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Assessment of Different Regimen of Oil Palm Leaf Extracts Against Crude Oil-Adulterated Feed Mediated Nephrotoxicity

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ABSTRACT

The health implications of crude oil and the protective effect of unprocessed oil palm leaves have been documented. This investigation was aimed at the assessment of different regimen of oil palm leaf extracts against crude oil-adulterated feed mediated nephrotoxicity. The study comprised the use of different solvent extracts (aqueous, ethanol, methanol, acetone, petroleum ether) and blended mixtures of oil palm leaf to assess the efficacy against crude oil imposed nephrotoxicity. The regimen adopted were pre-treated, co-treated and post-treated with the plant extracts before, during and after exposure to crude oil-adulterated feed for 28 days of experimentation. All groups comprised six rats each. The result implicated crude oil polluted feed in the alteration of kidney function markers compared to positive control. It also increased levels of kidney lipid peroxidation and induced reduction in antioxidants: superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) relative to control. Treatment with oil palm leave extracts reversed these trends. The pre-treatment of animals with the various extracts was more efficient in handling the metabolic challenges compared to the co-treatment and post-treatment. However, different solvent extracts of oil palm leaves and blended solvent extracts of the leave had efficacy on the altered kidney dysfunction parameters. Moreover, pretreatment of the animals with extracts, especially, the blended mixture exhibited more protective measure against crude oil-induced nephrotoxicity.

Keywords: Antioxidants, Crude oil, Nephrotoxicity, Oil palm leaves.

Introduction

The kidney is the bodies' major site of post xenobiotic filter that mediates excretion of several fluid wastes. It also acts as a center for the re-absorption of several elements such as food nutrients in form of protein and minerals.[1] An overworked kidney is bound to deteriorate in function over time thus the continual exposure of man and animals to toxic substances contributes a great deal to the kidneys overwork burden. One of such toxic substances that contributes to the kidneys overwork burden include several forms of petroleum products either directly through inhalation or exposure via the food chain through the consumption of various food sources and water from petroleum contaminated water environments.[2]

Today, the Niger-Delta region of Nigeria have lost most of its agricultural lands and water bodies due to contamination through oil exploration activities or stands a potential risk of being polluted.[3-6] It is on record that this is a public health challenge as many inhabitants have reportedly died of several forms of diseases especially diseases arising from mild to chronic kidney failures in inhabitants of the region.[7,8,9] With the reality of these disease occurrences and burden increasing daily, there is no doubt that man must find a way to significantly reduce the rising trend. Previous studies have shown that adequate nutrition and consumption of plants rich in phytochemicals could mediate the negative burden of petroleum mediated toxicity.[10] The oil palm plant whose leaf produces a large biomass of waste after its fruit and oil has been harvested and processed remains a potential adjuvant for this course. This is so because studies revealed that it is rich in phytochemicals and has several medicinal values.[11,12] However, recent studies by Achuba[13] revealed that incorporation of dried oil palm leave powder in crude oil-polluted meals significantly prevented the negative outcomes accompanying crude oil toxicity. This investigation was aimed at assessment of different regimen of oil palm leaf extracts against crude oil-adulterated feed mediated nephrotoxicity.

Materials and Methods

Collection of palm leaves, crude oil and reagents

Harvest of *Elaeis guineensis* leaves were done at Ovwor Mixed Secondary School in Ughelli, Delta State, Nigeria on May 11, 2016. This was immediately rinsed with water to remove all forms of dirt and taken to the laboratory. The leaves were identified at Forestry Research Institute of Nigeria, Ibadan where voucher number, F101173 was assigned. The crude oil, Escravos blend type, was obtained from Warri Refining and Petrochemical Company, Warri, Nigeria. The reagents used were analytical grade.

Experimental animals

Male albino rats (One hundred and twenty) were sourced from Faculty of Basic Medical sciences, Delta State University, Nigeria. The weights of the rats were taken and it ranged from 148 to 180 g. The experimental rats were maintained on grower's mash and exposed to laboratory environment for two weeks. This is to enable them acclimatize to the feed and the new environment. Ethical approval, with the number REC/FOS/19/01, for the use the animals was granted by the ethical committee, Faculty of Science, Delta State University Abraka, Nigeria.

Preparation of oil palm leaf extracts

Plant extraction was carried out using five solvents namely; water, ethanol, methanol, acetone and petroleum ether. Before extraction, the

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collected palm leaves were dried to constant weight and ground into smooth particles with the aid of an electric waring blender. A known amount of the leaf, 100 g was then macerated into 400 ml of the different solvents and allowed to stand for 48 hours in an air tight conical flask placed on magnetic stirrer. The aqueous extract at a temperature of 40^{o}C as described previously.[14] The other extracts, ethanol, methanol, acetone and petroleum ether extracts were prepared as previously described.[15,16] The homogenates were subjected to 48 hours standing period, and the various samples were filtered using a clean muslin cloth and the filtrate transferred to a rotary evaporator at 45^{0}C for concentration. The filtrates were further concentrated to dryness using a water bath at 50^{o}C and stored in a refrigerator until needed.

