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Effects of Palm Kernel Oil-treated Diet on Some Biochemical Parameters of Male Rats

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ABSTRACT: Male albino rats (*Rattus norvegicus*) were placed on guinea feed mixed with palm kernel oil (PKO) in ratio 10:1 for 35 days, using palm oil (PO) as a reference. Body weight gain, feed and water intake, organ (kidneys, spleen, heart and liver) weights, haematological and liver function indices of the rats were evaluated at the end of the experimental period. The oil-treated diets significantly ($p<0.05$) reduced the feed and water intake whereas the feed efficiency ratio (FER) increased. The oil-treated diets did not affect ($p>0.05$) the organ weights and haematological parameters, but increased ($p<0.05$) the serum aspartate aminotransferase activity and globulin concentration. Palm kernel oil-treated diet reduced ($p<0.05$) serum total bilirubin and albumin concentrations; the serum albumin concentration being lower than that of the serum globulin. The serum albumin/ globulin (A/G) ratios of the PKO- and PO-treated-diet rats were 0.74:1 and 1.21:1, respectively. In conclusion, the PKO-treated diet improved the nutrient adequacy of the diet, did not affect organ weights and haematological parameters, but decreased the serum A/G ratio of the rats.

KEYWORDS: Albumin:globulin, oil-treated diet, palm kernel oil, male rats.

1. Introduction

Palm kernel oil (PKO) is a fat (Wardlaw and Kessel, 2002) extracted industrially as a yellow coloured liquid from the kernel of the tropical palm tree (*Elaeis guineensis*) using mechanical press (Ugbogu *et al*., 2006) or with solvents like n-hexane (Akubugwo and Ugbogu, 2007). Traditionally, it is extracted as a dirty brown liquid by heating palm kernels in frying pan (Ekwenye and Ijeomah, 2005; Ugbogu *et al*., 2006) over firewood flames. This type of PKO is mixed with herbs in folklore medicine and used for the treatment of febrile seizures, infections and liniment for indolent tumours. The PKO extracted using the mechanical press method looks purer and has enjoyed wide industrial applications (Seth, 2004). Palm oil (PO) on the other hand is extracted from the fleshy mesocarp of the fruit of the tropical palm tree (Ugbogu *et al*., 2006). Wardlaw and Kessel (2002) reported that the major fatty acids in PKO are lauric acid (47.0%), myristic acid (16.4%), palmitic acid (8.1%) and oleic acid (11.4%). PO contains 43.5% palmitic acid, 36.6% oleic acid, 9.1% linoleic acid and 4.3% stearic acid (Wardlaw and Kessel, 2002). They also reported that PO contains β-carotene (a pro-vitamin A) while a tablespoonful of PKO or PO did not contain cholesterol.

The consumption of saturated oils, with low essential fatty acid contents like PKO, should be avoided or done in moderation in diet (Gopalan *et al*., 1992). The atherogenicity of PKO-containing diet has been reported by Ibegbulem and Chikezie (2012). It increased the serum concentrations of Low Density Lipoprotein (LDL) and decreased serum High Density Lipoprotein (HDL) levels, thereby increasing the total cholesterol/HDL and LDL/HDL ratios. Their PO-containing diet reversed the effects observed with the PKO-containing diet. The consumption of PO had been reported to cause drops in endogenous cholesterol levels (Pantazari and Ahmad, 2004). This confirmed PO as being non-atherogenic, as earlier reported by Gunstone *et al*. (1986). Diets high in saturated fat and cholesterol have also been reported to reduce LDL uptake by the liver cells (Wardlaw and Kessel, 2002). There have been limited reports on the possible effects of PKO-

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containing diets on organ weights, haematological and liver function indices.

Therefore, the present study examined the effects of PKO-containing diet on the body weight gain, feed and water intakes, organ weights, haematological parameters and liver function indices in male rats.

2. Materials and methods

2.1 Palm kernel oil, palm oil, feed, experimental rats and diagonistic kits

The refined PKO used in the study was purchased from a vegetable oil company located at Irete – Owerri, Nigeria. Fresh PO sample was purchased at the Nkwo-Ukwu Market, Ihiagwa, Nigeria. The unpelleted growers mash (guinea feed) was a product of Feeds and Flour Mill, Sapele, Nigeria. The twenty-four male albino rats (*Rattus norvegicus*) of the Wistar strain used as models in the study were obtained from the Animal Facility of the Department of Biochemistry, University of Port Harcourt, Port Harcourt, Nigeria. The assay kits were products of Randox® Laboratories Ltd., Co-Antrim, United Kingdom and BioSystems® S. A., Costa Brava, Bercelona, Spain. All the chemicals used were of analytical grade.

