

Consumption of Hydrogenated Versus Nonhydrogenated Vegetable Oils and Risk of Insulin Resistance and the Metabolic Syndrome Among Iranian Adult Women

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Remarkably rapid increase in the prevalence of the metabolic syndrome reflects the strong impact of lifestyle factors, including diet, on its etiology. In particular, dietary intake of fat may play a role in this regard. Different types of fat have been related to insulin resistance and the metabolic syndrome, but findings are inconsistent. One specific type of dietary fatty acid that has received increased attention is *trans* fatty acid (TFA); higher consumption was associated with increased risk of cardiovascular disease (1) and type 2 diabetes (2). Although some studies have reported a significant association between dietary fat intake and the metabolic syndrome (3,4), to our knowledge, such analyses have not been done separately for hydrogenated and nonhydrogenated vegetable oils. Hydrogenated vegetable oils (HVOs)—rich sources of both saturated fatty acids (SFAs) and TFAs—are extensively used for cooking in Iranian homes with average per-person intake of 14g per 1,000 kcal (5). In addition to large amounts of SFAs, these products have almost 33% of total fatty acids as TFAs. In other words, 4.2% of all calories consumed by Iranians are derived from TFAs, which is about twice the amount consumed in many developed countries (5). In the current study, we investigated whether high consumption of HVOs is contributing to a high

prevalence of the metabolic syndrome among Iranian women.

RESEARCH DESIGN AND METHODS

This cross-sectional study was conducted among Tehrani female teachers aged 40–60 years selected by a multistage cluster random sampling method. Detailed information about this sample and measurements have been provided elsewhere (6). Altogether, 486 women, with informed written consent, participated in the current study. Dietary intakes were assessed using a valid 168-item semiquantitative food frequency questionnaire (7). HVO is commonly used for cooking in Iran, and margarine was considered an HVO. Therefore, HVOs here include both fully and partially hydrogenated vegetable oils. Sunflower, corn, canola, soybean, and olive oils were defined as nonhydrogenated vegetable oils (non-HVOs). Anthropometric indexes were measured as described previously (8). A blood sample was drawn after 12-h overnight fasting, and biochemical assessment was done (6). Blood pressure was measured three times, and the mean of the three measurements was considered the participant's blood pressure. Additional information regarding age, physical activity, smoking habits, menopausal status, socioeconomic status, medical history, and cur-

rent use of medications was obtained using questionnaires. The metabolic syndrome was defined as recommended by the Adult Treatment Panel III (9). Insulin resistance was defined as the upper quartile of the homeostasis model assessment of insulin resistance score (10). To determine the associations, we used multivariable logistic regressions in different models.

RESULTS— Characteristics and dietary intakes of the study participants and multivariate-adjusted odds ratios (ORs) for the metabolic syndrome and insulin resistance are shown in Table 1. Those in the upper quintile of HVOs had higher age, BMI, and waist-to-hip ratio, were more likely to have insulin resistance, metabolic syndrome, and family history of diabetes, and had higher cholesterol intakes; whereas those in the upper quintile of non-HVOs had younger age, were slightly more physically active, less likely to have insulin resistance, and had lower intakes of energy as compared with those in the lowest quintiles. After control for age, energy intake, and other potential confounders, individuals in the highest quintile of HVOs were 2.48 (95% CI 1.51–4.92) and 2.71 (1.57–5.48) times more likely to have the metabolic syndrome and insulin resistance, respectively, compared with those in the lowest quintile, whereas those in the top quintile of non-HVOs had 44% (95% CI 3–74%) lower odds for insulin resistance compared with those in the lowest quintile. Further adjustment for dietary intakes had little impact on the associations. Additional control for BMI attenuated the associations, but they were still significant. No overall significant associations were seen between consumption of non-HVOs and the metabolic syndrome.

CONCLUSIONS— We observed a significant association between consumption of HVOs and risk of insulin resistance and the metabolic syndrome among a group of women in Iran. Consumption of

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Abbreviations: HVO, hydrogenated vegetable oil; SFA, saturated fatty acid; TFA, *trans* fatty acid.