Preparation of extracts for treatment

Exactly 2.5 g of each extract was dissolved in 25 mL of aqueous tween 80 (1% in 99 mL of distilled H_2O (v/v) to make a concentration of each extract to 0.1g/ml. The blended mixture was constituted by adding 0.5 g of each extract to make 2.5g. This was dissolved in 25 mL of aqueous tween 80 to create 100 mg/mL of the mixture.

Experimental design

Three categories of experiment were designed. They are pre-treatment (Pre-T), co-treatment (Co-T) and post- treatment (Post -T):

Pre-treatment (Pre-T): The rats were given 250 mg/kg body weight of the respective oil palm leaf extract via oral gavage for four weeks before exposure to crude oil contaminated feed (COCF) for another four weeks.

Co-treatment (Co-T): Rats were simultaneously exposed to COCF and treated with the respective 250 mg/kg body weight oil palm leaves extracts for four weeks.

Post- treatment (Post -T): The rats were fed COCF for 4 weeks and treated with 250 mg/kg body weight of oil palm leaves extracts for four weeks.

Treatment of animals

One hundred and twenty animals were used with six animals in groups 1 and 2 and 18 animals in groups 3-8 respectively. Each treatment regimen groups (3-8) was further sub-grouped into Pre-T, Co-T and Post-T consisting of six rats. While rats in the control group (Group I) served as control and were not exposed to any form of treatment except feed and water. The rats in group 2 were fed with COCF only as previously described.[16] However rats in group 3-7 were fed with crude oil contaminated food (COCF) and treated with 250 mg/kg body weight of aqueous, methanol, ethanol, acetone and petroleum ether, respectively of oil palm leaf extract. This was done according to the regimens of Pre-, Co- and Post- treatment sub-groups for four weeks. The rats in group 8 were treated with 250 mg/kg body weight of a blended mixture of (aqueous, methanol, ethanol, acetone and petroleum ether) extracts of palm oil leaves.

Sample collection and preparation

The rats were subjected to fasting overnight after treatment periods. This followed by scarification of the animals through cervical decapitation while under tranquility with chloroform. Sterile syringes were used for collection of blood into plain sample containers for biochemical analyses. The collected blood samples were centrifuged at 3500g for 15 minutes as reported previously.[16] The sera produced were collected for biochemical assays. Kidney wet tissue (0.5 g) was homogenized in 9.0 mL of normal saline with chilled mortar and pestle at 4^{o}C. The homogenates produced were equally centrifuged at 3500 g to yield supernatants that were stored at 4^{o}C in the refrigerator. The sera were used for various biochemical analyses using standard protocols within 48 hours

Determination of kidney function markers

Assay for serum and kidney electrolytes were carried out using standard Teco diagnostic kits based on standard methods highlighted. The concentrations of serum and kidney sodium levels were estimated by the methods of Henry[17] and Terri and Sesin,[18] respectively. The level of chloride was carried out directly using the method of Skeggs and Hochestrasser[19] with slight modification. Bicarbonate content was determined enzymatically by modification of the Forrester *et al*.[20] method. The concentration of calcium was estimated using the colorimetric method of Cali *et al.*[21]. Serum urea and creatinine nitrogen were both determined by method of Henry[17] based on a description provided in the Teco diagnostic kit manual.

Determination of peroxidation and kidney antioxidants levels

Lipid peroxidation, expressed as malondialdehyde (MDA) was determined as documented by Gutteridge and Wilkins,[22] while method of Ellman[23] was used to assay for reduced glutathione. The method of Misra and Fredorich[24] was used for super oxide dismutase (SOD) and Kaplan *et al*.[25] was used for Catalase, respectively.

Statistical Analysis

Data analysis was carried out using the statistical package of the social sciences (SPSS). The single factor analysis of variance (ANOVA) was used while group comparisons were done using the least significant difference (LSD) at 95% confidence interval ($p < 0.05$).

Results and Discussion

The effect of petroleum on kidney function parameters are well documented revealing a total deregulation and degeneration of functional indices such as increase in serum kidney function markers and decreased levels of antioxidant markers with concomitant rise in renal lipid peroxidation.[16] However, the protective, preventive and ameliorative efficiencies of various leave solvent extracts and blended mixtures of *E. guineensis* extracts is scarcely researched against food mediated crude oil toxicity in the kidney. Data from the present study reveal very interesting results based on three different models studied towards mitigation of adverse effects of consuming food tainted with petroleum. Figures 1 and 2 illustrate the effects of *E. guineensis* leaves extracts on some kidney function markers in rats fed crude oil adulterated feed. The level of these markers in the serum and kidney were significantly higher in control animals when compared to animals fed with petroleum treated feed. Generally, pre-treatment, co-treatment and post-treatment with *E. guineensis* leaves extract lowered the observed increase in kidney function markers reported in animals fed the adulterated feed. Comparatively, however, serum and kidney function markers were lower in the pretreatment compared to the co- and- post treatment regimens, respectively. The role of oil palm leaf extract in normalizing metabolic aberrations induced by crude oil adulterated fed was recently reported.[26] implying a possible alteration in the kidneys ability to selectively clear urea from the serum.[1, 16, 29]

