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Protective Activity of Palm Oil Extracts against Dichlorvos Poisoning in Rats: Biochemical Parameters and Mechanisms of Action

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ABSTRACT

Protective activity on biochemical parameters and possible mechanisms of action of palm oil extracts were investigated in orally Dichlorvos poisoned rats and the efficacy of treatments in the poisoned rats. The safety profile of the extracts was evaluated via acute toxicity studies which was done in two phases. To screen for phytochemicals, present in this extract. This study was carried out in Pharmacy and KIUTH laboratories, Kampala International University, Uganda. Wistar rats of both sexes, weighing 85g and above were used for the study. Selection of rats was done by simple random sampling. At the end of the administration, plasma biochemical parameters were measured as indices of organ toxicity. The red palm oil used in treatment of Dichlorvos poisoned rats caused a reduction in concentration of some of the biochemical parameters of the rats when given simultaneously compared to the kernel oil combinations. Administration of palm oil does not have prophylactic effect against the poison. However, it does seem to have similar protective effects against oxidative stress with ascorbic acid. In summary the administration of palm oil seemed to have limited ameliorative role against Dichlorvos poisoning in rats.

Keywords: Dichlorvos, palm oil, palm kernel oil, rats, acute toxicity, oxidative stress

INTRODUCTION

Pesticide poisoning accounts for about one-third of the world's mortality rate. Epidemiological and toxicological data suggest that many of these poisoning might be prevented if the accessibility and quality of care for poisoning could be improved [1]. Economically, the conventional drug for organophosphate poisoning which are anticholinergic agents such as atropine are expensive and cannot easily be afforded by the rural poor who are mostly vulnerable to organophosphate poisoning [2]

Oil palm *(Elaeis guineensis)* is believed to have originated from West Africa. The commercial value of the crop lies primarily in its oil. Oil palm is unique in that it produces two types of oil. The fleshy mesocarp produces palm oil and the kernel produces palm kernel oil.

Aside the nutritional properties, the healing properties of palm oil have been recognized for generations [3]. Until modern medicine arrived, red palm oil was the remedy of choice for nearly every illness in most parts of Africa [4,5]. When someone got sick, drinking a cupful of palm oil was a remedy of choice. Today, scientists are recognizing the value of red palm oil in the treatment and prevention of diseases. There have been a number of scientific studies that surport the medicinal use of palm oil. Rand et al [6] in the Netherlands first demonstrated that palm oil has an anti-clotting effect, and it's as antithrombotic as the highly unsaturated sunflower seed oil. A human study showed that tocotrienols (from palm oil) supplementation can reduce re-stenosis of patients with carotid atherosclerosis [7].

Tocopherol and its relative, tocotrienol in palm oil, demonstrates anti platelet aggregation properties in humans [8]. A study conducted by Rand *et al.*, [6] showed that a palm oil diet either increases the production of prostacyclin which inhibits blood-clotting or decreases the formation of thromboxane which induces blood-clotting.

Anticholinergic agents have long been used in the pharmacological treatment of organophosphate poisoning and other pesticide poisoning, the management of which is still a problem, particularly in developing countries. [9] These drugs produce significant and frequent side effects, depending on their mechanisms of action which may lead to minor complications such as skin flushing, hyperthermia, dry mucus membrane and no sweating, blurred vision, confusion, delirium and severe complications including renal toxicity progressing to nephrotic syndrome. Economically, they are expensive but affordable by the rural poor who are mostly mostly farmers and have ready access to such agricultural products. Rural farmers are highly vulnerable to organophosphate poisoning. Natural products are known to be used for

treatment of different ailments especially in developing countries. They have proven to have minimal side effects and are also cost- effective. Natural plants have been and remain the cornerstone of the health care especially in developing countries [10,11]

This study aimed to identify the phytochemicals present in *Elaeis guineensis* extracts, to determine the safety level (acute toxicity) of *Elaeis guinensis* extracts against orally ingested organophosphate, to identify whether *Elgaeis guineensis* extract (palm oil) has antidotal properties against organophosphate poisoning by evaluation of biochemical parameters, to determine the efficacy of *Elgaei sguineensis* in treatment of organophosphate toxicity and compare the effects of the two extracts and to evaluate the anti-poisoning and protective effects of *Elgaeis guineensis* extracts against organophosphate (Dichlorvos) poisoning in rats.

