ORIGINAL ARTICLE

Canola oil and olive oil impact on lipid profile and blood pressure in women with type 2 diabetes: a randomized, controlled trial

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Summary. Objective: A number of studies indicated that olive oil (OO) and canola oil (CO) have lipidlowering and blood pressure-lowering effects. This clinical trial was done to compare the effects of CO and OO on serum lipids and blood pressure in women with type 2 diabetes. Methods: This randomized controlled clinical trial was done on 77 type 2 diabetic women. 4 weeks before the intervention, lipid-lowering drugs intakes were cut under the supervision of an endocrinologist. The participants were randomly allocated into 2 intervention groups (Balanced diet + 30 grams/day OO or CO) and one control group (Balanced diet + 30 grams/day of sunflower oil (SFO)). Dietary intakes were assessed using three 24-hour food records at baseline and at weeks 4 and 8 of the interventions. At baseline and after 8 weeks, height, weight, waist circumference, systolic blood pressure (SBP), diastolic blood pressure (DBP), serum total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), very low-density lipoprotein (VLDL-C) and high-density lipoprotein cholesterol (HDL-C) were measured and the data were statistically analyzed by SPSS 19. Results: After treatment, SBP (p=0.02), TG (p=0.01) and VLDL-C (p=0.02) were significantly decreased in OO group. None of the variables had significant changes in CO or SFO groups. There were no significant differences in the blood pressure and lipid profile among 3 groups. Conclusion: Although we found no differences between the effects of CO, OO, and SFO, it seems that replacing CO and SFO by OO may have some beneficial effects on SBP, TG and VLDL-C in women with type 2 diabetes.

Key words: canola oil, olive oil, lipid profile, blood pressure, type 2 diabetes

Introduction

The global epidemic of type 2 diabetes is increasing rapidly (1) and the number of people with diabetes has doubled in the past decade (2). On the other hand, cardiovascular diseases (CVD) are the major cause of death in patients with type 2 diabetes, which covers about 60 percent of the patients (3).

Diabetics with high blood pressure (BP) are at high risk of CVD (4,5), also abnormal lipid metabolism is common among people with type 2 diabetes, which has significant effects on atherosclerosis and CVD risk (5,6).

It has been shown that the type of dietary fats has a more important role than the amount of it in the blood lipids and BP regulations (7,8). It is clear that the consumption of vegetable oils slows down the progression of chronic heart diseases (CHD). Accordingly, the consumption of vegetable oils are recommended (9). Also, the type of fatty acids such as Monounsaturated fatty acid (MUFA), Saturated fatty acid (SFA), Polyunsaturated fatty acid (PUFA) affects serum lipids and lipoproteins, which are related to the development of atherosclerosis and CVDs (8,10).

In some studies, it is reported that MUFA intake significantly decreases TG, TC and LDL-C levels, also

increases serum HDL-C (11, 12). Omega-3 fatty acids are effective in the regulation of the genes which play a role in controlling blood lipids (13). Animal model and human studies have shown that omega-3 fatty acids have beneficial effects on plasma lipids and lipoproteins (14,15). Besides, a meta-analysis has shown that omega-3 fatty acid intake can significantly reduce BP in hypertensive patients (16). Blood pressure-lowering effect of OO consumption via its high oleic acid content has been shown; as such, OO increases the oleic acid level of the membrane, regulating the membrane lipid structure and decreasing BP (12).

OO and CO are good sources of MUFA (17). CO contains 11% omega-3 PUFAs, 53-59% MUFA, 22% omega-6 PUFAs and 7.1% saturated fatty acids (SFA) (18–20) and its ratio of omega-6 to omega-3 is appropriate (20,21). OO contains 1% omega-3 PUFAs, 73.3% oleic acid (a MUFA), 7.9% omega-6 PUFAs and 13.5% SFA (21).

