PJN
ISSN 1680-5194

PAKISTAN JOURNAL OF NUTRITION

ANSInet
308 Lasani Town, Sargodha Road, Faisalabad - Pakistan
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Pakistan Journal of Nutrition 4 (2): 89-96, 2005
ISSN 1680-5194

Chronic Intake of Red Palm Olein and Palm Olein Produce Beneficial Effects on Plasma Lipid Profile in Rats

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Abstract: Palm olein (PO) and red palm olein (RPO) are rich in tocopherols and tocotrienols. In addition, RPO also contains a high content of carotene. This study was to determine the effect of chronic intake of diets containing palm oils, varying in their vitamin E and carotene contents, on lipid profile in rats. Weaning male Wistar rats were fed either 18% RPO, 18% PO or 18% vitamin E-stripped palm olein (SPO) for 12 weeks. Plasma total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL) and low density lipoprotein cholesterol (LDL) were measured at weeks 4, 8 and 12. Feeding the different types of palm oil did not affect TC and HDL from week 4 through week 12, but there were reductions in TG in all dietary groups at week 12 compared to week 4 but differences between groups were not observed. The RPO group had lower LDL at week 12 (vs weeks 4 and 8) but LDL was not reduced in the PO and SPO groups. TC/HDL was reduced in the RPO group at week 12 compared to both weeks 4 and 8, but the PO group only reduced this ratio at week 12 compared to week 4. This finding suggests that chronic feeding of diets high in palm oils did not cause any detrimental effects on blood lipid profile. In addition, red palm olein which is rich in antioxidants in the forms of vitamin E and carotene, showed better effect in terms of reduction in LDL and TC/HDL.

Key words: Palm oil, tocotrienol, tocopherol, blood lipid profile

Introduction

Palm oil is obtained from a tropical plant, *Elaies guineensis* and commonly used as a cooking oil in Malaysia, as well as in many parts of the world (Ong and Goh, 2000). It has an equal proportion of unsaturated and saturated fatty acids content. Palmitic (44%, saturated) and oleic (39%, monounsaturated) acids are two major fatty acids present in the oil. It also contains stearic (5%, saturated), linoleic (10%, polyunsaturated), linolenic (0.3%, polyunsaturated), lauric (0.1%, saturated) and myristic (0.1%, saturated) acids (Cottrell, 1991). High intake of palmitic acid in the diet was reported to increase plasma total cholesterol and low density lipoprotein cholesterol (Denke and Grundy, 1992), whereas oleic acid has a neutral effect on plasma cholesterol (Grundy, 1994). On the other hand, polyunsaturated fatty acids (PUFA) such as linoleic and linolenic acids, have hypocholesterolemic properties (Purushothama *et al*., 1994). Despite its high content of palmitic acid, many studies so far have documented that palm oil intake showed comparable effect on lipid profile to groundnut oil (high in oleic and linoleic acids) (Ghafoorunissa *et al*., 1995) and high oleic sunflower oil (Choudhury *et al*., 1997).

The refined, bleached and deodorized (RBD) palm oil, palm olein, is rich in natural antioxidants ie tocopherols and tocotrienols (tocols). However, the unbleached palm oil, so-called red palm olein is also abundant of α- and β-carotenes, in addition to the higher content of the tocols (Ong and Goh, 2000). The tocols and carotenes have been shown to exhibit good antioxidant properties (Kamisah *et al*., 2000; Panasenko *et al*., 2000) and enhance immune response (Gu *et al*., 1997; Wood *et al*., 1999) in many studies. The tocols have also been reported to have antitumor properties (Nesaretnam *et al*., 1998) and to be antithrombotic possibly by increasing the ratio of the prostacyclin/thromboxane (Nolan *et al*., 1995). Unlike tocopherol, tocotrienol is claimed as a potent inhibitor of 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase, a rate-limiting enzyme in cholesterol biosynthesis and thus, lowers serum cholesterol (Raederstorff *et al*., 2002).

Both crude and refined palm oils were shown to reduce total and low density lipoprotein cholesterols, as well as the ratio of total to high density lipoprotein cholesterols (Niyongabo *et al*., 1999; Zhang *et al*., 1997a). Supplementation of both α-tocopherol and β-carotene showed a beneficial effect on tissue cholesterol content and development of atherosclerotic lesions in rabbits fed an atherogenic diet for 8 weeks (Sulli *et al*., 1998). A

study by Kritchevsky *et al.* (2000) demonstrated that red palm olein was significantly less atherogenic than the RBD palm oil, supporting the hypothesis that carotenoids and vitamin E in the palm oil might protect against atherosclerosis, even though both edible oils had similar effects on serum and liver lipids.

Therefore, this study was designed to investigate the effect of chronic intake of palm oil-containing diets, which vary in their vitamin E and carotene contents on lipid profile in rats, to determine whether the cholesterol-lowering effect of the oils is contributed by their antioxidant contents.

