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Palm oil diet may benefit mildly hypercholesterolaemic Chinese adults

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The effects on serum lipids and platelet function of diets prepared with palm oil (PA) and peanut oil (PE) were studied in two groups of mild hypercholesterolaemic volunteers (serum TC between 5.5 - 7.0 mmol/L, aged 32-68). There were 15 men and 11 women in PE group and 16 men and 9 women in PA group. Dietary fat provided about 30% of total calories, and the test oil accounted for 60-65% of total dietary fat. During the 3 weeks of pretest period, diets were prepared with peanut oil, the local habitual cooking oil for all subjects. During the next 6 weeks the subjects in PA group consumed a diet prepared with palm oil while subjects in PE group continued to consume a diet prepared with peanut oil. Compared to the entry-level values, the concentrations of serum TC (total cholesterol), LDL-C (low density lipoprotein cholesterol), TC/HDL-C (high density lipoprotein cholesterol) ratio and plasma TXB_2/6-keto-$PGF_{1\alpha}$ ratio were significantly decreased in PA group (-6.5%, -9.0%, -11.5%, and -22.4%) while not appreciably altered in PE group by the end of the test. No significant change was observed on the whole blood platelet aggregation in both groups. In connection with the results from our previous study in normocholesterolaemic Chinese volunteers, palm oil, used as cooking oil in Chinese diet, will not lead to any adverse effect on blood lipids and thus will not increase CVD risks.

Introduction

Cardiovascular disease (CVD) is one of the major causes of death in developed countries and the death caused by CVD is increasing rapidly in China[1-4]. It is generally believed that high concentrations of serum total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) are positively related to the risk of CVD and that saturated fatty acids (SFAs) have the potential to increase the blood lipids and promote thrombosis.

Palm oil is the second largest volume of vegetable oil produced in the world. As it is highly saturated and contains about 50% palmitic acid, palm oil was discredited like saturated animal fats, such as butter, lard and tallow. The allegation that palm oil raises total serum cholesterol, thereby increasing the risk of coronary heart disease, however, was not based on actual experimental studies. Recently, studies in animals and in humans indicate that palm oil is quite different from other hypercholesterolaemic fats such as lard or coconut oil[5-7]. Therefore, the scientific community needs to conduct controlled studies on the effects of palm oil and its relation to cardiovascular disease and maintain a responsible perspective when reporting its findings or making recommendations concerning consumption of this oil.

The consumption of palm oil in China has increased rapidly in recent years[8], but the information about the relation of palm oil to health is very limited. A few papers show that palm oil maintains the normal growth of rats and causes a significant reduction of serum cholesterol in rabbits compared to lard[9]. The reports on palm oil in human studies are difficult to find in China. Therefore, it is necessary to undertake properly controlled studies on the effects of palm oil on blood lipids and on the risk of CVD. The previous study showed palm oil had no harmful effect on normal cholesterolaemic subjects in habitual Chinese diet[10]. This study observes the effects of palm oil on mildly hypercholesterolaemic volunteers.

Subjects and Methods

Subjects

After excluding known diabetes, hypertension and liver, renal and thyroid disorders, 31 males and 20 females, aged from 32-68 years of age, were selected as subjects. Their serum cholesterol concentration ranged from 5.5-7.0 mmol/L. All subjects worked either on a state owned farm or in a local plant producing micro electric motors. The body weight of the subjects ranged from 54.5 - 79.5 kg in males and from 42.0 - 66.5 kg in females. None of the subjects were taking medication known to affect lipid metabolism.

Diet

Experimental diet was composed of rice, flour, lean pork, chicken, bean curd, and some local green vegetables. The menu was developed around subjects' preferences and daily meals were prepared in habitual manner by a local cook under the direction of professionals to meet the experimental requirements. RBD palm oil was purchased from the Brother Oil Company, Singapore, while peanut oil was purchased from local edible oil company. According to the food inventory charges, average nutrient intakes were calculated based on the Chinese food composition table. The fatty acids profile and the main nutrients of the test diets were shown in Tables 1 and 2.

