Original Article

Effects of palm olein and olive oil on serum lipids in a Chinese population: a randomized, double-blind, cross-over trial

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Background and Objectives: As the most widely produced edible vegetable oil, palm oil is known as to contain a high level of saturated fatty acid, which was thought to adversely affect serum lipid profiles. However, recent studies have shown no influence or benefits of palm oil on serum lipids. The potential nutritional value of palm oil is attributed to the high mono-unsaturation at the crucial sn2-position of the oil's triacylglycerols, as with the so-called 'healthy' olive oil (OO). The aim of this study was to further test this hypothesis and evaluate the effects of consuming palm olein versus olive oil on serum lipid profiles in a Chinese population. **Methods and Study Design:** In total, 120 participants were recruited from a spinnery in Yixing city and randomly divided into two groups (palm olein or olive oil) to conduct a 2×2 crossover trial for 2 months' intervention with 2-week washout periods. Each participant was provided 48 g of test oil per day. At the end of each period, anthropometry, and blood lipid indices were measured to determine the effects of palm olein and olive oil. **Results:** Palm olein and olive oil consumption had no significantly different effect on BMI, on serum total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triacylglycerol (TG), Apo B, fasting glucose, or insulin concentrations (all $p>0.05$). **Conclusions:** In a dietary crossover trial, palm olein and olive oil had no recognisably different effects on body fatness or blood lipids in a healthy Chinese population.

Key Words: palm olein, olive oil, blood lipids, cross-over trial, population intervention

INTRODUCTION

According to a report from the United States Department of Agriculture on world markets and the trade of oilseeds, China was the third-largest importer of palm oil from 2012 to 2016, importing 12% to 15% of the world's palm oil. Of the 5500 to 6589 thousand metric tons of imported oil, approximately 60% was used in food products.[1] Palm oil, a vegetable oil obtained from the fruit of the palm trees (Elaeis guineensis), contains an equal proportion of unsaturated and saturated fatty acids, including 50% palmitic acid, 40% oleic acid, and 10% linoleic acid. Palmitic acid naturally occurs in animal and vegetable fats and is the main saturated fatty acid (SFA) in human milk fats.[2] Unlike most other edible oils that come as a single entity, palm oil is readily fractionated to provide a variety of oils with distinct solid fat content and melting points. Moreover, it can be applied to various food products, including liquid palm olein (PO) and the more solid palm stearin, by separating the upper and lower fractions during the dry (melt) fractionation of palm oil itself.[3] Palm oil is highly structured and contains predominantly oleic acid at the sn-2 position in major triacylglycerols (TGs), which accounts for the beneficial effects described in numerous nutritional studies. Notably, different dietary fatty acid compositions may affect serum lipid profiles. Numerous studies have confirmed the nutritional value of palm oil with the attribution of high monounsaturation at the crucial sn-2 position of the oil's TGs, indicating that the oil is as healthy as olive oil (OO).[4] The TG confor-

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mation in palm oil includes SFA at the sn-1 and sn-3 positions of the glycerol backbone, and unsaturated fatty acids at the sn-2 position in 75%–87% of these molecules. This TG conformation leads to the absorption of mation in palm oil includes SFA at the sn-1 and sn-3 positions of the glycerol backbone, and more unsaturated fats than saturated fats, as the fatty acid at the sn-2 position is absorbed preferentially to those at the sn-1 and sn-3 positions.[5] Additionally, it is well known that OO is the main source of fat in the Mediterranean diet, as well as a functional oil which not only contains several minor components with biological properties, but also has a high level of monounsaturated fatty acid (MUFA).[6]

Dyslipidemia, especially the elevated ratio of low density lipoprotein cholesterol (LDL-C) and the total cholesterol/ high density lipoprotein cholesterol (TC/HDL-C), is considered to be the most critical risk factor of cardiovascular disease (CVD).[7] Some recommendations aimed at lowering CVD morbidity and mortality suggest reducing the consumption of animal fats, which are rich in SFAs that can lead to an increase in blood TC and LDL-C,[8] and substituting them with polyunsaturated fatty acid (PUFA).

