The relation between dietary intake of vegetable oils and serum lipids and apolipoprotein levels in central Iran

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Abstract

BACKGROUND: The detrimental effects of partially hydrogenated vegetable oils (PHVOs) on apolipoproteins have been reported from several parts of the world. However, little data is available in this regard from the understudied region of the Middle East. The present study therefore tried to evaluate the association between type of vegetable oils and serum lipids and apolipoprotein levels among Iranians.

METHODS: In this cross-sectional study, data from 1772 people (795 men and 977 women) aged 19-81 years, who were selected with multistage cluster random sampling method from three cities of Isfahan, Najafabad and Arak in "Isfahan Healthy Heart Program" (IHHP) (Iran), was used. To assess participants' usual dietary intakes, a validated food frequency questionnaire was used. Hydrogenated vegetable oil (commonly consumed for cooking in Iran) and margarine were considered as the category of PHVOs. Soy, sunflower, corn, olive and canola oils were considered as non-HVOs. After an overnight fasting, serum cholesterol (total, low density lipoprotein (LDL) and high density lipoprotein (HDL) cholesterol) and triglyceride as well as apolipoproteins A and B were measured using standard methods.

RESULTS: Participants with the highest intakes of non-HVOs and PHVOs were younger and had lower weight than those with lowest intakes. High consumption of non-HVOs and PHVOs was associated with lower intakes of energy, carbohydrate, dietary fiber, and higher intakes of fruits, vegetables, meat, milk and grains. No overall significant differences were found in serum lipids and apolipoprotein levels across the quartiles of non-HVOs and PHVOs after controlling for potential confounding.

CONCLUSION: We did not find any significant associations between hydrogenated or non-hydrogenated vegetable oil and serum lipid and apolipoprotein levels. Thus, further studies are needed in this region to explore this association.

Keywords: Vegetable Oils, Cardiovascular Risk Factors, Lipids, Apolipoproteins, Diet.

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Introduction

High low-density lipoprotein cholesterol (LDL-C) and low high-density lipoprotein cholesterol (HDL-C) levels raise an individual's susceptibility for the progress of diabetes, cardiovascular diseases (CVDs), and other chronic diseases.[1] Increasing evidence demonstrates that all lipoprotein particles are not equally effective on cardiovascular risk.[2] Thus, function of these particles, rather than their own serum levels, may be a more accurate marker for predicting the risk of atherosclerosis.[2] Apolipoprotein A (apo A) is a main molecule carried on the surface of

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HDL-C particles. Low levels of this molecule are associated with higher cardiovascular risk.[3] On the other hand, each particle of LDL-C has one molecule of apo B on its surface regardless of triglyceride or cholesterol content.[4] Therefore, apo B levels might be considered as determinant atherogenic lipoprotein particles whose high levels are associated with higher cardiovascular risks.[5] Several investigations have found plasma apolipoprotein levels as a better predictor for cardiovascular risks than HDL-C or LDL-C levels.[6-8]

To reduce high LDL cholesterol levels, dietary intervention is generally accepted as a successful approach. Epidemiologic studies have indicated that a small change in lifestyle can significantly improve lipoprotein profile.[9,10] Dietary fat intake has long been at the center of studies assessing diet-CVD relations. Different types of fatty acids have been studied for their effects on serum lipoproteins. A recent meta-analysis demonstrated that LDL-C concentration and apo B levels significantly decreased when saturated fatty acids were replaced with unsaturated fatty acids in the diet.[11] Significant decreases in serum HDL-C concentration and apo A levels have also been reported.[11] Recent studies have primarily focused on *trans* fats and their detrimental effects on health. High intake of *trans* fatty acids results in higher LDL-C and lower HDL-C concentrations in comparison with unsaturated and saturated fatty acids.[12] High consumption of *trans* fatty acids was also associated with increased risk of developing CVD.[13]