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Table 1. Fatty acids profile of two test diets.

Fatty acid	Palm oil diet %	Peanut oil diet %
14:0	0.8	0.4
16:0	32.7	16.0
16:1	2.0	0.6
18:0	3.7	5.7
18:1	38.8	39.3
18:2	19.6	31.4
18:3	1.7	1.5
20:0	0.2	1.1
20:1	0.1	1.2
22:0	--	1.8

Table 2. Average daily nutrients intake of hypercholesterolaemic subjects on palm oil and peanut oil diets.

Test oil	Energy	Fat	Protein	Carbohydrate	Fibre	Cholesterol
	(MJ)	(g)	(g)	(g)	(g)	(mg)
PE	10.3	82	70	362	13	166
PA	10.6	85	76	366	14	163

Biochemical Analyses

Serum TC and TG (triglyceride) levels were determined by using enzymatic kits (Chinese Zhong Sheng High-Tech Bioengineering Company, ZS 89001) on Beckman 700s system auto analyser. HDL-C was assayed using enzymatic kits after a precipitation with phosphotungstic acid and magnesium chloride. LDL-C was calculated using the formula of Friedwald. Plasma TXB^2 and $PGF_{1\alpha}$ were determined by using ^{125}I radio-immunoassay kits purchased from radio-immunoassay laboratory, General Hospital of the People's Liberation Army of China. Platelet aggregation in whole blood was determined with a chronolog model 500vs aggregometer after introduction of collagen (obtained from Sigma chemical company) using the impedance method[11]. Collected in a 3.8% solution of sodium citrate (9 parts blood to 1 part citrate), each blood sample was assayed within an hour and the final concentration of collagen was 1 μg/mL blood.

Statistical analyses

The data were analysed with the SPSS/PC+ statistics program (V4.0, SPSS, Chicago, IL). The differences between the two test groups were assessed with t test (two-tailed). In all cases, statistical significance is $P < 0.05$ and data are presented in the text and tables as means ±SD.

Results

The effects of the two test cooking oils on plasma lipids are shown in Table 3. The average entry concentration of serum TC has not been completely matched in grouping due to some practical problems, but there exists no significant difference between two groups. It would be more reasonable to compare the difference between the entry and the end values of each group rather than the end values of PA and PE groups. As peanut oil is the habitual cooking oil for the subjects, all biochemical indices were not significantly influenced by PE diet. But compared with entry values, PA diet caused a significant reduction in serum TC (-6.5%, P<0.05), LDL-C (-9.0%, P<0.05) and TC/HDL-C ratio (-11.5%, P<0.05).

Table 3. Effects of test oil on serum lipids in hypercholesterolaemic Chinese.

Group		Blood Lipids (mmol/L)				
		TG	TC	HDL-C	LDL-C	TC/HDL-C
PE	0wk	1.40±0.52	5.88±0.41	1.23±0.27	4.34±0.45	4.78±1.10
	6wk	1.28±0.54	5.87±0.28	1.22±0.27	4.38±0.39	4.88±1.06
PA	0wk	1.54±0.50	6.11±0.44	1.22±0.17	4.55±0.44	5.05±0.81
	6wk	1.41±0.64	5.71±0.43*	1.28±0.19	4.14±0.36*	4.47±0.59*

Values are means±SD. n=26 in PE group, n=25 in PA group.
*Denotes a significant difference from entry value (P<0.05).

The average plasma concentration of TXB_2 was slightly increased while $PGF_{1\alpha}$ was slightly decreased in the PA diet group. Thus the plasma TXB_2 /$PGF_{1\alpha}$ ratio was significantly reduced (Table 4). The whole blood platelet aggregation was determined but no significant changes were observed in either group (Table 5).