Over the past two decades, some human studies have compared the effects of palm oil and OO on serum lipids profiles or the development of CVD. Notably, it has been reported that a dietary PO intervention did not change the serum TC, LDL-C, HDL-C, or TG concentrations when compared to a dietary OO intervention.[9,10] Moreover, Voon et al reported that no significant variations were observed in the plasma total homocysteine or the inflammatory markers when healthy Malaysian adults were given high-protein diets prepared with PO, coconut oil, or virgin OO; in addition, they noted that diets prepared with PO and OO had comparable non-hypercholesterolemic effects and lower levels of postprandial total cholesterol ($p<0.05$) than the OO diet.[11]

However, other research contradicts these findings. Montoya et al, reported that a SFA diet with high palm oil content (17% of total energy) increased the serum TC and LDL-C levels when compared with an MUFA diet with a predominance of oleic acid (18:1; 20.9% of total energy), an n-6 PUFA diet with a high percentage of sunflower oil (12.8% of total energy), or a PUFA diet (n-3) enriched with fish oil (4-4.5 g/day; 1.6% of total energy).[12] In another study, a MUFA-rich diet was found to reduce the concentration of LDL-C and significantly increase the resistance of LDL to oxidative modification, compared with a SFA diet with palm oil.[13] Tholstrup et al, indicated that lard increased the concentration of TC and LDL-C ($p<0.01$) when compared with dietary OO and PO; moreover, PO resulted in a lower plasma TG concentration than OO ($p<0.01$). The authors therefore concluded that the fatty acid in the sn position was not important when considering the effects of fatty acid on plasma cholesterol; nevertheless, finding a lower plasma TG concentration after the PO diet than after the OO diet was unexpected.[14]

As noted earlier, PO has a similar distribution of MUFA in the sn-2 position compared with OO. MUFA is known to improve blood lipid profiles for in people with metabolic syndrome, CVDs, and abdominal obesity, and in health non-obese individuals.[15-17] PO with MUFA almost exclusively in the sn-2 position may have similar effects on blood lipid profiles as does OO. Hence, this study investigated the effects of PO and OO on blood lipid profiles in a healthy Chinese population, which is understudied regarding the effects of PO and OO.

Overall, the effects of OO and PO on human serum lipid profile remain controversial. Further research is required to understand the impact of palm oil and OO on lipid metabolism in the human body, across a variety of populations. The present study contrasted the effects of PO and OO consumption on the human serum lipid profiles in a healthy Chinese population.

METHODS

The ideal sample size for this study was determined by serum TC levels, according to the epidemic formula $N = [2(Z_\alpha + Z_\beta)^2 \times \sigma^2] / d^2$ with a 95% CI and 5% margin of error, where $Z\alpha = 1.96$, $Z\beta = 1.64$, $\sigma = 0.65$ mmol/L (i.e., the estimated standard deviation of serum TC from our previous study), and $d = 0.50$ mmol/L (i.e., the estimated difference between the two groups from Montoya's study).[12] The calculation indicated that a minimum sample of 44 participants per group was required, and 60 participants were selected for each group.

Participant selection

At a textile mill in Yixing, Jiangsu province, China, 212 volunteers, whose ages ranged from 25 to 55 years old, were recruited. Their body weight, blood pressure, fasting serum lipid profile, blood cell count, and liver and kidney function indices were examined, and 120 of them met the following inclusion criteria:

(1) No personal or family history of CVD, diabetes, hypertension, hypothyroidism, hyperthyroidism, chronic renal disease, hepatitis, or cancer;
(2) The body mass index (BMI) between 18.5 and 24.9 kg/m^2;
(3) Not being pregnant or lactating;
(4) Serum TC level <6.20 mmol/L, LDL-C level <4.12 mmol/L, HDL-C level >1.04 mmol/L, and TG level <2.26 mmol/L (in accordance with the guidelines for dyslipidemia prevention and treatment for Chinese adults, 2007 edition[18]); and
(5) A fasting blood glucose (FBG) level <6.1 mmol/L (according to the guideline for type 2 diabetes prevention and treatment in China, 2013 edition[19]).