Materials and Methods

Animals, diets and study design: Weaning male Wistar rats (50-70 g) (Laboratory Animal Source Unit, Faculty of Medicine, Universiti Kebangsaan Malaysia) were either fed 18% red palm olein (RPO), palm olein (PO) or vitamin E-stripped palm olein (SPO) diet for 12 weeks. The rats were given free access to food and water. They were housed individually in polyethylene cages with mesh wire bottom. The rats were fasted overnight before blood was withdrawn via periorbital vein under ether anaesthesia, at weeks 4, 8 and 12 for plasma lipid profile determination. The experimental and animal handling procedures were approved by the Research Committee of Faculty of Medicine, Universiti Kebangsaan Malaysia.

Diets were prepared manually in the laboratory and mixed thoroughly using a mixer, and thereafter were pelleted. The diets were then left to dryness at room temperature overnight and stored at –20°C. Red palm olein was obtained from Malaysian Palm Oil Promotion Council (MPOPC) and palm olein (Cap Buruh brand) was obtained commercially. Individual components of the diet was purchased either from United States Biochemicals (Cleveland, OH, USA) or Sigma Chemical Co. (St. Louis, MO, USA). The composition of the diets was tabulated in Table 1. The source of vitamin E and carotene in the diets was solely from the added palm oils.

Table 1: Composition of the experimental diets (g/kg diet)

Component	SPO	PO	RPO
Sucrose	350	350	350
Vitamin-free casein	200	200	200
Corn starch	150	150	150
Cellulose	50	50	50
[a]Salt mixture	35	35	35
[b]Vitamin E-free vitamin mixture (AIN 76)	10	10	10
[c]Vitamin E-stripped corn oil	20	20	20
DL-Methionine	3	3	3
Choline bitartrate	2	2	2
[c]Vitamin E-stripped palm olein	180	-	-
Palm olein	-	180	-
Red palm olein	-	-	180

SPO, stripped palm olein; PO, palm olein; RPO, red palm olein.

[a]Containing (%): $(NH_4)_6Mo_7O_{24}.4H_2O$, 0.003; $CaCO_3$, 29.3; $CaHPO_4.2H_2O$, 0.43; $CuSO_4$, 0.156; $FeC_6H_5O_7$, 0.632; $MgSO_4.7H_2O$, 9.98; $MnSO_4.H_2O$, 0.121; KI, 0.0005; KH_2PO_4, 34.31; NaCl, 25.06; Na_2SeO_3, 0.002 and $ZnCl_2$, 0.02.

[b]Containing (g/kg): thiamine HCl, 0.6; riboflavin, 06; nicotinic acid, 3; calcium pantothenate, 1.6; pyridoxine HCl, 0.7; folic acid 0.2; biotin, 0.02; vitamin B-12, 0.001; retinyl palmitate, 1.6 (250,000 IU/g); Vitamin D_3, 0.25 (400,000 IU/g); vitamin K_2, 0.005 and finely powdered sucrose, 992.9.

[c]Vitamin E-stripped palm olein and vitamin E-stripped corn oil were prepared following the method of Gapor *et al* (1989).

Plasma lipid profile analysis: Plasma total cholesterol (TC), triglyceride (TG) and high density lipoprotein cholesterol (HDL) were determined by enzymatic technique using commercial kits (Boehringer Mannheim GmbH, Germany). Plasma HDL was analysed after sodium phosphotungstate-Mg^{2+} precipitation of apoB and apoE containing lipoproteins, after which the HDL content of the supernatant was measured by the same cholesterol enzymatic kit. Plasma low density lipoprotein cholesterol (LDL) was calculated from TC, HDL and TG values using the Friedwald equation (Friedwald *et al.*, 1972).

Analysis of vitamin E: Vitamin E in the oils, diet and liver were extracted as previously described (Podda *et al.*, 1996) with some modifications. Briefly, 100 mg sample were homogenized in a tube containing 50 µl ethanolic butylated hydroxytoluene (10 mg/ml) and 1 ml distilled water. One ml of sodium dodecyl sulfate (0.1 M) was then added into the homogenates. After addition of 1 ml ethanol, the homogenates were extracted with 3 ml hexane. An appropriate aliquot was dried down using vacuum concentrater (Heto Lab Equipment, Denmark) and reconstituted in hexane.

The vitamin E in hexane lipid extract (20 µl sample) was analysed using an analytical high performance liquid chromatography (HPLC; Gilson 714). The stationary phase was a 250 mm Spherisorb 5 silica normal phase column, internal diameter 4.6 mm and particle size 5 µm, protected by a guard column (2 mm x 4.6 id mm). The mobile phase was hexane : isopropanol (99:1) at a flow rate of 1.5 ml/min. The column effluent was monitored with a fluorescence detector (Spectra System FL2000), set at 295 nm (excitation wavelength) and 330 nm (emission wavelength).

