Mal J Nutr 8(2) : 137-156, 2002

The Effect of Increased Consumption of Edible Palm Oil on the Nutritional Status, Lipid Profiles and Lipid Peroxidation Among Malaysian Aborigines

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ABSTRACT

This study was conducted to determine the effects of increased edible palm oil consumption on community health status in the aboriginal communities in Tual Post (treatment group) and Sinderut Post (control group), Kuala Lipis, Pahang. Nutritional status, blood pressure, lipid profiles, fasting blood glucose (FBG), vitamin E (alpha-tocopherol) levels and lipid peroxidation product (malonaldehyde) levels were taken as indicators of health status. This is a pre-and post-controlled community trial in which similar variables were measured in each group. Every family of 2–6 household members was given 2–5 kg cooking palm oil per month for a period of 18 months. All subjects were measured for height (cm), weight (kg) and waist-hip ratio (WHR). For calorie intake measurement, house-to-house interviews were conducted using 24-hour dietary recall method. Blood pressure, percent body fat, lipid profiles, namely total cholesterol, high density lipoprotein cholesterol, triglyceride and fasting blood glucose (FBG) were also measured. Vitamin E (alpha-tocopherol) levels and lipid peroxidation products (MDA) were also determined. There was a significant increase ($p<0.05$) in percent body fat (28.1%) and calorie intake (17.2%) following palm oil consumption. The proportion of fat intake as an energy source also increased from 4.6% to 33.9%. There was a reduction in the systolic blood pressure following consumption ($p<0.05$). However, diastolic blood pressure did not change. A significant decrease ($p<0.05$) was observed in total cholesterol, low density lipoprotein and triglyceride. No particular pattern in fasting blood glucose levels was observed among the indigenous inhabitants following palm oil consumption. There was a significant increase in alpha-tocopherol levels ($p<0.0001$) and a decrease in MDA levels ($p<0.0001$) following consumption. In conclusion, high consumption of edible palm oil for 18 months was found to be not harmful to health. For the Malaysian aborigines, it serves as a good source of fat and energy.

INTRODUCTION

Oil palm, *Elaeis guineensis*, is believed to have originated from West Guinea. It was brought to Malaya in the early 20th century and later palm oil became a major commodity and industry in Malaysia. Edible palm oil is extracted from the mesocarp of the oil palm fruit and is different from palm kernel oil in terms of fatty acid profile as well as its chemical and physical characteristics (Gurr & Harwood, 1991; Cottrell, 1991). In Malaysia and in many other countries, palm oil is in the form of palm olein.

Humans require fat for energy, growth and normal functions of the body. Palm oil is considered to be a good source of fat because it contains an equal distribution of saturated and unsaturated fatty acids comprising 44% palmitic acid, 5% stearic acid, 40% oleic acid, 10% linoleic acid (essential) and 0.4% alpha linolenic acid (essential). It also contains natural antioxidants such as tocotrienols which are important in the body defense system against free radicals (Esterbauer *et al*., 1991).

The first clinical trial on the effect of edible palm oil consumption on human lipid profiles was reported by Ahrens *et al*. (1957). Several studies followed this trial. In most of these studies, it was found that consumption of palm oil did not increase the total cholesterol (TC), low density lipoptotein (LDL) cholesterol and triglyceride (TG) but increased high density lipoprotein (HDL) cholesterol (Lim *et al*., 1988).

In Malaysia, community-based studies related to the effect of palm oil consumption are lacking. Most studies were conducted in animals or human volunteers. In this study, the aborigines were chosen as subjects because their palm oil consumption is still low. The aim of the study was to observe the effects of increased consumption of edible palm oil on the nutritional status, lipid profiles and lipid peroxidation among the aborigines in Tual Post, Kuala Lipis, Pahang.

MATERIALS AND METHODS

Background of study area

Tual Post, Kuala Lipis, Pahang

Tual Post is an aboriginal settlement located approximately 70 km from the town of Raub and 220 km from the city of Kuala Lumpur (Figure 1). The area is surrounded by jungle and has two villages located at the valley of Titiwangsa Dividing Range, which is approximately a three-hour journey by a four-wheel vehicle from Raub. The villages are lead by two ‘Batin’ and have a total population of 411 (224 males and 187 females). They are from the Semai (Senoi) tribe and most of them can speak Malay (the national language) in addition to their own language. Their main activities are farming such as planting cassava and hill rice, hunting, fishing and collecting jungle products such as rattan, ‘petai’, jungle fruits, medicinal herbs/roots, etc. They also hunt animals with blow-pipes, using poisoned darts and catch animals in traps. Most families earned less than RM 250 per month. Only a small percentage of the villagers have had any formal education. Most of them are animists by religion. Their houses are made of bamboos and wood with roofs made of rumbia or bertam leaves. Currently, the government has provided them with wooden houses. They get their water supply by using a gravity feed system. Due to limited water supply, the villagers also depend on a nearby river which unfortunately was recently polluted due to logging activities.

Sinderut Post, Kuala Lipis, Pahang.

Sinderut Post is an aboriginal settlement comprising 12 villages and 1,064 residents (505 females and 559 males). It is also located at the valley in the Titiwangsa Dividing Range, approximately

100 km from the town of Raub. This area can be accessed either by helicopter or by a four-wheeled vehicle. As in the case of Tual Post, there is no electricity in this area and communication is by means of the wireless. Their main activities are also farming, hunting, fishing and collecting jungle products.