METHODOLOGY

Extraction of kernel oil

The palm fruits that served as sample for analysis was obtained from Nakasero market in Kampala Uganda. The fruits were thoroughly screened to remove the bad ones and stones. The kernel nuts was collected, washed dried under sun, for 2 hours and cracked. The dried clean nuts were milled using an electric blender. The milled nuts were boiled and the floating oil was scooped from the top.

Extraction of red oil

The ripe oil palm fruits were boiled in a pot for 2hours.A pulp mass will produce by pounding the boiled fruits. The pulp was immersed in water and stirred thoroughly. The fibres and the seeds were filtered out using a basket as a sieve. The filtrate or infusion was poured into an aluminum cooking pot and boiled for five hours. The heated mixture was allowed to cool. The palm oil which set on top of the aqueous portion of the boiled filtrate was scooped into a fresh container. The collected oil was heated gently for ten minutes to remove traces of water.

Phytochemical analysis

Chemical tests was carried for preliminary phytochemical screening of palm fruit using standard procedure by Sofowora [12], Trease and Evans [13] and Barbone [14].

Procurement of animals for the study

The study was conducted on mature male and female experimental albino rats A total of 87 rats were used. Fifty (55) rats were used for the anti- poisoning study while 32 were used for acute toxicity tests of both extract. Mature and healthy rats weighing 85g and above were used for the study excluding pregnant rats and those weighing less 85g. The animals were randomly selected for each group.

Acute toxicity studies

It was conducted according to a modified method of Akhila et al [15] (with the use of rats instead of mice and inclusion of control group).

Anti-poisoning studies

The animals were categorized into 11 groups involving 2 different treatments, whereby eachgroup has 5 rats.

Group 1- received poison for one week and red oil for one week

Group 2- received red palm oil after 30mins poison was administered

Group 3- received poison after 30mins red palm oil was administered Group 4- received poison and red palm oil simultaneously. Group 5- received poison for one week and kernel oil for one week Group 6- received kernel oil after 30mins poison was administered. Group 7- received poison after 30mins kernel oil was administered. Group 9- received poison alone for two weeks.

Group 10- received vitamin C (Ascorbic acid) 30mins after administration of poison (positive control for protection against Dichlorvos-induced oxidative stress and the process will last for a period of two weeks).

Group 11- received distilled water for two weeks.

Volume to be administered was calculated using this formula,

$$\text{Volume} = \frac{\text{weight of rat (g)} \times \text{dose (mg/kg)}}{\text{Concentration of extract (mg/ml)} \times 1000}$$

The dose that was administered of Dichlorvos given orally during this study represents 1/20 Of the LDS0 (25mg/kg)

4ml per body weight (kg) of each extract was administered to each rat daily

Ascorbic acid was given at a dose of 100mg/kg which gives a protection against the toxicity and used like a positive control for protection against Dichlorvos-induced oxidative stress.

Biochemical Studies

After administration of the treatment with the palm oil extracts and poison on the last day, the rats was sacrificed painlessly using chloroform in their respective groups and biochemical assays such as AST, ALT, ALP, GGT, Bilirubin, Total Protein, Albumin levels were carried out to determine the effectiveness of the two extracts with administration of different treatment and poison in the different groups.

Standard commercial test kits obtained from appropriate sources were used to estimate Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Alkaline phosphatase (ALP), and Albumin, Gamma Glutamyl Transferase (GGT), and total bilirubin (Bil T)

Reitmann and Franklin [16], Tietz [17]

Data Analysis

Data was analyzed using one way analysis of variance using SPSS version 16.0 (ANOVA). Differences between two means were detected using the student's t-test.

Ethical considerations

Approval was sought and obtained from the KIU-WC Ethics Committee to undertake the study. Experiment was conducted according to the guide for the care and use of laboratory animals and ethical guidelines for investigation of experimental pain in conscious animals [18].