In addition, the levels of electrolyte in the kidney (Table 2) showed that crude oil intoxication significantly ($p < 0.05$) decreases the level of Na^+, Ca^{2+}, Cl^- and CO_3^{2-} and increases K^+ level in the kidney of rats exposed to crude oil adulterated feed when compared with the control (rats fed normal feed). Increases were observed in Na^+, Ca^{2+}, Cl^- and CO_3^{2-} following pre-treatment, co-treatment and post-treatment with the extracts of *E. guineensis* relative to group 2 except for co-treatment group 3 (Cl^- and CO_3^{2-}) which was not statistically significant, and K^+ level which decreased significantly. The crude oil-adulterated feed-mediated decrease in serum and kidney electrolytes (Na^+, Ca^{2+}, K^+ and Cl^-) in this study is in line with earlier reports by Achuba[16] that reported altered serum electrolyte levels in rats consuming petroleum tainted food. Low serum and kidney electrolyte levels have been linked to possible malabsorption of nutrients and minerals from the diets, conditions leading to poor hydration and acute kidney diseases.[27] The implication of this is that crude oil contaminated diet consumption may have contributed to poor level of mineral reabsorption and retention of body fluids which are the major transporters for these electrolytes within the body for eventual removal when they are in excess. This argument is supported by earlier reports that have implicated petroleum toxicity in high level of appetite loss and unavailability of food nutrient components.[28] It also agrees with the report of Asagba,[1] that the presence of toxic

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substances such as cadmium and other substances alike mediates high level of appetite loss. A study by Ogbeke *et al*.[29] has earlier reported that serum and kidney bicarbonate are essential for the improvement and stabilization of the kidneys buffering system. Thus the observed crude petroleum mediated drop in serum and kidney bicarbonate in tables 1 and 2 may be indicative of the kidneys loss of its potential of maintaining the normal acid-base balance for normal physiological function of the kidney[29]. The ability of *E. guineensis* to upregulate the bicarbonate concentrations is indicative of the plant extract ability to improve renal function which was corroborated by Achuba[30] harping on the ability of the crude plant powder to improve renal oxidative stress parameters in rats consuming crude oil polluted meals. A careful observation of the three treatment regimens reveals that the pre-treatment regimen had better reversibility potentials of all altered renal indices in rats that consumed petroleum tainted diets. The possible reason for this may be due to the fact that earlier consumption or treatment of the rats with the plant extracts have contributed to boosting antioxidant defense through free radical scavenging properties of the active metabolites in the plant extracts. It also implies that in cases of an already established toxicity that the plant extracts consumption or treatment may significantly contribute to ameliorating the negative outcomes of renal malfunction. Results presented in Table 3 illustrate that consumption of crude oil adulterated feed reduced levels of malondialdehyde (MDA) and antioxidant enzyme (CAT and SOD) activities as well as kidney reduced glutathione (GSH). However, these reductions were normalized by the Pre-treatment, co-treatment and post-treatment with *E. guineensis* leaves extracts. The observed rise in lipid peroxidation and the concomitant reduction in antioxidant enzymes (SOD; CAT) and reduced glutathione is similar to increase in renal oxidative stress status and reduced levels of enzymatic and non-enzymatic antioxidants reported by Azeez *et al*.[31], Achuba [13] and Okpoghono *et al*.[32] These parameters are widely established markers of kidney functionality while the ability of the *E. guineensis* extracts to ameliorate the rising trend of these parameters at the pre-treatment, co-treatment and post-treatment regimens is indicative of the ability of the plant extract to confer protection on the kidney as well as prevent possible alteration of normal kidney physiology due to petroleum toxicity.[33,34]

Table 1: Alteration of serum electrolyte levels of rats fed COCF treated with *Elaeis guineensis* leaves extracts