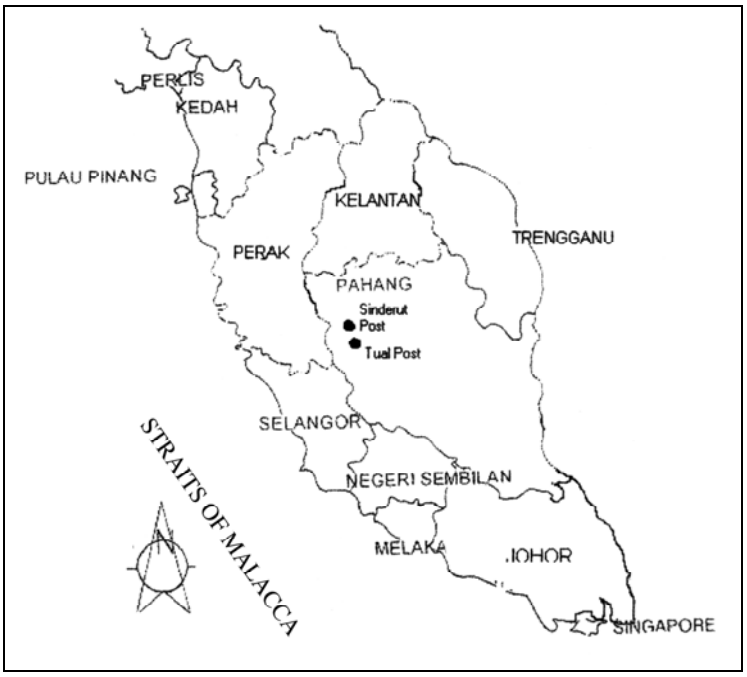

Figure 1. Map of Peninsular Malaysia showing study areas

Population and sampling

The study was approved by the Universiti Kebangsaan Malaysia research ethical committee and by the Department of Aboriginal Affairs, Malaysia. The selection of study areas was based on the pattern of consumption of edible palm oil and logistics – these criteria lead to the selection of Tual Post as the study area and Sinderut Post as the control.

All household members aged four years and above were interviewed for their socio-economic and nutritional status. A physical examination was conducted and basic anthropometric measurements (weight and height) were taken of every subject. A total of 10–20 ml blood samples were taken for the measurement of FBG, TC, HDL, LDL, TG, MDA and an antioxidant (alpha-tocopherol). The measurement of alpha-tocopherol levels and lipid peroxidation products (malonaldehyde) was conducted in the laboratory of the Biomedical Department, Faculty of Allied Health Sciences, Universiti Kebangsaan Malaysia, whilst the measurement of lipid profiles such as TC, TG, HDL, FBG and percent body fat was conducted at the clinic during each visit.

All villagers were invited to attend the clinic for examinations, during which time the edible palm oil was distributed to each family. There were altogether four study visits in the intervention group (Figure 2). As the control group, all villagers in Sinderut Post, aged four years and above, were invited to the clinic.

Study design

This study is a pre-and post-controlled community trial conducted at two settlements in Kuala Lipis, Pahang between April 1995 and September 1996. All members in the two communities had an equal opportunity to be involved in the study. The samples were all household members aged four years and above. Pregnant women and those who had recently migrated into the treatment group area were excluded from the study. All villagers who came to the clinic for baseline and subsequent examinations were selected as study subjects. A total of one baseline and three follow-up visits in the 3rd, 9th and 18th month of palm oil supplementation were conducted in the treatment group. Meanwhile, the control subjects were visited twice i.e. during the early and final stages of the study.