RESULTS

Acute Toxicity

Acute toxicity and lethality of *Elaeis guineensis* extracts administered orally up to 10,000 mg/kgto rats was tested and found to cause no death in the two phases of the test. Thus, the LD_{50} of *Elaeis guineensis* extract in rats was estimated to be greater than 10,000 mg/kg.

Phytochemical Analysis

The fruit extracts were screened for the presence of tannins, phlobatanins, saponins, flavonoids, terpenoids, anthraquinones, cardiac glycosides, alkaloids and carotenoid and tocopherols. The presence of constituents tested were recorded as present (+) or absent (-).

Phytochemical	Red palm oil	Palm kernel oil
Alkaloids	-	+
Carotenoid	+	-
Flavonoids	+	+
Phlobatannins	-	-
Tocopherol	+	-
Phenol	+	-
Saponins	+	+
Steroids	+	+
Tannins	-	-
Terpenoids	+	+

The result of the phytochemical analysis of Elaeis guineensis extracts indicates that carotenoid, flavonoids, tocopherol, phenol, saponins, steroids and terpenoids were present in palm oil; alkaloids, flavonoids, Saponins, steroids and terpenoids. Terpenoids, steroids, flavanoids and saponins were present in both red palm and kernel oils in moderately high concentration. While alkaloids, tannins and, phlobatannins were absent in palm oil, carotenoids, phlobata1mins, tocopherol, phenol and taimins were absent in palm kerneloil.

Biochemical assay

Biochemical assay of kernel oil results (Table 1)

The ALT results in descending order indicate the highest value in group F (poison after 30minsof kernel oil) followed by group C (simultaneous kernel oil + poison), then groups A (positive control), H (poison 1 st week+ kernel oil 2ndweek), X (poison only), D (poison+ kernel oil after 30mins of poison) and Y (negative control (distilled water)) being the lowest.

The AST results in descending order indicate the highest value in group H (poison I stweek + kernel oil 1st week) followed by group C (simultaneous kernel oil + poison) then groups Y (negative control), poison only), A (positive control), F (poison after 30 mins of kernel oil) and D (kernel oil after 30mins of poison) being the lowest.

The ALP results in descending order indicate the highest value in group C (simultaneous kernel oil + poison) followed by group H (poison I st week + kernel oil 2ndweek) and groups X (poison only), A (positive control), F (poison after 30 mins of kernel oil), D (kernel oil after 30mins of poison) and Y (negative control) being the lowest.

GOT results in descending order indicate the highest value in group D (kernel oil after 30mins of poison) followed by group X (poison only), the group F (poison after 30 mins of kernel oil), H (poison 1st week + kernel oil 1 week), A (positive control), Y (negative control) and C (simultaneous kernel oil+ poison) being the lowest.

TP results in descending order indicate the highest value in group Y (negative control) followed by group C (simultaneous kernel oil + poison), then groups A (positive control), H (poison Ist week+ kernel oil 2nd week), X (poison only), F (poison after 30 mins of kernel oil) and D (kernel oil after 30mins of poison) being the lowest.

ALB results in descending order indicate the highest value in group A (positive control) followed by group X (poison only), then groups F (poison after 30 mins of kernel oil), Y (negative control), C (simultaneous kernel oil + poison), H (poison 1 st week + kernel oil 2nd week), and D (kernel oil after 30mins of poison) being the lowest. D-Bil results in descending order indicate the highest value in group Y (negative control), followed by group F (poison after 30 mins of kernel oil) and then groups A (positive control), D (kernel oil after 30mins of poison), H (poison 1 st week + kernel oil 2nd week), X (poison only) and C (simultaneous kernel oil + poison) being the last group.

Biochemical assay of red palm oil results (Table 2)

The ALT results in descending order indicate the highest value in group A (positive control) followed by group I (poison 1st week + palm oil 2nd week), followed by group X (poison only) then group Y (negative control, Distilled water), group B (simultaneous palm oil + poison), G (poison after 30 mins of palm oil) and group E (palm oil after 30 mins of poison) which is the lowest value.