Dietary intake of Iranian population provides a unique opportunity to assess harmful effects of *trans* fats. Iranians take 4.2% of their energy from *trans* fatty acids that is almost twice as the amount of intake in developed countries.[14] Until recently, more than 75% of total vegetable oils consumed in Iran were partially hydrogenated vegetable oils (PHVOs)[14] that are rich in saturated and trans fatty acids. This kind of dietary intake could explain the higher prevalence of CVD risk factors, such as high serum triglyceride levels and low HDL-C concentrations, among Iranians than in western populations.[15] Previous investigations from Iran have shown significant associations between consumption of partially hydrogenated fats and inflammation[16] as well as the metabolic syndrome[17] and CVD risk factors.[18] The detrimental effects of PHVOs on apolipoproteins have been reported from several parts of the world.[11,12] However, little data is available in this regard from the understudied region of the Middle East. This is particularly important considering that the picture of CVD and its risk factors is somewhat different in this part of the world as compared with other regions. While elevated serum total cholesterol and LDL-C levels are the prevalent CVD risk factors in western population, high serum triglyceride and low serum HDL-C levels seem to be the major risk factors in the Middle East.[19] Therefore, assessing vegetable oils in relation to serum apolipoprotein levels might provide additional information about this discrepancy. The objective of the present study was to evaluate the association between type of vegetable oils, serum lipid and apolipoprotein levels among Iranians.

Materials and Methods

Participants

This cross-sectional study was performed in the framework of Isfahan Healthy Heart Program (IHHP), Iran. The IHHP was a 5-6 year comprehensive integrated community-based program for CVD prevention and control. It started in 1999 and finished in 2006.[20,21] Isfahan Cardiovascular Research Centre and Isfahan Provincial Health Office cooperatively conducted this program. Totally, 12514 men and women aged above 19 years old from three cities of Isfahan, Najafabad and Arak in Iran were selected. Multistage cluster random sampling method was used to select subjects. Detailed information about the sampling process has been provided elsewhere.[20,21] The current study was performed based on data from the first phase of IHHP. We included IHHP participants whose dietary data, serum lipid profiles and apolipoproteins as well as data for confounding variables were available. Therefore, 1772 individuals (795 men and 977 women) aged 19-81 years were included in the present study. A written informed consent was obtained from each participant when they arrived at the clinic after full explanation of the study protocol. This study was approved by the Ethics Committee of Isfahan Cardiovascular Research Center and other relevant national organizations.

Assessment of dietary intake

Usual dietary intakes of participants were assessed by trained technicians using a food frequency questionnaire containing 49 food items usually consumed by Iranians. The Medical Education Development Center had confirmed the validity of this questionnaire before being used.[20] For each food item, participants reported portion sizes and consumption frequency in the previous year. The items were recorded on daily (e.g. bread), weekly (e.g. rice, meat) and monthly (e.g. fish) basis. For data analysis, all food items were converted to daily consumption by dividing weekly consumption by 7 and monthly consumption by 30. Hydrogenated vegetable oil (commonly consumed for cooking in

Iran) and margarine were considered as the category of PHVOs. Soy, sunflower, corn, olive and canola oils were considered as non-HVOs category.

Assessment of lipids and apolipoproteins

Participants were asked to fast for about 12 hours prior to blood sampling. The blood samples collected at each center in the three cities were frozen at -20°C until assayed within 72 hours in the central laboratory of Isfahan Cardiovascular Research Center. Serum total cholesterol and triglyceride levels were measured by enzymatic colorimetric method. HDL-C levels were determined after dextran sulphate-magnesium chloride precipitation of non-HDL cholesterol. LDL-C levels were calculated using the Friedewald equation.[22] Serum apolipoproteins (apo A and apo B) were measured by available commercial kits (Pars Azmon Co, Tehran, Iran).

Assessment of other variables

Socioeconomic and demographic data (sex, age, education, and occupation), medical and family history, smoking behavior, physical activity (using Baecke Questionnaire of Habitual Physical Activity)[23] and medication use were collected by trained interviewers in a face-to-face method. Height was measured using a metal ruler while the participants were barefoot. In addition, weight was measured in light clothing using a calibrated scale. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Waist and hip circumferences were measured as the smallest circumference at the costal margin and at the greatest trochanter, respectively.