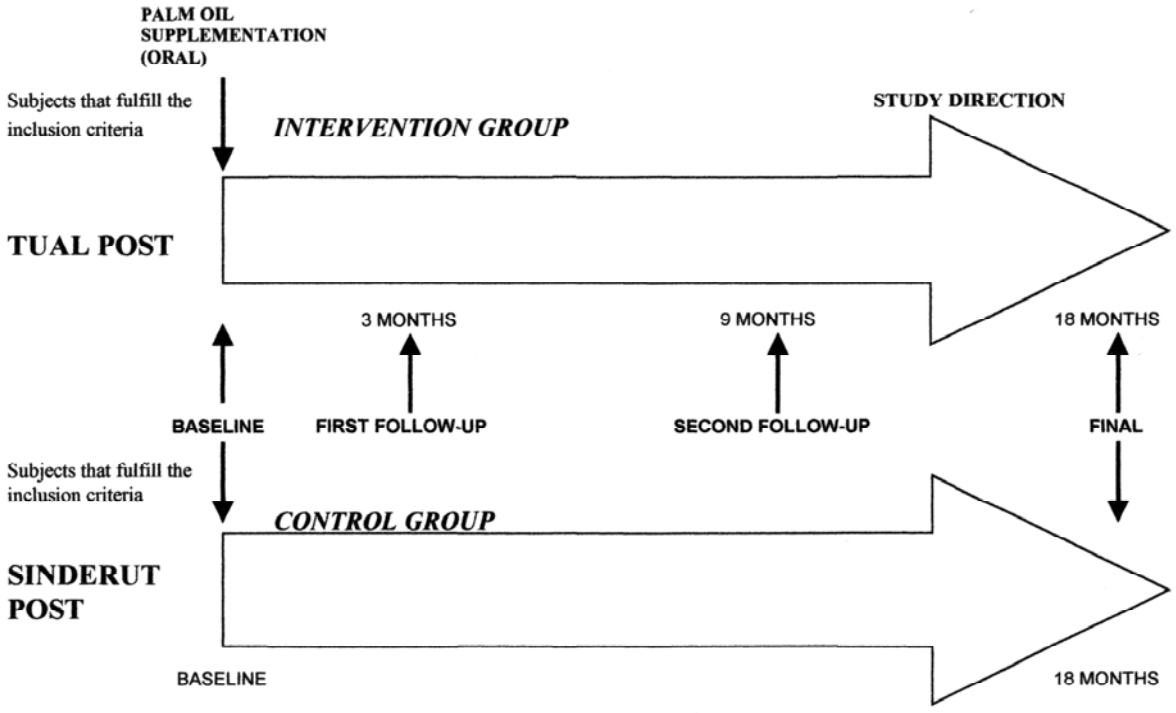

Figure 2. Palm oil intervention design

'Buruh' brand palm oil (Lam Soon (M) Ltd.) was supplied monthly to every family in the treatment group. A palm oil pack of 5 kg was given to households of four to six members whilst a palm oil pack of 2 kg was given to households with one to three members. During palm oil distribution, the remaining palm oil from the previous usage was measured to determine the amount consumed by the household and to observe subject compliance. The villagers were advised on the mode of consumption of the palm oil and also given cooking demonstrations.

Data collection

The study used questionnaires that had been tested and validated during the pilot study. The questionnaires were filled by four trained interviewers comprising two dietitians and two research assistants. Villagers were required to fast from 8:00 pm the previous day until blood samples were taken the next morning at the clinic.

Biochemical test

The levels of TC, TG, HDL and FBG were measured using Reflotron machine (Boehringer Mannheim) with Reflotron Precinom control. LDL levels were measured using the formula of Frieldweld, Levy & Frederickson (1972). Blood glucose levels were classified according to the WHO classsification (WHO, 1980). TC of less than 5.2 mmol/L is considered normal, 5.2 to 6.4 mmol/L as mild hypercholesterolemia and 6.5 mmol/L and above as high risk. HDL levels of higher than 0.91 mmol/L and TG levels of lower than 2.3 mmol/L were taken as normal. The levels of alpha-tocopherol-vitamin E were measured in duplicate using a modified method of Bieri (Bieri, Tolliver & Catagani, 1979) and classified according to Machlin classification (Machlin, 1990). Alpha-tocopherol levels were analyzed using HPLC, whilst the measurement of malonaldehyde (MDA) levels which is the lipid peroxidation product, was conducted using Shimadzu UV-160A Spectrophotometer based on the method by Pryor, Stanley & Blair (1976), Ledwozyw *et al*. (1986) and Janero & Burghardt (1989).

Clinical examination

Height was measured using Microtoise tool. A daily calibrated portable spring balance (SECA brand) was used to measure weight. Body mass index (BMI) was according to WHO classification (WHO, 1995). Percent body fat was measured using portable TANITA Bodyfat Analyzer model TBF-105, which is a patented 'foot pad' design that sends a low, safe electrical current through the body to measure its composition. Body composition is calculated mathematically based upon the speed at which the signal passes through the body. Waist-hip ratio (WHR) was measured using flexible tape based on the method by Jones *et al*. (1986) and classified according to Bjorntop (Bjorntop, 1987). Systolic and diastolic blood pressure and pulse rate were measured using digital BP set model HEM-400 of Omron brand. Blood pressure and pulse rate were taken from the left arm of the subjects while sitting. Measurements were taken three times with only mean values being recorded. Blood pressure classification method was based on WHO (WHO, 1978).

Dietary intake

Daily calorie intake was measured by 24-hour dietary recall interviews. Subjects were reminded two days before the interview which was conducted at the house of the subject. The percentage of energy fractions was calculated for the baseline, second and third visits. The calculations were done using Diet 4 programme which was produced by the Institute of Medical Research (IMR), Malaysia. Food frequency interviews were conducted only during the baseline visit.

Data analysis

Data were entered into the microcomputer using STATISTICA software version 4.5 and analysed using the analysis of variance (ANOVA) test, Student-t test, chi-suare test and multiple linear regression analysis. All values were measured at $p<0.05$ significance level.

RESULTS

Demographics

The baseline demographic characteristics of the treatment and control groups are listed in Table 1. Both groups were similar with respect to sex, age, weight, height, BMI, WHR, daily calorie intake, diastolic blood pressure, fasting blood glucose, MDA and alpha-tocopherol except as noted in Table 1.

Nutritional status

A significant increase in the daily calorie intake and percent body fat was noted among the subjects following increased consumption of edible palm oil (Table 2a). Fat intake increased from 4.6% at baseline to 33.9% during the final visit, and an increase in calorie intake was observed among females ($p<0.05$). Percent body fat was also found to have increased in both genders following palm oil consumption. The control group, however, showed no difference in the daily calorie intake and waist-hip ratio, whilst a significant reduction in the BMI and percent body fat was observed among the males in this group (Table 2b).