The AST levels in descending order indicate the highest value in group I (poison 1st week + palm oil 2nd week) followed by group X (poison only) then groups A (positive control, Ascorbic acid), B (simultaneous palm oil + poison), G (palm oil after 30 mins of poison), Y (negative control, Distilled water) and group E (poison after 30 mins of palm oil) the lowest.

ALP levels in descending order indicate the highest value in group I (poison Ist week palm oil 2nd week) followed by group X (poison only) then groups A (positive control, Ascorbic acid), G (palm oil after 30mins poison), E (poison after 30mins palm oil), Y (negative control, Distilled water) and the lowest group B (simultaneous Poison+ palm oil).

GGT levels in descending order indicate the highest value in group E (poison after 30 mins palm oil) group I followed by group I (poison Ist week palm oil 2nd week), then groups X (poison only), G (palm oil after 30mins poison), B (simultaneous poison + palm oil), A (positive control, Ascorbic acid) and the lowest been group Y (negative control, Distilled water).

TP levels in descending order indicate the highest value in group Y (negative control, Distilled water), followed by group A (positive control, Ascorbic acid), then groups I (poison 1st week palm oil 2nd week), X (poison only), G (palm oil after 30mins poison), B (simultaneous poison + palm oil) and group E (poison after 30mins palm oil) being the lowest.

ALB levels in descending order indicate the highest value in group A (positive control, Ascorbic acid), followed by group G (palm oil after 30mins poison), then groups X (poison only), Y (negative control, Distilled water), I (poison Ist week palm oil 2nd week), B (simultaneous poison + palm oil) and E (poison after 30mins palm oil) being the lowest. D-Bil levels in descending order indicate the highest value in group B (simultaneous poison + palm oil) then group A (positive control), followed by groups E (poison after 30mins palm oil), I (poison 1st week palm oil 2ndweek), G (palm oil after 30mins poison), X (poison only) and Y (negative control, Distilled water) being the lowest.

Comparing results in table 1 and table 2

ALP levels in group A (positive control, Ascorbic acid) and C (kernel oil + poison) increased more highly compared to the group B (simultaneous palm oil+ poison) which showed a decrease in ALP levels. The ALP in group B decreased compared to the negative control group (table I).

The ALP activity was statistically significant ($p==0.036<0.05$) in Group H (poison Ist week kernel oil 2ndweek) compared to group B (palm oil + poison).

Between groups D (poison after 30mins kernel oil) and E (poison after 30mins palm oil), there was no statistical difference, but effects shown in group E (poison after 30mins palm oil) had slightly decreased biochemical parameters when compared to negative control (group Y).

There was no statistically significant difference observed in among the groups in the TP levels. There was a significant increase in the ALT level of F (kernel oil after 30mins poison) compared to G (palm oil after 30mins poison). The ALT level of G (palm oil after 30mins poison) decreased compared to the control group. There is no significant difference between the biochemical parameters of H (poison Ist week kernel oil 2nd week) and! (poison 1st week palm oil 2nd week) and are increased compared to the control group.

In this study, decrease in the albumin level was noticed in group D (poison after 30mins kernel oil) and E (poison after 30mins palm oil). The levels of plasma hepato-specific enzymes such as ALT, AST, ALP and GGT, TP, ALB and bilirubin were significantly increased in dimethoate intoxicated rats when compared with the control groups.

Table 1: Anti poisoning effects of kernel oil on rats' biochemical profiles Dichlorvos® induced poisoning

