Original Article

The association of dietary patterns and the incidence of insulin resistance after a 3-year follow-up: Tehran lipid and glucose study

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Background and Objectives: The aim of this study was to investigate the relationship between major dietary patterns and the risk of insulin resistance (IR) among an urban Iranian population. **Methods and Study design**: In this longitudinal study, 802 adult men and women were studied within the framework of Tehran Lipid and Glucose Study. Fasting serum insulin and glucose were measured at baseline and again after a 3-year of follow-up. The usual dietary intakes were assessed using a validated 168 item semi-quantitative food frequency questionnaire and major dietary patterns were obtained using principal component analysis. Logistic regression models were used to estimate the occurrence of IR across tertiles of dietary patterns with adjustment for potential confounding variables. **Results:** Mean age of participants was 39.0±11.2 years and 45.5% were men. Three major dietary patterns including the Western, traditional and healthy were extracted, which explained 25.3% of total variance in food intake. The healthy dietary pattern, loaded heavily on intake of vegetable oils, fresh and dried fruits, low-fat dairy, nuts and seeds, was accompanied with a reduced risk of insulin resistance by 51% (OR=0.49, 95% CI=0.30-0.81), and 81% (OR=0.19, 95% CI=0.10-0.36), in the second and third tertile, respectively (*p* trend=0.001). In the presence of all dietary pattern scores in the logistic regression model, a 45% reduced risk of IR was observed per 1 unit increase in healthy dietary pattern score. **Conclusion:** These findings confirmed the protective effect of a plant-based, low-fat dietary pattern against the development of insulin resistance as a main risk factor of type 2 diabetes and metabolic disorders.

Key Words: insulin, insulin resistance, type 2 diabetes, dietary pattern, principal component analysis

INTRODUCTION

Insulin resistance, defined as the inability of insulin to optimally stimulate transport of glucose into the peripheral tissues, especially the skeletal muscle and adipose tissues, is considered as the main important contributor in the pathogenesis of metabolic syndrome, type 2diabetes and cardiovascular disease.[1,2]

There has been growing interest regarding the association of dietary factors and development of metabolic disorders, especially type 2 diabetes and insulin resistance.[3] Some studies have tried to indicate the role of different dietary factors in the development of insulin resistance.[4-8] Several Western investigations have focused on Western and prudent/healthy dietary patterns associated with risk of diabetes, metabolic disorders and cardiovascular disease;[9-11] however there is limited evidence in relation to dietary patterns and insulin homeostasis especially among Asian populations. The Western dietary pattern with higher load of meats, refined grains, snacks and fast food products has been found to be a main contributor of metabolic disorders while a systematic review and meta-analysis of epidemiological studies and clinical trials con-

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firmed that adherence to some traditional dietary patterns, such as the Mediterranean diet, are associated with reduced risk of metabolic syndrome.[12,13]

Despite rapid transition of traditional to Western dietary patterns among Asians, different dietary cultures lead to substantial differences between dietary patterns among Asian and Western populations, and consequently, inconsistent associations with metabolic risk factors, making it difficult to compare studies and generalize findings to all populations. On the other hand, there is a potential interaction between social, demographics and genetic determinants and dietary patterns which should be considered in relation to insulin resistance and related metabolic disorders.[14]

Current studies which have investigated the association of dietary patterns with insulin resistance mostly had a cross-sectional settings and this controversial issue has not yet been investigated in the framework of a prospective longitudinal examination; such a setting could probably help to better justify and provide causality regarding the association between dietary pattern and insulin homeostasis. To address this gap and clarify the incidence of insulin resistance in exposure to different dietary patterns among Iranian population, we conducted this study an adult men and women participants of the TLGS study during a 3-year follow-up.

