Hypertension is related to the degradation of dietary frying oils[1–3]

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ABSTRACT

Background: The family kitchen resembles an uncontrolled laboratory experiment, and some discrepancies in the relation between the risk of hypertension and dietary fat may be partly due to the manipulation to which the fats were subjected.

Objective: We investigated whether deterioration in the quality of the cooking oils in the family household contributes to the risk of high blood pressure.

Design: The study was cross-sectional. Anthropometric measurements were obtained for 1226 persons aged 18–65 y who were selected randomly from the municipal census of Pizarra, Spain. An oral-glucose-tolerance test was given to 1020 of these persons. Samples of the cooking oil being used were taken from the kitchens of a random subset of 538 persons. The concentrations of polar compounds and polymers were used as markers of the deterioration of the oils. The strength of association between variables was measured by calculating the odds ratio from logistic models.

Results: Hypertension was strongly associated with obesity and was influenced by sex, diabetes, and age. The presence of excess polar compounds in the cooking oil and the use of sunflower oil were related to the risk of hypertension, whereas the concentration of monounsaturated fatty acids in the serum phospholipids was negatively related to this risk. These associations remained after inclusion in the models of age, sex, obesity, and the presence of carbohydrate metabolism disorder.

Conclusions: The risk of hypertension is positively and independently associated with the intake of cooking oil polar compounds and inversely related to blood concentrations of monounsaturated fatty acids. *Am J Clin Nutr* 2003;78:1092–7.

KEY WORDS Cross-sectional study, general population, hypertension, cooking oils, frying oils, oil polar compounds, monounsaturated fatty acids

INTRODUCTION

Hypertension is a common health problem in developed countries (1). The prevalence of hypertension in the adult population of Spain is ≈45% (2), although it varies significantly according to age, sex, or obesity. Numerous dietary factors have been implicated in the pathogenesis of hypertension, and measures to control blood pressure have included reducing the intakes of salt and alcohol, as well as energy intake, in obese persons and increasing the dietary intake of potassium, magnesium, or calcium (3). Epidemiologic studies of dietary fats showed no clear relation between blood pressure and the total amount of fat consumed (4). A positive association between the estimated amount of dietary saturated fat and blood pressure was reported in some epidemiologic studies (5), but not in others (4, 6, 7). In an interventional study, a reduction in dietary total and saturated fat lowered blood pressure in both normotensive and hypertensive subjects (8). Epidemiologic and experimental studies of the relation between monounsaturated fatty acids (MUFAs) or n–6 polyunsaturated fatty acids and blood pressure have proved inconsistent, with some showing a relation (9–12) but others not doing so (6, 13–16). Although a few epidemiologic studies with small sample populations suggested that an increased intake of MUFAs is inversely related to blood pressure (7, 17), other studies with larger sample populations were unable to find this correlation (6, 13, 18). However, epidemiologic and controlled studies do suggest that n–3 fatty acids may be associated with a reduction in blood pressure (5, 14, 19, 20).

During the process of frying, the oils and fats are heated to high temperatures at the same time that they are exposed to the air, which results in a complex series of reactions that generate a wide spectrum of new components, both volatile and nonvolatile, that may have important physiologic effects. From the nutritional viewpoint, the nonvolatile products of degradation are more important, because they remain in the oil, are absorbed by the food, and are later consumed. Among these nonvolatile products are the polymers and the polar compounds (PCs) (21, 22). The biological effects of fats submitted to various conditions of thermo-oxidation were studied in animals, and the results were contradictory (23). We are unaware of any controlled studies in humans of the biological effects of the intake of fats that were subjected to repeated culinary use.

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The family kitchen resembles an uncontrolled laboratory experiment, so that some of the discrepancies found in the relation between the risk of hypertension and dietary fat may be partly due to the fact that studies have considered only the intake of fats but not the manipulation to which the fats were subjected. We studied the quality of the frying oil in the family kitchen to determine whether its deterioration contributes to the risk of hypertension in the general population.