Statistical analysis: Results are expressed as mean with their standard error. Statistical analysis was performed by one way ANOVA followed by Tukey's multiple comparison test. Values of $P<0.05$ were

Table 2: Vitamin E content (mg/kg) in palm oils

	Oil type		
	Stripped palm olein (SPO)	Palm olein (PO)	Red palm olein (RPO)
α-Tocopherol	ND	124.00±0.46#	257.20± 7.00¶
α-Tocotrienol	ND	123.20±0.72#	199.90±0.42¶
γ-Tocopherol	ND	0.34±0.02*	0.37±0.02*
γ-Tocotrienol	ND	97.21±0.55#	148.40±0.63¶
δ-Tocotrienol	ND	86.13±0.97#	106.50±1.79¶
Total	0.00±0.00	430.88±1.65#	712.37±15.24¶

Values are mean ± standard error (n=6). *Significantly different from SPO group (P<0.05). #Significantly different from SPO and RPO groups (P<0.05). ¶Significantly different from SPO and PO groups (P<0.05). ND; not detectable.

Table 3: Vitamin E content (mg/kg) in different types of palm oil-containing diet

	Dietary		
	Stripped palm olein (SPO)	Palm olein (PO)	Red palm olein (RPO)
α-Tocopherol	ND	22.95±1.53#	39.98±2.11¶
α-Tocotrienol	ND	23.81±1.58#	30.61±1.93¶
γ-Tocopherol	ND	0.06±0.02*	0.06±0.01*
γ-Tocotrienol	0.26±0.14	20.49±0.52#	23.78±0.98¶
δ-Tocotrienol	0.33±0.14	17.48±0.40#	18.65±1.06¶
Total	0.59±0.12	84.79±3.74#	113.08±5.52¶

Values are mean ± standard error (n=6). *Significantly different from SPO group (P<0.05). #Significantly different from SPO and RPO groups (P<0.05). ¶Significantly different from SPO and PO groups (P<0.05). ND; not detectable.

considered statistically significant. All statistical analyses were performed using GraphPad Prism 2.1 software (1997; GraphPad Software Inc., San Diego, CA, USA).

Results

Tocols levels in the oils and diets: Five vitamin E isomers were detected in the palm oils i.e. α- and γ-tocopherols, as well as α-, γ- and δ-tocotrienols (Table 2). Overall, red palm olein had the highest content of vitamin E, whilst stripped palm olein was shown to contain very little vitamin E. α-Tocopherol was the major component of vitamin E in both red palm olein and palm olein, followed by α-, γ- and δ-tocotrienols. The tocotrienols represented 64% and 71% of the total vitamin E in the red palm olein and palm olein respectively.

Table 3 shows vitamin E content in the synthetic diet that had been prepared. As expected, red palm olein diet (RPO) had the highest content of vitamin E, followed by palm olein diet (PO), whereas almost completely absent in stripped palm olein (SPO).

Plasma lipid profile: The results of plasma lipid profile determined at three time intervals (weeks 4, 8 and 12) in rats that were fed different types of palm oil is shown in Fig. 1. Total cholesterol (TC) and high density lipoprotein cholesterol (HDL) levels were unaffected in plasma rats in all three groups throughout the study. However, plasma triglyceride (TG) was decreased in all treatment groups at week 12, as compared to week 4, but no differences were seen between the groups.

Feeding palm olein (PO) and stripped palm olein (SPO) diets did not influence rat plasma low density lipoprotein cholesterol (LDL) from week 4 through week 12. Nevertheless, feeding 18% red palm olein for 12 weeks did reduce this lipoprotein level compared to weeks 4 and 8, but was not significantly different from the LDL levels of PO and SPO (at week 12).

Both dietary PO and RPO reduced TC/HDL after 12 weeks of treatment compared to the ratio calculated at week 4. However, reduction by dietary RPO was significantly different compared to both weeks 4 and 8, compared to dietary PO (Fig. 2).

Hepatic vitamin E content: Only three vitamin E isomers i.e. α-tocopherol, α- and γ-tocotrienols were detected in the liver after 12 weeks of treatment (Fig. 3). All treatments resulted in significant increases in hepatic vitamin E (α-tocopherol, α- and γ-tocotrienols) content. More than 90% of the vitamin E detected was in the form of α-tocopherol, whilst the rest (10%) was α- and γ-tocotrienols.

Discussion

Vitamin E analysis done on the tested oils confirmed that red palm olein (RPO) contained a higher amount of vitamin E than palm olein (PO). α-Tocopherol was the