MATERIALS AND METHODS

Study population

This study was conducted within the framework of the Tehran Lipid and Glucose Study (TLGS), an ongoing community-based prospective study being conducted to investigate and prevent non-communicable diseases, in a representative sample in the district 13 of Tehran, the capital city of Iran.[15] This study was conducted in adult men and women with complete data on demographics, anthropometrics, biochemical and dietary measurements, who participated in the third (2006-2008) and fourth (2009-2011) TLGS examinations. Participants with diagnosed type 2 diabetes at baseline examination, subjects who had implausible energy intakes (<800 kcal/d or ≥4200 kcal/d) or were on specific diets, or had no follow-up information on anthropometrics and biochemical measurements at the follow-up examination, were excluded from the study. Finally, after exclusion of IR^+ participants (HOMA-IR ≥3.2) at baseline, data of 802 participants was included in the final analysis. The mean duration of the follow-up was approximately 3 years.

Written informed consent was obtained from all participants and the study protocol was approved by the Ethics Research Council of the Research Institute for Endocrine Sciences, Shahid Beheshti University of Medical Sciences.

Demographic, anthropometric and blood pressure measures

Demographics, anthropometrics, and biochemical measures were evaluated at baseline and after 3-years of follow-up. Trained interviewers collected information using pretested questionnaires. Smoking status data was obtained during face-to-face interviews. Physical activity level was assessed using the Kriska et al[16] questionnaire to obtain frequency and time spent on light, moderate, high and very high intensity activities according to a list of common daily life activities over the past year. Physical activity levels were expressed as metabolic equivalent hours per week (METs h/wk).

Weight was measured to the nearest 100 g using digital scales, while the subjects were minimally clothed, without shoes. Height was measured to the nearest 0.5 cm, in a standing position without shoes, using a tape measure. Body mass index was calculated as weight (kg) divided by square of the height (m^2). Waist circumference (WC) was measured to the nearest 0.1 cm (at anatomical landmarks), at the widest portion, over light clothing, using a soft, tape measure, without any pressure to the body. Blood pressure was measured by a trained physician; briefly, after 15 minutes rest, in a sitting position, blood pressure was measured twice on the right arm, using a standard mercury sphygmomanometer with at least a 30 seconds interval. Subject's blood pressure was estimated based on average of the two measurements.

Biochemical measures

Fasting blood samples were taken after 12-14 h, from all study participants at baseline and after 3-years of follow-up. Fasting serum insulin (FSI) was determined by the electro chemiluminescence immunoasaay (ECLIA), using Roche Diagnostics kits and the Roche/Hitachi Cobas e-411 analyzer (GmbH, Mannheim, Germany). The intra- and inter-assay coefficients of variation for insulin were 1.2 and 3.5 %, respectively. Fasting serum glucose (FSG) was measured by the enzymatic colorimetric method using glucose oxidase. Inter- and intra-assay coefficient of variation of glucose assays was <5%. Enzymatic colorimetric analysis with glycerol phosphate oxidase was used to measure triglyceride (TG) levels. High-density lipoprotein cholesterol (HDL-C) was measured after precipitation of apolipoprotein B containing lipoproteins with phosphotungstic acid. Analyses were performed using Pars Azmoon kits (Pars Azmoon Inc., Tehran, Iran) and a Selectra 2 auto-analyzer (Vital Scientific, Spankeren, Netherlands). Inter- and intra-assay coefficients of variation of all assays were <5%.

Definition of terms

Homeostatic model assessment of insulin resistance, β-cell function and insulin sensitivity were defined as follows:

HOMA-IR=fasting insulin (μU/mL)×fasting glucose (mmol/L)/22.5

HOMA-B=20×fasting insulin (μU/mL)/fasting glucose (mmol/L)-3.5

HOMA-S=1/HOMA-IR×100

These indexes have been developed as simple, inexpensive, and validated alternative tools for assessment of insulin resistance in epidemiological studies.[17,18]

In the present study, hyperinsulinemia was defined as fasting serum insulin ≥11.13 and 9.16 μU/mL in women and men, respectively.[19] Insulin resistance was also defined as HOMA-IR ≥3.2.[20]