SUBJECTS AND METHODS

The study was undertaken in Pizarra, a small city in the province of Malaga in southern Spain. Details of the study design and characteristics of the subjects in the study were reported previously (24). A total of 1226 persons aged 18–65 y were selected randomly from the municipal census, and this selection provided a definitive sample error of 4.7% for the prevalence of hypertension. All persons who were institutionalized, for whatever reason, were excluded from the study, as were pregnant women and persons with a severe clinical illness or psychological disorder. The subjects were requested by mail to attend their local health center for a medical examination. Those who failed to attend their first appointment were sent a second letter scheduling another appointment, and all those still not attending were visited at home to ascertain the reason. The final participation was 78.5%, and the final sample distribution by age and sex did not differ significantly from the population distribution (25).

All subjects were informed of the nature of the study, and they gave their written consent to participate. Likewise, all subjects and their family doctors were informed of the clinical results. The study was approved by the Ethics and Clinical Investigation Committee of Carlos Haya Hospital.

Procedures

All participants were interviewed and underwent a physical examination according to standard procedures. All the examinations were performed by the same investigators and dietitians. Standardized measurements (26) were taken of weight, height, body mass index (in kg/m^2), blood pressure, waist and abdominal circumferences, waist-to-hip ratio, and sagittal diameter. The blood pressure was measured twice with a mercury sphygmomanometer with an interval of 5 min between measurements. An oral-glucose-tolerance test was given to 1020 persons, and blood samples were taken at baseline and 120 min after the oral-glucose-tolerance test; the serum was stored at −70 °C until further study.

During the course of the interviews about dietary habits that were conducted in the homes of a random subset of 538 subjects, a sample was taken of the oil being used. To avoid the oil's being swapped for newer oil before the sampling, the family was not told of the intention to request a sample of their oil until the time of the investigator's visit. All participants authorized the collection of these samples of oil from their kitchens.

Composition and quality of the cooking oil

Fatty acids were analyzed by gas chromatography after derivatization to fatty acid methyl esters according to the standard methods of the International Union of Pure and Applied Chemistry (27). An HP 6890 chromatograph on an HP Innowax polyethylene glycol capillary column (30 m × 0.25 mm internal diameter; film thickness 0.25 μm) (Hewlett-Packard, Palo Alto, CA) was used.

PCs were quantified by adsorption chromatography, and triacylglycerol polymers were quantified by high-performance size-exclusion chromatography according to the standard method of the International Union of Pure and Applied Chemistry (27, 28). After analysis, samples were classified according to 2 criteria: fatty acid composition and concentration of PCs. Because only olive and sunflower oils are commercially available in Spain for domestic use, 3 groups of oils were defined: oils having a proportion of linoleic acid $>$ 50% were classified as sunflower oil, oils having $<$ 25% linoleic acid were classified as olive oil, and oils having 25–50% linoleic acid were classified as mixtures. Two groups of PCs were defined, depending on whether their concentration was higher or lower than 20%. This value was selected as indicative of significant degradation, because official regulations establish that frying fats and oils for human consumption must be discarded when PC concentrations reach ≈25% (29). Because there was a close correlation between PCs and polymers ($r = 0.93$), the analysis of the results was undertaken in PCs.

Blood serum measurements

Glucose concentrations were measured at baseline and 120 min after the oral-glucose-tolerance test. Baseline insulinemia was also measured in each serum sample by radioimmunoassay, and the degree of insulin resistance was calculated by the homeostasis model assessment method (30). The fatty acid composition of the serum phospholipids was calculated by extraction of the serum fat with the use of chloroform and methanol at a ratio of 2 to 1 and with the use of butylated hydroxytoluene at 0.025% (31) and by separation of phospholipids with the use of thin-layer chromatography. Fatty acid methyl esters were formed by heating the extracted fat for 30 min with 0.61 mol H_2SO_4/L in anhydrous methanol. After extraction with hexane, the fatty acid methyl esters were analyzed in a Hewlett-Packard chromatograph (Hewlett-Packard) that was equipped with a flame ionization detector and that used a BPX75 fused-silica capillary column (SGE, Villebon, France).

