



## Plasma lipid profile of Wistar albino rats fed palm oil-supplemented diets

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### ABSTRACT

The effect of palm oil-supplemented diet on plasma lipid profile of Wistar albino rats was investigated by feeding the experimental animals with 20% palm oil-supplemented diets for 12 weeks. The plasma levels of Total cholesterol (TC), Triglycerides (TG), Low-density lipoprotein (LDL), and High-density lipoprotein (HDL) were determined at 4-, 8-, and 12-week intervals. The results showed significant ( $p < 0.05$ ) increases in TC (from  $2.44 \pm 0.44$  to  $3.41 \pm 0.41$ ), TG (from  $1.72 \pm 0.51$  to  $2.46 \pm 0.19$ ), and LDL (from  $0.87 \pm 0.21$  to  $1.33 \pm 0.30$ ) at the 4<sup>th</sup> week. However, the level of HDL ( $0.97 \pm 0.06$ ) did not show significant difference from control value ( $0.87 \pm 0.21$ ). The TC/HDL ratio increased non-significantly from  $3.12 \pm 0.29$  to  $3.55 \pm 0.29$ . At the 12<sup>th</sup> week, significant decreases ( $p < 0.05$ ) were observed for TC ( $1.50 \pm 0.08$ ), LDL ( $0.28 \pm 0.06$ ), while no significant difference was noted for TG and HDL. The TC/HDL ratio decreased significantly ( $p < 0.05$ ) to  $2.56 \pm 0.13$ . Analysis of organ weights showed significant ( $p < 0.05$ ) increase for lungs and liver while no significant differences were noted for heart and kidneys at the 12<sup>th</sup> week of feeding the diets. The findings of the present study suggest that palm oil may be beneficial as its intake produces decreases in TC/HDL, a useful index for possible cardiovascular problems in individuals.

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**Keywords:** Palm oil, cardiovascular, HDL, LDL, Total cholesterol, Triglycerides.

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### INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death worldwide (Zhang et al., 1997a), with majority of these deaths in developing regions of the world such as sub-Saharan Africa. Elevated concentrations of plasma total cholesterol (TC) and low density lipoproteins (LDL) have proved to be among the risk factors in the development of CVD. Thus, foods that promote these conditions have been object of several investigations to ascertain their ability to enhance CVD.

Palm oil, a reddish oil, is obtained from the fruit of the oil palm (*Elais guineensis*). The oil palm originated from tropical Africa but is now also cultivated in Southeast Asia and South America. In these areas, the oil is commonly used in cooking (Ong and Goh, 2000). Palm oil has been shown to be rich in vitamins, antioxidants and other phytonutrients, in addition to an equal proportion of unsaturated and saturated fatty acids (Sundram, 1997). While red palm oil has been recognized as an excellent source of carotenes

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and tocopherols which serve as antioxidants (Ong and Goh, 2000, Kamisah et al., 2005, and Panasenko et al., 2000) and other nutrients, some questions had remained on its use due to the high content of saturated fat (Keys et al., 1986; Ng et al., 1991; Hornstra et al., 1991; Ghafoorunissa et al., 1995; Choudhry et al., 1997). Some studies reported increased plasma total cholesterol and lowered low-density lipoprotein cholesterol following high intake of palmitic acid, a major component of palm oil (Denke and Grundy, 1992; Osim et al., 1996), while others reported decreases in HDL-C, LDL-C and TC (Chen and Fad, 1992; Kamisah et al., 2005; Adedeji et al., 2008). Other studies showed hypocholesterolemic effect of intake of other components of palm oil, the polyunsaturated fatty acids such as linoleic and linolenic acids (Purushothama et al., 1994). No increase in total cholesterol was observed in rabbits fed 0.1% cholesterol diet (Kritchevsky et al., 2000) while increases on total cholesterol and high density lipoprotein were reported in rabbits (Kamsiah and Nafeeza, 1997). Palm oil, as part of a low-fat diet (<30% energy) was shown to be effective in maintaining desirable plasma cholesterol and lipoprotein cholesterol levels (Sundram, 1990). In addition, a beneficial effect on the plasma lipid profile of rats was described by Kamisah et al., (2005). Many other studies provided support for positive contribution of palmitic acid, a major component of palm oil, to hypercholesterolemic effect (Denke and Grundy, 1992; Cuesta et al., 1998).

There are thus conflicting results of studies that investigated the role of dietary palm oil on lipid indices of experimental rats, and the objective of the present study was to evaluate the effect of dietary palm oil from Rivers State of Nigeria on the plasma lipid profile of the experimental animals with a view to ascertaining what role it could play in possible incidence of CVD in the area of study.

## MATERIALS AND METHODS

### Animals, diets and study plan

Weaning male Wistar albino rats (45.5-65.0 g) from the animal house of the Department of Biochemistry, University of Port Harcourt were used for this study. They were fed *ad libitum* with either commercial rat feeds (Top Feeds) or 20% palm oil-supplemented feed, and had free access to water. The rats were housed in large cages in groups of 6, and maintained in a well ventilated room. The standard guidelines for the use of experimental animals (including applying human actions during sacrifice) were adhered to.