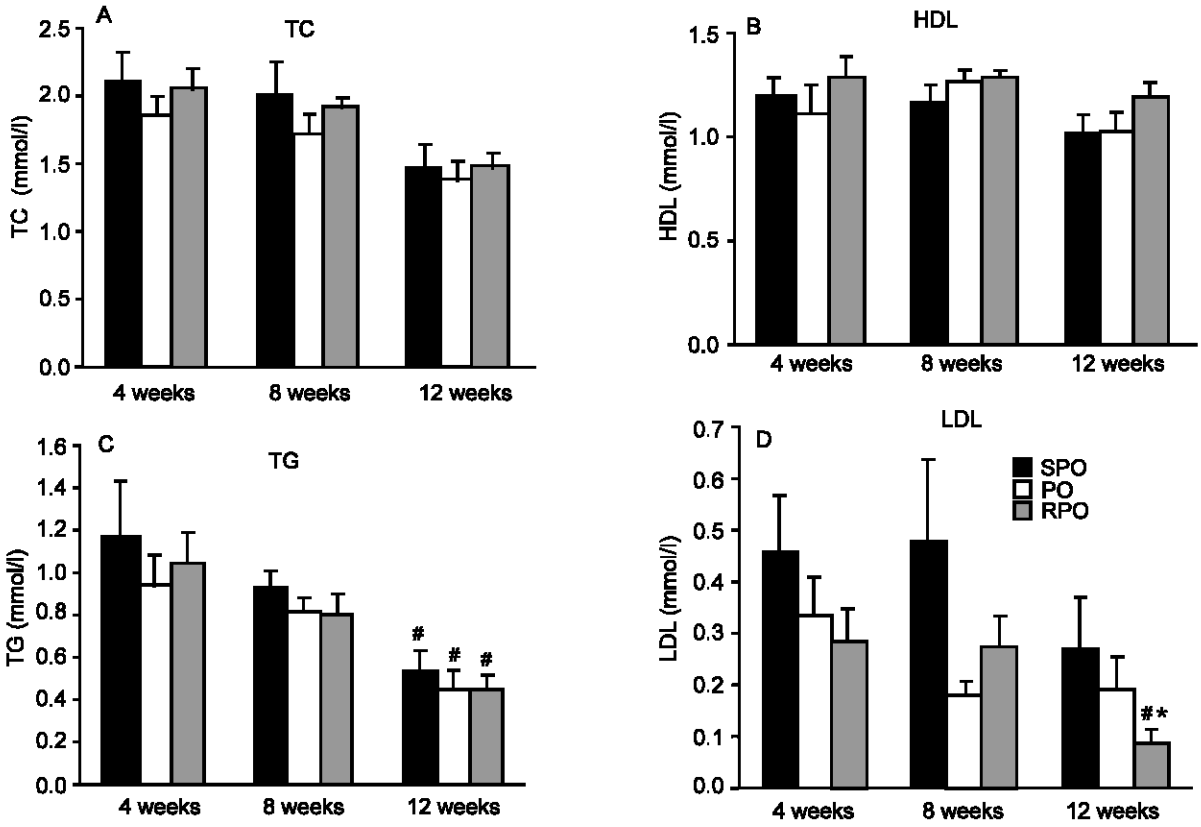

Fig. 1: Plasma lipid profile (mmol/L) in rats fed different type of palm oil (18%) for 4, 8 and 12 weeks. Bars are mean ± SEM (n=8-10). (A) Total cholesterol levels. (B) High density lipoprotein cholesterol levels. (C) Triglyceride levels. (D) Low density lipoprotein cholesterol levels. #Significantly different compared to week 4 ($P<0.05$). *Significantly different compared to week 8 ($P<0.05$).

major isomer detected for tocopherol in the palm oils (PO and RPO). In the diets prepared, both PO and RPO diets showed similar patterns of vitamin E content to the oils, with the RPO diet having the highest level of total vitamin E as compared to both PO and SPO diets, with the SPO diet containing little vitamin E.

Body weight and weight gain showed that they were similar for all diet groups (data not shown), indicating that diets were adequate and food consumption was similar. Chronic feeding of high fat (20% w/w) palm oil for 12 weeks did not affect both plasma total cholesterol (TC) and high density lipoprotein cholesterol (HDL). There is possibility that a significant decrease in TC may be seen with a longer treatment duration.

All the tested oils had a similar fatty acid composition but varied in their antioxidant levels. Higher contents of both vitamin E and carotenes in RPO did not have any further influence on TC and HDL. However, in contrast, Sulli *et al.* (1998) showed both α-tocopherol and β-carotene supplementation reduced plasma cholesterol in hypercholesterolemic rabbits after 8 weeks. This may be due to the higher dosage of α-tocopherol (5000 mg/kg diet) and β-carotene (25 mg/kg body weight, intravenously) used in the study, as compared to our study (113 mg vitamin E /kg diet). β-Carotene level was not measured in this study, but Malaysian red palm olein has been reported to contain about 500 mg/kg carotenoids, with approximately 250 mg/kg β-carotene and 200 mg/kg α-carotene (Kritchevsky *et al.* 2000). Thus, the RPO diet may contain about 80 mg carotenes per kg diet.

Many reports have been published about the effect of palm oil on cholesterol levels. Osim *et al.* (1996) claimed that chronic consumption of a diet containing 15% palm oil for 18 weeks increased total cholesterol but no differences were seen in low and high density lipoprotein cholesterols compared to the control group. However, many other studies have demonstrated that when palm oil was used to replace a major part of other fats in a traditional diet, it did not increase serum cholesterol or affect HDL (Zhang et al., 1997a; Zhang et al., 1997b).