Studies have shown that consumption of diets rich in OO, which contains important phenolic compounds, has a remarkable ability in reducing cholester-ol level and platelet aggregation and is inversely associated with risk of CHD (22). Given that the dysfunction of lipid metabolism is one of the most important

Table 1. Fatty acids composition of consumed oils

Fatty acids	Olive oil	Canola oil	Sunflower oil
C16	11.2	6.5	7.8
C18	2.9	2.5	4.9
C20	0	0.2	0.4
C22	0	0	0.9
C18:1	72.5	59.4	27.6
C18:2	11	21.3	58
C18:3	1	9.9	0
C20:1	0	0.2	0.4
SFA∑	14.1	9.2	14
MUFA∑	72.5	59.6	28
PUFA∑	12	31.2	58

All values are % of total fatty acids, SFA: Saturated fatty acid, MUFA: Monounsaturated fatty acid, PUFA: Polyunsaturated fatty acid

complications in patients with type 2 diabetes, and that the impact of different type of oils and their components on lipid profile and BP in diabetic patients, this clinical trial was done to compare the effects of OO and CO consumption on lipid profile and BP in type 2 diabetic women.

Material and methods

Patients

This study was held from July 2015 to November 2015. 81 females over 50 years old with type 2 diabetes and an average body mass index (BMI) of 28 kg/m² were recruited. Participants were selected from Motahhari clinic in Shiraz, according these inclusion criteria:

Female gender, records of type 2 diabetes of at least 6 months, and the routine use of SFO. Patients who need insulin and/or lipid-lowering drugs; patients with thyroid disorders, kidney and liver diseases, CVD; participating in other studies in the past 6 months; taking non-steroidal immunosuppressant, cyclosporine and warfarin; smokers, alcohol consumption, and who have TG > 400 (mg/dL) and/or LDL > 200 (mg/dL) were not included to the study.

Study design

This is a single-center, parallel group, randomized controlled clinical trial. This study is approved by the Ethics Committee of Shiraz University of Medical Sciences (IR.SUMS.REC.1394.27) and is recorded in the Iranian Registry of Clinical Trials (IRCT2015062722818N1).

All study protocols were introduced into the patients then written consents were taken. The sample size was estimated based on a previous study by POWER SSC software (23) and with consideration of the mean difference between independent groups by assuming the probability of Type 1 error (α) equal to 0.05, the power of (β -1) equal to 80 %, the mean difference (μ 1- μ 2) equal to 0.35 and standard deviation (σ) equal to 0.40. After adding 25% dropout rate, 25 persons per each group was considered.

Intakes of lipid-lowering drugs were discontinued under the supervision of an endocrinologist 4

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weeks before the intervention. Then, by using balanced block method patients were randomly allocated into 3 groups.

Using Estimated Energy Requirement (EER) equation, weight maintenance (55% carbohydrate, 18% protein and 27% fat) diet was designated for each participant. With Each diet contained 30 grams per day of vegetable oils (SFO, CO and OO) and patients were asked to add it to their salads or their boiled foods by using a small measuring cup.

Anthropometric measurements and assessment of dietary intake

At baseline and at the end of the intervention, anthropometric indices were obtained by measuring height, weight, and waist circumference.

Patients' weights were measured in light clothes, and without shoes with an accuracy of 100 grams by a digital balance (BF11 OMRON made in France). Height was measured with an accuracy of 0.5 centimeter by a non-stretchable tape measure. Then BMI was calculated as Weight (kg)/ (Height (m)* Height (m).

At baseline, week 4 and week 8 of the intervention, 3 days 24-hour record and physical activity record were filled by participants. Participants were asked not to change the recommended diet, medications and daily physical activity during the intervention.

Blood Pressure and Biochemical evaluation of blood

BP was measured by using a mercury manometer after 10-15 minute relaxation in the sitting position and away from any excitement before and after intervention. BP was measured twice with an interval of 10 minutes, then the mean of 2 measurements was recorded.

Five milliliter blood sample was taken after 12 to 14 hours fasting and was held for 15 to 20 minutes at room temperature, and then it was centrifuged for 5 minutes at 300 rpm. Serums were kept on -76°C until further analysis. TC, TG, HDL-C, VLDL-C and LDL-C were measured by the colorimetric methods by Auto Analyzer Biochemical Model BT1500 device (Pars Azmoon kit, Iran). Data were taken twice, before and after intervention.

Statistical Analysis

24-hour food records were analyzed by Nutritionist IV software. Data were analyzed by SPSS 19. P values less than 0.05 considered significant.