Clinical profiles

Blood pressure evaluation showed a significant reduction ($p<0.05$) in systolic blood pressure following palm oil consumption (Table 3) in both genders. The diastolic blood pressure, however, did not show significant changes in both treatment and control groups following palm oil consumption. As for FBG, a significant increase was observed in both genders in the treatment group up to the second follow-up visit before lower values were recorded again during the final visit. As for the control group, no significant changes were observed in all the clinical variables studied.

Lipid profiles

TC was found to have decreased among the female subjects following palm oil consumption ($p<0.05$) (Table 4). No change was observed in the control group. As for HDL, a significant increase was noted in both genders following palm oil consumption ($p<0.0001$). The control group, however, showed a significant increase in HDL among the male subjects ($p<0.05$). LDL was found to have decreased in the treatment group in both males ($p<0.05$) and females ($p<0.0001$) following palm oil consumption. No significant change was observed in the control group. Meanwhile, there was no change in TG levels in both the treatment and control groups

following palm oil consumption. In this study, palm oil consumption was found to increase HDL and reduce LDL and TC levels.

Table 1. Clinical characteristics of study subjects

Clinical characteristics	Tual Post community (Intervention group)	Sinderut Post community (Control group)
Gender		
Male	99 (48.5%)	190 (58.1%)
Female	105 (51.5%)	137 (41.9%)
Age (year)	24.32 ± 16.58 (n = 204)	22.81 ± 15.15 (n = 327)
Weight (kg)	34.82 ± 13.62 (n = 204)	32.43 ± 12.53 (n = 327)
Height (cm)	133.46 ± 20.41 (n = 204)	131.23 ± 18.81 (n = 327)
Nutritional status		
Percent body fat (%)*	12.53 ± 7.89 (n = 111)	15.79 ± 5.82 (n = 184)
Body mass index (BMI) (kg/m^2)	18.49 ± 3.14 (n = 202)	18.00 ± 2.98 (n = 211)
Waist-hip ratio (WHR)	0.85 ± 0.07 (n = 183)	0.86 ± 0.07 (n = 212)
Daily calorie intake (kcal)	910 ± 363 (n = 188)	922 ± 456 (n = 153)
Blood pressure		
Systolic (SBP) (mm Hg)*	109 ± 12 (n = 134)	106 ± 14 (n = 178)
Diastolic (DBP) (mm Hg)	64 ± 11 (n = 132)	63 ± 13 (n = 178)
Lipid profiles		
Total cholesterol (TC) (mmol/L)*	3.68 ± 0.92 (n = 203)	3.18 ± 0.78 (n = 201)
High-density lipoprotein (HDL) (mmol/L)*	0.49 ± 0.23 (n = 201)	0.57 ± 0.20 (n = 180)
Low-density lipoprotein (LDL) (mmol/L)*	2.62 ± 0.92 (n = 200)	2.17 ± 0.78 (n = 171)
Triglyceride (TG) (mmol/L)*	1.18 ± 0.52 (n = 204)	0.99 ± 0.44 (n = 212)
Fasting blood glucose (FBG) (mmol/L)	4.50 ± 0.45 (n = 153)	4.67 ± 0.74 (n = 33)
Free radicals and antioxidant		
Malonaldehyde (MDA) (nmol/mg protein)	1.55 ± 0.88 (n = 108)	1.54 ± 0.75 (n = 97)
Alpha-tocopherol (AFT) (mg/dL)	0.46 ± 0.43 (n = 123)	0.37 ± 0.17 (n = 101)

Data are mean ± SD values; *significant difference between the treatment and control groups, p<0.05; n, number of subjects.

Table 2a. Nutritional indices following increased consumption of edible palm oil

Factors	Consumption of edible palm oil					
	Baseline visit	1st follow-up visit	2nd follow-up visit	Final visit	Statistical test	p-value
Energy intake						
Treatment						
Total calorie (kcal)	910.9 (n=188)	-	1076.4 (n=146)	1100.2 (n=146)	-	-
Protein (%)	6.9 (n=188)	-	6.5 (n=146)	6.6 (n=142)	-	-
Fat (%)	4.6 (n=188)	-	29.8 (n=146)	33.9 (n=142)	-	-
Carbohydrate (%)	88.5 (n=188)	-	63.7 (n=146)	59.5 (n=142)	-	-
Control						
Total calorie (kcal)	922.9 (n=153)	-	-	888.4 (n=193)	-	-
Protein (%)	7.5 (n=153)	-	-	5.3 (n=193)	-	-
Fat (%)	4.1 (n=153)	-	-	5.1 (n=193)	-	-
Carbohydrate (%)	88.5 (n=153)	-	-	88.5 (n=193)	-	-
Daily calorie intake (kcal)						
Male	961±390 (n=91)	-	1095±598 (n=73)	1139±543^{x} (n=71)	ANOVA	NS
Treatment						
Control	963±485 (n=95)	-	-	933±473 (n=106)	t-test	NS
Female						
Treatment	863±330 (n=97)	-	1057±485 (n=73)	1060±481^{x} (n=71)	ANOVA	NS
Control	857±398 (n=58)	-	-	834±357 (n=87)	t-test	p<0.05
Percent body fat (%)						
Male						
Treatment	7.38±4.56^{x} (n=47)	13.94±3.08^{a} (n=68)	12.19 ± 3.82^{a} (n=82)	14.72±3.89^{a,x} (n=84)	ANOVA	p<0.0001
Control	13.23±3.25 (n=103)	-	-	10.90±3.07 (n=98)	t-test	p<0.0001
Female						
Treatment	16.31±7.69^{x} (n=64)	20.69±6.52^{a} (n=59)	18.98 ± 6.26^{a} (n=80)	20.67±6.63^{a,x} (n=70)	ANOVA	p<0.05
Control	19.05±6.70 (n=81)	-	-	17.45±6.10 (n=89)	t-test	NS
Waist-hip ratio (WHR)						
Male						
Treatment	0.86±0.08 (n=97)	0.87±0.07 (n=76)	0.88±0.87 (n=95)	0.87±0.72 (n=89)	ANOVA	NS
Control	0.88±0.07 (n=114)	-	-	0.86±0.08 (n=106)	t-test	NS