Red palm oil (14% diet) was reported not to elevate TC in rabbits fed low cholesterol diet (0.1%), but increased the TC level when the animals were fed a higher percentage of cholesterol (0.2%), compared to refined,

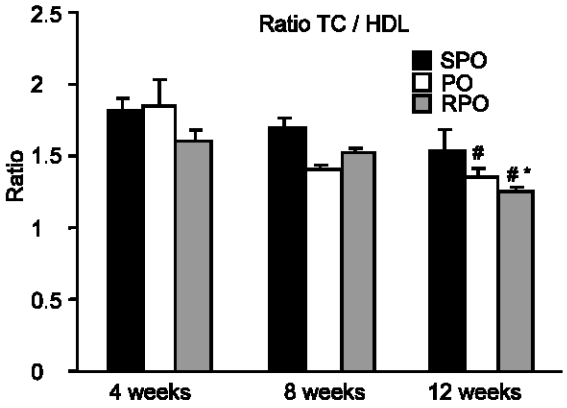

Fig. 2: The ratio of TC/HDL in rats fed different dietary palm oils for 4, 8 and 12 weeks. Bars are mean ±SEM (n=8-10). #Significantly different compared to week 4 (P<0.05). *Significantly different compared to week 8 (P<0.05)

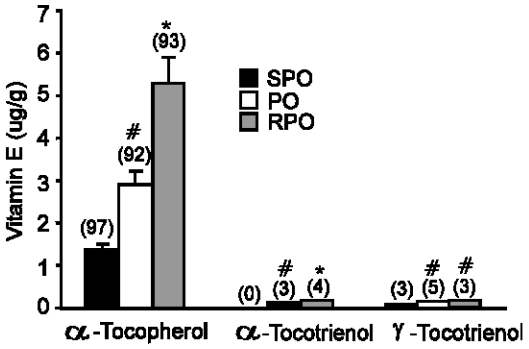

Fig. 3: Hepatic vitamin E isomers (α-tocopherol, α-tocotrienol and γ-tocotrienol) concentrations in rats fed different type of palm oil (18%) for 12 weeks. Bars are mean ± SEM (n=8-10). Numbers in parentheses indicate fractions (%) of the individual isomer detected from each group of treatment. #Significantly different compared to SPO group (P<0.05). *Significantly different compared to SPO and PO groups (P<0.05).

bleached and deodorized (RBD) palm oil. In the study, red palm oil-fed rabbits also had a significantly lower severity of thoracic aorta atherosclerosis as compared to RBD-palm oil group (Kritchevsky *et al.*, 2000). It was also demonstrated that RPO (20% w/w) raised serum TC and HDL in rabbits determined at weeks 6 and 12 of treatments, but the ratio of TC/HDL in this group was comparable with the control group after 12 weeks of study (Kamsiah and Nafeeza, 1997).

Palmitic acid (16:0) which is abundant in palm oil, has been claimed in many studies to contribute to hypercholesterolemic effect of the fat (Denke and Grundy, 1992; Cuesta *et al.*, 1998). However, in our study, the results obtained suggest that palmitic acid in the oil did not cause an increase in plasma TC. The atherogenic potential of the palmitic acid is attributed to the presence of the acid at sn-2 position. It has been reported that palm oil contains more than 40% palmitic acid, but the actual amount of the fatty acid at the sn-2 position is only 2.6% (Kritchevsky *et al.*, 2000). Fats containing palmitic acid at the sn-2 position are absorbed more completely than other fats (Tomarelli *et al.*, 1968) and triglycerides containing a saturated fatty acid at sn-2 are cleared more slowly from the circulation (Lien *et al.*, 1997). In the palm oil, palmitic acid is predominantly located at the sn-1 and sn-3 positions (Small, 1991), locations that are believed to be less hypercholesterolemic (Renaud *et al.*, 1995). This may explain the hypocholesterolemic effect of this oil. A few studies have reported that palmitic acid only becomes hypercholesterolemic in the presence of dietary cholesterol (Khosla and Hayes, 1993; Wijendran *et al.*, 2003) when LDL receptors are suppressed (Renaud *et al.*, 1995). Along with cholesterol intake, palmitic acid elevates plasma TC but does not affect plasma TG, HDL and TC/HDL, relative to 18:0-rich diet (Wijendran *et al.*, 2003). Furthermore, palm oil has only a minimal content of hypercholesterolemic intermediate chain fatty acids ie myristic (14:0) and lauric (12:0) acids, and none of atherogenic short chain fatty acids, caproic (6:0), caprilic (8:0) or capric (10:0) acids (Cottrell, 1991).

In contrast to TC and HDL, plasma triglyceride (TG) levels fell with the increasing duration of treatment in all groups, but significant reductions were only seen after 12 weeks of treatment as compared to week 4. Decreased plasma TG concentrations with palm oil diets are considered as beneficial since elevated TG levels constitute an independent risk factor for coronary heart disease in man (Yarnell *et al.*, 2001). No differences were observed in TG levels in all treatment groups at week 12, suggesting that this TG-lowering effect was not contributed by the antioxidant vitamins that were present in the PO or RPO. The effects of palm oils (PO and RPO as well as SPO) on plasma TG in this study was in agreement with another study which reported that rats consumed palm oil (10% fat) for 3 weeks had lower serum and liver TG than corn oil-fed group, but had no effect on TC. They observed liver TG levels fell with increasing dietary palmitic acid (Kritchevsky *et al.*, 2001).