All participants who met the requirements were divided into two groups using the random number table method, with 60 participants randomly assigned to each group.

This study was conducted according to the guidelines detailed in the Declaration of Helsinki, and all of the procedures involving human participants were approved by the Ethics Committee for Clinical Research of Zhongda Hospital (affiliated with Southeast University, approval number is 2014ZDSYLL0540). Written informed consent was obtained from all participants, and we also registered with the Chinese Clinical Trial Registry (http://www.chictr.org.cn/, registration number ChiCTR-ICR-14005587).

Study design and diet

A 2×2 crossover design was used, and the technical protocol chart is depicted in Figure 1. The trial was carried

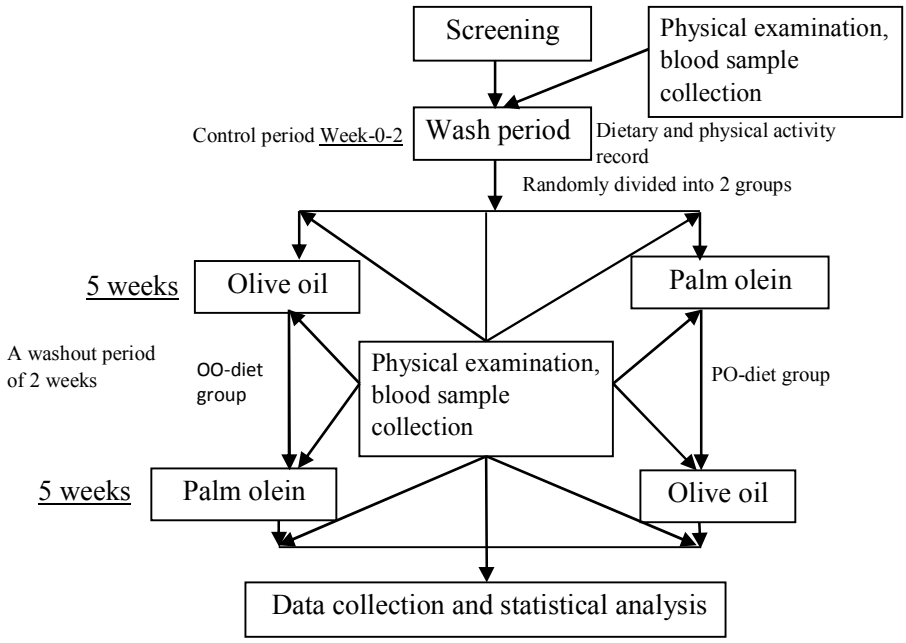

Figure 1. Flowchart of subject recruitment and experiment process

out in a sequence of four stages: a 2-week run-in period, followed by 5-week interventional period, a 2-week washout period, and then another 5-week interventional period. During the run-in period, the total dietary energy was controlled and a baseline diet was provided according to the recommendation of the Chinese Dietary Reference Intakes (DRIs); the same was done for the washout period. The two groups respectively followed the crossover of the PO diet and the OO diet to preserve the sample size and control for individual variance. Before starting the experiment, everyone involved (including researchers, participants and chefs) received training. All participants were requested to eat their meals in the factory canteen, as well as submit to a simple physical examination and blood collection for a defined period; eating-out was prohibited. Throughout the experimental, the researchers encouraged the participants to finish the provided food. The researchers also noted participants' presence at every meal, and the participants were required to deliver any leftover food to researchers after each meal for every 5 days' food weighing. The chefs in the factory canteen were trained to prepare meals in accordance with our requirements, and the experimental test fats were OO and PO, which are used as the cooking oil in a typical Chinese diet.