Classification criteria

Classification of persons with diabetes and carbohydrate metabolism disorders was made according to the 1998 American Diabetes Association criteria (32). Subjects were considered obese if their BMI was $>$ 30 (33) and were considered hypertense if they were receiving specific pharmacologic therapy or if their systolic blood pressure was $>$ 140 mm Hg and their diastolic blood pressure was $>$ 90 (34) at the second measurement.

Statistical analysis

The prevalence data, with 95% CIs, are expressed as the standardized prevalence for age of the European population according to World Health Organization recommendations (35). Contrast hypothesis testing with continuous variables was done by using one-factor or two-factor ANOVA (post hoc comparisons were made by Tukey's test), and the chi-square test was used for qualitative variables. The strength of association between variables was measured by calculating the odds ratio from logistic models. The CIs of the odds ratio were calcu-

TABLE 1
General description of the study subjects, according to sex and the presence or absence of hypertension[1]

	Men		Women		P^2		
	No hypertension (n = 207)	Hypertension (n = 175)	No hypertension (n = 406)	Hypertension (n = 232)	Sex	Hypertension	Interaction
Age (y)	37.5 ± 14.0[3]	43.6 ± 13.8	34.5 ± 11.3	40.0 ± 12.8	0.3	0.001	0.001
BMI	26.1 ± 3.6	28.7 ± 4.4	25.6 ± 4.3	31.2 ± 5.9	0.1	0.001	0.001
Waist-to-hip ratio	0.93 ± 0.07	0.97 ± 0.06	0.85 ± 0.07	0.92 ± 0.09	0.001	0.001	0.5
Sagittal diameter (cm)	21.4 ± 3.2	23.9 ± 3.7	20.2 ± 3.4	24.4 ± 3.8	0.001	0.001	0.1
Systolic blood pressure (mm Hg)	124.3 ± 9.5	147.8 ± 14.1	120.4 ± 10.8	151.2 ± 17.8	0.001	0.001	0.001
Diastolic blood pressure (mm Hg)	76.7 ± 9.1	90.1 ± 10.9	75.3 ± 8.1	90.6 ± 10.3	0.001	0.001	0.4
Prevalence of obesity (%)	36.7	61.2[4]	16.5	55.6[5]	—	—	—
Abnormal OGTT (%)[6]	16.7	40.8[7]	15.5	40.6[8]	—	—	—

[1] OGTT, oral-glucose-tolerance test.
[2] Two-factor ANOVA adjusted for age.
[3] $\bar{x}$ ± SD.
[4] $OR_{\text{Hypertension (yes or no)/obesity (yes or no), age (y)}} = 3.59$ (95% CI: 1.63, 5.81).
[5] $OR_{\text{Hypertension (yes or no)/obesity (yes or no), age (y)}} = 3.26$ (95% CI: 2.13, 4.95).
[6] Abnormal OGTT: basal glycemia > 110 mg/dL or glycemia post-OGTT > 140 mg/dL.
[7] $OR_{\text{Hypertension (yes or no)/nonnormal OGTT (yes or no), obesity (yes or no), age (y)}} = 2.84$ (95% CI: 1.71, 4.66).
[8] $OR_{\text{Hypertension (yes or no)/nonnormal OGTT (yes or no), obesity (yes or no), age (y)}} = 1.61$ (95% CI: 1.03, 2.48).

lated by Miettinen's method (36). Inclusion of variables in the multivariate models was made according to the recommendations of Kleimbaun et al (37). In all cases the rejection level for a null hypothesis was $\alpha = 0.05$ for 2 tails. Analyses were made by using SPSS software (version 10; SPSS Inc, Chicago).