Palm oil reportedly increased plasma TC, LDL and HDL concentrations but decreased TG level (ie both beneficial and detrimental effects) in Scottish volunteers. However, these changes were small considering the high proportion of palm oil in the diet (26% of energy) and were similar to those in the 'wild' group (consumed habitual diet only) which would have contained no more than 3% of energy as palm oil (Mutalib *et al.*, 1999). A study done by the same group discovered that palm oil-

enriched diets reduced pre-prandial plasma Lp(a) (Mutalib *et al.*, 2002), one of the major independent risk factors for heart disease (Sandkamp *et al.*, 1990).

In the present study, dietary RPO lowered plasma LDL concentration significantly after 12 weeks feeding compared to weeks 4 and 8. In the PO-fed group, a decreasing trend in LDL concentration was seen with increasing feeding period, but the changes were not significant. A higher content of bioactive components (tocopherol, tocotrienol and carotenoids) in RPO may play an important role in reducing the lipoprotein level. Several studies have observed a significant reduction (Zhang *et al.*, 1997a; Zhang *et al.*, 1997b) or rise (Cuesta *et al.*, 1998; Mutalib *et al.*, 1999) in plasma LDL with palm oil dietary intake, while others reported no such effect (Bosch *et al.*, 2002; van Jaarsveld and Benade, 2002). Though palm oil increased LDL concentrations, it afforded better protection of the LDL particles against lipoperoxidation (Cuesta *et al.*, 1998) and significantly reduced the risk for developing early lesions in peripheral arteries and aortas (van Jaarsveld *et al.*, 2002).

In our study, reductions in the TC and HDL ratio, which is a more useful index of atherogenicity, of the dietary PO (vs week 4) and RPO (vs weeks 4 and 8) observed after 12 weeks of treatment, were suggested to be attributed to the high antioxidant contents of the oils. Higher levels of vitamin E and carotenes in RPO afforded further reduction in the index. Similar effect of palm oil on the ratio was also reported by other investigators (Zhang *et al.*, 1997b). Previous studies have shown that dietary palm oil had comparable effect on TC/HDL ratio in normo- and hypercholesterolemic subjects (Zhang *et al.*, 1997a; Zhang *et al.*, 1997b), as well as in aged women (Cuesta *et al.*, 1998) to dietary peanut oil or oleic acid-rich sunflower, respectively.

α-Tocopherol was the major isomer detected in the liver after 12 weeks of dietary treatment. Although the PO and RPO diets had a quite similar proportion of tocotrienols to α-tocopherol, the concentrations of these individual tocotrienol isomers were not as high as of the α-tocopherol. One possible explanation for the lack of bioavailability of tocotrienol in vivo, may be due to poor absorption or rapid clearance from plasma (Fairus *et al.*, 2004). The latter is likely due to a low affinity of tocotrienols to hepatic α-tocopherol transfer protein (Hosomi *et al.*, 1997). Tocotrienol, especially the γ-tocotrienol possesses hypocholesterolemic activity (Raederstorff *et al.*, 2002), which may be attributable to posttranscriptional suppression of HMG CoA reductase, and a concomitant upregulation of the LDL receptor (Parker *et al.*, 1993). α-Tocopherol has been reported to attenuate the inhibitory effects of tocotrienols on HMG CoA reductase (Qureshi *et al.*, 1996). However, this effect of the α-tocopherol was not observed in this study. The reduction in LDL level as well as in TC/HDL ratio were the greatest in the RPO group, even though the group had the highest hepatic concentration of α-tocopherol. This suggests that α-tocopherol also have a role in lowering the plasma cholesterol level.

In conclusion, the results of the study suggest that the hypocholesterolemic effects of the palm oils are attributable to their balanced fatty acid composition and high content of antioxidant vitamins. Therefore, the effects that palm olein and red palm olein have on blood lipids is different and that palm oil should not be categorized as a saturated vegetable oil in the same group as coconut oil.

Acknowledgement

This study was funded by the Government of Malaysia through the Ministry of Science, Technology and Environment (IRPA Grant 03-07-03-0089).

References

Bosch, V., A. Anular, J. Medina, N. Ortiz and R. Apitz, 2002. Changes in plasma lipoproteins after the use of palm oil in the diet of a group healthy adults. Arch. Latinoam. Nutr., 52: 145-150.

Choudhury, N., A.S. Truswell and Y. McNeil, 1997. Comparison of plasma lipids and vitamin E in young and middle-aged subjects on potato crisps fried in palmolein and highly oleic sunflower oil. Ann. Nutr. Metab., 41: 137-148.