Statistical methods

Statistical analyses were performed by $SPSS_{13.0}$ (SPSS Inc., Chicago, IL). Participants were categorized based on quartiles of PHVOs and non-HVOs intake. General characteristics of the participants across quartiles of PHVOs and non-HVOs intake were assessed by one-way analysis of variance (ANOVA) and chi-square test, where appropriate. Age- and energy-adjusted dietary intakes of participants across quartiles of PHVOs and non-HVOs intake were compared by the analysis of covariance (ANCOVA). Multivariate adjusted means of serum lipids and apolipoproteins across quartiles of PHVOs and non-HVOs intake were obtained by general linear model in different models. First, we adjusted the model for sex, age and total energy intake. The analyses were further adjusted for smoking, physical activity, dietary intakes of meat, grains, pulses, fruits, vegetables, and dairy and the mutual effects of HVOs or Non-HVOs. In an additional model, we included BMI to explore if the associations were mediated through obesity. Multivariate linear regression models with covariates as mentioned above were also constructed to further assess the associations. P values of less than 0.05 were considered significant.

Results

The characteristics of the participants across the quartiles of PHVOs and non-HVOs consumption are shown in Table 1. Individuals in top quartile of both non-HVOs and PHVOs were younger and tended to have lower weight and BMI. They were less likely to be males as compared to those in the bottom quartiles. No significant differences were found in the distribution of current smokers and educated subjects across the quartile categories of both non-HVOs and PHVOs intake.

Dietary intakes of the participants across the quartiles of non-HVOs and PHVOs intake are provided in Table 2. High consumption of both non-HVOs and PHVOs were associated with lower intake of energy, carbohydrate and dietary fiber. Individuals in the highest quartiles of both non-HVOs and PHVOs intake took higher percentage of their energy from fat as compared to those in the lowest quartiles Crude and multivariate-adjusted means of cardiovascular risk factors across the quartiles of non-HVOs and PHVOs intake are indicated in Table 3. In the crude models, we observed significant inverse associations between intake of PHVOs and serum triglyceride, cholesterol and apo B levels, i.e those in the highest quartile of PHVOs had lower serum triglyceride, cholesterol and apo B levels. However, after adjustment for sex, age and total energy intake, the associations disappeared. Further control for other potential confounders, including dietary intakes, did not alter the associations. Even after additional adjustment for BMI, we did not find any significant associations between PHVOs intake and serum lipid and apolipoprotein levels. Higher intake of non-HVOs was associated with lower serum triglyceride levels. This association disappeared after control for sex, age and total energy intake. Further control for other potential confounders revealed no significant association between dietary intakes of non-HVOs and serum lipid and lipoprotein levels.

To explore the linear associations between non-HVOs and PHVOs intake and metabolic variables, we applied linear regression models (Table 4). Again, in the crude model, we found a significant inverse relationship between PHVOs and serum triglyceride and cholesterol levels. However, after adjustment for confounders, no significant associations were detected between consumption of either non-HVOs or PHVOs with the metabolic variables.

Table 1. Characteristics of the participants by quartiles (Q) of partially hydrogenated (PHVOs) and non-hydrogenated vegetable oils (non-HVOs)[1]

	Quartiles of Non-HVOs[2]				P	Quartiles of PHVOs[3]				P
	Q[1]	Q[2]	Q[3]	Q[4]		Q[1]	Q[2]	Q[3]	Q[4]	
Age (y)	41.20 ± 15.60	38.60± 15.00	36.40 ± 13.50	33.9 ± 13.20	< 0.05	41.2 0± 15.30	38.50 ± 15.40	36.30 ± 13.00	34.20 ± 13.60	< 0.05
Weight (kg)	70.10 ± 12.90	68.80 ± 13.30	68.20 ± 13.00	66.50 ± 12.20	< 0.05	69.80 ± 12.90	69.10 ± 13.40	68.30 ± 13.10	66.60 ± 12.00	< 0.05
BMI (kg/m^2)	26.2 ± 4.6	26.5 ± 4.90	25.7 ± 5.20	25.5 ± 5.20	0.06	26.5 ± 4.50	26.4 ± 5.0	25.8 ± 5.3	25.2 ± 4.8	< 0.05
Male %	76.3	2.4	40.80	49.20	< 0.05	77.0	5.9	46.2	38.4	< 0.05
Physical activity (met.min/day)	870.60 ± 500	802.20 ± 479	875.00 ± 515	819.10 ± 465	0.24	845.50 ± 502	813.4 ± 482	851.80 ± 497	856.2 ± 4820	0.76
Smoking (%)	15.10	6.80	11.20	12.00	0.09	10.80	12.70	11.60	9.20	0.64
Education (%)										
0-5 years	35.900	59.20	38.50	35.80	0.14	45.00	43.10	45.90	35.0	0.22
6-12 years	35.90	38.00	48.80	51.30		43.40	48.80	44.70	55.10	
>12 years	10.20	2.90	12.70	12.90		11.50	8.10	9.40	9.90	