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Table 1—Characteristics and dietary intakes of the study participants and risk of insulin resistance and the metabolic syndrome across quintiles of hydrogenated and non-hydrogenated vegetable oils

	Hydrogenated oils quintiles					P for trend†	Non-hydrogenated oils quintiles					P for trend†
	1 (lowest)	3	5 (highest)	1 (lowest)	3		5 (highest)					
Age (years)	45 ± 6	47 ± 5	53 ± 7	51 ± 6	49 ± 7	<0.01	51 ± 6	49 ± 7	46 ± 6	<0.05		
BMI (kg/m ²)	26.8 ± 3.3	27.3 ± 4.0	28.2 ± 3.7	27.3 ± 3.8	27.6 ± 4.0	<0.05	27.3 ± 3.8	27.6 ± 4.0	26.9 ± 3.3	0.39		
Waist-to-hip ratio	0.86 ± 0.08	0.87 ± 0.08	0.89 ± 0.08	0.88 ± 0.07	0.86 ± 0.09	<0.05	0.88 ± 0.07	0.86 ± 0.09	0.87 ± 0.08	0.52		
Physical activity (MET h/week)	14.8 ± 11.3	14.4 ± 10.2	14.9 ± 10.8	14.1 ± 10.6	14.3 ± 11.3	0.40	14.1 ± 10.6	14.3 ± 11.3	15.5 ± 11.7	<0.05		
Family history of diabetes (%)	9	7	11	9	10	<0.05	9	10	10	0.25		
Family history of stroke (%)	1	1	2	1	1	0.76	1	1	2	0.61		
Current daily smokers (%)	0	0	2	1	0	0.34	1	0	1	0.81		
Obese (%)‡	32	33	37	34	36	0.19	34	36	33	0.44		
Postmenopausal (%)	37	44	61	52	54	<0.01	52	54	43	0.18		
Current estrogen use (%)	24	25	28	24	21	0.11	24	21	29	0.09		
Insulin resistance (%)	16	21	36	31	22	<0.01	31	22	19	<0.01		
Metabolic syndrome (%)	20	26	39	30	31	<0.01	30	31	28	0.3		
Nutrients												
Total energy (kcal/day)	2481	2449	2395	2352	2428	0.18	2352	2428	2206	<0.05		
Carbohydrate (% of total energy)	58	60	58	60	57	0.29	60	57	59	0.11		
Protein (% of total energy)	13	13	13	12	13	0.97	12	13	12	0.65		
Fat (% of total energy)	29	27	29	28	30	0.55	28	30	29	0.51		
Cholesterol (mg/day)	157	173	191	188	196	<0.05	188	196	202	0.08		
Dietary fiber (g/day)	14	16	15	15	13	0.20	15	13	16	0.44		
Foods (g/day)												
Total hydrogenated oils	14	27	43	36	32	<0.01	36	32	15	<0.01		
Margarine	4	7	7	6	5	0.09	6	5	3	<0.05		
Hydrogenated oils for cooking	11	22	34	32	28	<0.01	32	28	13	<0.01		
Nonhydrogenated oils	31	22	13	9	18	<0.05	9	18	39	<0.01		
Fruits	216	235	221	242	209	0.24	242	209	219	0.15		
Vegetables	194	209	219	188	171	0.19	188	171	242	<0.05		
Meat and fish	69	84	93	79	86	0.12	79	86	98	<0.05		
Whole grains	116	109	124	125	106	0.30	125	106	111	0.19		
Refined grains	201	193	212	198	186	0.29	198	186	205	0.13		
Dairy	141	189	197	126	179	<0.01	126	179	209	<0.01		

Quintiles of hydrogenated vegetable oils

	5	4	3	2	1	OR for metabolic syndromes
<0.01	2.64 (1.38–5.02)	1.83 (0.94–3.54)	1.42 (0.72–2.78)	1.13 (0.56–2.26)	1.00	Crude
<0.01	2.48 (1.51–4.92)	1.75 (0.98–3.41)	1.40 (0.75–2.73)	1.15 (0.57–2.20)	1.00	Model III
<0.01	2.39 (1.57–4.98)	1.69 (1.00–3.39)	1.35 (0.78–2.72)	1.15 (0.59–2.17)	1.00	Model III*
<0.01	2.07 (1.62–4.71)	1.59 (1.06–3.43)	1.20 (0.80–2.61)	1.11 (0.60–2.15)	1.00	Model III#
						OR for insulin resistance**
<0.01	2.85 (1.45–5.60)	1.85 (0.91–3.72)	1.31 (0.49–3.47)	1.07 (0.50–2.26)	1.00	Crude
<0.01	2.71 (1.57–5.48)	1.81 (0.93–3.69)	1.27 (0.57–3.39)	1.05 (0.53–2.29)	1.00	Model I
<0.01	2.62 (1.55–5.41)	1.75 (0.98–3.61)	1.20 (0.61–3.33)	0.99 (0.59–2.25)	1.00	Model II
<0.01	2.21 (1.59–5.33)	1.68 (1.03–3.55)	1.09 (0.59–3.25)	0.99 (0.67–2.20)	1.00	Model III