Normal distribution of variables was assessed using Kolmogorov-Smirnov test. Paired-Samples T-Test was used to compare the anthropometric measurements, energy, dietary intakes, lipid profile and BP at baseline and week 8 of the intervention in each group. Oneway ANOVA was used to compare mean changes of dietary intake, blood lipids and BP among the three groups, and then Post-Hoc test was used for further analysis.

Result

Of 81 participants, one in the OO group (not following the dietary regimen), one in the CO group (need for Insulin) and two in the SFO group (need for blood lipids lowering drugs) were excluded, and 77 of them completed the study (Figure 1). Participants reported no side effects associated with the consumption of the oils.

General characteristics, anthropometric status, and the dietary intake of participants at baseline are shown in Table 2. No significant differences in energy, macronutrients distribution, and fatty acid intake, weight, waist circumference, BMI and physical activ-

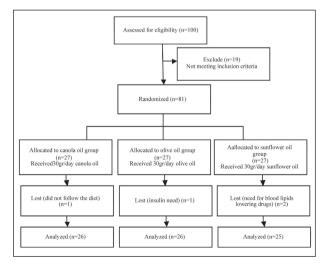


Figure 1. Participants flow diagram throughout the study

Table 2. General characteristics, anthropometric status, physical activity and dietary intake of	e of participants at base	ine.
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Variables	Olive Oil	Canola Oil	Sunflower Oil	p-Value*
Age (Year)	59±7	58±6	57±5	0.63
Height (cm)	155±5	156±4	155±4	0.67
Weight (kg)	69.8±14	70.7±7.8	68±9.9	0.68
BMI (kg/m²)	28.7±4.8	28.9±3.6	28.1±3.8	0.76
Waist Circumference (cm)	95.2±10.5	98.8±6.8	95.6±9.7	0.31
Physical Activity (METh/day)	27.7±2	27.5±2	28.6±2.2	0.20
Fat (%Energy)	29.4±3.6	27.7±4.5	28.9±5.4	0.39
Protein (%Energy)	16.8±2.4	17.5±2.9	17±2.1	0.58
Carbohydrate (%Energy)	55.7±5.4	56.1±6.5	55.2±7.1	0.89
Energy (kcal/day)	1586.6±337.3	1642.3±299.0	1542.6±262.4	0.50
Cholesterol (mg/day)	211.2±71.0	206.3±72.1	231.6±83.7	0.46
SFA (% of total energy)	13.6±3.6	13.4±3.1	13.0±4.2	0.85
MUFA (% of total energy)	9.6±3.8	10.0±3.8	12.1±2.6	0.08
PUFA (% of total energy)	22.7±3.9	22.4±8.5	18.3±1.5	0.06
Dietary fiber (% of total energy)	14.9±5.6	18.0±5.8	13.99±3.9	0.07
Soluble fiber (% of total energy)	0.4±0.3	0.4±0.2	0.39±0.3	0.54

BMI: body mass index, SFAs: saturated fatty acids, MUFAs: monounsaturated fatty acids, PUFAs: polyunsaturated fatty acids. All values are mean ± Standard deviation. * One way ANOVA.

ity were observed in the control and the intervention groups.

Dietary intakes of participants during the intervention are given in Table 3. No significant differences were observed in energy and fiber intakes, macronutrient distributions and physical activities of three groups. MUFA (P=0.001) and PUFA (P=0.001), intakes had significant differences among the three groups.

Comparisons of the mean changes in blood lipids and BP among the three groups are illustrated in Table 4. There were no significant differences in TG, TC, LDL-C, VLDL-C, HDL-C, SBP and DBP levels of the three groups. In the inter-group analysis, reduction of TG (P=0.01) and VLDL-C (P=0.02) were significant just in OO group. Reduction of serum TC, LDL-C, HDL-C, and BP were not significant in all groups.

Discussion

The results of our study showed that there were no significant differences among the effects of OO, CO or SFO consumption on lipid profile or BP in women with diabetes, however OO consumption led to significant reduction of serum TG and VLDL-C.

Based on the previous studies, effects of different kind of oils on blood lipids are controversial. TG and VLDL-C levels increased by consumption of OO instead of CO and SFO (24) while the opposite results (25,26) and no TG level changes were also observed (11). In Gustafsson and Nigam studies, consumption of CO led to significant serum TG and VLDL-C reductions (27,28). In Jones study, DHA-enriched higholeic acid canola oil improves TG (29). OO and CO are rich sources of MUFA (17). Consumption of MUFA

Table 3. Anthropometric status, physical activity and dietary intake of participants during the intervention.