RESULTS

The mean (± SD) age of the study population was 39.9 ± 3.8 y, and 46.1% (95% CI: 41.2%, 51.0%) of the men and 36.9% (95% CI: 33.1%, 40.6%) of the women had elevated blood pressure or were receiving antihypertensive therapy. For both men and women, the mean age of subjects with hypertension was significantly higher than that of subjects without hypertension (**Table 1**), with a clear slope in the whole population between age and the systolic ($r = 0.42$, $P < 0.001$) and diastolic ($r = 0.38$, $P < 0.001$) blood pressures. After adjustment for age, the body mass index, waist-to-hip ratio, and sagittal diameter were significantly greater in men and women with hypertension than in those without hypertension (Table 1). The prevalence of obesity was significantly greater in men and women with hypertension than in those without hypertension. After adjusting for age, the risk (odds ratio) of hypertension was 3.59 ($P < 0.001$) in obese men and 3.26 ($P < 0.001$) in obese women. Some degree of carbohydrate metabolism disorder (ie, impaired fasting glucose, impaired glucose tolerance, or diabetes mellitus) was present in 24.0% (95% CI: 21.38%, 26.61%) of the population. The risk (odds ratio) of hypertension in persons with carbohydrate metabolism disorder, after adjustment for age and obesity, was 2.84 ($P < 0.001$) in men and 1.61 ($P = 0.03$) in women (Table 1).

The plasma phospholipid fatty acids varied according to the type of cooking oil used (**Figure 1**). The concentration of saturated fatty acids was greatest in those who used only sunflower oil ($P = 0.05$; Figure 1); the concentration of MUFAs was greatest in those who used only olive oil ($P < 0.001$; Figure 1); and the concentration of n–6 fatty acids was greatest in those who used sunflower oil or mixed oil ($P < 0.001$; Figure 1). There was no significant variation in either the eicosapentaenoic acid or the docosahexaenoic acid (n–3 fatty acids) in the phospholipids according to the type of cooking oil used (Figure 1). The concentration of the different families of fatty acids according to age and the presence of hypertension and the corresponding analysis of variance results are shown in **Figure 2**. There was a significant variation in the concentration of saturated fatty acids in the plasma phospholipids according to age, but not according to the presence of hypertension. MUFAs increased with age and varied significantly according to the presence of hypertension. There was no variability in n–6 fatty acids according to age or hypertension. There was no variability in n–3 fatty acids according to hypertension, but there was variability according to age.

Ten percent (95% CI: 7.5%, 12.5%) of the samples of cooking oil analyzed contained > 20% PCs, which indicated excessive use of the oil. However, this proportion varied according to the type of cooking oil used, as high PC concentrations were present in just 6.2% of the samples of olive oil compared with 11.9% or 16.2% ($P = 0.005$) of the samples of sunflower oil alone or of the mixed samples, respectively.

The presence of excess PCs and the use of sunflower oil alone were associated with a risk of hypertension (logistic regression analysis in **Table 2**, models 1, 2, and 3), whereas the concentration of MUFAs in the phospholipids was negatively associated with the risk of hypertension (Table 2, model 4). The strength of the association between excess PCs in the oil, the concentration of MUFAs in the serum phospholipids, and the risk of hypertension remained after inclusion in the models of variables strongly associated with hypertension, such as age, sex, and obesity, or the presence of some carbohydrate metabolism disorder (Table 2, model 5). Other variables tested but not included in the model because of a lack of significance or because of a lack of influence on the odds ratio of the PCs included the consumption of fish per week, the homeostasis model assessment method for insulin resistance, alcohol intake, level of education, and n–3 and n–6 fatty acid composition of the plasma phospholipids.