Experimental group	Na^+ (mEq/L)	K^+ (mEq/L)	Ca^{2-} (mEq/L)	Cl^- (mEq/L)	CO_3^{2-} (nmol/L)
Group 1	176.17 ± 4.73 [a]	4.46 ± 0.35 [a]	150.32 ± 2.32[a]	134.35 ± 3.16 [a]	60.34 ± 3.78 [a]
Group 2	113.65 ± 10.18 [b]	18.09 ± 4.66[b]	80.57 ± 11.03[b]	64.50 ± 5.98 [b]	21.04 ± 7.65 [b]
Pre-treatment					
Group 3	142.52 ± 2.27 [c]	14.49 ± 5.00 [c]	110.00 ± 2.91[c]	92.45 ± 1.58 [c]	30.46 ± 7.90 [c]
Group 4	129.11 ± 12.26 [d]	8.46 ± 5.20 [d]	128.07 ± 2.915[d]	112.00 ± 1.58 [d]	54.39 ± 2.94 [a]
Group 5	153.14 ± 2.50 [e]	14.40 ± 8.82 [c]	115.25 ± 3.80[c]	102.00 ± 1.58[e]	64.18 ± 5.82 [a]
Group 6	160.12 ± 3.79 [e]	5.70 ± 7.51[a]	124.00 ± 1.58[d]	117.00 ± 1.58[d]	53.34 ± 2.25 [a]
Group 7	164.56 ± 3.72 [e]	8.39 ± 8.31[d]	130.00 ± 5.14[d]	130.00 ± 6.67[a]	51.36 ± 8.03 [a]
Group 8	168.52 ± 71.09[e,a]	7.59 ± 5.14 [d]	140.00 ± 3.80[e]	131.00 ± 4.00[a]	58.54 ± 4.57 [a]
Co-treatment					
Group 3	125.79 ± 9.45 [d]	17.48 ± 1.52[b]	90.49 ± 8.37[f]	74.74 ± 8.74[c]	22.44 ± 6.66[c,d]
Group 4	144.14 ± 15.91 [c]	11.53 ± 1.49[c]	109.24 ± 13.62[c]	98.40 ± 12.15[c]	35.55 ± 9.85[c]
Group 5	136.45 ± 5.67[c,d]	15.43 ± 3.27[c]	99.93 ± 13.23[c,f]	85.27 ± 1.28[d]	26.57 ± 9.06[c,d]
Group 6	142.29 ± 16.74 [c]	13.49 ± 2.48[c]	104.60 ± 15.23[e]	90.41 ± 11.18[e]	34.51 ± 7.17[c]
Group 7	147.59 ± 8.43 [c]	12.96 ± 9.34[cd]	108.69 ± 13.87[e]	103.65 ± 10.51[e]	37.57 ± 8.82[c]
Group 8	157.44 ± 15.54 [e]	9.38 ± 2.84[d]	121.59 ± 16.03[d]	111.33 ± 9.76[f]	38.54 ± 12.29[c]
Post-treatment					
Group 3	135.34 ± 8.23[c,d]	16.19 ± 5.94 [b]	100.17 ± 8.93[b]	84.60 ± 9.80 [f]	31.26 ± 6.44 [c]
Group 4	153.60 ± 6.92 [d]	10.28 ± 4.75[c]	119.51 ± 5.40 [c]	105.39 ± 7.73 [e]	45.43 ± 6.34 [e]
Group 5	145.34 ± 6.50 [e]	13.79 ± 7.09[d]	104.36 ± 6.19 [d]	96.38 ± 7.45 [c]	35.24 ± 6.32 [c]
Group 6	151.26 ± 6.68 [e]	12.25 ± 4.93[d]	115.50 ± 17.40 [c]	101.23 ± 12.1 [e]	44.45 ± 5.92 [e]
Group 7	158.23 ± 7.39 [e]	10.44 ± 5.96[c,d]	123.44 ± 24.8 [d]	114.66 ± 5.15 [d]	46.43 ± 3.33[a,e]
Group 8	166.43 ± 7.39[a,e]	8.72 ± 5.67[c]	131.41 ± 16.911 [e]	121.61 ± 3.35[g]	49.72 ± 17.82[a,e]

Values are represented in mean ± SD. N = 5. Mean values with different superscript alphabet in the same column differ significantly at $p < 0.05$.

Table 2: Electrolyte levels in the kidney of rats fed COCF treated with *Elaeis guineensis* leaves extracts

Experimental group	Na^+ (mEq/L)	K^+ (mEq/L)	Ca^{2-} (mEq/L)	Cl^- (mEq/L)	CO_3^{2-} (nmol/L)
Group 1	185.25 ± 3.52 [a]	7.24 ± 2.93 [a]	142.24 ± 16.09[a]	141.21 ± 3.57 [a]	130.66 ± 14.34 [a]
Group 2	110.14 ± 12.19 [b]	36.82 ± 9.74 [b]	90.57 ± 15.48[b]	31.32 ± 4.41 [b]	22.23 ± 05.93 [b]
Pre-treatment					