Variables	Olive Oil	Canola Oil	Sunflower Oil	p-Value*
Weight (kg)	69.5±14.1	70.7±8	67.8±9.8	0.63
BMI (kg/m²)	28.6±4.9	28.9±3.7	28±3.7	0.71
Waist Circumference (cm)	94.9±10.6	98.9±7.3	95.2±9.3	0.23
Physical Activity (METh/day)	27.7±2.1	27.6±1.8	28.2±1.9	0.67
Fat (%Energy)	28.3±3.9	27.8±2.9	28±4.5	0.91
Protein (%Energy)	16.8±2.1	16.5±1.4	16.8±1.7	0.82
Carbohydrate (%Energy)	56.9±5.6	57.3±4.2	56.6±5.6	0.89
Energy (kcal/day)	1614.8±267.0	1620.8±252.0	1614.9±316.7	1.00
Cholesterol (mg/day))	197.3±76.4	183.1±49.8	178.5±65.5	0.56
SFA (% of total energy)	13.4±2.9	12.0±2.3	12.9±3.1	0.20
MUFA (% of total energy)	24.57±2.6	21.7±3.0	12.3±2.3	0.00
PUFA (% of total energy)	6.8±2.1	11.3±2.0	19.0±1.4	0.00
Dietary fiber (% of total energy)	18.5±5.5	17.7±4.7	16.3±4.2	0.28
Soluble fiber (% of total energy)	0.5±0.2	0.53±0.2	0.4±0.2	0.28

BMI: body mass index, SFAs: saturated fatty acids, MUFAs: monounsaturated fatty acids, PUFAs: polyunsaturated fatty acids. All values are mean ± Standard deviation. * One way ANOVA

increases TG entrance into the bloodstream, also makes its clearance faster (30) which in our study probably it is the reason of significant TG reduction after consumption of OO and moderate TG reduction in CO.

Although we found no significant differences in serum TC and LDL-C among the OO, CO and SFO groups. TC and LDL-C levels increased in OO and decreased in CO non-significantly. It is reported that compared to SFO, consumption of OO did not make significant reductions in serum TC and LDL-C (11,25), while opposite results are also reported (26,31). In Lichtensten study, serum TC decreased after consumption of OO or CO enriched diet (32). Nydahl and coworkers reported that TC, LDL-C and LDL-C to HDL-C ratio, reduced after the consumption of OO and CO (33), while we found different results because of using different methodologies and/ or low concentration of blood lipids at baseline.

After substitution of omega 6 PUFAs with MUFAs Griffin et al found no changes in serum TG,

TC and LDL-C, but LDL-C was rich in oleic acid and subsequently its linoleic acid content was reduced, which could reduce cholesterol ester to free cholesterol ratio in LDL-C, so helps to regulate the cellular cholesterol synthesis De Novo as an important factor against atherosclerosis (34). These results probably happened in the current study; however, LDL-C structures were not analyzed because of the financial limitations.

HDL-C had no significant differences among the OO, CO and SFO groups in the current study. In agreement with our finding several studies reported that compared to SFO, OO and CO made no significant changes in HDL-C levels (11,31,33) while some others reported a significant increase in HDL-C after consumption of OO (24,34). And also, In Jones study, DHA-enriched high-oleic acid canola oil improves HDL-C (29).

In low-fat diet, PUFA has not adversely effected on HDL-C (35). So the energy of fat can be one of the