Biochemical parameters ± SEM for Kernel oil

Groups	ALT (IU/L)	AST (IU/L)	ALP (IU/L)	r-GT (IU/L)	TPP (g/L)	ALB (g/L)	D-Bil-D (µmol/L)
A	193.7± 30.2	223.7± 10.2	552.6±52.5	1.7± 1.4	72.2± 1.3	37.3± 1.1	9.5± 2.3
C	228.6± 59.6	269.0± 89.4	770.1±22.*	1.6± 0.5	73.8± 73.8	33.6± 2.5	5.9 ± 0.5
D	133.8± 38.2	216.8± 56.0	436.0± 84	2.9± 0.6	56.1± 15.5	23.5± 6.6	9.1± 2.1
F	259.3± 11.4	217.9± 17.6	481.9±32.7	2.2± 0.8	67.6± 1.8	35.3± 0.6	10.9± 1.4
H	186.0± 21.8	305.3± 13.4	728.4±140*	2.0± 0.5	72.2± 1.1	31.6± 0.5	8.1± 1.3
X	181.8± 9.5	247.8± 18.3	601.6±98.1	2.7± 0.3	68.0± 1.3	36.2± 0.6	6.7± 0.6
Y	130.1± 19.9	263.8± 28.6	364.9±65.5	1.6± 0.7	75.0± 5.4	35.0± 1.5	15.6±3.3

*Statistically significant at p :S 0.05

The levels of ALP increased significantly in group C compared to that of group 8, P= 0.037

Key: A (Positive control, Ascorbic acid), C (Kernel oil+ poison), D (poison after 30mins kerneloil), F (kernel oil after 30mins poison), H (poison 1st week kernel oil 2nd week), X (poison for two weeks) and Y (Distilled water for two weeks).

Table 2: Anti poisoning effects of red palm oil on rats' biochemical profiles Dichlorvos®induced poisoning.

	Biochemical parameters ± SEM for palm oil						
groups	ALT (IU/L)	AST (IU/L)	r-GT ALP (IU/L) (IU/L)		TP (g /L)	ALB (g/L)	D-Bil-D (µmol /L
A	193.7± 30.2	223.7± 10.2 1.4	552.6±52.5	1.7±	72.2± 1.3	37.3± 1.1	9.5± 2.:
B	110.00	213.30	194.7*	2.1	67.6	32.4	I0.57
C	82.8± 110.3	176.1± 52.8 1.4	377.5±168	4.5±	49.415.6	24.7± 7.7	9.5 ±I.S
D	123.4 ± 11.4	206.1 ± 20.2 0.5	497.1±81.7	2.6 ±	67.5 ± 3.7	36.5 ± 0.8	6.8± 0.i
E	182.4± 19.2	266.6± 13.3 0.3	612.4±98.1	2.9±	70.4± 2.9	34.2± 2.1	8.5±I.
F	181.8± 9.5	247.8± 18.3 0.3	601.6±98.1	2.7±	68.0± 1.3	36.2±0.6 1	6.7±0.c

*Statistically significant at p :S 0.05. The ALP activity was statistically significant(p=0.036) in Group H compared to group B.

Key: A (Positive control, Ascorbic acid), B (palm oil+poison), E (Poison after 30 mins palm oil), G (palm oil after 30 mins poison), I (poison 1st week palm oil 2nd week), X (poison for two weeks) and **Y** (Distilled water for two weeks).

DISCUSSION

The safety of drugs and plant products for human use can be determined using toxicological evaluation which is usually carried out in various experimental animals to predict toxicity and to provide guidelines for selecting a safe dose in humans. [19] The acute toxicity studies showed that the *Elaeis guineensis* has a high safety profile when given orally with an LD_{50} value greater than 10,000mg/kg body weight. Therefore, the medicinal palm fruit extracts in its local formulation can be categorized as relatively non-toxic based on the scale proposed by Akhila, et al [15]

The result of the phytochemical analysis of *Elaeis guineensis* extracts indicates that terpenoids, steroids, flavonoids and saponins are in both kernel and red palm oil are in moderately high concentration while phenol, cardiac glycosides, tannins, phlobatannins, anthraquinone are absent in kernel oil and palm oil except phenol which is present in palm oil and it is abundant in carotenoid and tocopherol. The presence of terpenoids and steroids may be related to their non- polar nature which obviously favoured their increased concentration in the oil. The absence of cardiac glycoside may be related to their expected tendency to partition away from the oil due to their lipid insoluble nature [20]. The presence of flavonoid indicates that the oil will be good for management of cardiovascular diseases and oxidative stress because flavonoids are potent biological antioxidants [21]. Alkaloids have been used as central nervous system stimulants, topical anaesthetics in ophthalmology, powerful pain relievers, anti-pyretic action, among other uses, as such the use of this oil can serve these purposes. Saponins aid in reducing cholesterol levels by forming complexes with cholesterol and bile acids which prevent them from being absorbed through the small intestine hence lowers the cholesterol levels in the liver and blood. Saponins also serve as antioxidants as they prevent degradation of DNA and also helps to

reduce colon damage and risk of cancer [22]. The presence of terpenoids accounts for its use as an anti-diabetic agent [23]. Terpenoids also act as antibiotics and they are heart friendly.