Both the PO and OO were purchased from the market, and the fatty acid compositions of the two oils were determined using gas chromatograph (Agilent 6890N, USA). The distribution of fatty acids in the sn-2 position was detected by ^{13}C NMR spectroscopy; specifically, ^{13}C NMR spectra were recorded using a JEOL LA-400 MHz spectrometer, fitted with a 5-mm-id dual probe $^{13}C/^{1}H$ and carefully tuned to a recording frequency of 100.40 MHz. The temperature of the probe was set to 298.15 K. The proton decoupled spectra were acquired using an inverse gated heterodecoupling sequence, for which a spectral width of 1500 Hz (where the acyl chain carbonyl carbons resonate), 8192 data points, a 90-degree pulse width, and 5.5 s of acquisition time were employed. The total repetition delay was set at 27.4 s to achieve a 99.9% recovery of z-magnetization, and free induction decay was processed in an exponential window. No zero-filling or artificial cosmetic-valued apodization was done during data processing to ensure unbiased quantitative information. However, curve fitting was performed, using a coefficient mixture ratio of 1:1 between Lorentzian and Gaussian functions and a nonlinear least-square procedure for optimization, to achieve the optimal values of the line shape parameters. All spectra were acquired with 128 scans. Standard Alice JEOL processing software was used.

A questionnaire was used to inquire about the participants' physical activity levels according to the DRIs. During the experimental stages, participants were asked to maintain their regular physical activity level, which was easy to achieve because of their fixed working arrangements. The participants were provided with the test food that was cooked with either PO or OO for three meals per day, although they did not know which test oil they had consumed. The daily dietary plan was designed by the researchers, and the menu were the same for both groups. The participants ate their three meals at the factory canteen. According to the daily recipe and the number of participants, the required amounts of the oils were calculated and individually provided to the chefs for each meal. The researchers supervised the chefs daily to use the oils according to instructions.

The energy from fat comprised approximately 30% of the total dietary energy. In addition, the intake of dietary cholesterol was controlled to be no more than 300 mg/day, and the total fat intake was not to exceed 80 g including 48 g of test oil. The researchers recorded the participants' dining status daily.

Dietary surveys were carried out at the end of each of the four stages, and the weighted food record method was used to obtain the participants' nutrients intakes.

Physical examination, blood sample collection, and detection indices

Physical examinations were conducted and fasting blood samples were measured five times: at the beginning and the end of run-in period, and at the end of each subsequent stage. Blood samples were collected after the participants had fasted for at least 12 h, and sent to Zhongda Hospital for blood index analysis. Specifically, the blood samples were collected from the antecubital vein and placed into both EDTA tubes (used for the routine blood tests) and separation gel tubes (used for blood biochemical tests). Serum was isolated from the blood at 4000 rpm for 4 min before detection. Routine blood tests (e.g., red blood cell, hemoglobin (Hb), white blood cell count, platelet count, hematocrit, mean corpuscular volume, mean corpuscular Hb, mean corpuscular Hb concentration, and red blood cell volume distribution width) were tested by an automatic hematology analyzer (XE-2100, Sysmex, Japan). The blood biochemical indices, including serum lipid profiles (TC, TG, LDL-C, HDL-C, Apolipoprotein (Apo) A1, and Apo B levels in serum) and serum glucose, were detected using an automated biochemical analyzer (LX20, Beckman, Germany), and serum insulin was identified using a radioimmunoassay (HTA Company, Peking, China). The HOMA-IR was calculated using the formula: homeostasis model assessment-insulin resistance (HOMA-IR) = [fasting glucose (mU/L) × fasting insulin (mmol/L)] / 22.5.[11] Finally, physical examination indices, including height, weight, waist circumference, hip circumference, and blood pressure, were collected by professionals at the physical examination center.