[1] Data is presented as means ± standard deviation unless indicated.; [2] Non- hydrogenated vegetable oils: Soy, sunflower, corn, olive and canola oils; [3] Partially hydrogenated vegetable oils: Hydrogenated vegetable oil (commonly consumed for cooking in Iran) and margarine; [4] Obtained from ANOVA for continuous variables and chi-square for categorical variables

Table 2. Dietary intakes of the participants by quartiles (Q) of partially hydrogenated (PHVOs) and non-hydrogenated vegetable oils (non-HVOs)[1]

	Non-HVOs[2]				P	PHVOs[3]				P
	Q[1]	Q[2]	Q[3]	Q[4]		Q[1]	Q[2]	Q[3]	Q[4]	
Total energy (kcal/d)	2175.50 ± 896	1729.30 ± 940	2287.10 ± 854	1986.20 ± 902	< 0.001	2100.60 ± 894	1821.30 ± 1006	2253.50 ± 809	1984.40 ± 917	< 0.001
Carbohydrate (% of energy)	61.0 ± 12.6	62.2 ± 10.3	59.2 ± 11.0	59.3 ± 9.4	< 0.001	60.9 ± 12.3	62.0 ± 10.5	59.1 ± 11.0	56.60 ± 9.50	< 0.05
Protein (% of energy)	13.6 ± 3.6	13.5 ± 3.4	12.9 ± 3.0	13.1 ± 3.2	0.07	13.5 ± 3.6	13.5 ± 3.4	13.1 ± 3.0	13.00 ± 3.20	0.18
Fat (% of energy)	27.2 ± 10.2	27.3 ± 9.3	30.0 ± 9.1	29.9 ± 8.2	< 0.001	27.4 ± 10.1	27.4 ± 9.4	29.8 ± 9.2	29.90 ± 8.30	< 0.001
Dietary fiber	18.6 ± 13.2	14.7 ± 8.4	19.3 ± 9.6	16.6 ± 9.1	< 0.001	18.5 ± 13.2	15.0 ± 8.8	19.2 ± 9.3	16.50 ± 9.10	< 0.001
Food groups (times/wk)										
Fruit	6.80 ± 4.00	7.10 ± 4.50	7.40 ± 4.40	8.50 ± 4.60	< 0.001	7.10 ± 4.50	6.80 ± 3.90	7.30 ± 4.20	8.70 ± 4.90	< 0.001
Vegetables	6.20 ± 3.40	6.20 ± 3.60	6.50 ± 3.80	7.50 ± 4.50	< 0.001	6.40 ± 3.40	6.00 ± 3.50	6.50 ± 3.90	7.40 ± 4.40	< 0.001
Meat	6.10 ± 2.90	6.20 ± 3.20	7.00 ± 3.40	7.50 ± 3.90	< 0.001	6.30 ± 3.00	6.10 ± 3.10	7.00 ± 3.40	7.50 ± 3.80	< 0.001
Milk	0.30 ± 0.80	0.40 ± 1.10	0.60 ± 1.90	1.10 ± 2.60	< 0.001	0.30 ± 0.80	0.40 ± 1.20	0.60 ± 1.90	1.10 ± 2.60	< 0.001
Grains	22.40 ± 6.20	22.90 ± 6.30	24.20 ± 5.60	24.5 0± 6.10	< 0.001	22.30 ± 6.40	23.10 ± 6.10	24.40 ± 5.70	24.40 ± 6.00	< 0.001
Non-HVOs	4.80 ± 4.80	2.20 ± 3.50	2.10 ± 4.10	1.60 ± 3.60	< 0.001	4.50 ± 4.30	2.30 ± 3.70	2.40 ± 4.30	1.90 ± 3.30	< 0.001
PHVOs	3.60 ± 2.80	7.00 ± 1.50	11.83.4	14.20 ± 3.20	< 0.001	4.40 ± 2.70	8.50 ± 1.90	14.10 ± 1.50	19.30 ± 4.40	< 0.001