Blood is a sensitive index of changes of an animal to any environmental pollutants and it is well known that toxic stress of any nature would show conspicuous and significant changes in the blood parameters. The present study revealed that administration of Dichlvos® to rats at aconstant dose for 14 days like other poisons produces significant changes in serum biochemical parameters [24,25]. Dichlorvos® binds to plasma protein, where it causes alterations in high number of enzymes; it can also perturb protein synthesis in hepatocytes [20]. Increasing the activities of AST and ALT in the blood sera was most likely the consequence of hepatotoxic effects of Dichlorvos. The Dichlorvos entering the body by oral gavages is delivered to the liver through the portal blood circulation where the greatest part of it remains stored. The accumulated poison in the liver can act by directly damaging the hepatocytes, primarily by destroying the cell membrane which results in the increased release of cytosolic enzymes AST and ALT into the circulation.

The elevation of AST and ALT levels may be due to pathological changes such as necrosis of hepatocytes, which causes increase in the permeability of the cell membranes, resulting in the release oftransaminases in the blood stream. The observed damage may be due to the fact that the liver being the first target of acute toxicity and the first organ exposed to everything that is absorbed in the small intestine, may metabolize foreign substances to highly reactive metabolites which may be hepatotoxic. In addition, because of the small duration of treatment, the alterations might be incipient and reversible, and not pronounced enough to change significantly serum ALT levels. A significant reduction in ALP may be attributed to the decrease in osteoblastic activity of bone, since the ALP is formed in the osteoblasts. An elevated direct bilirubin may be associated with hepatocellular disease. GGT with elevation of other liver enzymes may be indicative of liver damage. A decreased serum albumin level may result from a decreased ability to synthesize protein and indicative of liver disease. The two general causes of alterations of serum total protein are change in the volume of plasma water and a change in the concentration of one or more of the specific proteins in the plasma. The red palm oil used in treatment of oxidative stress caused by Dichlorvos caused a reduction in the biochemical parameters of the rats when given simultaneously and after few minutes of poisoning compared to the kernel oil combinations and does not have prophylactic effect against the poison. This could be due to the fact that the antioxidants present in the palm oil are available for mopping up of free radicals released by the poison when given simultaneously or few minutes after poison ingestion rather than taking the oil as prophylaxis because the antioxidants would have been broken down and used up by the body and leaving little or nothing to fight free radicals produced by the poison. However, when compared to the treatment with ascorbic acid have similar protective effects against oxidative stress which may be due to an inhibition of reactive oxygen species (ROS). Oxidative prope11y of palm fruit is related to the large amounts of tocopherols and carotenoids [5] which act through different mechanisms. Synergistic effects were evidenced with combinations of carotenoids and tocopherols which are both present in red palm oil.

CONCLUSION

It could be concluded that simultaneous administration of red palm oil and Dichlorvos poison and administration of red palm oil 30 minutes after treatment with Dichlorvos poison possessed a statistically significant anti-poisoning effects and protective value as a potent antioxidant (Twumasi *et al*., 2014) against a Dichlorvos sensitive biochemical variables (AST, ALT, ALP and GGT). It could be concluded that administering palm oil 30 mins before poison, palm oil after a week of poison, kernel oil+ poison simultaneously, kernel oil 30 mins after poison, poison 30 mins before kernel oil and also kernel oil after one week of poison have little or no protective and anti--poisoning effects against Dichlorvos poison.

Recommendation

Further work needs to be done on the haematological and histopathological parameters in rats to identify the extent of damage and repair and also to isolate and purify the active principles involved in the antioxidant activity of this plant.

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