Female						
Treatment	0.84±0.06 (n=86)	0.84±0.05 (n=79)	0.83±0.06[b] (n=87)	0.86±0.06[x] (n=78)	ANOVA	P<0.05
Control	0.85±0.07 (n=98)	-	-	0.83±0.07 (n=97)	t-test	NS
Body mass index (kg/m²)						
Male						
Treatment	18.58±3.13 (n=99)	18.97±2.96 (n=76)	17.81±2.78 (n=94)	18.45±2.85[x] (n=94)	ANOVA	NS
Control	17.96±2.73 (n=113)	-	-	16.94±2.77 (n=106)	t-test	P<0.05
Female						
Treatment	18.39±3.17 (n=103)	18.78±3.29 (n=78)	17.98±3.18 (n=90)	18.26±2.86[x] (n=73)	ANOVA	NS
Control	18.04±3.27 (n=98)	-	-	17.28±2.82 (n=96)	t-test	NS

Data are mean ± SD values; [x]significant difference between the treatment and control groups, p<0.05; [a]significant difference compared to the baseline, p<0.05; [b]significant difference compared to the final visit, p<0.05; n, number of subjects; NS, not significant.

Table 3. Clinical profiles following increased consumption of edible palm oil

Factors	Consumption of edible palm oil					
	Baseline visit	1st follow-up visit	2nd follow-up visit	Final visit	Statistical test	p-value
Systolic blood pressure (mm Hg)						
Male						
Treatment	113±13[x] (n=60)	112±18[b] (n=70)	110±14[b] (n=84)	105±22[a] (n=88)	ANOVA	P<0.05
Control	108±14 (n=92)	-	-	105±20 (n=106)	t-test	NS
Female						
Treatment	106±11 (n=74)	107±19 (n=74)	106±13 (n=86)	99±15[x] (n=80)	ANOVA	P<0.05
Control	104±14 (n=86)	-	-	107±17 (n=98)	t-test	NS
Diastolic blood pressure (mm Hg)						
Male						
Treatment	68±11[x] (n=60)	68±17 (n=70)	66±12 (n=85)	68±16 (n=87)	ANOVA	NS
Control	64±12 (n=92)	-	-	66±14 (n=106)	t-test	NS
Female						
Treatment	61±10 (n=72)	64±18 (n=74)	64±11 (n=86)	64±14 (n=80)	ANOVA	NS
Control	62±14 (n=86)	-	-	65±14 (n=98)	t-test	NS
Fasting blood glucose (FBG) (mmol/L)						
Male						
Treatment	4.45±0.42 (n=71)	4.69±0.68[a,b] (n=43)	4.89±0.69[a,b] (n=72)	4.41±0.43 (n=30)	ANOVA	P<0.05
Control	4.56±0.65 (n=25)	-	-	4.64±0.66 (n=53)	t-test	NS
Female						
Treatment	4.53±0.48[x] (n=82)	4.61±0.59 (n=42)	5.11±0.89[a,b,c] (n=69)	4.37±0.34[x] (n=30)	ANOVA	P<0.0001
Control	4.99±0.96 (n=8)	-	-	4.72±0.60 (n=52)	t-test	NS

Data are mean ± SD values; [x]significant difference between the treatment and control groups, $p<0.05$; [a]significant difference compared to the baseline, $p<0.05$; [b]significant difference compare to the final visit, $p<0.05$; [c]significant difference compared to 1st follow-up visit, $p<0.05$; n, number of subjects; NS, not significant.

Lipid peroxidation

A significant increase in alpha-tocopherol levels was observed in both males ($p<0.0001$) and females ($p<0.0001$) in the treatment group following palm oil consumption (Figure 3). However, the increase was also noted in both genders in the control group following the study. The significant increase in the control group was contributed by the adult age group (>18 years old). However, a more pronounced increase was observed in the treatment group ($p<0.05$). The health implication of increased alpha-tocopherol levels is that it will increase the protective effect of body cells against the activity of free radicals and reduce the risk of cancer and cardiovascular diseases. There was also a significant decrease in MDA levels in the treatment group following palm oil consumption ($p<0.05$) (Figure 4). The reduction was observed in both genders. Meanwhile, no significant changes in MDA levels were observed in the control group. In this study, palm oil consumption did not increase lipid peroxidation product (MDA) but reduced MDA levels compared to the controls.