		Olive Oil	Oil			Canola Oil);I		S	Suntlower Oil		Betweer	Between Group
	Before	After	Changes	P-Value*	Before	After	Changes P-Value*	P-Value*	Before	After	Changes	Changes p-Value* p-Value**	p-Value**
SBP (mmHg)	133.5±19.7	133.5±19.7 127.8±19.6 -5.7	-5.7±12	0.02	128.50±18.05	127.15±12.74	-1.3462±12	0.61	125.28±14.52	128.50±18.05 127.15±12.74 -1.3462±12 0.61 125.28±14.52 124.32±14.24 -0.96±12	-0.96±12	0.70	0.33
DBP (mmHa)	80.5±12.3	80.5±12.3 79.4±9.9	-1.1±8	0.55	79.54±7.77	79.54±7.77 78.62±6.92 -0.9231±6	-0.9231±6	0.49	79.00±7.91	79.92±7.85	0.92±7	0.54	0.61
TG (mg/dL)	149.4±63.2	149.4±63.2 129±58.6 -20.4±34	-20.4±34	0.01	$148.27 \pm 81.17 \ 131.92 \pm 61.06 \ -16.3461 \pm 50 \ 0.11 \ 139.04 \pm 57.91 \ 133.72 \pm 54.40$	131.92±61.06	-16.3461±50	0.11	139.04±57.91	133.72±54.40	-5.32±51	0.57	0.64
TC (mg/dL)	164.5±34.4	164.5±34.4 171.5±39.7	7±35	0.32	163.23±33.68	155.54±29.13	-7.6923±32	0.24	172.00±51.19	163.23±33.68 155.54±29.13 -7.6923±32 0.24 172.00±51.19 170.52±40.74 -1.48±29	-1.48±29	0.84	0.26
HDL -C (mg/dL) 40.6±10.6 41±8.3	40.6±10.6	41±8.3	0.4±9	0.81	39.88±8.16	38.54±7.78 -1.3461±6	-1.3461±6	0.31	40.80±7.73	40.96±6.37	0.16 ± 6	0.91	29.0
LDL -C (mg/dL) 81.5±26.8 90.7±28.8 9.2±25	81.5±26.8	90.7±28.8	9.2±25	0.1	80.08±23.91	80.08±23.91 79.73±20.55 -0.3461±22 0.94	-0.3461±22	0.94	86.32±30.36	86.32±30.36 90.32±27.97 4.00±23	4.00±23	0.40	0.27
VLDL -C (mg/dL) 31.7±14.8 25.8±11.8 -5.9±8	3 1.7±14.8	25.8±11.8	-5.9±8	0.02	26.77±12.75	26.77±12.75 26.42±12.16 -0.3461±10 0.90	-0.3461±10	06.0	27.68±11.79	27.68±11.79 26.68±10.88	-1.00±9	09:0	0.27
LDL -C/HDL -C 2.1±0.9	2.1±0.9	2.3±0.9	0.2±0.9	0.4	2.09±0.69	2.18±0.67 0.0946±0.6 0.43	0.0946±0.6	0.43	2.16±0.68	2.25±0.71	0.10 ± 0.5	0.38	0.95
TC/HDL -C	4.2±1.2	4.2±1.2 4.3±1.5 0.1±1.3	0.1±1.3	0.77	4.26±1.23	4.32±1.13	0.063±1	0.75	4.26±1.11	4.32±1.11	0.050±0.9	0.79	0.99

Table 4. Comparison of serum lipids and BP changes among the three groups before and after intervention.

HDL-C. high-density lipoprotein cholesteral LDL-C. low-density lipoprotein cholesteral VLDL-C: very low-density lipoprotein cholesteral. SBP: systolic blood pressure, DBP: Diastolic blood pressure, TG: triglyceride, TC: total cholesterol, The Value of Paired-Samples T-Test, **The Value of ANOVA Test,

factors that affect HDL-C. In this study, the average amount of energy derived from fats was 28.6 percent. So, MUFA increase and PUFA decrease in OO and CO groups compare to SFO group were not enough for a significant increase in HDL-C during 8 weeks of the treatment. So, by considering low-fat and low-energy dietaries and normal amount of HDL-C at the beginning of the treatment, no sensible effect on HDL-C had happened.

Besides, SBP and DBP did not differe significantly among the three groups, but SBP reduced significantly in OO group. Based on the previous studies, the following results were made; long-term consumption of OO reduced SBP and DBP (4,36) also positive effects of CO consumption on SBP and DBP were reported (29,37), while neutral results were also observed (38). Probably, length of study, methodology, amount of consumed oils and participants' health status are the reasons that the results of the current study is not exactly similar to the previous studies.

In conclusion, although we found no differences between the effects of CO, OO, and SFO on BP and the lipid profile of the participants, it seems that replacing of SFO by OO may have some trivial beneficial effects on SBP, TG and VLDL-C in women with type 2 diabetes.

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