2.2 Analysis of oils and feeds

Acid value (AV), iodine value (IV) and saponification value (SV) of the oils were evaluated using the procedures described by AOAC (1990). Percentage rancidity and percentage unsaturation were calculated by multiplying the AV/ SV ratio by 100 and multiplying the IV/ SV ratio by 100 respectively. Analyses of the feeds for their proximate compositions were evaluated using the procedures of AOAC (1990). Their energy contents were computed as described by Wardlaw and Kessel (2002).

2.3 Formulation of experimental diet

Supplemental fat from PKO and PO were introduced and mixed, respectively, as concentrated energy sources at the oil (ml) to guinea feed (g) ratio of 1/10 as described by Ibegbulem and Chikezie (2012).

2.4 Rat feeding study

The experimental rats weighing 220 ± 5.3 g were randomly assigned into three groups of eight rats each and housed in wire-screened stainless steel cages with troughs for feed and water. They were placed under the same room temperature and circadian rhythm and acclimatized for four days. The test and control rats were placed on the oil treated and untreated guinea feeds, respectively, *ad libitum* for 35 days. The total feed consumed (TFCd) and total volume of water drunk (TVWDk) by each rat was also noted. All procedures were approved by the Ethics Committee of the Department of Biochemistry, Federal University of Technology, Owerri, Nigeria.

2.5 Collection of blood and preparation of sera

At the end of the study, each rat was re-weighed then anaesthetized in chloroform vapour. Incisions were made into the thoracic cavity and blood collected from the pumping heart using a 10 ml hypodermic syringe and needle. An aliquot (1.0 ml) of the blood was transferred into a sequestering bottle containing ethylene diamine tetra-acetic acid (EDTA) as anticoagulant. The remaining (5.0 ml) was allowed to clot in a sample vial at room temperature. The clotted blood samples were centrifuged at 3000 x *g* for 5 minutes and the serum recovered by aspiration with Pasteur pipette.

2.6 Determination of haematological parameters

The haematological parameters were evaluated as described for haemoglobin (Bain and Bates, 2002), packed cell volume (PCV), mean corpuscular haemoglobin concentration (MCHC) and white blood cell (WBC) count (Baker *et al*., 2001).

2.7 Determination of liver function indices

Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were evaluated using a diagnostic kit according to the Manufacturer's instructions while serum total bilirubin, total protein and albumin contents were evaluated according to the instruction

contained in the kit manual. Globulin concentration was computed as the difference between the serum total protein and albumin contents.

2.8 Weight gain and feed efficiency ratio

The total weight gained (TWGd) by the rat was calculated as the difference between the final body and initial body weights while the feed efficiency (FER) ratio was computed as the ratio of the TWGd/ TFCd.

2.9 Statistical analysis

Results were presented as mean ± SD of eight determinations. Data were analysed using the two-tailed student's t – distribution test of significance and one-way analysis of variance (ANOVA) at $p \leq 0.05$.

3. Results

The acid value, iodine value, percentage rancidity and percentage unsaturation of the PO were significantly ($p<0.05$) higher than those of the PKO. The saponification value of the PKO was, however, significantly ($p<0.05$) higher than that of PO (Table 1). The oils significantly ($p<0.05$) increased the crude protein, crude fat, crude ash, total carbohydrates and energy contents of the diets. The energy content of the palm oil-treated diet was, however, not significantly ($p>0.05$) higher than that of the untreated diet (Table 2). Furthermore, the oils did not significantly ($p>0.05$) reduce the total weight gained by the rats. It also shows that the total quantity of feed and volume of water consumed were also significantly ($p<0.05$) reduced whereas the feed efficiency ratios of the diets increased significantly (Table 3). The oil-treated diets did not significantly ($p>0.05$) affect the weights of the kidney, spleen, heart and liver of the animals as well as the levels of PCV, Hb, MCH and WBC of the animals (Table 5). The oil-treated diets significantly ($p<0.05$) increased the AST activity whereas the ALT activity was not altered significantly (Table 6). The AST/ ALT ratio and serum globulin levels increased significantly. The PKO-treated diet significantly ($p<0.05$) increased serum total protein levels whereas it decreased the serum total bilirubin, albumin and albumin/ globulin (A/G) ratio. The PO-treated diet did not significantly ($p>0.05$) affect the serum total bilirubin, total protein and albumin levels whereas the A/G ratio decreased significantly (Table 6).