Dietary assessment

A 168-item food frequency questionnaire (FFQ) was used

at first examination to assess typical food intake over the previous year. The validity and reliability of the FFQ were previously assessed in a random sample, by comparing the data from two FFQs, completed 1 y apart and comparing the data from the FFQs and 12 dietary recalls, respectively. The validity and reliability of the FFQ for total dietary fat were acceptable; the correlation coefficients between the FFQ and multiple 24 recalls were 0.59 and 0.38 and those between the two FFQs were 0.43 and 0.42 in male and female subjects, respectively.[21] Study of reliability, comparative validity and stability of dietary patterns derived from the FFQ also showed that there was a reasonable reliability and validity of the dietary patterns among the population over time.[11] Trained dietitians asked participants to designate their intake frequency for each food item consumed during the past year on a daily, weekly, or monthly basis. Portion sizes of consumed foods reported in household measures were then converted to grams. Energy and nutrient content of foods and beverages were analyzed using the US Department of Agriculture Food Composition Table (FCT) because the Iranian FCT is incomplete, and has limited data on nutrient content of raw foods and beverages.[22] Finally, dietary intakes of participants, including dietary energy and energy density, macronutrients, micronutrients, and food groups were determined. Twenty-two separate food groups were defined for factor analysis, according to nutrient profiles and data available.

Statistical methods

After adjustment of food groups for total energy intake using residual methods, principal component analysis (PCA) with varimax rotation based on 22 food groups was used to determine major dietary patterns. Considering eigenvalues >1, the scree plot and the interpretability of the factors, 3 factors were obtained. Food groups with an absolute component loading ≥0.30 were selected to describe the dietary pattern although all food items contributed to the calculation of dietary pattern score. The Kaiser-Mayer-Olkin statistic, considered as measure of sampling adequacy, was 0.74, which indicating a good appropriateness of factor analysis. The dietary patterns derived were defined according to our interpretation of the data and of the data available. Factor scores of the participants were calculated using sum of multiplying the intake of the standardized food groups by their respective factor loadings on each pattern. Dietary pattern scores were categorized into tertiles.

Demographics, anthropometrics and biochemical values of the participants with and without insulin resistance were compared by independent sample t-test for continuous variables or chi square test for dichotomous variables.

A univariate analysis was performed for each potential confounding variable including age (y), sex (men/women), smoking (yes/no),BMI (kg/m^2), using of medications (yes/no), physical activity (MET-h/week), total energy intakes (kcal/d), dietary intakes of total fats (% of energy), carbohydrates (% of energy), and protein (% of energy); variables with p_E<0.2 in the univariate analysis were selected for the multivariable models; p_E (p value for entry) determines which variables should be included in the final multivariable model. To clarify the association of dietary pattern scores and the occurrence of insulin resistance after 3 years, logistic regression models were used with adjustment of potential confounders. The first quartile of dietary patterns score was considered a reference in all multivariate models. Test of trend across tertiles of dietary pattern scores was conducted in logistic regression models using the median value of each respective tertile as a continuous variable in the models.

To assess potential interaction effect of dietary patterns scores on risk of IR, tertiles of each pattern score (low, middle and high) were cross-tabulated into a nine level categorical variable and the ORs were calculated across the categories in the multivariate model with adjustment of all potential confounding variables ('low-low' category was considered as the reference in the analysis). We also calculated the risk of IR per 1 unit change score for each pattern in the presence of all patterns score in the fully adjusted regression model.

All statistical analysis were conducted using SPSS (Version 16.0; Chicago, IL), and p values <0.05 were considered significant.