Table 4. Effect of test oil on plasma level of TXB_2 and 6-keto-$PGF_{1\alpha}$ (M ± SD)

Group		TXB_2 (pg/ml)	6-keto-$PGF_{1\alpha}$ (pg/ml)	TXB_2/ 6-keto-$PGF_{1\alpha}$
PE	0 wk	83.3±44.7	42.9±14.5	1.85±1.07
	6 wk	87.8±38.0	50.6±18.3	1.74±0.76
PA	0 wk	80.9±18.9	45.9±11.1	1.83±0.49
	6 wk	71.3±26.1	51.2±16.4	1.42±0.45*

Values are means ± SD. N = 18 per group.
*Denotes significant difference from entry value (P<0.05).

Table 5. Effect of test oil on whole blood platelet aggregation.

Group	n	Blood platelet aggregation (oms)	
		0 wk	6 wk
PE	7	13.4±2.1	14.5±3.4
PA	9	11.4±3.5	13.1±3.0

Discussion

A substantial body of data implies that dietary saturated fat tends to increase serum cholesterol concentrations and promote thrombosis[12-13]. Analysis of some saturated fats, such as palm oil, lard, butter and coconut oil reveals that each has distinct profiles and exert different metabolic effects. Recently, studies from humans and experimental animals show that palm oil, despite a high concentration of SFA (mainly palmitic acid), does not increase serum cholesterol concentrations or thrombotic tendencies[14,15].

The Chinese diet contains more vegetables but less animal foods than the western diet. The average per capita intake of cholesterol is less than 300mg/d in most urban area and less than 200mg/d in rural areas. Our former study showed that palm oil used in Chinese diet did not increase serum cholesterol concentration in normocholesterolaemic subjects[10]. Some studies show that palmitic acid appears to increase plasma cholesterol in hypercholesterolaemic subjects[16]. The results in the present study show that in comparison with PE diet, PA diet induced a reduction of serum TC, LDL-C and TC/HDL-C ratio. This is consistent with results from other laboratories that palmitic acid appears to be non-hypercholesterolaemic when dietary cholesterol intake is low[15,17].

Since the 1950s, numerous studies in humans and in animals have investigated the effects of dietary fat

saturation on cholesterolaemia. Keys and Hegsted, respectively, transformed these early results into mathematical regression equations that have been used to predict the average change in serum cholesterol that might be expected for a given change in the percentage of energy consumed from a specific class of fatty acids[16,18]. But Lowenstein showed that African pastoral tribes had a low serum cholesterol concentration and a low incidence of coronary heart disease (CHD) mortality despite having high milk-fat intake[19]. Pronczuk reported that although tallow and lard contained appreciable amounts of SFAs, they were not much more hypercholesterolaemic then corn oil[20]. This prompted investigators to question the generally held belief that the 12-16C fatty acids were equivalent in terms of their cholesterol-raising ability. A study and regression analysis showed that myristic acid (l4:0) is four times more potent than palmitic acid in raising serum cholesterol[21]. Hayes et al reported that the exchange of dietary 16:0 for 12:0+14:0 caused a decrease in the plasma cholesterol when dietary total saturated, monounsaturated (MUFAs), and polyunsaturated fatty acids (PUFAs) were held constant[22]. The same result was obtained in normocholesterolaemic humans, even with 300mg of cholesterol in the diet[23]. These results clearly suggest that palmitic acid, the major and most controversial saturated fatty acid in palm oil, was not cholesterolaemic but neutral and the widely held belief that all saturated fatty acids were the same was invalid.

Oleic acid is another major fatty acid (about 40%) in palm oil and was formerly considered neutral. Epidemiologic studies on Mediterranean populations who consume substantial amounts of olive oil (high content of oleic acid) showed that the mortality for CHD in these populations was low[24]. A significant inverse relationship between red blood cell phosphatidylcholine (RBC-PC) oleate and CVD mortality, particularly CHD was also found in China[25]. Some animal and human studies also suggest[26] that oleic acid has cholesterol-lowering potential as does linoleic acid and has the benefit of lowering LDL-C without decreasing HDL-C levels[27-29]. An American group reported that the combination of 16:0+18:1 had some beneficial impact on enhancing HDL-C and LDL-C receptor mRNA abundance in hamsters[30]. But more work needs to be done to confirm these findings.