The test diet was prepared manually by mixing 200 g of palm oil with 800 g of commercial rat feed. The mixed diets were kept at room temperature to dry overnight and stored in polyethylene bags.

### Collection of plasma samples

Blood was collected via cardiac puncture from four (4) experimental rats at zero-, 4-, 8, and 12-week intervals into heparinized screw-cap bottles with prompt mixing. The blood was then centrifuged at 10,000 rpm for 5 minutes and the supernatant plasma utilized for estimation of the various parameters investigated.

### Plasma lipid profile analysis

The Total Cholesterol (TC) in plasma was determined using the enzymatic end-point method of Siedel et al. (1983). Plasma Triglyceride (TG) was measured by the enzymatic colorimetric procedure based on the method of Tietz (1990) using commercial kit from Randox Laboratories. HDL-cholesterol was determined after the precipitation procedure of Jacobs et al. (1990). Plasma LDL was calculated applying the Friedwald equation (Friedwald et al. 1972).

### Vitamin analysis

The tintometric method of Pearson (1976) was applied to determine Vitamin A

while AOCS Official method (1986) was used for the measurement of Vitamin E in the palm oil sample.

### Chemical analysis

The free fatty acid (FFA), acid value, peroxide value and saponification value were determined by the AOCS Official method (1986).

### Statistical analysis

The results were analyzed using one-way ANOVA followed by student's t-test. Values of  $p < 0.05$  were considered significant.

### RESULTS

The concentrations of the different plasma lipid at zero-, 4-, 8- and 12-week of feeding the experimental animals with control and palm oil-supplemented diets are shown in Table 1. The Total cholesterol (TC) level showed initial increase at the 4<sup>th</sup> week but

decreased by the 12<sup>th</sup> week when compared with the control values. Similar pattern was obtained for Triglyceride (TG). However, the High density lipoprotein (HDL) level remained at about same level until the determination on the 12 weeks which was significantly lower than the control value. The same trend was observed for Low density lipoprotein (LDL).

The results obtained for body weight gain are shown in Table 2. There was progressive increase in body weight for both control and test groups and no difference between both groups at the various time intervals determined. The organ weights data (g/kg body wt) in Table 3 showed increasing values with increased duration of feeding with the test diets. The various chemical characteristics and vitamins A and E content of the palm oil sample used in this study are shown in Table 4.

**Table 1:** Plasma lipid concentrations of rats fed palm oil-supplemented diets for 4, 8 and 12 weeks.

Conditions		Parameters measured (mmol/l)				
		TC	TG	HDL	LDL	TC/HDL
0 week	Control	2.44±0.44	1.72±0.51	0.81±0.10	0.87±0.21	3.12±0.29
4 weeks	Control	2.57±0.20	1.63±0.12	0.91±0.04	0.93±0.25	2.84±0.28
	Test	3.41±0.41 <sup>a</sup>	2.46±0.18 <sup>a</sup>	0.97±0.06	1.33±0.30 <sup>a</sup>	3.55±0.29
8 weeks	Control	2.59±0.08	1.53±0.06	0.89±0.01	1.01±0.09	2.92±0.07
	Test	3.08±0.63 <sup>a</sup>	1.57±0.43	0.90±0.10	1.45±0.40	3.41±0.33
12 weeks	Control	2.63±0.13	1.80±0.05	0.85±0.05	0.96±0.12	3.11±0.17
	Test	1.50±0.08 <sup>a</sup>	1.93±0.67	0.59±0.05 <sup>a</sup>	0.28±0.06 <sup>a</sup>	2.56±0.13

Values represent mean ± SD for three determinations, and n=4. (TC: Total cholesterol, TG: Triglycerides, HDL: High density lipoprotein, LDL: Low density lipoprotein); and values denoted by superscript a are significantly different ( $p < 0.05$ ) from the control.

**Table 2:** Body weight determinations for rats fed palm oil-supplemented diets for 4, 8 and 12 weeks.

Condition	Body weight (g)			
	0 week	4 weeks	8 weeks	12 weeks
Control	49.25±1.75	77.50±16.58	108.75±16.52	180.00±22.73
+ Palm oil	50.00±1.50	76.67±10.60	107.80±15.60	181.20±19.89

Values are presented as mean ± SD for three determinations, and n=4.

**Table 3:** Organ weights (g/kg body wt) for rats fed palm oil-supplemented diets for 4, 8 and 12 weeks.

	0 week	4 weeks	8weeks	12 weeks
Lungs (g)	0.33±0.01	0.51±0.22	0.81±0.27	1.37±0.02
Heart (g)	0.18±0.01	0.24±0.02	0.32±0.05	0.31±0.03
Kidney (g)	0.30±0.01	0.39±0.03	0.61±0.03	0.93±0.02
Liver (g)	1.33±0.07	2.07±0.35	2.97±0.78	3.70±0.28

Values are presented as mean ± SD for three determinations, and n=4.