RESULTS

Mean age of participants was 39.0±11.2 years and 45.5% were men. Mean FSI and HOMA-IR was 8.9±5.0 μU/mL and 1.93±1.22 at baseline, respectively. Baseline characteristics of the participants with and without insulin resistance are presented in Table 1. Overall, participants who developed insulin resistance during the follow up, also had lower physical activity as well as higher BMI, waist circumference, systolic and diastolic blood pressure, FSI, FSG, TG at baseline. A higher prevalence of hyperglycemia, and a higher HOMA-IR and HOMA-B value along with a lower HOMA-S value was observed at baseline in subjects with development of insulin resistance. At baseline, there was no significant difference in total calories, carbohydrates, total fats and protein intakes between the participants but the healthy dietary score was significantly higher in healthy, compared with insulin resistant subjects.

According to factor analysis, 3 major dietary patterns including the Western, traditional and healthy were extracted, which overall explained 25.3% of total variance in food intake (Table 2). The Western dietary pattern was characterized by higher intakes of fast foods, salty snacks, mayonnaises, soft drinks, and confectioneries, while refined grains, potato, eggs, fish and poultry, legumes, hydrogenated fats, confectioneries, red meats, tea and coffee constituted the traditional dietary pattern. The healthy dietary pattern was loaded heavily on intakes of vegetable oils, fresh and dried fruits, low-fat dairy, nuts and seeds.

Logistic regression analysis showed no statistically significant association between Western and traditional dietary patterns with the development of insulin resistance after 3 years (Table 3).

In both the crude model and the age-, sex- and BMI-adjusted model, a higher score of healthy dietary pattern was associated with a reduced risk of insulin resistance. After additional adjustment of physical activity, smoking and medications, the highest compared with the lowest tertile of healthy dietary pattern score was accompanied with a substantially reduced risk of insulin resistance

Table 1. Baseline characteristics of the participants

	Insulin resistance (n=802)		*p* value
	-	+	
Age at baseline (y)	38.8±11.3	39.7±11.2	0.39
Men (%)	45.5	50.4	0.22
Smoking (%)	10.1	6.3	0.24
Physical activity (MET-h/week)	37.5±2.2	28.4±3.2	0.023
Body mass index (kg/m^2)	26.2±4.5	29.9±5.1	0.001
Waist circumference (cm)	87.3±12.6	97.2±12.3	0.001
Systolic blood pressure (mm Hg)	108±14.9	113±14.1	0.001
Diastolic blood pressure (mm Hg)	72.1±10.3	76.6±9.7	0.001
Serum triglycerides (mg/dL)	124±71	181±96	0.001
HDL-C (mg/dL)	42.9±9.5	38.1±9.5	0.001
Fasting serum insulin (mg/dL)	7.7±3.5	14.5±6.9	0.001
Fasting serum glucose (mg/dL)	85.2±7.9	91.3±10.3	0.001
HOMA-IR	1.64±0.83	3.31±1.77	0.001
HOMA-B	136±72	207±122	0.001
HOMA-S	78.1±45.1	37.9±21.8	0.001
Hyperglycemia (%)	5.9	19.7	0.001
Hyperinsulinemia (%)	24.7	27.0	0.31
Dietary pattern scores			
Western	-0.03±0.95	0.07±1.1	0.28
Traditional	0.03±0.99	0.09±1.0	0.53
Healthy	0.02±0.97	-0.30±1.0	0.001
Energy intakes (Kcal)	2260±700	2353±798	0.17
Carbohydrates (% of energy)	57.6±7.1	58.8±7.8	0.06
Total fats (% of energy)	31.4±6.9	30.3±7.8	0.09
Protein (% of energy)	13.5±2.4	13.4±2.1	0.69

Data are mean±SD.