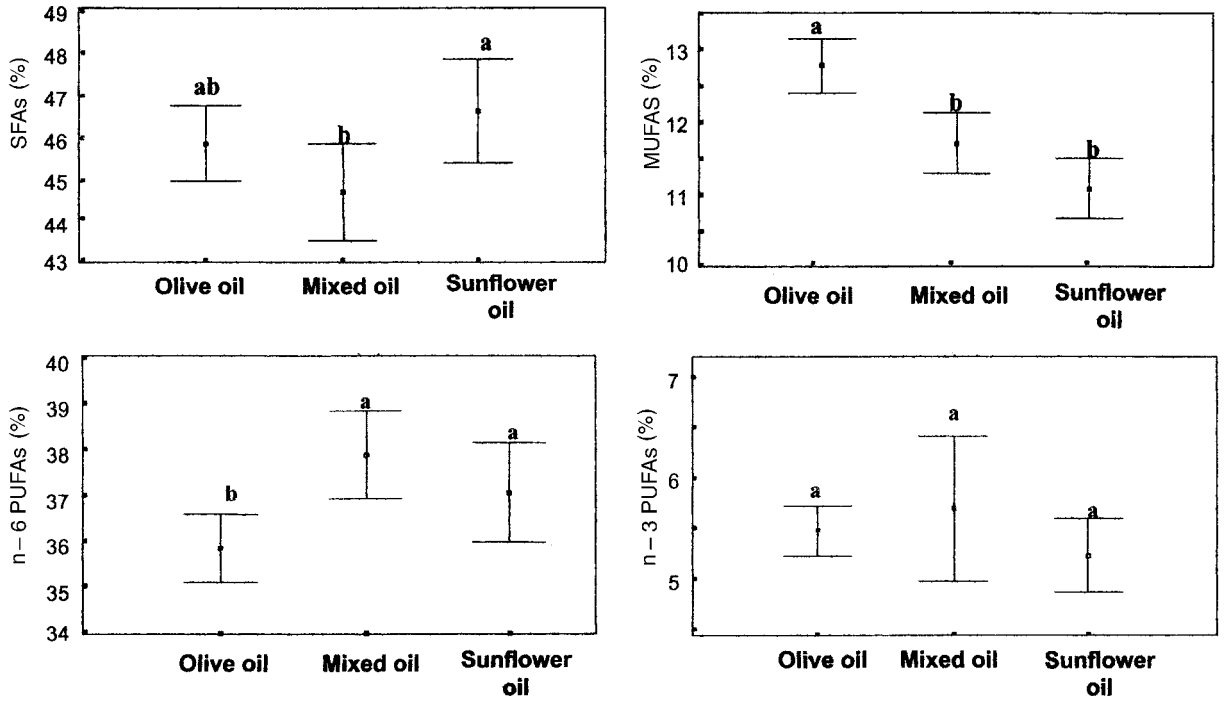

FIGURE 1. Mean (95% CI) fatty acid composition of the plasma phospholipids according to the type of cooking oil used. Means with different superscript letters are significantly different, $P < 0.05$ (ANOVA and post hoc Tukey's test). SFAs, saturated fatty acids (14:0 + 18:0 + 16:0); MUFAs, monounsaturated fatty acids (16:1, 18:1n–9); n−6 PUFAs, n−6 polyunsaturated fatty acids (18:2n–6 + 24:0n–6); n−3 PUFAs, n−3 polyunsaturated fatty acids (22:5n–3 + 22:6n–3).

DISCUSSION

The prevalence of obesity found in this study is similar to that found in other studies conducted in Spain (2). The presence of hypertension was strongly related to the presence of obesity in our study, as in others (38), and it was influenced by sex, the presence of diabetes, and especially by age. The main finding of this study is that the risk of hypertension was positively and independently related to the intake of products resulting from the degradation of vegetable oils during the cooking process in the family household and inversely related to the concentration of MUFAs in the serum phospholipids.