[1] Data is presented as means ± standard deviation. All values are adjusted for sex, age and energy intake; [2] Non-hydrogenated vegetable oils: Soy, sunflower, corn, olive and canola oils
[3] Partially hydrogenated vegetable oils: Hydrogenated vegetable oil (commonly consumed for cooking in Iran) and margarine; [4] Obtained from ANCOVA

Table 3. Cardiovascular risk factors by quartiles (Q) of partially hydrogenated (PHVOs) and non-hydrogenated vegetable oils (non-HVOs)[1]

	Non-HVOs[2]				P	PHVOs[3]				P
	Q[1]	Q[2]	Q[3]	Q[4]		Q[1]	Q[2]	Q[3]	Q[4]	
FBS										
Crude	83.6 ± 1.2	81.6 ± 1.3	82.5 ± 1.7	81.1 ± 1.6	0.68	84.2 ± 1.3	80.4 ± 1.0	83.8 ± 1.8	80.5 ± 1.6	0.13
Model 1[5]	80.3 ± 1.6	83.3 ± 1.6	83.3 ± 1.4	81.9 ± 1.4	0.56	82.8 ± 1.4	80.1 ± 1.4	84.2 ± 1.4	81.8 ± 1.4	0.25
Model 2[6]	80.2 ± 1.6	83.3 ± 1.6	83.5 ± 1.6	82.3 ± 1.5	0.49	82.9 ± 1.5	80.3 ± 1.5	84.4 ± 1.5	81.6 ± 1.5	0.25
Model 3[7]	80.2 ± 1.7	83.4 ± 1.7	83.5 ± 1.5	82.3 ± 1.6	0.51	82.8 ± 1.5	80.2 ± 1.5	84.4 ± 1.5	82.0 ± 1.5	0.26
Triglyceride										
Crude	185.4 ± 7.4	167.4 ± 5.9	167.2 ± 7.8	154.3 ± 7.7	< 0.05	185.8 ± 6.7	166.4 ± 6.7	164.9 ± 7.7	157.3 ± 7.6	< 0.05
Model 1	169.6 ± 7.8	177.6 ± 7.8	166.9 ± 7.1	161.3 ± 7.1	0.48	181.2 ± 7.1	162.1 ± 7.1	169.5 ± 7.1	161.6 ± 7.1	0.18
Model 2	169.2 ± 7.9	179.8 ± 8.0	166.9 ± 7.1	158.6 ± 7.4	0.31	181.0 ± 7.0	156.5 ± 7.0	168.6 ± 6.9	167.4 ± 7.1	0.10
Model 3	170.6 ± 7.8	178.1 ± 7.8	167.0 ± 7.0	158.8 ± 7.2	0.35	181.9 ± 6.9	157.7 ± 7.0	168.9 ± 7.0	168.4 ± 7.0	0.11
Cholesterol										
Crude	198.9 ± 3.0	197.6 ± 3.0	190.8 ± 3.2	190.0 ± 3.0	0.08	200.1 ± 3.0	196.8 ± 2.8	188.1 ± 3.2	192.2 ± 3.0	< 0.05
Model 1	194.8 ± 3.2	194.0 ± 3.2	194.4 ± 2.9	194.4 ± 2.9	0.99	196.9 ± 2.9	194.7 ± 2.9	190.5 ± 2.9	195.2 ± 2.9	0.47
Model 2	195.8 ± 3.3	194.6 ± 3.3	194.6 ± 3.0	193.4 ± 3.1	0.96	197.5 ± 3.0	195.4 ± 3.0	190.6 ± 3.0	194.6 ± 3.0	0.44
Model 3	196.7 ± 3.2	193.8 ± 3.2	193.8 ± 2.9	194.0 ± 3.0	0.91	196.9 ± 2.9	193.7 ± 2.9	190.7 ± 2.9	196.6 ± 2.9	0.38