Table 1: Some physicochemical properties of the oils[‡]

Parameters	Sample		Coconut oil[§]
	Palm kernel oil	Palm oil	
Acid value (mg KOH/ g fat)	34.67 ± 0.02^{a}	48.76 ± 0.02^{b}	2.5 – 6
Iodine value (mg KOH /g fat)	40.32 ± 0.04^{a}	81.22 ± 0.02^{b}	7 – 10
Saponification value (mg KOH/ g fat)	204.53 ± 0.03^{a}	188.27 ± 0.03^{b}	254 – 262
Rancidity (%)	16.95 ± 0.03^{a}	25.90 ± 0.01^{b}	0.98 – 2.29*
Unsaturation (%)	19.74 ± 0.02^{a}	43.14 ± 0.01^{b}	2.76 – 3.82*

[‡]Values are mean ± SD of triplicate determinations; Values on the same row bearing the same superscript letters are not significantly different ($p<0.05$); [§]Plummer (1971); *Computed.

Table 2: Proximate composition (%) of untreated, palm kernel oil- and palm oil-treated diets‡

Diet	Parameter					
	Moisture	Crude protein	Crude fat	Crude ash	Total carbohydrates	Energy content (kcal/ 100 g)
Untreated	3.41 ± 0.10[a]	13.32 ± 0.04[a]	11.58 ± 0.07[a]	6.08 ± 0.17[a]	65.62 ± 0.21[a]	419.94 ± 0.33[a]
PKO-treated	3.42 ± 0.12[a]	14.19 ± 0.14[b]	13.98 ± 0.09[b]	7.22 ± 0.17[b]	61.20 ± 0.39[b]	427.34 ± 0.24[b]
PO-treated	3.51 ± 0.10[a]	20.72 ± 0.16[c]	13.47 ± 0.10[c]	9.53 ± 0.11[c]	54.53 ± 2.12[c]	422.34 ± 6.99[a,b]

‡Values are mean SD of duplicate determinations.
Values on the same column bearing the same superscript letters are not significantly different (p>0.05).

Table 3: Effects of palm kernel oil- and palm oil-based diets on feed efficiency ratio‡

Group	Parameters			
	TWGd (g)	TFCd (g)	TVWDk (ml)	FER
Control	54.50 ± 9.89[a]	384.00 ± 61.87[b]	405.00 ± 5.70[c]	0.14 ± 0.07[d]
PKO	58.77 ± 8.78[a]	281.09 ± 9.69[c]	315.00 ± 8.20[d]	0.21 ± 0.11[f]
PO	52.48 ± 5.36[a]	265.43 ± 7.70[c]	310.15 ± 6.34[d]	0.20 ± 0.08[f]

‡Values are mean ± SD of eight observations.
PKO = palm kernel oil; PO = palm oil; TWGd = total weight gained; TFCd = total feed consumed; TVWDk = total volume of water drunk; FER = feed efficiency ratio.
Values on the same column bearing the same superscript letters are not significantly different (p>0.05).

Table 4: Effects of palm kernel oil- and palm oil-based diets on organ weights‡

Group	Weight of Organs (g)			
	Kidneys	Spleen	Heart	Liver
Control	0.93 ± 0.04[a]	0.43 ± 0.07[b]	0.35 ± 0.03[c]	4.66 ± 0.29[d]
PKO	0.93 ± 0.37[a]	0.40 ± 0.06[b]	0.40 ± 0.06[c]	4.82 ± 0.37[d]
PO	0.95 ± 0.08[a]	0.44 ± 0.04[b]	0.41 ± 0.04[c]	4.79 ± 0.30[d]

‡Values are mean ± SD of eight observations; PKO = palm kernel oil; PO = palm oil.
Values on the same column bearing the same superscript letters are not significantly different (p>0.05).