Correction for baseline differences

In the baseline visit, the treatment group showed lower percent body fat and HDL, and higher systolic blood pressure, TC, LDL and TG, compared to the control group ($p<0.05$) (Table 5). After controlling for baseline measures, increased palm oil consumption was still found to have a significant effect on reducing TC ($p<0.0001$), LDL ($p<0.0001$) and systolic blood pressure ($p<0.0001$), as well as increasing HDL ($p<0.05$) and percent body fat ($p<0.0001$) levels. However, palm oil consumption did not affect TG levels and BMI at the end of intervention.

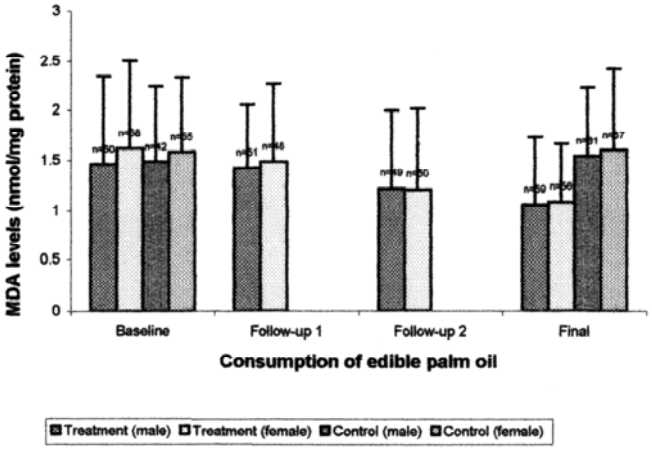

Figure 3. MDA levels following increased consumption of edible palm oil

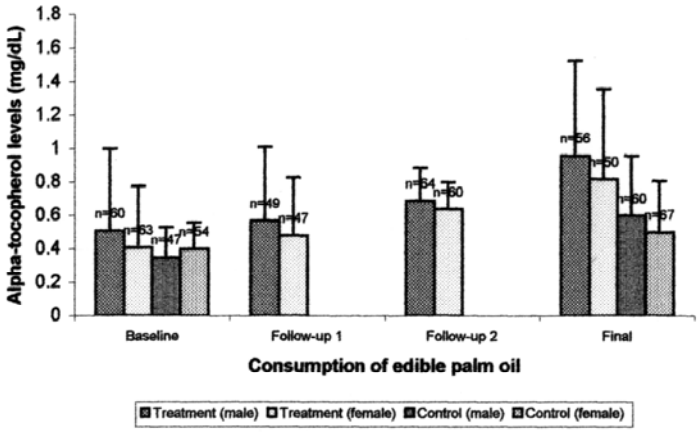

Figure 4. Alpha-tocopherol levels following increased consumption of edible palm oil

DISCUSSION

The study results on baseline cholesterol (before intervention) among the aborigines in Tual Post and Sinderut Post showed that the levels were very low, that is, in the range of 3.16 to 3.48 mmol/L (n = 205) among the males and 3.21 to 3.84 mmol/L (n = 199) among the females. These results are also similar to the study conducted by Osman Ali (1992) on several aboriginal populations in Malaysia. In his study, TC levels among the aboriginal population in Lanai Post and Betau Post, Kuala Lipis, Pahang and in Bukit Lanjan, Petaling, Kuala Lumpur among subjects aged 7 years and above were 2.5 to 3.8 mmol/L for males (n = 266) and 3.5 to 4.5 mmol/L for females (n = 257). Compared to other communities in the world, the Malaysian aborigines still have low TC levels. TC levels among the Indian population in Kerala, India (20 – 60 years old, males and females, n = 218) who traditionally used coconut oil was 5.122 mmol/L (Arumughan *et al*., 1996). TC levels among 15 healthy Finland female subjects aged 19 to 34 years old was 4.86 mmol/L (Schwab *et al*., 1995). Ethnic difference in TC levels is probably influenced by lifestyle factors such as physical activity, dietary pattern and food preparation method, attitude and environment.

Fat intake among the study population was very low (4.1 to 4.6% fat as an energy source) before intervention and increased to 28.9 to 33.9% following palm oil consumption. Low energy intake among the aborigines is associated with the high carbohydrate content of their food sources (tapioca and rice) and low fat. In this study, palm oil consumption successfully increased the total calorie intake from 891 to 1,112 kcal (baseline) to 1,082 to 1,265 kcal (final) in the adult group. The results indicate the beneficial effect of palm oil consumption in increasing daily calorie intake. The consumption of palm oil, however, did not increase the BMI. This shows that palm oil does not increase the risk of developing cardiovascular disease (Poulter, 1993). A study done by Suk, In & Jin (1989) with 21 healthy male Korean subjects (25 – 40 years old) following 20 days of palm oil restricted diet showed that there was no change in mean BMI at the final visit compared to the baseline.