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Group 3	150.08 ± 39.49 [c]	19.52 ± 9.44 [c]	117.29 ± 8.68[c]	101.32 ± 7.18 [c]	90.21 ± 9.91 [c]
Group 4	168.40 ± 15.31 [d]	12.46 ± 4.03 [d]	139.16 ± 7.84[a]	125.14 ± 7.91 [d]	115.17 ± 11.75 [d]
Group 5	152.15 ± 7.18 [c]	16.24 ± 4.48 [c]	130.34 ± 7.94[d]	111.09 ± 39.50 [e]	101.28 ± 9.12 [e]
Group 6	168.18 ± 14.96 [d]	14.40 ± 4.53[c,d]	132.23 ± 8.56[a,d]	115.26 ± 11.05 [e]	106.40 ± 19.81[e]
Group 7	170.06 ± 22.36[a,d]	11.78 ± 3.21 [a]	140.15 ± 7.91[a]	126.15 ± 8.25 [d]	117.32 ± 12.83 [d]
Group 8	183.02 ± 14.23 [a]	6.49 ± 3.06 [a]	142.09 ± 7.21[a]	139.07 ± 7.28 [a]	128.51 ± 7.34 [a]
Co-treatment					
Group 3	148.49 ± 36.83 [c]	22.37 ± 7.79 [c]	100.47 ± 23.39[f]	36.60 ± 4.31[b]	27.38 ± 10.03[b]
Group 4	151.41 ± 25.34 [c]	30.36 ± 7.81 [e]	120.48 ± 14.93[c]	48.53 ± 3.63[c,d]	38.68 ± 05.96 [f]
Group 5	135.97 ± 7.34 [e]	20.25 ± 7.75 [c]	111.63 ± 32.81[c,e]	41.34 ± 12.76 [c]	31.76 ± 07.20 [f]
Group 6	140.79 ± 52.92 [e]	18.61 ± 6.08 [c]	116.31 ± 33.83[c]	44.54 ± 1.25 [c]	37.21 ± 07.93 [f]
Group 7	152.09 ± 27.65 [c]	15.52 ± 4.08 [c]	121.33 ± 13.32 [c]	50.25 ± 3.87 [d]	40.44 ± 06.51[f]
Group 8	156.49 ± 18.96 [c]	10.34 ± 3.88 [a]	124.82 ± 22.73 [c]	57.21 ± 8.23 [a]	69.60 ± 12.01 [h]
Post-treatment					
Group 3	139.33 ± 5.99 [c]	20.58 ± 3.92 [c]	110.48 ± 19.96[c]	80.48 ± 29.211 [c]	45.32 ± 7.96 [f]
Group 4	160.20 ± 5.59 [d]	15.24 ± 2.32 [c]	130.17 ± 12.31[d]	105.20 ± 18.43 [c]	57.42 ± 6.28 [g]
Group 5	144.51 ± 3.71 [e]	18.49 ± 4.79 [c]	120.36 ± 5.67[e]	90.54 ± 07.82 [e]	50.42 ± 5.86 [g]
Group 6	150.52 ± 5.78 [e]	16.40 ± 5.41 [c]	125.57 ± 5.67[e]	97.52 ± 07.99 [e]	53.36 ± 9.59 [g]
Group 7	161.28 ± 6.20 [d]	13.36 ± 4.63[a,c]	130.53 ± 3.24[a,d]	108.40 ± 10.21 [c]	60.19 ± 7.91 [g]
Group 8	175.45 ± 3.27 [a]	8.42 ± 5.20 [a]	136.46 ± 13.71[a,d]	120.22 ± 18.43[d,e]	72.61 ± 10.82[h]

Values are represented in mean ± SD. N = 5. Mean values with different superscript alphabet in the same column differ significantly at $p < 0.05$

Table 3: Changes in Oxidative Stress indices of rats fed crude oil contaminated feed treated with *E. guineensis* leaves extracts

Group	MDA (μmolml^{-1})	CAT (μmolml^{-1}g^{-1}tissue)	SOD (Unitg^{-1}tissue)	GSH (μmolmg^{-1}protein)
Group 1	2.50 ± 2.12[a]	85.90±8.78 [a]	44.34 ± 7.65 [a]	10.20 ± 4.16[a]
Group 2	12.11 ± 3.12[b]	22.28 ± 6.23 [b]	12.28 ± 318 [b]	0.30± 0.54[b]
Pre-treatment				
Group 3	8.24 ± 2.56[c]	53.24 ±7.36 [c]	24.39 ± 7.36 [c]	2.90 ± 0.15[c]
Group 4	5.57 ± 2.13[d]	74.40 ±7.70 [d]	37.44 ± 3.49 [d]	7.17 ± 1.23[a]
Group 5	6.33 ± 2.34[cd]	63.39 ±9.37 [e]	29.31 ± 4.25 [c]	3.91 ± 1.53[c]
Group 6	4.42 ± 2.39[d]	71.30±14.40 [d]	37.28 ± 6.21 [d]	6.06 ± 0.58[a]
Group 7	3.21 ± 1.54[a]	81.90 ±7.81 [a]	38.38 ± 13.07[a,d]	8.26 ± 2.60[a]
Group 8	2.32 ± 0.43[a]	85.12 ± 11.19 [a]	42.14 ± 12.16 [a]	9.31 ± 2.48[a]
Co-treatment				
Group 3	10.3 ± 3.99[b]	25.36 ± 3.23 [b]	16.52 ± 5.53 [b]	0.41± 0.23[b]
Group 4	5.37 ± 2.55[d]	44.45 ± 2.56 [e]	29.50 ± 7.23 [c]	2.13± 0.82[c]
Group 5	8.19 ± 2.11[c]	30.37 ± 12.23 [f]	21.35 ± 4.71[b,e]	0.77± 0.31[b]
Group 6	6.48 ± 0.34[cd]	38.24 ± 9.12 [f]	28.44 ± 6.24 [c]	1.04± 0.73[b]
Group 7	4.28 ± 0.37[d]	53.45 ± 7.123 [c]	31.50 ± 7.83[d,c]	3.20 ± .043[c]
Group 8	3.27 ± 0.34[a]	61.62 ± 12.43 [e]	34.31 ± 9.45 [d]	4.35 ± 1.92[c,d]
Post-treatment				
Group 3	9.32 ± 0.45[b]	48.35 ± 3.94 [c]	18.51 ± 2.66[b,e]	0.50 ± 0.43[b]
Group 4	6.37 ± 2.34[d]	69.22 ± 6.93[d,e]	32.29 ± 7.37 [d]	3.00 ± 1.82[c]
Group 5	7.39 ± 2.78[cd]	58.30 ± 6.00 [e]	28.27 ± 5.08 [c]	0.91 ± 0.21[b]
Group 6	6.28 ± 0.92[d]	65.25 ± 7.23 [e]	31.38 ± 4.32[d,c]	2.71 ± 0.72[c]
Group 7	4.77 ± 0.46[d]	78.16 ± 7.43[a,d]	33.17 ± 6.16[d,c]	4.40 ± 0.32[c,d]
Group 8	4.17 ± 0.23[d]	80.31 ± 4.67 [a]	36.16 ± 5.69 [d]	5.45 ± 0.72[a,c,d]

Values are represented in mean ± SD. N = 5. Mean values with different superscript alphabet in the same column differ significantly at $p < 0.05$.