Vegetable oils occupy a very important place in the human diet. Numerous epidemiologic, clinical, and experimental studies have highlighted the biological importance of fats, especially the saturated and polyunsaturated n–6 fats used for human consumption (39, 40). The observations concerning olive oil are more recent, although the Seven Countries Study showed an inverse relation between consumption of MUFAs and the incidence and prevalence of cardiovascular disease (41). Prospective studies indicate that a high intake of fats, especially fats rich in saturated fatty acids, contributes to the risk of abnormal glucose tolerance and type 2 diabetes, and that fish, potatoes, and vegetables can have a protective effect (42). However, the influence on human health of fats that are altered by cooking is largely unknown. The initial advantages reported for unsaturated fats over saturated fats have been partly tarnished by the risks associated with the inadequate use of unsaturated fats (39). It is now well known that industrial handling, such as hydrogenation of n–6–rich oils, completely changes their biological properties, even to the extent of reversing their biological effect (21). We are also learning how diets rich in n–3 and n–6 polyunsaturated fats entail a greater risk of oxidation of some plasma lipoproteins (43). Nevertheless, numerous contradictions still exist between epidemiologic, clinical, and experimental studies. Some of these discrepancies may be the consequence of a lack of awareness of what happens to the oil between its production and its final consumption (eg, mixtures of different oils for cooking or frying) (44). The lower oxidation capacity of MUFAs, together with their other known biological effects, means that olive oil is one of the fats with the most advantages for human consumption (39). Indeed, the risk of hypertension in our study population was lower in those who used olive oil for frying, as in general it degrades less, and the concentration of MUFAs in the serum phospholipids was inversely related to the presence of hypertension.

During the process of frying with oils, new compounds are formed as a result of oxidation, polymerization, and hydrolysis, which substantially modify the composition of the oil and its nutritional quality (21–23). Such is the case of the PCs, which have a greater polarity than do triacylglycerols. The biological effects of fats submitted to different conditions of thermooxidation have been studied in animals, and the results have varied (23). We are unaware of any interventional study in humans to evaluate the effect of the degradation of cooking oils. Although most epidemiologic studies show no clear relation between dietary fats and blood pressure, some have found an association between the intake of saturated fats and mean blood pressure (15, 45) and a negative relation to the intake of polyunsaturated fats (15). Randomized controlled trials also leave unclear the question of dietary lipid-blood pressure relations, primarily because of design limitations (15, 45). Some studies have been

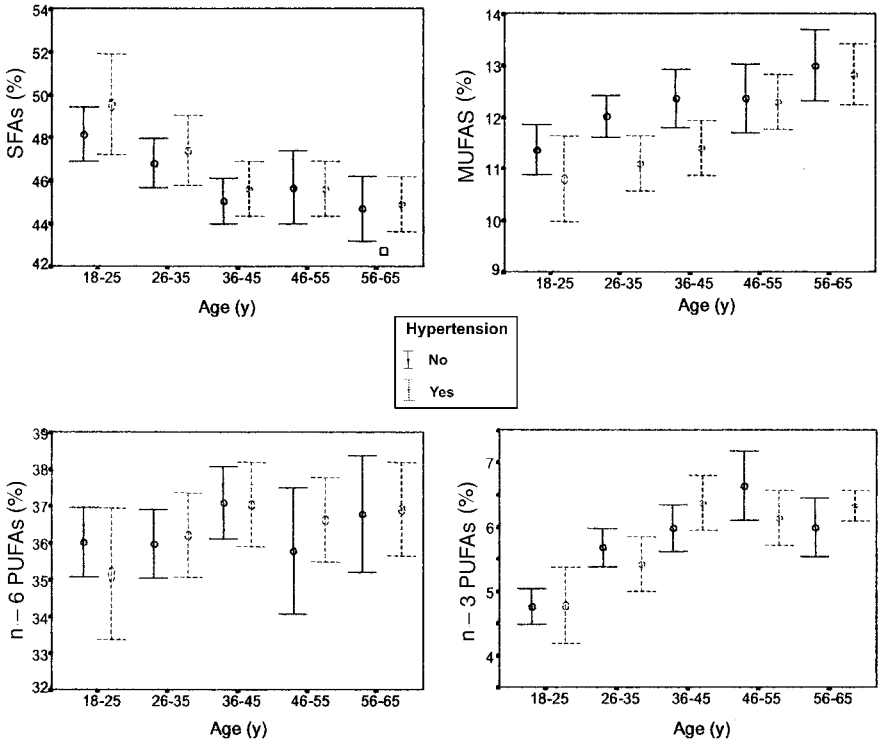

FIGURE 2. Mean (95% CI) concentrations in the plasma phospholipids of saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs), n–6 polyunsaturated fatty acids (PUFAs), and n–3 PUFAs according to age and the presence or absence of hypertension. Two-factor ANOVA for SFAs: age, $P < 0.001$; hypertension, NS; hypertension by age, NS. Two-factor ANOVA for MUFAs: age, $P < 0.001$; hypertension, $P < 0.01$; hypertension by age, NS. Two-factor ANOVA for n–6 PUFAs: age, NS; hypertension, NS; hypertension by age, NS. Two-factor ANOVA for n–3 PUFAs: age, $P < 0.001$; hypertension, NS; hypertension by age, NS.