**Table 4:** Chemical characteristics and vitamin levels of palm oil sample used in study.

Parameters	Values
Saponification value (mgKOH/g)	205.00±10.49
Iodine value (Wijs)	55.00± 4.60
% Free fatty acids	22.02±4.50
Peroxide value (meq/kg)	17.95± 3.72
Vitamin A ( g/g)	650.10±18.53
Vitamin E (U/l)	97.19±4.89

Values represent mean ± SD of three determinations.

## DISCUSSION

In this study, body weight and organ weight data were similar between the control and test groups suggesting that the diets were adequate with similar food consumption by the two groups. An earlier report showed that when palm oil was used to replace a major part of other fats in traditional diets of Chinese male adults, it did not increase serum cholesterol or affect HDL (Zhang et al., 2003). Also, chronic consumption of high fat (20% w/w of palm oil) for 12 weeks did not affect both plasma TC and HDL (Kamisah et al., 2005). The TC level showed initial increase by the 4<sup>th</sup> week in our study, a finding that had been noted for rabbits fed 20% (w/w) red palm oil at 6- and 12-weeks of feeding (Kamsiah and Nafeza, 1997). This could be ascribed to the response of the test animals to the experimental diet during the short period of intake. However, at the end of week 12, our findings showed that the plasma TC of the test group was significantly lower when compared with the control, a finding that disagrees with earlier reports as regards plasma TC (Denke and Grundy, 1992; Osim et al., 1996). The present findings are in line

with the observation by Edem (2002) that although palm oil-based diets induced higher blood cholesterol than do corn and other oils, the prolonged consumption of palm oil caused the endogenous cholesterol level to drop. Furthermore, our findings support the report by Sulli et al. (1998) that the supplementation of diet with  $\alpha$ -tocopherol and  $\beta$ -carotene (major components of red palm oil) reduced plasma cholesterol in hypercholesterolemic rabbits after 8 weeks. Also, the finding of significant reduction in TC level disagrees with the observation by Osim et al. (1996) that increased TC but no difference in low and high density lipoproteins occurred with chronic consumption of 15% palm oil for 18 weeks.

Elevated plasma triglycerides (TG) had been implicated in the development of CVD and such elevation is associated with obesity, pro-inflammatory and pro-thrombic biomarkers and type II diabetes which predispose to CVD (Hodis et al., 1999). The plasma TG in this study decreased significantly in the test animals after 12 weeks of feeding with the test diet an effect considered beneficial to man in relation to risk

factor for coronary heart disease (Yarnell et al. 2001). Similar decreases had been reported by other workers (Kritchevsky et al., 2001; Kamisah et al., 2005).

There are conflicting reports on the effect of palm oil on LDL levels. Some workers observed increase (Cuesta et al. 1998; Mutalib et al., 1999; Mutalib et al., 2002), others report decrease (Zhang et al., 1997a; Zhang et al., 1997b), and yet others no effect (Bosch et al. 2002; van Jaasveld and Benade, 2002). In this report, initial increase in LDL was seen after 4 weeks but then a significant decrease occurred at the 12<sup>th</sup> week for test animals when compared with the control groups. Our findings agree with the reports by Kamisah et al (2005) and Karaji-Bani et al (2006).

The initial increase in LDL level in the 8<sup>th</sup> week declined to a significantly lower level than the control at the 12<sup>th</sup> week in this study, a finding that agrees with other workers (Kamisah et al., 2005; Karaji-Bani et al., 2006). However, other reports of increase in LDL had been made (Cuesta et al., 1998; Mutalib et al., 1999; Mutalib et al., 2002). Increased level of LDL had been documented to contribute to hyperlipidaemia, progression of atherosclerosis and cardiovascular disease (Lawn, 1992; Nwanjo and Oze, 2007). It is possible that the levels of Vitamins A and E in the palm oil used in the present study could be relevant in the observed decline in LDL level at the 12<sup>th</sup> week.

The HDL levels did not show any appreciable change over the period of the experiment, in agreement with other reports (Lawn, 1992; Zhang et al., 1997a & b; Kamisah et al., 2005; Adedeji et al., 2008). It had been reported that HDL- cholesterol does not contribute to atherosclerosis as it favours the delivery of cholesterol from peripheral sites to the liver for elimination (Lawn, 1992). The TC/HDL and LDL/HDL ratios correlate with cardiovascular diseases and are thus useful indices for atherogenicity (Kamisah et al., 2005; Oladipo et al., 2005). The TC/HDL ratio in the present study showed an initial rise by the 8<sup>th</sup> week but

declined by the 12<sup>th</sup> week. This reduction in TC/HDL ratio had been suggested to be due to high antioxidant level in the oils. Similar observations had been made by other workers (Zhang et al., 1997b; Kamisah et al., 2005). Our findings in the present study with palm oil grown in Rivers state, in the South-Eastern part of Nigeria supports the view that the oil has beneficial effect on the predisposition of sustained intake to the plasma lipid profile as it relates to TC levels. This occurrence could lend support to the view by some workers (Zang et al., 1997b; Zang et al., 2003; Lawn, 1992; Kamisah et al., 2005; Adedeji et al., 2008) that intake of palm oil does not contribute to the aetiology of cardiovascular disease, precipitated from alterations in plasma TC concentrations and TC/HDL ratio.

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