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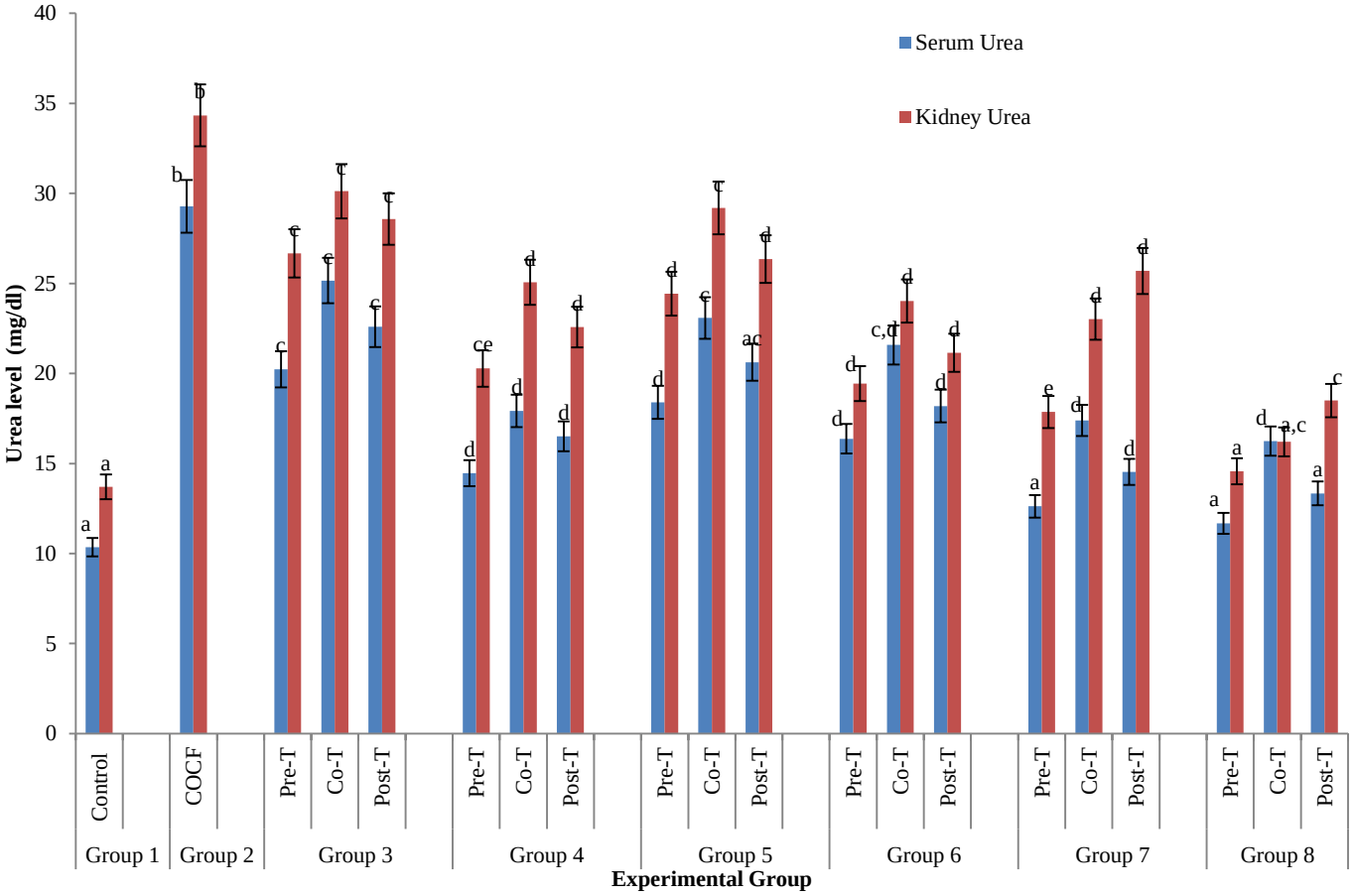

Figure 1: Effect of *E. guineensis* leaves extracts on serum and kidney urea levels of rats fed crude oil contaminated feed
Bars of same parameter with different superscript letter differ significantly at $p < 0.05$. n = 6

