2.95 ± 0.73^{a}	3.32 ± 0.65^{a}	2.91 ± 1.07^{a}	2.92 ± 0.99^{a}	3.19 ± 0.67^{a}	3.09 ± 0.95^{a}
%Change		(12)	(1)	(-1)	(8)	(5)
SERUM	4.89 ± 1.09^{a}	6.28 ± 1.39^{a}	5.12 ± 1.63^{a}	6.27 ± 1.99^{a}	6.10 ± 1.95^{a}	5.49 ± 2.22^{a}
%Change		(28)	(5)	(47)	(25)	(12)
24h						
LIVER	3.57 ± 0.95^{a}	14.69 ± 1.78^{b}	6.23 ± 1.44^{c}	4.04 ± 2.32^{ac}	9.74 ± 2.02^{d}	4.43 ± 1.46^{a}
%Change		(311)	(75)	(13)	(173)	(24)
KIDNEY	2.30 ± 0.74^{a}	11.15 ± 0.70^{b}	6.81 ± 1.81^{c}	5.30 ± 1.04^{c}	7.07 ± 0.85^{c}	4.66 ± 1.35^{c}
%Change		(383)	(196)	(130)2	(207)	(103)
HEART	2.22 ± 0.38^{a}	9.4 ± 1.12^{b}	5.32 ± 1.35^{c}	3.13 ± 0.80^{a}	5.36 ± 0.97^{b}	2.43 ± 0.59^{a}
%Change		(323)	(140)	(41)	(141)	(9)
BRAIN	4.39 ± 1.04^{a}	7.10 ± 1.28^{b}	4.26 ± 0.84^{a}	4.80 ± 0.88^{a}	4.49 ± 1.15^{a}	4.96 ± 1.72^{a}
%Change		(62)	(-3)	(9)	(2)	(13)
MUSCLE	2.36 ± 0.53^{a}	3.92 ± 0.47^{a}	2.86 ± 0.81^{a}	3.38 ± 0.97^{a}	3.81 ± 1.05^{a}	3.38 ± 0.62^{a}
%Change		(66)	(21)	(43)	(61)	(43)
SERUM	4.35 ± 0.57^{a}	8.18 ± 1.58^{b}	5.48 ± 1.26^{a}	6.57 ± 1.57^{a}	6.33 ± 1.19^{a}	6.83 ± 1.19^{a}
%Change		(88)	(26)	(51)	(46)	(57)
48h						
LIVER	3.67 ± 0.93^{a}	16.2 ± 1.58^{b}	7.45 ± 1.37^{ac}	6.05 ± 2.80^{c}	10.50 ± 2.12^{c}	5.20 ± 1.56^{a}
%Change		(341)	(103)	(65)	(186)	(42)
KIDNEY	2.45 ± 0.66^{a}	12.22 ± 1.00^{b}	7.05 ± 1.72^{c}	5.30 ± 0.87^{c}	7.45 ± 0.91^{c}	5.36 ± 1.41^{c}
%Change		(399)	(188)	(204)	(204)	(119)
HEART	2.58 ± 0.66^{a}	9.85 ± 1.20^{b}	5.80 ± 1.10^{c}	3.38 ± 0.59^{a}	5.71 ± 0.63^{c}	2.90 ± 0.68^{a}
%Change		(282)	(125)	(31)	(121)	(12)
BRAIN	4.30 ± 0.93^{a}	7.57 ± 1.31^{b}	4.78 ± 0.53^{a}	4.93 ± 0.89^{a}	5.05 ± 0.91^{a}	4.85 ± 1.15^{a}
%Change		(76)	(11)	(15)	(17)	(13)
MUSCLE	2.60 ± 0.28^{a}	4.71 ± 0.62^{b}	3.11 ± 0.68^{a}	3.48 ± 0.90^{a}	3.11 ± 0.68^{a}	3.60 ± 0.66^{a}
%Change		(81)	(20)	(34)	(20)	(38)
SERUM	4.35 ± 0.62^{a}	9.03 ± 2.10^{b}	6.02 ± 2.10^{a}	6.65 ± 1.33^{ab}	6.53 ± 1.04^{ab}	7.10 ± 0.90^{ab}
%Change		(108)	(38)	(53)	(50)	(38)