Quintiles of non-hydrogenated vegetable oils

	5	4	3	2	1	OR for metabolic syndrome
0.71	0.90 (0.48–1.67)	0.72 (0.38–1.36)	1.04 (0.57–1.89)	0.77 (0.40–1.45)	1.00	Crude
0.82	0.99 (0.54–1.61)	0.75 (0.40–1.35)	1.07 (0.55–1.84)	0.77 (0.42–1.48)	1.00	Model I
0.73	1.10 (0.51–1.58)	0.81 (0.44–1.32)	1.07 (0.58–1.80)	0.84 (0.49–1.41)	1.00	Model II
0.54	1.18 (0.54–1.55)	0.86 (0.49–1.29)	1.09 (0.59–1.78)	0.89 (0.54–1.37)	1.00	Model III
						OR for insulin resistance
0.01	0.50 (0.26–0.99)	0.54 (0.28–1.04)	0.61 (0.31–1.16)	0.86 (0.46–1.59)	1.00	Crude
0.02	0.56 (0.26–0.97)	0.55 (0.31–1.03)	0.65 (0.33–1.15)	0.87 (0.48–1.56)	1.00	Model I
0.04	0.60 (0.28–0.93)	0.61 (0.35–1.00)	0.64 (0.34–1.17)	0.89 (0.44–1.56)	1.00	Model II
0.14	0.68 (0.32–0.94)	0.68 (0.37–0.97)	0.68 (0.36–1.15)	0.92 (0.46–1.55)	1.00	Model III

Data are means ± SD and OR (95% CI) unless otherwise indicated. Reported means of nutrient and food intakes were adjusted for age and total energy intake. †By using linear regression. P values for ORs obtained from Mantel-Haenszel extension χ^2 test. ‡Obesity: BMI ≥ 30 kg/m². §Metabolic syndrome was defined as the presence of three or more of the following components: 1) abdominal adiposity (waist circumference >88 cm), 2) low serum HDL cholesterol (<50 mg/dl), 3) high serum triglyceride levels (≥ 150 mg/dl), 4) elevated blood pressure ($\geq 130/85$ mmHg), and 5) abnormal glucose homeostasis (fasting plasma glucose level ≥ 110 mg/dl). ¶Adjusted for age (years), energy intake (kcal/day), cigarette smoking (yes or no), physical activity (continuous), current estrogen use (yes or no), menopausal status (yes or no), socioeconomic status (categorical), and family history of diabetes and stroke (yes or no). ††Additionally adjusted for dietary intakes including consumption of fruits, vegetables, meats and fish, whole- and refined-grains, dairy, and mutual effects of hydrogenated and nonhydrogenated vegetable oils (all as continuous). ‡‡Additionally adjusted for BMI (continuous). *Insulin resistance was estimated on the basis of fasting glucose and insulin levels, using the homeostasis model assessment for insulin resistance (HOMA-IR) method and was defined as the upper quartile of the HOMA-IR scores.

non-HVOs was inversely associated with insulin resistance but not with the metabolic syndrome. To our knowledge, this is the first investigation in which consumption of HVOs and non-HVOs was directly related to insulin resistance and the metabolic syndrome in a developing country.

HVOs are used extensively in Iran. Commercial hydrogenation of vegetable oils results in production of TFAs, higher intakes of which have been documented to increase the risk of cardiovascular disease (11), diabetes (12), and elevated inflammatory biomarkers (13). Some investigators have reported no association between TFA intakes and insulin resistance (14,15). Others have shown ethnic differences in the relationship between dietary fat intake and insulin resistance (16). The Food and Drug Administration has ruled that TFA content of packaged foods marketed in the U.S. must appear on the Nutrition Facts panel, as of 1 January 2006. The TFA content of HVOs used in Iranian households is 23–36% (5). Given the importance of insulin resistance in the development of diabetes and heart disease, establishing appropriate levels of fat in the diet is an important clinical goal.

Although metabolic syndrome is highly prevalent in the world, limited data are available assessing the contribution of HVOs to such a high prevalence. However, numerous studies have reported the adverse effects of TFAs on serum lipid profiles (17). Vega-López et al. (18) have recently shown that partially hydrogenated soybean oils, compared with soybean and canola oils, adversely altered the lipoprotein profile in moderately hyperlipidemic subjects. In Iran, it has been estimated that between 11 and 39% of coronary heart disease events might be prevented by near-elimination of TFAs (5). Consistent with results of the present study, HVOs have been reported to be associated with insulin resistance; however, the clarity of this finding remains to be identified. Adverse effects of HVOs in this study could be attributed to both TFA and SFA content of these products. A favorable association of non-HVOs and insulin resistance could be explained by the beneficial effects of polyunsaturated fatty acid on glucose metabolism (19).

In conclusion, the present findings indicate that higher intakes of HVOs were associated with a greater risk of the metabolic syndrome and insulin resistance, whereas higher intakes of non-HVOs, in the range of

energy requirements, were associated with a lower risk of insulin resistance.

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