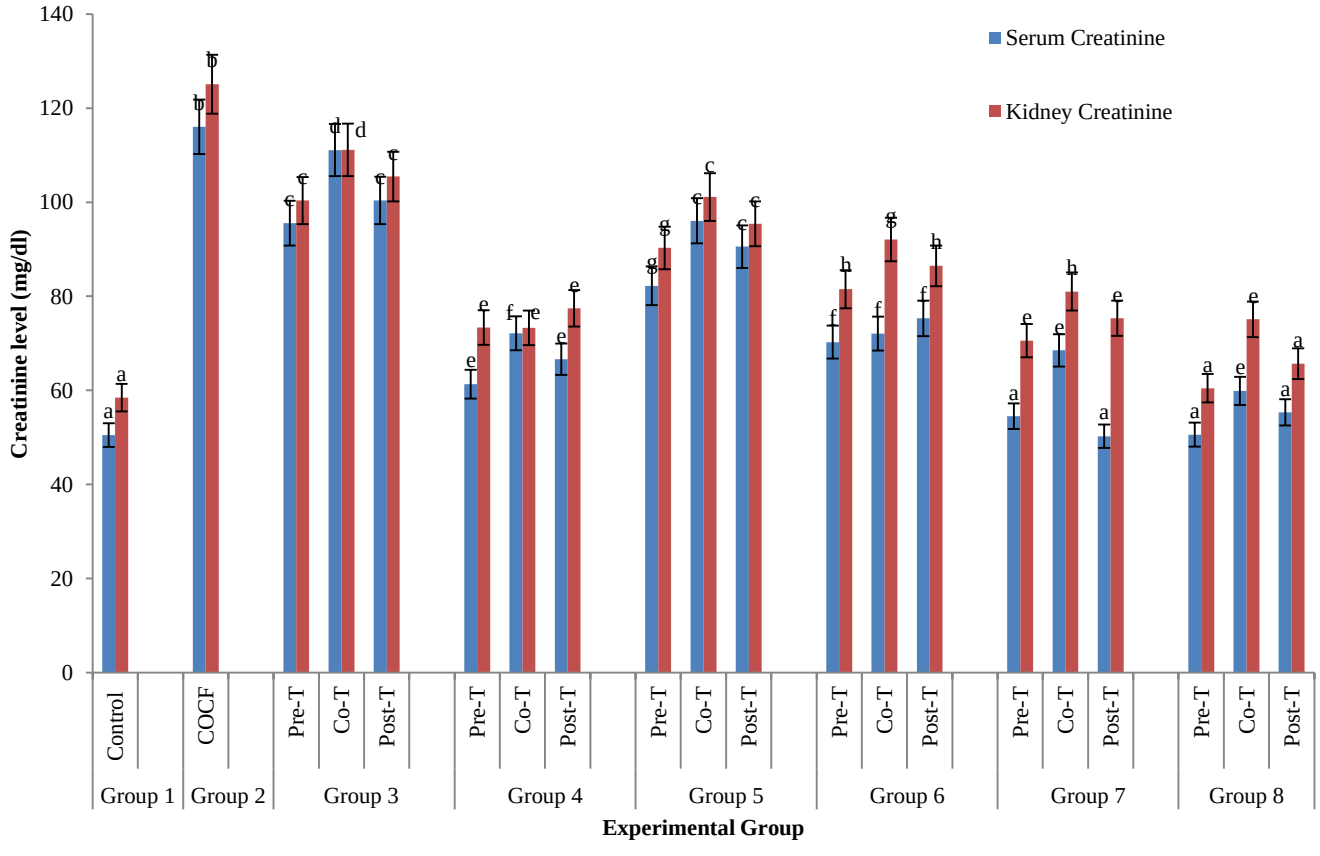

Figure 2: Effect of *E. guineensis* leaves extracts on serum and kidney creatinine levels of rats fed COCF.
Bars of same parameter with different superscript letter differ significantly at $p < 0.05$. n = 6

Conclusion

It is concluded that the administration of the various solvent extracts (aqueous, ethanol, methanol, acetone, petroleum ether) and blended solvent extract mixture of *E. guineensis* had the potency of reversing and modulating the reported kidney dysfunctions. Also the study indicated that the pre-treatment of animals with the various extracts had more efficient in handling the metabolic challenge compared to the co-treatment and post-treatment regimens targeted at preventing and ameliorating the metabolic challenge occasioned by crude petroleum toxicity

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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References