LDL-C										
Crude	117.1 ± 2.5	116.7 ± 2.5	114.4 ± 2.8	113.1 ± 2.5	0.66	117.1 ± 2.5	117.4 ± 2.3	111.7 ± 2.6	115.0 ± 2.5	0.37
Model 1	116.3 ± 2.8	112.4 ± 2.8	115.4 ± 2.5	117.4 ± 2.5	0.61	114.9 ± 2.5	116.4 ± 2.5	113.1 ± 2.5	113.1 ± 2.5	0.72
Model 2	116.5 ± 2.8	112.5 ± 2.8	115.5 ± 2.6	117.5 ± 2.7	0.66	115.6 ± 2.6	117.0 ± 2.5	113.4 ± 2.5	115.9 ± 2.6	0.77
Model 3	117.3 ± 2.8	112.0 ± 2.8	114.7 ± 2.6	117.9 ± 2.6	0.45	115.3 ± 2.5	115.9 ± 2.5	113.4 ± 2.5	117.2 ± 2.6	0.76
HDL-C										
Crude	45.2 ± 0.7	46.9 ± 0.6	45.2 ± 0.7	46.8 ± .06	0.09	46.2 ± 0.7	46.3 ± 0.7	45.1 ± 0.7	46.7 ± 0.6	0.37
Model 1	45.3 ± 0.7	46.8 ± 0.7	45.7 ± 0.6	46.7 ± 0.6	0.48	46.0 ± 0.7	46.1 ± 0.6	45.3 ± 0.6	46.8 ± 0.7	0.48
Model 2	45.4 ± 0.7	46.7 ± 0.7	45.7 ± 0.7	46.3 ± 0.7	0.60	46.1 ± 0.7	46.1 ± 0.7	45.4 ± 0.6	46.5 ± 0.7	0.68
Model 3	45.3 ± 0.7	46.8 ± 0.7	45.8 ± 0.7	46.3 ± 0.7	0.56	46.2 ± 0.7	46.2 ± 0.7	45.3 ± 0.6	46.4 ± 0.7	0.70
Apo A										
Crude	153.9 ± 2.2	150.2 ± 2.0	156.1 ± 2.7	157.1 ± 2.5	0.17	156.7 ± 9.2	148.9 ± 2.0	155.5 ± 2.6	156.3 ± 2.5	0.07
Model 1	154.2 ± 2.6	155.6 ± 2.6	154.9 ± 2.4	152.7 ± 2.4	0.87	155.8 ± 2.4	148.8 ± 2.4	155.7 ± 2.4	157.0 ± 2.4	0.06
Model 2	154.3 ± 2.7	156.0 ± 2.7	154.8 ± 2.4	153.0 ± 2.5	0.89	156.6 ± 2.4	149.9 ± 2.4	155.9 ± 2.4	155.6 ± 2.4	0.18
Model 3	154.6 ± 2.7	155.8 ± 2.7	154.7 ± 2.4	153.2 ± 2.5	0.92	156.7 ± 2.4	149.7 ± 2.4	155.9 ± 2.4	155.8 ± 2.5	0.15
Apo B										
Crude	119.4 ± 2.1	113.7 ± 2.4	115.1 ± 1.9	114.2 ± 2.0	0.21	120.8 ± 2.1	111.1 ± 2.0	116.3 ± 2.3	114.2 ± 1.9	< 0.05
Model 1	115.0 ± 2.3	116.1 ± 2.3	117.3 ± 2.0	113.8 ± 2.1	0.68	118.3 ± 2.1	114.1 ± 2.0	117.5 ± 2.0	116.6 ± 2.1	0.12
Model 2	115.5 ± 2.3	116.9 ± 2.3	117.0 ± 2.1	113.5 ± 2.2	0.66	119.0 ± 2.1	117.8 ± 2.1	118.4 ± 2.1	117.7 ± 2.1	0.25
Model 3	115.7 ± 2.3	116.4 ± 2.3	116.3 ± 2.0	113.9 ± 2.1	0.84	118.7 ± 2.0	113.2 ± 2.0	117.5 ± 2.0	116.9 ± 2.1	0.19