Table 2. Component loadings for major dietary patterns

	Dietary patterns		
	Western	Traditional	Healthy
Refined grains	-	0.39	-0.24
Fast foods	0.57	-	-
Potato	-	0.57	-
Salty snacks	0.59	-	0.21
Mayonnaise	0.51	-	-
Soft drinks	0.64	-	-
Eggs	-	0.45	-
Vegetables	-	0.25	0.53
Whole grains	-0.28	0.24	-
Fruits (fresh and dried)	-	-	0.63
White meats (fish and poultry)	-	0.30	0.23
High-fat dairy	0.28	-	0.27
Low-fat dairy	-	-	0.45
Jams and compotes	-	-	-
Vegetable oils	-	-	0.45
Hydrogenated oils	0.26	0.34	-
Confectioneries	0.33	0.35	-
Red meats	-	0.45	-
Organ meats	0.23	-	-
Tea and coffee	-	0.40	-
Nuts and seeds	-	-	0.53
Legumes	-	0.50	0.22
Variance (%)[†]	12.6	6.7	5.9

Values are factor loadings of food patterns measured by factor analysis (n=802).
Absolute value <±0.2 are excluded from the from for simplicity.
[†]Eigenvalues >1.

(OR=0.34, 95% CI= 0.19-0.58, *p* for trend= 0.004). In the final model, additional adjustment for total calorie, dietary intakes of carbohydrates, fats and protein, the risk of insulin resistance was reduced by 51% (OR=0.49, 95% CI= 0.30-0.81), and 81% (OR=0.19, 95% CI= 0.10-0.36), in the second and third tertiles, respectively (*p* for

Table 3. The odds ratio (95% CI) of insulin resistance across tertile categories of dietary patterns

Dietary patterns	T1	T2	T3	*p* for trend
Western				
Crude model	1.00 (ref.)	1.23 (0.78-1.96)	1.33 (0.84-2.11)	0.73
Model 1	1.00 (ref.)	1.19 (0.72-1.95)	1.42 (0.86-2.35)	0.88
Model 2	1.00 (ref.)	1.20 (0.72-1.98)	1.44 (0.86-2.39)	0.89
Model 3	1.00 (ref.)	1.30 (0.76-2.17)	1.58 (0.89-2.73)	0.93
Traditional				
Crude model	1.00 (ref.)	1.21 (0.77-1.91)	1.03 (0.65-1.63)	0.14
Model 1	1.00 (ref.)	1.19 (0.75-1.87)	1.00 (0.63-1.60)	0.11
Model 2	1.00 (ref.)	1.13 (0.62-1.85)	1.07 (0.69-1.87)	0.11
Model 3	1.00 (ref.)	1.07 (0.64-1.78)	0.90 (0.45-1.65)	0.13
Healthy				
Crude model	1.00 (ref.)	0.71 (0.46-1.08)	0.44 (0.27-0.72)	0.004
Model 1	1.00 (ref.)	0.63 (0.39-1.00)	0.34 (0.20-0.58)	0.009
Model 2	1.00 (ref.)	0.65 (0.40-1.03)	0.34 (0.19-0.58)	0.004
Model 3	1.00 (ref.)	0.49 (0.30-0.81)	0.19 (0.10-0.36)	0.001

Logistic regression models were used. Model 1: adjusted for age (years), sex (men/women), body mass index (kg/m^2); Model 2: additional adjustment for physical activity (MET-h/week), smoking (yes/no), and using of medications (yes/no); Model 3: additional adjustment for total energy intakes (kcal/d), dietary intakes of total fats (% of energy), carbohydrates (% of energy), and protein (% of energy).