TABLE 2
Logistic regresion analysis of the risk of hypertension according to the presence of polar compounds (PCs) in the cooking oil ($n = 538$)[1]

Model and independent variables	β	β error	Odds ratio	95% CI	P
1					
PCs ≥ 20%	0.75	0.30	2.13	1.16, 3.85	0.01
2					
OO compared with SO frying oil	0.49	0.21	1.64	1.08, 2.48	0.01
3					
PCs ≥ 20%	0.70	0.31	2.02	1.08, 3.74	0.02
OO compared with SO frying oil	0.47	0.21	1.61	1.05, 2.43	0.02
4					
PCs ≥ 20%	0.86	0.32	2.36	1.12, 4.48	0.008
MUFA phospholipids	−0.073	0.036	0.92	0.86, 0.99	0.05
5					
PCs ≥ 20%	0.84	0.36	2.33	1.12, 4.75	0.02
OO compared with SO frying oil	0.36	0.27	1.44	0.83, 2.45	0.17
MUFA phospholipids	−0.105	0.046	0.89	0.82, 0.98	0.02
Age	0.040	0.009	1.04	1.02, 1.05	0.001
Obesity	1.18	0.25	3.27	1.97, 5.36	0.001
Sex	0.34	0.24	1.41	0.86, 2.27	0.15
Abnormal OGTT	0.56	0.26	1.76	1.04, 2.94	0.03

[1] OO, olive oil; SO, sunflower oil; MUFA, monounsaturated fatty acid; OGTT, oral-glucose-tolerance test. Dependent variable: hypertension (0 = no, 1 = yes). Independent variables: PCs ≥ 20% (O = PCs < 20%; I = PCs ≥ 20%); OO compared with SO frying oil: 0 = OO or mixture, 1 = SO alone; age (y). Continuous variables: sex (0 = male, 1 = female); obesity BMI ≥ 30 (0 = no; 1 = yes); MUFA in plasma phospholipids (%); abnormal OGTT (1 = impaired glucose tolerance or diabetes mellitus; O = normal OGTT).

unable to show any effect on blood pressure due to a change in the type of dietary fat (15, 45). Earlier studies by our group showed that the fatty acid composition of the plasma phospholipids is a good marker of the type of dietary fatty acid (46, 47), and those results are confirmed in this study in that the persons who consumed olive oil had the highest concentration of MUFAs in their serum phospholipids. The negative association found in our study between the concentration of MUFAs in the phospholipids and the risk of hypertension lends support to the beneficial effect of MUFAs on blood pressure reported in the past 5 y (10, 12). This effect is strengthened by the greater resistance of olive oil to denaturalization, as witnessed by the lower proportion of PCs in those subjects using olive oil.

In summary, this study controlled the degree of degradation of commonly used oils by measuring their PCs in samples obtained from the family kitchen and identified the type of oil consumed by chromatographic analysis of the fatty acids rather than by reports of the oil purchased. Furthermore, the fatty acid composition of the plasma phospholipids was used as a biological marker of the intake of a particular type of fatty acid. The results show that degradation due to the reuse of vegetable oils, especially sunflower oil, is an independent risk factor for hypertension and that the serum concentration of MUFAs is associated negatively with this risk.

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FS and IE were responsible for study design. GR-M, MCD, JMGA, MB, MSRdA, FT, JMG-Z, EG-F, and SG-R were responsible for data collection. FS and GR-M were responsible for data analysis. All authors were responsible for writing the manuscript. None of the authors had any conflict of interest, either financial or personal, with this study.

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