Cottrell, R.C., 1991. Introduction: nutritional aspects of palm oil. Am. J. Clin. Nutr., 53: 989S-1009S.

Cuesta, C., S. Rodenas, M.C. Merinero, S. Rodriguez-Gil and F.J. Sanchez-Muniz, 1998. Lipoprotein profiles and serum peroxide levels of aged women consuming palm olein or oleic acid-rich sunflower oil diets. Eur. J. Clin. Nutr., 52: 675-683.

Denke, M. and S.M. Grundy, 1992. Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins. Am. J. Clin. Nutr., 56: 895-898.

Fairus, S., M.N. Rosnah, H.M. Cheng and K. Sundram, 2004. Metabolic fate of palm tocotrienols in human postprandial plasma model. Asia Pac. J. Clin. Nutr., 13: S77.

Friedwald, W.T., R.I. Levy and D.S. Fredrickson, 1972. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin. Chem., 18: 499-502.

Gapor, M.T., A.S.H. Ong, A. Kato, H. Watanabe and T. Kawada, 1989. Antioxidant activities of palm vitamin E with a special reference to tocotrienols. Elaies, 1: 63-67.

Ghafoorunissa, S.A., V. Reddy and B. Sesikaran, 1995. Palmolein and groundnut oils have comparable effects on blood lipids and platelet aggregation in healthy Indian subjects. Lipids, 30: 1163-1169.

Grundy, S.M., 1994. Influence of stearic acid on cholesterol metabolism relative to other long-chain fatty acids. Am. J. Clin. Nutr., 60: 986S-990S.

Gu, J.Y., A. Tsujita, Y. Wakizono, K. Yamada and M. Sugano, 1997. Combined effects of sesamin with α-tocopherol or tocotrienols on lipid and immune indices in Brown-Norway rats. Nutr. Res., 17: 339-350.

Hosomi, A., M. Arita, Y. Sato, C. Kiyose, T. Ueda, O. Igarashi, H. Arai and K. Inoue, 1997. Affinity for α-tocopherol transfer protein as a determinant of the biological activities of vitamin E analogues. FEBS Lett., 409: 105-108.

Kamisah, Y., A. Adam, W.Z. Wan Ngah, A. Gapor, B.A.K. Khalid and A. Marzuki, 2000. Tocotrienol and tocopherol were protective against xanthine plus xanthine oxidase induced oxidative stress. Asia Pac. J. Pharmacol.14: 111-116.

Kamsiah, J. and M.I. Nafeeza, 1997. Evaluation of the effects of red palm oil on serum lipid profiles, lipid peroxidation and atherogenesis. Malays. J. Biochem. Mol. Biol., 1: 32-35.

Khosla, P. and K.C. Hayes, 1993. Dietary palmitic acid raises plasma low density lipoprotein cholesterol relative to oleic acid only at a high intake of cholesterol. Biochem. Biophys. Acta, 1210: 13-22.

Kritchevsky, D., S.A. Tepper and D.M. Klurfield, 2001. Serum and liver lipids in rats fed mixtures of corn and palm oils ± cholesterol. Nutr. Res., 21: 191-197.

Kritchevsky, D., S.A. Tepper, A. Kuksis, S. Wright and S.K. Czarnecki, 2000. Cholesterol vehicle in experimental atherosclerosis: Refined, Bleached, Deodorized (RBD) palm oil, randomized palm oil and red palm oil. Nutr. Res., 20: 887-892.

Lien, E.L., F.G. Boyle, R. Yuhas, R.M. Tomarelli and P. Quinlan, 1997. The effect of triglyceride positional on fatty acid absorption in rats. J. Ped. Gasteroenterol. Nutr., 25: 167-174.

Mutalib, M.S.A., H. Khaza'ai, H. Peace, P. Whiting and K.W.J. Wahle, 2002. Palm oil-enriched diets reduced plasma Lp(a) in volunteers abnormally high concentrations: involvement of decreased triglyceride-rich apo(a). Nutr. Res., 22: 769-784.

Mutalib, M.S.A., K.W.J. Wahle, G.G. Duthie, P. Whiting, H. Peace and A. Jenkinson, 1999. The effect of dietary palm oil, hydrogenated rape and soya oil on indices of coronary heart disease risk in healthy Scottish volunteers. Nutr. Res., 19: 335-348.

Nesaretnam, K., Stephens, R. Dils and P. Darbre, 1998. Tocotrienols inhibit the growth of human breast cancer cells irrespective of estrogen receptor status. Lipids, 33: 461-469.

Niyongabo, A., A. Youyou, C.L. Leger, B. Descomps, A. Ammouche and M. Bellal, 1999. Effects of dietary crude palm oil, fish oil and their association on cholesterol and lipoprotein contents in rats which could be beneficial in humans. Int. J. Vitam. Nutr. Res., 69: 330-306.

Nolan, M.R., S. Kennedy, W.J. Blanchflower and D.G. Kennedy, 1995. Lipid peroxidation, prostacyclin and thromboxane A2 in pigs depleted of vitamin E and selenium and supplemented with linseed oil. Br. J. Nutr., 74: 369-380.