Table 4. Lipid profiles following increased consumption of edible palm oil

Factors	Consumption of edible palm oil					
	Baseline visit	1st follow-up visit	2nd follow-up visit	Final visit	Statistical test	p-value
Total cholesterol (mmol/L)						
Male						
Treatment	3.48±0.84[x] (n=98)	3.40±0.79 (n=72)	3.38±0.40 (n=95)	3.22±0.47 (n=83)	ANOVA	NS
Control	3.16±0.73 (n=107)	-	-	3.09±0.56 (n=107)	t-test	NS
Female						
Treatment	3.84±0.97[x] (n=105)	3.70±0.91[b] (n=77)	3.66±0.98[b] (n=95)	3.34±0.55[a,x] (n=77)	ANOVA	P<0.05
Control	3.21±0.83 (n=94)	-	-	3.12±0.69 (n=98)	t-test	NS
HDL (mmol/L)						
Male						
Treatment	0.49±0.26 (n=98)	0.54±0.16 (n=67)	0.69±0.22[a,b,c] (n=93)	0.64±0.27[a] (n=80)	ANOVA	P<0.0001
Control	0.54±0.14 (n=91)	-	-	0.61±0.33 (n=107)	t-test	P<0.05
Female						
Treatment	0.49±0.21[x] (n=89)	0.06±0.17[a,b] (n=74)	0.83±0.37[a,c] (n=95)	0.75±0.32[a] (n=80)	ANOVA	P<0.0001
Control	0.61±0.24 (n=89)	-	-	0.68±0.35 (n=98)	-test	NS
LDL (mmol/L)						
Male						
Treatment	2.47±0.88[x] (n=97)	2.37±0.73[b] (n=65)	2.15±0.63[a] (n=93)	2.10±0.55 (n=80)	ANOVA	P<0.05
Control	2.21±0.71 (n=85)	-	-	2.04±0.60 (n=105)	t-test	NS
Female						
Treatment	2.76±0.94[x] (n=103)	2.54±0.83[b] (n=74)	2.32±0.77[a] (n=94)	2.10±0.61[a] (n=76)	ANOVA	P<0.0001
Control	2.12±0.85 (n=86)	-	-	1.98±0.70 (n=97)	t-test	NS
TG (mmol/L)						
Male						
Treatment	1.12±0.37[x] (n=99)	0.99±0.29 (n=76)	1.13±0.50 (n=95)	1.06±0.44 (n=83)	ANOVA	NS
Control	0.97±0.45 (n=114)	-	-	1.07±0.44 (n=107)	t-test	NS
Female						
Treatment	1.24±0.62[x] (n=105)	1.23±0.72 (n=80)	1.26±0.80 (n=95)	1.07±0.47 (n=77)	ANOVA	NS
Control	1.02±0.46 (n=98)	-	-	1.06±0.41 (n=98)	t-test	NS

Data are mean ± SD values; [x]significant difference between the treatment and control groups, p<0.05; [a]significant difference compared to the baseline, p<0.05; [b]significant difference compare to the final visit, p<0.05; [c]significant difference compared to 1st follow-up visit, p<0.05; n, number of subjects; NS, not significant.

Table 5. Multiple regression analysis to show effect of intervention on lipid profiles, nutritional status and systolic blood pressure.

Outcome	R^2	F	Independent variables	Regression coefficient (β)	t-value	p-value
Total cholesterol	0.3056	36.972	Baseline TC	0.0994**	5.11651	P<0.001
			Sex	-0.1023*	-3.3221	P<0.05
			Intervention	0.2612*	-8.2996	P<0.0001
			Constant	3.1851**	35.1095	P<0.0001
HDL	0.2320	18.956	Baseline HDL	0.1637*	3.4543	P<0.05
			Sex	0.1097**	6.7822	P<0.0001
			Age	0.0012*	2.6297	P<0.05
			Intervention	-0.0363*	2.2037	P<0.05
			Constant	0.2720**	6.5050	P<0.0001
LDL	0.1938	30.410	Sex	-0.1357**	-5.438	P<0.0001
			Intervention	0.1487**	-5.928	P<0.0001
			Constant	2.4102**	43.0302	P<0.0001
TG	0.1194	11.386	Baseline TG	-0.01248	-3.9007	P<0.05
			Sex	0.0105**	4.0118	P<0.0001
			Age	0.0002*	2.4942	P<0.05
			Constant	0.8040**	150.2708	P<0.0001
BMI	0.734	137.78	Baseline BMI	0.5234**	13.5066	P<0.0001
			Sex	-0.8376**	-4.1500	P<0.0001
			Age	0.0208*	3.7596	P<0.05
			Constant	5.4110**	8.3356	P<0.0001
% BF	0.6793	77.574	Baseline %BF	0.4754**	7.0868	P<0.0001
			Sex	4.5810**	5.0613	P<0.0001
			Intervention	-7.4365**	-9.1435	P<0.0001
			Constant	14.6737**	8.9746	P<0.0001
SBP	0.3324	31.251	Baseline SBP	0.3204**	3.7189	P<0.05
			Age	0.4725**	8.7207	P<0.0001
			Intervention	-7.4365**	4.1871	P<0.0001
			Constant	14.6737**	4.7446	P<0.0001

*β significant at p<0.05; **β significant at p<0.0001

Level of TC is the risk factor for cardiovascular disease (Woft, 1994; Blackburn, 1994). Palm oil consumption, however, was able to reduce TC levels in this study. Similar results were obtained by other studies in Canada (Cook *et al*., 1996), USA (Khosla & Sundram, 1996), Pakistan (Khan, Ahmad & Ahamd, 1996), Hungary (Biro *et al*., 1996), India (Arumughan *et al*., 1996), China (Zhang *et al*., 1996) and Malaysia (Marzuki *et al*., 1991; Ng *et al*., 1991). This study also found that palm oil consumption increased HDL levels and reduced LDL and TG levels (Lim *et al*., 1988; Arumughan *et al*., 1996). The effect of palm oil intake on percent body fat has not been studied before. In this study, palm oil consumption was found to increase percent body fat among the aborigines without changing BMI. The normal percent body fat for males and females was 12 to 18% and 18 to 24%, respectively (Ganong, 1989). Based on these studies, our

observations indicate that, palm oil intake was able to increase percent body fat of the male subjects, that is, from low levels at baseline to normal levels at the final visit. This shows that palm oil consumption is able to increase body fat status among the aborigines in Tual Post, Kuala Lipis, Pahang.