[1] Data is presented as means ± standard error; [2] Non- hydrogenated vegetable oils: Soy, sunflower, corn, olive and canola oils
[3] Partially hydrogenated vegetable oils: Hydrogenated vegetable oil (commonly consumed for cooking in Iran) and margarine; [4] Obtained from ANCOVA
[5] Adjusted for sex, age and total energy intake; [6] Further adjusted for smoking, physical activity, dietary intakes of meat, grains, pulses, fruits, vegetables, dairy, HVOs and No-HVOs
[7] Additionally controlled for BMI; LDL-C: Low-density lipoprotein cholesterol; HDL: High-density lipoprotein cholesterol

Table 4. Regression coefficients for the association of partially hydrogenated (PHVOs) and non-hydrogenated vegetable oils (non-HVOs) with serum lipid and apolipoproteins (apo A and apo B) levels

	Non-HVOs[1]			P	PHVOs[2]			P
	β	95% CI	R^2		β	95% CI	R^2	
FBS								
Crude	0.04	-4.31–0.01	0.00	0.26	-0.78	-2.11–0.54	0.00	0.24
Model 1 [3]	0.02	-4.35–0.12	0.05	0.53	0.11	-1.24–1.43	0.06	0.89
Model 2 [4]	0.03	-4.14–0.14	0.06	0.39	0.04	-1.31–1.38	0.06	0.96
Model 3 [5]	0.03	-4.68–0.14	0.07	0.41	0.18	-1.18–1.54	0.06	0.79
Triglyceride								
Crude	-0.01	-2.74–0.65	0.00	0.60	-8.72	-15.11–-2.43	0.00	< 0.05
Model 1	-0.04	-2.72–0.54	0.07	0.22	-5.10	-11.41–1.26	0.06	0.11
Model 2	-0.04	-2.32–0.93	0.07	0.17	-4.97	-11.43–1.52	0.07	0.13
Model 3	-0.05	-2.44–0.80	0.14	0.15	-3.00	-9.35–3.34	0.13	0.35
Cholesterol								
Crude	0.04	-0.70–0.64	0.00	0.17	-3.20	-5.90–-0.51	0.00	< 0.05
Model 1	-0.00	-0.71–0.64	0.11	0.96	-0.91	-3.54–1.73	0.11	0.49
Model 2	-0.02	-0.71–0.71	0.11	0.61	-1.37	-4.09–1.34	0.11	0.32
Model 3	-0.02	-0.73–0.65	0.13	0.57	-0.44	-3.08–2.19	0.18	0.74
LDL-C								
Crude	0.06	-0.31–0.91	0.00	0.07	-1.21	-3.46–1.05	0.00	0.30
Model 1	0.02	-0.29–0.92	0.06	0.50	0.22	-1.99–2.45	0.66	0.84
Model 2	0.02	-0.25–0.94	0.07	0.59	-0.29	-2.62–2.02	0.07	0.80
Model 3	0.02	-0.24–0.93	0.11	0.65	0.32	-1.97–2.61	0.11	0.78
HDL-C								
Crude	0.05	-0.19–0.10	0.00	0.14	0.03	-0.55–0.62	0.00	0.91
Model 1	0.02	-0.22–0.12	0.04	0.59	0.15	-0.43–0.74	0.03	0.61
Model 2	0.02	-0.21–0.12	0.04	0.61	0.03	-0.57–0.63	0.04	0.92
Model 3	0.02	-0.21–0.12	0.05	0.59	-0.04	-0.65–0.57	0.04	0.89
Apo A								
Crude	-0.002	-1.01–0.14	0.00	0.99	0.54	-1.52–2.63	0.00	0.61
Model 1	-0.002	-1.01–0.11	0.02	0.58	1.07	-1.04–3.17	0.021	0.32
Model 2	-0.02	-1.11–0.03	0.03	0.64	0.29	-1.89–2.47	0.02	0.79
Model 3	-0.02	-1.14–0.02	0.03	0.63	0.31	-1.89–2.50	0.03	0.78
Apo B								
Crude	0.02	-0.59–0.31	0.00	0.59	-1.48	-3.33–0.37	0.00	0.12
Model 1	-0.01	-0.62–0.31	0.05	0.75	-0.33	-2.16–1.50	0.05	0.72
Model 2	-0.02	-0.61–0.33	0.06	0.55	-0.94	-2.85–0.96	0.06	0.33
Model 3	-0.02	-0.62–0.33	0.11	0.56	-0.32	-2.19–1.55	0.11	0.73