Statistics

Data were analyzed using SPSS 19.0 to determine significant differences between the two groups. Levene's normality test was used to review the normal distribution of data, and logarithmic transformations were used when appropriate. Gaussian distribution data were expressed as means ± SDs and skewed distribution data were expressed as the median and quartile. Following the methods for crossover design, the first step was to test the interaction between the PO diet group and the OO diet group for different indices, and the results showed that there were no interaction between them. Subsequently, a standard t-test was used, because there were only two experimental oils; specifically, the PO and OO diet effects was examined by adopting a two-tailed paired t-test, modified for the crossover design. Statistical significance was set at $p<0.05$.

RESULTS

Participant characteristics

Of the 120 participants who initially joined the study, 100 of them (47 men and 53 women) completed the study; notably, 11 participants were unwilling to provide the required number of blood sample, five withdrew without offering a reason, and four left the factory. The baseline characteristics of the final 100 participants are listed in Table 1.

On average, the participants were 40 years old with 18.5< BMI <24.9, TC <6.2 mmol/L, TG <2.26 mmol/L and FBG <6.1 mmol/L, respectively. None had any metabolic syndromes or a medical history of CVD according to the medical and lifestyle questionnaires. Additionally, no significant differences were found between the two groups regarding BMI, routine blood test indices (data not listed), blood glucose, insulin, or blood lipids. Thus, it was concluded that the participants were suitable for this study.

Dietary status

Each participant consumed approximately 48 g test oil daily. The calculated dietary intakes of energy, protein, fat, carbohydrates, cholesterol, as well as the contribution rates of protein, fat, and carbohydrate toward total energy, are provided in Table 2. No significant differences in the dietary intakes of energy, macronutrient composition (i.e., percentage of energy from protein, fat, and carbohydrates) were observed between the PO and OO diet groups during the four stages. Table 3 presented a comparison the blood biochemical indices and demographics between the PO and OO groups during the run-in and the washout periods. The blood lipid profiles (triglyceride, total cholesterol, HDL, and LDL) indicated that there were no

Table 1. The baseline information of recruited subjects ($\bar{x}$/M, 25th, 75th)

	PO-Diet (n=48) (Palm Olein-Diet)	OO-Diet (n=52) (Olive Oil-Diet)	*p*
Age† (years)	39.2±10.0	41.3±8.36	0.251
Sex‡ (male/female)	25/23	23/30	0.250
BMI† (kg/m^2)	22.4±2.23	22.0±1.90	0.432
TG† (mmol/L)	0.91±0.29	0.91±0.28	0.946
TC† (mmol/L)	4.30±0.64	4.41±0.64	0.422
HDL† (mmol/L)	1.19±0.17	1.23±0.22	0.288
LDL† (mmol/L)	2.47±0.44	2.55±0.38	0.363
Apo A1† (g/L)	1.15±0.23	1.18±0.24	0.523
Apo B† (gL)	0.75±0.11	0.79±0.09	0.109
Glucose† (mmol/L)	4.38±0.73	4.50±0.70	0.431
Insulin§ (uIU/mL)	6.01 (4.34, 8.29)	6.76 (5.19, 2.05)	0.129
HOMA-IR§	1.18 (0.77, 1.75)	1.32 (0.97, 1.78)	0.136

BMI: body mass index; TG: triacylglycerol; TC: total cholesterol; HDL: high density lipoprotein; LDL: low density lipoprotein; Apo: apolipoprotein; HOMA-IR: homeostasis model assessment-insulin resistance.
†Comparison between two groups, t-test.
‡χ2-test.