1. Asagba SO. Cadmium in our food and drinking water; should we be worried? 70th in the series of inaugural lectures of the Delta State University, Abraka. 14th February, 2019; Delta State University Press, 2019.
2. Chinedu E and Chukwuemeka CK. Oil spillage and heavy metals toxicity risk in the Niger Delta, Nigeria. J Health Pollut. 2018; 8(19):180905.
3. Oyedeji A, Adebiyi A, Omotoyinbo M, Ogunkunle C. Effect of crude oil-contaminated soil on germination and growth performance of *Abelmoschus esculentus* L. Moench-A widely cultivated vegetable crop in Nigeria. Am J Plant Sci. 2012; 3(10):1451-1454.
4. Akpokodje J and Salau S .Oil pollution and agricultural productivity in the Niger Delta of Nigeria. Environ Econ 2015; 6(4):68-75.
5. Osuagwu ES and Olaifa E. Effects of oil spills on fish production in the Niger Delta. PLoS ONE 2018; 13(10):e0205114.
6. Ohanmu EO, Bako SP, Ohanmu E, Ohanmu SO. Environmental implications, properties and attributes of crude oil in the oil-producing states of Nigeria. Ecol. 2019; 9:1-9.
7. Adekola J, Fischbacher-Smith M, Fischbacher-Smith D, Adekola. Health risks from environmental degradation in the Niger Delta, Nigeria. Environ Planning C: Politics Space. 2017; 35(2):334-354.
8. Nriagu J, Udofia EA, Ekong I, Ebuk G. Health Risks Associated with Oil Pollution in the Niger Delta, Nigeria. Int J Environ Res Pub Health. 2016; 13:346.
9. Ordinioha B and Brisibe S. The human health implications of crude oil spills in the Niger delta, Nigeria: An interpretation of published studies. Nig Med J. 2013; 54:10-16.
10. Achuba FI. Protective role of Elaesis guineensis leaves against crude oil tainted diet–induced hematotoxicity in Wistar rats. Iranian J Toxicol. 2019; 13(4):1-4.
11. Yin NS, Abdullah S, Phin CK. Phytochemical constituents from leaves of *Elaeis guineensis* and their antioxidant and antimicrobial activities. Int J Pharm Pharm Sci. 2013; 5:137-140.
12. Mohamed S. Oil palm leaf: A new functional food ingredient for health and disease prevention. J Food Proc Technol. 2014; 5(2):300-306.
13. Achuba FI. Powdered oil palm (*Elaesis guineensis* Jacq) leaf as remedy for hydrocarbon induced liver damage in rats. Nig J Pharm Appl Sci Res. 2018; 7(3):89-95.
14. George BO, Okpoghono J, Osioma E, Aina OO. Changes in oxidative indices in *Plasmodium berghei* mice treated with aqueous extract of *Aframomum sceptrum*. Front Sci. 2012; 2(1):6-9.
15. Okpoghono J, Achuba FI, George BO. Protective effects of Monodora myristica extracts on crude petroleum oil-contaminated catfish (*Clarias gariepinus*) diet in rats. Int J Vet Sci Med. 2018; 6:117-122.
16. Achuba FI. Role of bitter leaf (*Vernonia amygdalina*) extract in prevention of renal toxicity induced by crude petroleum contaminated diets in rats. Int J Vet Sci Med. 2018; 6:172-177.
17. Henry RF. Clinical Chemistry Principle and Techniques. Harper and Row Hagerstein. 1974. 2 p.
18. Terri AE and Sesin PG. Determination of serum potassium by using sodium tetraphenylboron method. Am J Clin Pathol. 1958; 29(1):86-90.
19. Skeggs LT and Hochestrasser H. Multiple automatic sequential analysis. Clin Chem. 1964; 10:918-936.
20. Forrester RL, Wataji LJ, Silverman DA, Pierre KJ. Enzymatic method for the determination of CO_2 in serum. Clin Chem. 1976; 22:243-249.
21. Cali JP, Mandel J, Moore LJ. A reference method for the determination of calcium in serum. N.S.B., Sp Pub. 1972. 36 p.
22. Gutteridge JMC and Wilkins C. Copper dependent hydroxyl radical damage to ascorbic acid. formation of a thiobarbiturie acid reactive product. FEBS Lett 1982; 137:327-340.
23. Ellman GL. Tissue sulfhydryl groups. Arch Biochem and Biophys. 1959; 82:70-77.
24. Misra HP and Fridovich I. The role of superoxide anion in the autooxidation of epinephrine and a sample assay for superoxide dismutase. J Bio Chem. 1972; 247:3170-3175.
25. Kaplan A, Dembiec D, Cohen G, Marcus J. Measurement of catalase activity in tissue extracts. Anal Biochem. 1972; 34:30-38.
26. Onakurhefe P, Achuba FI, George BO. Pretreatment with Oil Palm Leaf Extracts Confers Protection against Crude Oil Adulterated Food (COAF). Trop J Nat Prod Res. 2020; 4(9):643-648.
27. Caney S. Management and treatment of chronic kidney disease in cats. In Practice. 2016; 38:10-13.
28. Chilcott RP. Compendium of chemical hazards: Kerosene (fuel oil). A publication of WHO. 2006; Available in https://www.who.int/ipcs/emergencies/kerosene.pdf
29. Ogbeke GI, George BO, Ichipi-Ifukor PC. *Aframomum sceptrum* modulation of renal function in monosodium glutamate (MSG) induced toxicity. UK J Pharm Biosci. 2016; 4(4):54-60
30. Achuba FI. Protective influence of *Elaesis guineensis* leaf in diet on petroleum-mediated kidney damage in rat. Nig J Pharm Appl Sci Res. 2018; 7(2):33-38.
31. Azeez OM, Akhigbe RE, Anigbogu CN. Oxidative status in rat kidney exposed to petroleum hydrocarbons. J Nat Sci Biol Med. 2013; 4:149-54.
32. Okpoghono J, George BO, Achuba FI. Assessment of antioxidant indices after incorporating crude oil contaminated catfish and African nutmeg (*Monodora myristica*) extracts into rat diet. J Appl Sci Environ Manag. 2018; 22(2):197-202.
33. Ita SO and Edagha IA . Renal protective effect of antioxidant vitamins C and E against crude oil-induced nephrotoxicity. Merit Res J Med Medic Sci. 2016; 4(9):425-431.
34. Onakurhefe P, Achuba FI, George BO, Okpoghono J. Effect of *Elaeis guineensis* (Jacq) leaf extracts on crude oil-induced genotoxicity in Wistar albino rats. Sci Afr. 2020; 7:e00280.