A study done by Suk *et al*. (1989) also showed that palm oil supplementation did not change systolic and diastolic blood pressure at the final visit compared to the baseline. This agrees with the current study on aborigines where palm oil supplementation did not significantly change systolic and diastolic blood pressure following intervention. This shows that palm oil consumption does not increase the risk of cardiovascular disease. After controlling the confounding factors (age, sex and baseline systolic blood pressure), it was found that palm oil consumption still had a significant influence on the reduction of systolic blood pressure among the aborigines.

Packer (1991) reported that vitamin E in palm oil has many beneficial effects. It can act as an anticancer agent, reduce phospholipase A2 activity and has hypercholesterolemic effects. There is also evidence that it can delay the aging process, reduce arthritic symptoms, reduce platelet aggregation rate, delay the development of cataract, increase physical performance and protect the lungs against injury from the free radicals of cigarette smoke and pollution. Palm oil is known to have antioxidant activity and has a protective effect from free radicals attack and lipid peroxidation. This is because, palm oil contains natural antioxidant materials – tocopherols and tocotrienols. This study showed that subjects increased their serum alpha-tocopherol levels to as much as 48.3% following 18 months of palm oil consumption. Confounding factors such as vitamin E originating from leaves, roots, and stalks of jungle plant (Machlin, 1990) consumed by the aborigines might have also influenced the increase in alpha-tocopherol levels.

Palm oil also contains 10.6 to 11.4% linoleic acid (18:2) which is an essential fatty acid. A cross-sectional study done by Ohrvall *et al*. (1994) among a healthy population (n = 103) showed that essential fatty acid deficiency increases serum MDA levels. This shows that apart from not increasing cholesterol levels, linoleic fatty acid in palm oil also does not increase serum MDA levels. Low serum antioxidant and high levels of MDA are believed to increase the risk of developing atherosclerosis (Iribarren *et al*., 1997; Benzie, 1996) and coronary heart disease (Meraji *et al*., 1997; Wen *et al*., 1996). A study done by Torun *et al*. (1995) found that mean levels of MDA were higher among cancer patients compared to controls. Mean levels of alpha-tocopherol in cancer patients was also lower than the controls. A clinical trial study done by Meraji *et al*. (1997) also showed that vitamin E supplementation was able to reduce serum MDA levels. It is believed that vitamin E acts by inhibiting lipid peroxidation which in turn reduces MDA production (Haglund *et al*., 1991).

Several previous studies have shown that palm oil consumption does not increase FBG. Therefore, it was assumed that palm oil intake did not increase glucose tolerance disturbance. This is because, based on previous epidemiologic studies, saturated fat intake might have an association with glucose tolerance disturbance (Schwab *et al*., 1995). A study done by Qureshi *et al*. (1996) found that palm oil supplementation reduced glucose levels as much as 12%, and Arumughan *et al*. (1996) also showed that palm oil intake for 8 weeks did not affect fasting blood glucose levels. This shows that there is no increase in insulin resistance following palm oil

consumption. A study among the aborigines showed that palm oil consumption did not increase fasting blood glucose levels above the normal range (4.22–6.11 mmol/L). This study shows that palm oil consumption does not disturb the important function of insulin hormone in the body system.

Palm oil has high levels of tocopherols and tocotrienols. Although tocotrienols exist in higher amounts compared to tocopherols in palm oil, the biological activity of alpha-tocopherols is higher than that of tocotrienols. Alpha-tocopherol has a higher protective effect compared to tocotrienols in specific conditions, and the effectiveness of tocotrienols as in vivo antioxidants is still questionable (Choudhury, Tan & Truswell, 1995). In general, it is believed that this study has succeeded in establishing the protective effect of antioxidants in palm oil manifested by reducing lipid peroxidation products as much as 29% among the aborigines. Either tocopherols or tocotrienols or combinations of both in palm oil might be able to protect body cells from the action of free radicals and consequently to increase the health status of the aborigines.

CONCLUSION

The daily consumption of olein palm oil for 18 months among the Malaysian aborigines did not increase the risk factors for coronary heart disease. On the contrary, it was found to improve the health and nutritional status of the aborigines, besides providing fat as an energy source. Continuous consumption of palm oil did not affect mean BMI, WHR and FBG; it increased antioxidant levels (alpha-tocopherol) and reduced lipid peroxidation products (MDA). However, increased consumption of palm oil must be in moderation for individuals who are prone to obesity. It is suggested that long-term studies be done among the urban and urban-fringe populations for comparative purposes.

ACKNOWLEDGEMENTS

This study was made possible through funding from UKM Research Code F 18/95. The authors would like to thank the Department of Aboriginal Affairs of Kuala Lipis and Gombak branches for their cooperation. Gratitude is also expressed to the endocrine technologists at Universiti Kebangsaan Malaysia for their technical assistance.

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