Table 2. Dietary nutrient intake measured for four sessions[†] per capita

	PO run-in	OO run-in	PO first	OO first	PO washout	OO washout	PO second	OO second	p[‡]
Energy (kcal)	2.57×10^3	2.58×10^3	2.28×10^3	228×10^3	2.48×10^3	2.43×10^3	2.454×10^3	2.45×10^3	0.882
Protein (g/d)	56.0	68.2	62.2	60.4	71.5	74.5	69.4	73.5	0.396
Fat (g/d)	80.8	80.9	78.2	71.2	79.8	80.5	80.2	79.1	0.531
Carbohydrate (g/d)	404	395	333	349	368	352	361	362	0.909
Cholesterol (mg/d)	67.8	131	158	240	214	142	109	138	0.548
Protein (% total energy)	8.7	10.6	10.9	10.6	11.5	12.3	11.3	12.0	0.367
Fat (% total energy)	28.3	28.2	30.8	28.2	28.9	29.8	29.4	29.1	0.459
Carbohydrate (% total energy)	62.9	61.2	58.3	61.3	59.3	58.0	58.9	59.1	0.971
experimental oil (%total energy)	16.8	16.7	18.9	19.0	17.4	17.8	17.6	17.6	0.882

PO: palm olein; OO: olive oil.
[†]Four sessions studied, namely run-in period, first experimental period, washout period and a second cross-over experimental period, separately.
[‡]Comparison between two groups of four times of dietary macro-nutrient intake with significance when $p<0.05$.

Table 3. Demographics and blood biochemical indices of comparison between PO and OO diet group after the run-in and washout period ($\bar{x}\pm s$)

	Run-in			Washout		
	PO-Diet	OO-Diet	p[†]	PO-Diet	OO-Diet	p[†]
BMI (kg/m^2)	22.4±2.38	22.1±1.97	0.513	22.0±1.95	22.2±2.59	0.591
WHR	0.82±0.06	0.83±0.06	0.455	0.83±0.06	0.84±0.06	0.747
Glucose (mmol/L)	4.11±0.50	4.18±0.57	0.542	4.11±0.44	4.19±0.66	0.491
TG (mmol/L)	1.04±0.43	0.90±0.30	0.056	0.90±0.41	0.96±0.41	0.499
TC (mmol/L)	4.38±0.71	4.45±0.68	0.593	4.47±0.61	4.24±0.62	0.069
HDL (mmol/L)	1.20±0.16	1.28±0.25	0.068	1.23±0.12	1.19±0.17	0.149
LDL (mmol/L)	2.52±0.52	2.49±0.42	0.782	2.55±0.45	2.48±0.54	0.481
Apo a1 (g/L)	1.13±0.14	1.20±0.19	0.040	1.17±0.12	1.17±0.16	0.824
Apo b(gL)	0.77±0.11	0.78±0.10	0.790	0.76±0.10	0.74±0.13	0.336
Insulin (uIU/mL)	4.23 (2.39, 6.60)	6.01 (3.89, 8.22)	0.005	5.57 (4.12, 8.42)	6.10 (4.04, 8.79)	0.754
HOMA-IR	0.82 (0.40, 1.27)	1.07 (0.68, 1.59)	0.009	1.06 (0.78, 1.59)	1.15 (0.70, 1.65)	0.777

PO: palm olein; OO: olive oil; BMI: body mass index; WHR: waist-to-hip ratio; TG: triacylglycerol; TC: total cholesterol; HDL: high density lipoprotein; LDL: low density lipoprotein; Apo: apolipoprotein; HOMA-IR: homeostasis model assessment-insulin resistance.
[†]Nonparametric test: Mann-Whitney test.

significant differences between the PO group and OO group during the run-in and washout periods (p>0.05).

Fatty acid composition of PO and OO

The composition of fatty acid in PO and OO is detailed in Table 4. Although the proportion of oleic acid in OO is higher than that in PO, the main distribution of fatty acids at the sn-2 position is PUFA and MUFA for PO and OO, respectively.

BMI and serum variables

Table 5 shows the mean values for BMI and serum variables. Before the statistical analysis, the interactions between the different blood biochemical indices were explored using SPSS; no significant interaction was found. Subsequently, the two-tailed paired t-test, modified for the crossover design, was adopted to compare the serum content (i.e., amount of TG, TC, LDL, HDL, Apo A1, Apo B, insulin, and glucose) and BMI between the PO and OO diet, no significant difference was noted.