Analyses of accumulating data show that 85% of the observed variation in serum cholesterol could be explained solely on the basis of 14:0 and 18:2 when dietary cholesterol intake was 300mg or less[17]. In the present study, the content 14:0 is less than 1.0% in PA diet and the level of 18:2 actually exceeded the threshold levels required to counter the cholesterol-raising effects of 12 and 14-carbon SFAs (Table 1).

It is generally believed that SFAs promote thrombosis[13] and that PUFAs of n-6 family present in vegetable oils have proaggregatory thrombotic effects while the n-3 PUFAs of seafoods increase bleeding time[31,32]. Cook demonstrated that a balance of saturated and unsaturated fatty acids is important to enzymes responsible for synthesis of crucial membrane components and substrates for eicosanoid formation (20:4n-6, 20:5n-3 and 22:6n-3)[33]. Garg *et al* showed that partial replacement of dietary 18:2n-6 by tallow, which contains about 51% stearic acid (18:0) and 29% palmitic acid (16:0), can accelerate the conversion of 18:3n-3 to 20:5n-3 and maximise the inhibition of the conversion of 18:2n-6 to 20:4n-6[34]. This may partly explain the fact that although palm oil contains nearly 50% palmitic acid, it does not increase arterial thrombotic tendencies and even tends to decrease platelet aggregation compared to the effect of safflower oil (SA) which contains about 70% linoleic acid 18:2n-6[35]. On the other hand, some studies show that oleic acid (18:1n-9) has potential to decrease thrombotic tendencies[29,36].

All of these may be reasons why the replacement of habitual peanut oil with palm oil in diet causes a significant reduction in TXB_2/PGF_{1_α} ratio. Contrary to our expectancy, the whole blood platelet aggregation rate was not significantly affected by palm oil. This is probably because of wide range of fluctuation between individuals and the limited number of subjects.

In conclusion, in comparison to peanut oil, PA has a hypocholesterolaemic and antithrombotic effect on mild hypercholesterolaemic Chinese adults. In connection with the results from our former study in normocholesterolaemic Chinese volunteers, we feel confident to say that the large amounts of PA entering into Chinese diet will not lead to any adverse effect on blood lipids, thus will not increase CVD risks.

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食用棕榈油对中国血胆固醇水平偏高成人血脂和血小板功能的影响

摘要

作者研究了以棕榈油和花生油烹调的膳食对血胆固醇水平在5.5~ 7.0mmol/L, 年龄在32-68岁之间成年人血脂和血小板功能的影响。膳食总热能的30%由膳食脂肪提供, 其中的60~ 65%由实验用油提供。实验开始前三周为预备期, 其间所有受试者的膳食都以当地日常食用的花生油烹调。实验期6周, 15名男性和11名女性为一组, 继续食用以花生油烹调的膳食(称PE组); 16名男性和9名女性为一组, 食用以棕榈油烹调的膳食(称PA组)。结果表明, 与起始水平相比, 实验结束时PA组受试者血清总胆固醇(TC), 低密度脂蛋白胆固醇(LDL-C)浓度, 血清总胆固醇与高密度脂蛋白胆固醇(HDL-C)的比值(TC/HDL-C)以及血浆TXB_2/6-keto-$PGF_{1\alpha}$ 比值都出现了显著性下降(分别降低了6.5%, 9.0%, 11.5%和11.5%); 而PE组受试者的上述几项生化指标均未见显著变化。两组受试者的血小板凝集率在实验期间均未见显著变化。结合有关棕榈油对中国健康青年男子血脂水平影响的研究结果, 作者认为棕榈油做为中国居民膳食中的日常用油不会对血脂水平产生不利影响, 也不会增加发生心血管疾病的危险性。

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