All values are expressed as Mean ± SD, values sharing different letters of the alphabet as superscript on the same row are significantly different. Values expressed in bracket indicate percentage change relative to control.
A* See Table 1 footnote for interpretation of abbreviations

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Of all the extracts used in the pre-treatment of the animals before cadmium administration, the bleached extract (BE group E) had the least level of efficacy in protecting the rats from alteration in the stability of AO and SO activities relative to control while CPO and SGE contributed more to the sequestration of their induction. The possible justification for the above claims based on the results indicates that the pre-treatment with palm oil and palm oil fractions limited the effect of Cd on serum and tissue SO activity, but comparatively no consistent pattern was discernible however, the examination of the percentage changes indicate that in most cases the AO and SO activity in Cd administered rats pre-treated with CPO and SGE had values almost comparable to control. Hence, the study reveals that Cd increases AO activity, while palm oil and palm oil extracts confers protection of rats from these negative effects.

Another credence to the claims made above may not be far from the reduced level of antioxidants of the bleached extracts used in the study previously reported by Ichipi-Ifukor *et al*.[20] Evidence in literature indicate the major constituent of bleached palm oil extract were free fatty acids. This claim is also supported by the study of Idoko *et al*.,[30] who found that bleaching of palm oil contributes greatly to the destruction of its primary antioxidants. This submission is further given credence to by Ani *et al*.,[31,32] that the consumption of thermally oxidized palm oil contributed greatly in the reduction of haematological parameters, increase in serum creatinine and urea as well as increased anaemia.

A careful examination of the results reveals that in terms of organs, AO and SO activities were more in the liver followed by the kidney, heart and serum with the least occurring in the brain and muscle tissues Because as revealed, activities of brain SO was not significantly ($P \geq 0.05$) altered after all periods of exposure across all experimental groups. Muscle and Serum SO activities after 12 h showed a statistically significant ($P \leq 0.05$) increase in Cd (group B) compared to control (group A) while rats pre-administered SGE (group D) and BE (group F) showed no significant ($P \geq 0.05$) changes in muscle SO relative to Cd only group (B) but the activity of the enzyme was significantly increased compared to control (group A) (Table 1). SO activity of Cd exposed rats administered palm oil and palm oil extracts (groups C-F) remained statistically ($P \geq 0.05$) unchanged except for those administered BE relative to control (group A) but were significantly reduced relative to Cd (group B). In the same vein, no significant change was observed in the brain, muscle and serum AO activity across all experimental groups irrespective of the duration except in (group B after 24 h and 48 h) time interval.

This pattern of distribution has been previously reported by Klassen *et al*.[33] Asagba[28] also reported that cadmium induced rise in AO and SO activities were usually elevated in the liver and attributed it to the high induction of Methalothionein (MT) within the liver architecture compared to other organs while the serum is not a cell hence no gene exists that may induce the expression of AO and SO making their activities in the serum mostly rely on endogenous leakages from other tissues. In addition, cadmium induced activation of these enzymes in the liver are said to contribute enormously to cadmium transport and retention within the liver and other tissues because the MT-Cd complex functions as an adjuvant/transporter during Cd toxicity.[5,34-36] Having known the liver as a major site of xenobiotic metabolism and the kidney as a major site for filtration of toxicants and re-absorption of essential fluid mineral elements it gives further credence to the high activity of these enzymes in the liver and kidney relative to other tissues.[2,5-6] Compared to the brain, the selective permeability due to the blood-brain barrier may be the possible justification for the limited AO and SO enzyme activities occasioned by reduction of cadmium concentrations entering into the brain cells to induce their noxious effects which may necessitate the enzyme activities.[27,37,38] The muscle on the other hand being a major absorptive tissue may also not have been affected much because Cd tissue absorption takes a relative longer time before its deleterious effects could be felt.[20]

Conclusion

Based on the findings presented in this study, it is reasonable to conclude that cadmium intoxication in Wistar rats has inductive capabilities of the oxidative enzymes within 12 h, while the reduction in these activities tends to set in after 24-48 h. However, the pre-treatment of rats with palm oil and fractions of palm oil contributed significantly to the reduction of the observed oxidative enzyme activities.

Conflict of interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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