DISCUSSION

The main findings of this experiment was that PO has a neutral effect on serum lipids in a healthy Chinese population, which is similar to the effects of OO. Neither PO- nor OO-inclusive diets raised blood cholesterol concentrations, which is consistent with some previous reports.[9,10] However, these results also contrast with Tholstrup's study, which determined that lard increased the total cholesterol and LDL-C in a sample of 19-64 year-old healthy Danish men compared with OO or PO intake (p<0.0001), although PO resulted in a lower plasma TG concentration than OO (p<0.01).[14] Although it is impossible to provide a definitive reason for this contrast, we propose that the variation may because that Tholstrup's study had no washout period before the cross-process. Many animal studies also suggest that palm oil as OO have similar effects on serum lipids profiles. One study indicated that a high fat diet (45% energy) mainly containing palm oil and OO for 8 weeks had the same effects on body weight and the accumulation of both myocellular TG and diacylglycerol in C57BL/6 mice.[20]

Other potential explanations for the neutral effects between the PO and OO intervention exist. The proportions of oleic and palmitic acid in the PO we used were 48.91/100 g and 27.46/100 g respectively, while those in the OO were 77.33/100 g and 9.67/100 g. Although the palmitic acid concentration in PO was higher than that in OO, 67.1% of the fatty acid in the sn-2 position was oleic acid. The positional distribution of fatty acids in the TG structure is believed to affect lipid metabolism.[21] Lien et al reported that when the palmitic acid at the sn-2 site in TG increases, the absorption of palmitic acid in fed experimental rats also increases, demonstrating that the palmitic acid at the triglyceride sn-2 position had excellent absorption characteristics compared with fat; notably, most palmitic acid was located at the sn-1 and sn-3 positions.[22] The same conclusion was drawn following in a feeding study on pigs.[23]

Because most of the fatty acids at the sn-2 position in the TG of palm oil are oleic, more oleic acids are absorbed and less palmitic acids are absorbed at sn-1 or sn-3 positions. Numerous studies have confirmed the nutritional value of palm oil as a result of the high monounsaturation at the crucial sn-2 position in the TGs, which makes it as healthy as OO.4 In 1997, Zhang discussed an intervention experiment in which 120 healthy men aged 18-25 years old were randomly divided into four groups for dietary interventions with palm oil, soybean oil, lard or peanut oil. Each intervention lasted for 6 consecutive weeks after a run-in period of 3 weeks.[24] The serum TC and LDL-C levels of the palm oil group were reduced by 6.7% and 13.1% (p<0.05), respectively, after dietary intervention, whereas the levels in the lard group were elevated by 22.8% and 30.7% (p<0.05), respectively.

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Table 4. The distribution of fatty acid between PO and OO in different glycerol binding position and structure

Fatty acid (g/100g)	PO-Diet	OO-Diet
Lauric acid	0.27	0
Myristic acid	0.88	0
Palmitic acid	27.46	9.67
Palmitoleic acid	0.24	0.59
Stearic acid	2.79	2.81
Oleic acid	48.9	77.3
Linoleic acid	13.87	3.74
α-linolenic acid	0.21	0.55
Arachidic acid	0.23	0.34
Cis-11-Eicosenoic acid	0.14	0.14
1,3-position† (% total fatty acid)		
Saturated fatty acid	51.3	22.1
Monounsaturated fatty acid (cis-9 monoene)	41.9	65.4
Monounsaturated (cis-11 monoene)	---	6.1
Polyunsaturated fatty acid	6.8	6.4
2-position† (% total fatty acid)		
Saturated fatty acid	4.3	---
Monounsaturated fatty acid (cis-9 monoene)	67.1	88.0
Monounsaturated (cis-11 monoene)	---	---
Polyunsaturated fatty acid	28.6	12.0
overall† (% total fatty acid)		

†Composition is quoted in mol %; ---: not detected.