[1] Non- hydrogenated vegetable oils: Soy, sunflower, corn, olive and canola oils; [2] Partially hydrogenated vegetable oils: Hydrogenated vegetable oil (commonly consumed for cooking in Iran) and margarine; [3] Adjusted for sex, age and total energy intake; [4] Further adjusted for smoking, physical activity, dietary intakes of meat, grains, pulses, fruits, vegetables, dairy, HVOs and Non-HVOs; [5] Additionally controlled for BMI; FBS: Fasting blood sugar; LDL-C: Low-density lipoprotein cholesterol; HDL: High-density lipoprotein cholesterol

Discussion

In the current cross-sectional study, we failed to find any significant relationships between consumption of non-HVOs, serum lipid profiles and apolipoprotein levels among a group of adults from Isfahan. Although consumption of PHVOs was inversely related to serum total cholesterol and triglyceride levels in unadjusted models, further control for potential confounders made these associations disappear. For other cardiovascular risk factors and PHVOs, there were not any relationships, neither before nor after controlling for potential confounders.

Finding no significant associations between cardiovascular risk factors and hydrogenated or non-hydrogenated vegetable oils in our study is in contrast to the previously reported findings. However, some investigators reported the same findings. For instance, Mattson et al. performed a case-control study to compare consumption of hydrogenated fat, containing high amount of trans, with non-hydrogenated fat intake. They could not show significant changes in plasma cholesterol or triglyceride levels.[24] In another study, replacement of partially-hydrogenated oil with non-HVOs had no significant effects on serum HDL-C or apo A concentrations.[25] These findings are not in line with several others. In a meta-analysis by Mozaffarian et al., replacement of trans fatty acids with saturated fatty acids (SFAs), monounsaturated fatty acids. (MUFAs) or polyunsaturated fatty acids (PUFAs), decreased serum total cholesterol and HDL-C levels.[26] Replacement of PHVOs with non HVOs has also been reported to reduce coronary heart disease (CHD) incidence.[14] Even in Iranian women, high intake of hydrogenated oils were suggested to be significantly associated with cardiovascular risk factors, whereas consumption of non-hydrogenated oils was inversely associated with cardiovascular risk factors.[27] Such results have also been found by others.[18,28,29] The discrepancy between our findings with those of others might be explained by differences in populations assessed, type of PHVOs intake, random errors and biases.[24] Another reason for this difference might be related to the food frequency questionnaire (FFQ) we used in our study. In the current study, we used a 49-item FFQ. This short FFQ might have been unable to capture usual dietary intakes in the study population. However, the validity of this questionnaire was confirmed by the Medical Education Development Center, which may not be a classical validation study. On the other hand, due to lack of a complete food composition database for Iranian food, we were unable to assess dietary intakes of individual fatty acids. It must also be kept in mind that we considered home consumption of PHVOs and non-HVOs that may lead to underestimation of the total intake and would therefore influence the associations examined. Moreover, we did not collect information about cooking methods which could have affected the quality and nature of nutrients and therefore the consequent association. Although we controlled the analysis for several potential confounders, the effect of residual confounding cannot be excluded. Cross-sectional design of the current study might also help explaining the discrepancy in findings. Lack of a significant association in the current study needs further investigations. Hence, the association between cardiovascular risk factors and consumption of PHVOs or non-HVOs remains to be confirmed in prospective studies with the use of validated questionnaires to reduce random errors.

In conclusion, we did not find a significant relation between consumption of HVOs and non-HNOs and cardiovascular risks in this population.

Conflict of Interests

Authors have no conflict of interests.

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