Table 5: Effects of palm kernel oil- and palm oil-based diets on haematological parameters‡

Group	Parameters			
	PCV (%)	Hb (g/ dl)	MCHC	WBC (number/ L)
Control	34.66 ± 1.69[a]	9.00 ± 0.45[b]	0.26±0.14[c]	4900.00 ± 200.00[d]
PKO	31.33 ± 1.24[a]	9.73 ± 0.74[b]	0.31±0.12[c]	4533.00 ± 207.00[d]
PO	31.00 ± 5.35[a]	7.53 ± 0.74[b]	0.24±0.09[c]	4883.00 ± 1003.60[d]

‡Values are mean ± SD of eight observations; PCV = packed cell volume; Hb = haemoglobin; MCHC = mean corpuscular cell haemoglobin; WBC = white blood cell; PKO = palm kernel oil; PO = palm oil.
Values on the same column bearing the same superscript letters are not significantly different (p>0.05).

Table 6: Effects of palm kernel oil- and palm oil-based diets on liver function parameters‡

Group	Parameter							
	AST (U/L)	ALT (U/L)	AST/ ALT ratio	Total bilirubin (mg/ dL)	Total protein (g/ L)	Albumin (g/ L)	Globulin (g/L)	A/G ratio
Control	20.50 ± 0.82[a]	22.75 ± 1.75[a]	0.90 ± 0.13[a]	1.11 ± 0.00[a]	56.05 ± 3.30[a]	35.50 ± 0.89[a]	20.55 ± 2.38[a]	1.73 ± 0.36[a]
PKO	26.50 ± 0.50[b]	26.50 ± 8.50[a]	1.00 ±0.14[b]	0.88 ± 0.11[b]	59.40 ± 0.00[b]	25.20 ± 0.00[b]	34.20 ± 0.00[b]	0.74 ± 0.00[b]
PO	27.33 ± 6.54[b]	27.50 ± 6.54[a]	0.99 ±0.15[b]	1.19 ± 0.18[a]	66.00 ± 0.00[a]	36.13 ± 2.40[a]	29.87 ± 0.75[c]	1.21 ± 0.30[c]

‡Results are mean ± SD of eight observations; AST = aspartate aminotransferase; ALT = alanine aminotransferase, A = albumin; G = globulin; PKO = palm kernel oil; PO = palm oil.
Values on the same column bearing the same superscript letters are not significantly different ($p>0.05$).

4. Discussion

The PO used in this study was more rancid than the PKO (Table 1). The percentage occurrence of double bonds per average fatty acid chain length was also higher in the PO. This indicated that PKO was a more saturated fat. Though the percentage rancidity of the oils was low, the assay method used for measuring acid value may not be a good analytical tool. This is because the base used in such assay may also react, and neutralize, organic acids in oils thereby increasing the titre value. Atawodi *et al*. (2007) reported the presence of ρ - hydroxybenzoic acid, vanillic, syringic and ferulic acids in red PO. Their levels of unsaturated fatty acids compared favourably with those reported for PO and PKO (Wardlaw and Kessel, 2002). Saponification and iodine values of oil are inherent property, while free fatty acid level or acid value largely depends on the state of its rancidity. The acid and iodine values of the oils were higher than those of coconut oil while their saponification values were lower than that of coconut oil (Table 1). It meant that the oils were more rancid and contained more mono- and polyunsaturated fatty acids than the coconut oil.

The higher energy values of the oil-treated diets (Table 2) may have promoted satiety and improved the nutrient adequacy (the FER) of the diets (Table 3). While the reduced feed intake may have been compensated for by the concentrated energy in the oils, the reduced water intake may have been compensated for by the high turnover of metabolic water produced from fat oxidation (Church and Pond, 1988). Palm oil- and PKO-containing diets are known to be of higher energy values than normal diets (Akpanabiatu and Edem, 2006). Ibegbulem and Chikezie (2012) also reported that the energy contents of their diets were increased by 19.40 to 20.03 %.