Table 5. The comparison of PO and OO treatment on blood biochemical indexes ($\bar{x}\pm s$)

	PO-Diet n=48	OO-Diet n=52	$p^{\dagger}$	PO-Diet n=100	OO-Diet n=100	$p^{\dagger}$
TG (mmol/L)	1.04±0.43	0.90±0.30	0.056	0.93±0.40	0.94±0.39	0.836
TC (mmol/L)	4.38±0.71	4.45±0.68	0.593	4.34±0.69	4.36±0.68	0.861
HDL (mmol/L)	1.20±0.16	1.28±0.25	0.068	1.21±0.17	1.22±0.20	0.963
LDL(mmol/L)	2.52±0.52	2.49±0.42	0.782	2.48±0.50	2.51±0.50	0.703
Apo A1 (g/L)	1.13±0.14	1.20±0.19	0.040	1.21±0.21	1.17±0.18	0.176
Apo B (gL)	0.77±0.11	0.78±0.10	0.790	0.76±0.13	0.79±0.11	0.162
Glucose (mmol/L)	4.11±0.50	4.18±0.57	0.542	4.18±0.64	4.12±0.62	0.455
Insulin (uIU/mL)‡	4.23 (2.39, 6.60)	6.01 (3.89, 8.22)	0.005	5.50 (3.71, 7.45)	5.36 (3.75, 7.49)	0.199
HOMA-IR‡	0.8 2(0.40, 1.27)	1.07 (0.68, 1.59)	0.009	0.97 (0.65, 1.52)	0.95 (0.63, 1.40)	0.178

PO: palm olein; OO: olive oil; TG: triacylglycerol; TC: total cholesterol; HDL: high density lipoprotein; LDL: low density lipoprotein; Apo: apolipoprotein; HOMA-IR: homeostasis model assessment-insulin resistance.

†Comparison between two groups, t-test.

‡All values are median (25th percentile, 75th percentile), nonparametric test: Mann-Whitney test.

blood lipids profiles, the sample sizes varied from 21 to 33 participants,[9,10,14] which are smaller than ours. In the present study, 100 participants completed the entire trial, which was more than required according to our power analysis. Although other cross-sectional studies have notably shortcomings, such as a carry-over effect that can influence the accuracy of results. We implemented a washout period to eliminate this effect. After comparing the demographics and blood biochemical indices at the end of the run-in and washout periods, we found no remarkable distinctions in blood lipid profiles between the PO and OO groups. However, the serum insulin and HOMA-IR indices were with significantly different at the end of the run-in period. One possible reason is that the diet was meticulously controlled according to energy consumption calculations during the run-in period; thus, the participants' diet habits were altered from their previous eating habits. However, the crossover design of this study may eliminate the individual differences between the two groups because of the cross intervention of both PO and OO.

Recently, a review that focused on the relationships between published human nutrition studies and the predicted values of serum cholesterol concentrations based on total fatty acid composition and those at sn-2 position on TGs was published. The researchers concluded that sn-2 position appeared to determine the effects of PO, OO, cocoa butter, sunflower seed oil, corn oil, soybean oil, grape seed oil, groundnut oil and rice bran oil diets on serum cholesterol level, rather than the total fatty acids.25 However, considering the high SFA contents, according to the present study results, PO does not seem to influence blood lipid profiles, which may be explained by sn-2 position theory as mentioned.

Overall, our dietary crossover trial indicated that PO and OO had no significant impact on body fatness or blood lipids in a healthy Chinese population. We have confirmed several previous reports, especially regarding the 'sn-2 position theory'.[9-11] However, we only examined BMI and blood lipids profiles, and did not consider related inflammatory factors or other possible health outcomes; thus, further research is needed to develop our findings.

Conclusion

The present study supports previous findings that the effect of PO on total serum cholesterol and LDL-C in healthy individuals with normal plasma cholesterol, along with total body fatness, is neutral, compared with that of OO.

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AUTHOR DISCLOSURES

The authors declare no conflict of interest.

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