Ong, A.S.H. and S.H. Goh, 2000. Palm oil: a healthful and cost-effective dietary component. Food Nutr. Bull., 23: 11-22.

Osim, E.E., D.U. Owu and K.M. Etta, 1996. Arterial pressure and lipid profile in rats following chronic ingestion of palm oil diets. Afr. J. Med. Sci., 25: 335-340.

Panasenko, O.M., V.S. Sharov, K. Briviba and H. Sies, 2000. Interaction of peroxynitrite with carotenoids in human low density lipoproteins. Arch. Biochem. Biophys., 373: 302-305.

Parker, R.A., B.C. Pearce, R.W. Clark, D.A. Gordon and J.J. Wright, 1993. Tocotrienols regulate cholesterol production in mammalian cells by posttranscriptional suppression of 3-hydroxy-3-methylglutaryl-coenzyme A reductase. J. Biol. Chem., 268: 11230-11238.

Podda, M., C. Weber, M.G. Traber and L. Packer, 1996. Simultaneous determination of tissue tocopherols, tocotrienols, ubiquinols and ubiquinones. J. Lipid Res., 37: 893-901.

Purushothama, S., K. Narasimhamurthy, P.L. Raina and K. Hariharan, 1994. A study of plasma and liver lipid profile of rats fed palm oil or safflower oil along with cholesterol. Nutr. Res., 14: 255-269.

Qureshi, A.A., B.C. Pearce, R.M. Nor, A. Gapor, D.M. Peterson and C.E. Elson, 1996. Dietary α-tocopherol attenuates the impact of γ-tocotrienol on hepatic 3-hydroxy-3-methylglutaryl coenzyme A reductase activity in chickens. J. Nutr., 126: 389–394.

Raederstorff, D.E., V. Elste, C. Aebischer and P. Weber, 2002. Effect of either γ-tocotrienol or a tocotrienol mixture on plasma lipid profile in hamster. Ann. Nutr. Metab., 46: 17-23.

Renaud, S.C., J.C. Ruf and D. Petithory, 1995. The positional distribution of fatty acids in palm olein and lard influences their biologic effects in rats. J. Nutr., 125: 229-237.

Sandkamp, M., H. Funke, H. Schulte, E. Kohler and G. Assmann, 1990. Lipoprotein (a) is an independent risk factor for myocardial infarction at young age. Clin. Chem., 36: 20-23.

Small, D.M., 1991. The effects of glyceride structure on absorption and metabolism. Annu. Rev. Nutr., 11: 413-434.

Sulli, K.C., J. Sun, D.W. Giraud, R.A Moxley and J.A. Driskell, 1998. Effects of β-carotene and α-tocopherol on the levels of tissue cholesterol and triglyceride in hypercholesterolemic rabbits. J. Nutr. Biochem., 9: 344-350.

Tomarelli, R.M., B.J. Meyer, J.R. Weaber and F.W. Bernhart, 1968. Effects of positional distribution on the absorption of fatty acids of human milk and infant formulas. J. Nutr., 95: 583-590.

van Jaarsveld, P.J. and A.J. Benade, 2002. Effect of palm olein oil in a moderate-fat diet on low density lipoprotein composition in non-human primates. Asia Pac. J. Clin. Nutr., 11: S416-S423.

van Jaarsveld, P.J., C.M. Smuts and A.J. Benade, 2002. Effect of palm olein oil in a moderate-fat diet on plasma lipoprotein profile and aortic atherosclerosis in non-human primates. Asia Pac. J. Clin. Nutr., 11: S424-S432.

Wijendran, V., A. Pronczuk, C. Bertoli and K.C. Hayes, 2003. Dietary trans-18:1 raises plasma triglycerides and VLDL cholesterol when replacing either 16:0 or 18:0 in gerbils. J. Nutr. Biochem., 14: 584-590.

Wood, S.M., C. Beckham, A. Yosioka, H. Darban and R.R. Watson, 1999. β-Carotene and selenium supplementation enhances immune response in aged humans. Int. Med., 2: 85-92.

Yarnell, J.W., C.C. Patterson, P.M. Sweetnam, H.F. Thomas, D. Bainton, P.C. Elwood, C.H. Bolton and N.E. Miller, 2001. Do total and high density lipoprotein cholesterol and triglycerides act independently in the prediction of ischemic heart disease? Ten-year follow-up of Caerphilly and Speedwell cohorts. Arterioscler. Thromb. Vasc. Biol., 21: 1340-1345.

Zhang, J., P. Wang, C.R. Wang, X.S. Chen and K. Ge, 1997a. Nonhypercholesterolemic effects of a palm oil diet in Chinese adults. J. Nutr., 127: 509S-513S.

Zhang, J., C.R. Wang, J.H. Dai, X.S. Chen and K. Ge, 1997b. Palm oil diet may benefit mildly hypercholesterolaemic Chinese adults. Asia Pac. J.Clin. Nutr., 6: 22-25.