When the PKO- and PO-treated diets were administered to the rats, the organ weights (markers of their pathological conditions) and the haematological indices were not affected (Tables 4 and 5). The oil-treated diets, however, caused hepatocellular and/ or cardiocellular damage (Table 6). The AST activities of the test rats may have indicated cardiocellular damage, because higher levels were found in the heart and thus suggest pathology of the heart (Hawcrot, 1987; Stroev and Makarova, 1989). The AST/ALT ratio of the rats placed on the PKO-treated diets, however, suggest no hepatocellular and/ or cardiocellular damage. On the other hand, the AST/ALT ratio of the rats maintained on the PO-treated diet indicated hepatocellular damage. Higher ALT levels were found in the liver and were associated with hepatocellular damage (Hawcrot, 1987; Stroev and Makarova, 1989). The tissue damage may have occurred due to the rancid states of the oils. The decrease in serum total bilirubin of rats placed on PKO-treated diet may have been due to increased excretion of bilirubin by the liver or reduced haemolysis of red blood cells by the spleen. However, the Hb level was not affected by the PKO (Table 5). The ability of the liver to synthesize proteins was also affected by the oil-containing diets. Synthesis of serum albumin in

the rats that were placed on the PKO-treated diet may have been decreased probably due to enhanced synthesis of serum globulins.

Palm kernel oil has a high saturated fatty acid (SFA) content (Pantazari and Ahmad, 2004) that may reduce the receptor-mediated uptake of LDL by the liver. It also has a low polyunsaturated fatty acid (PUFA)/ SFA ratio (Wardlaw and Kessel, 2002). These may promote increased serum total cholesterol content (Chaney, 2006). Both factors may then decrease cellular uptake of serum LDL and total cholesterol thereby increasing the time required to clear them from blood. Ibegbulem and Chikezie (2012) reported high serum LDL levels in the rats placed on PKO-treated diets. Increase in blood lipids may also account for the elevated serum globulin concentration, as observed in the present study, leading to a reduction or reversal of the albumin/globulin (A/G) ratio. Serum albumin concentrations are normally higher than globulin (Mathotra, 1989; Devlin, 2006), thereby giving an A/G ratio that is always greater than unity. Mathotra (1989) reported that the ratio was 2 to 1; with alteration and reversal occurring due to reduction in serum albumin and/ or elevation in serum globulin. The PO-treated diet reduced the A/G ratio while the PKO-treated diet reversed it by more than two-folds (Table 6). The serum A/G ratios of the control, PKO- and PO-treated-diet rats were effectively 1.73:1, 0.74:1 and 1.21:1, respectively. Serum albumin levels decrease in cirrhosis (with increase in serum cholesterol), nephritic syndrome, malnutrition and malignancies (Mathotra, 1989; Neale, 1997) and the A/G ratio are normally reversed in cases of cirrhosis with jaundice (Mathotra, 1989). The experimental rats placed on PKO-treated diets could not have developed cirrhosis, as it occurs after several years (Mathotra, 1989; Neale, 1997; Devlin, 2006). They could also not have been jaundiced because there was no accumulation of serum total bilirubin as shown in Table 6.

The increase in serum globulin on consumption of PO-treated diet buttressed the point that increases in dietary fat and/ or serum lipid contents could enhance serum globulin mediated blood lipid transport. The globulin types that were synthesized may have been more of the α- and β-globulin types, which are required for the transportation of lipids in serum, rather than the γ-globulins that are immunologically inclined. The test rats did not encounter any immunological challenge, as their total white blood cell counts were not significantly ($p>0.05$) affected (Table 4). More white blood cells are normally synthesized and mobilized when the immune system is challenged. A further study on the types of globulins synthesized is suggested. The lower serum globulin concentration of rats that were placed on the PO-treated diet, when compared with their PKO counterparts, also showed that increases in dietary unsaturated fatty acids and PUFA lead to reduced serum lipid clearance time. Palm oil contains less saturated fatty acids than PKO as confirmed in this study (Table 1) and reported by Wardlaw and Kessel (2002).

The serum total protein level of the oil-treated diets (Table 6) also showed that the integrity of the kidneys was not compromised as there was no case of hypoproteinemia. This supported the result of the organ's weight presented in Table 4.

In conclusion, the PKO-treated diet improved the nutrient adequacy of the diet, did not affect the organ weights and haematological parameters, but reversed the serum A/G ratio of the rats. The long term effect of consuming PKO as a vegetable oil may be the development of cirrhosis of the liver with increase in serum cholesterol contents, as it had been reported to be atherogenic.

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