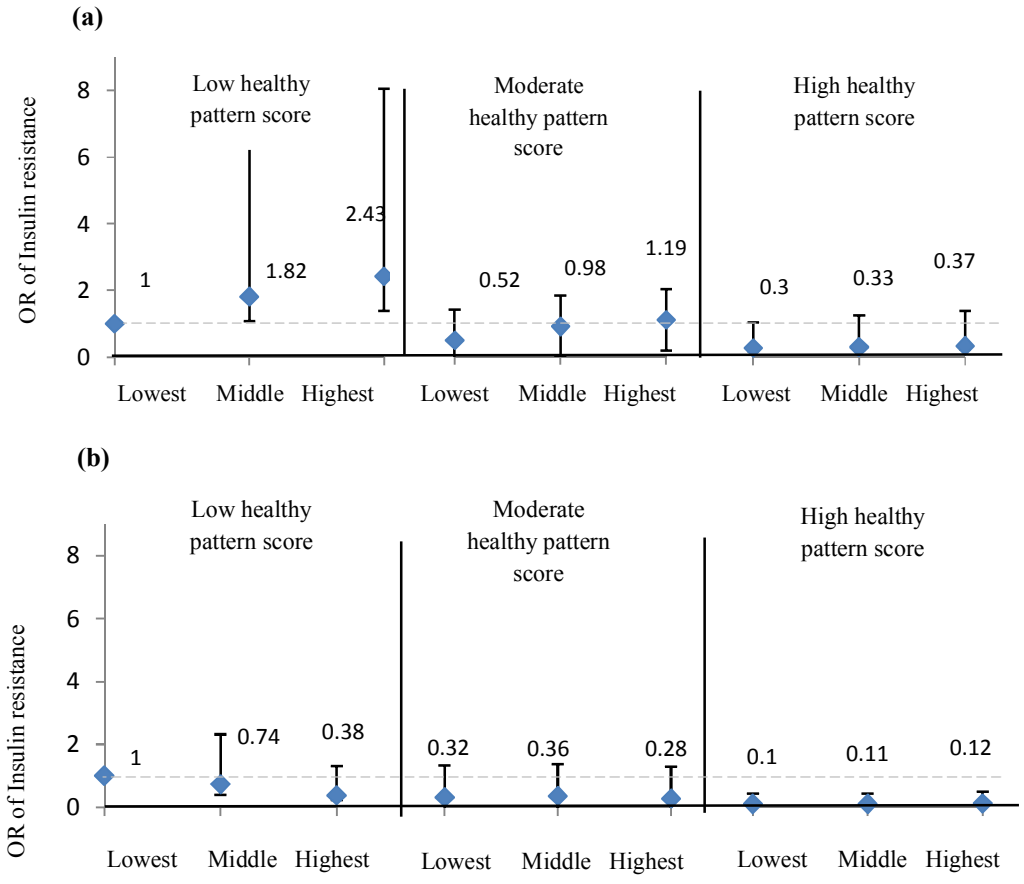

Figure 1. Tertiles of Western dietary scores. a) Odds ratios (ORs) of combined classification of healthy and Western dietary pattern scores for the incidence of insulin resistance; b) ORs of combined classification of healthy and traditional dietary pattern scores for the incidence of insulin resistance (logistic regression models were used with adjustment of age (years), sex (men/women), body mass index (kg/m2), physical activity (MET-h/week), smoking (yes/no), using of medications (yes/no), total energy intakes (kcal/d), dietary intakes of total fats (% of energy), carbohydrates (% of energy), and protein (% of energy).

trend= 0.001).

To assess potential interaction effect of dietary pattern scores in the development of IR, categories of healthy and the Western or traditional dietary patterns were simultaneously interred in the logistic regression models in the presence of all the potential risk factors for IR (Figure1). The lowest healthy pattern score along with the highest Western pattern score was accompanied with considerably increased in the risk of IR (OR=2.43, 95% CI=1.05-5.63); in contrast, highest compared with the lowest healthy pattern score in combination with the lowest score of Western pattern reduced the risk of IR (OR=0.3,

95% CI=0.11-0.83).Highest compared with the lowest category of healthy pattern score in combination with the three categories of traditional pattern score significantly reduced the risk of IR. Moreover, when the scores of three dietary patterns were simultaneously included as continuous variables in the final logistic regression model, the chance of IR was 1.11 (95% CI=0.88-1.39), 0.97 (95% CI=0.79-1.22), and 0.55 (95% CI=0.41-0.73) for Western, traditional and healthy pattern scores, respectively.

DISCUSSION

In this population-based prospective study of adult men and women, followed for a median of 3 years, we demonstrated that the healthy dietary pattern, characterized by higher load of vegetable oils, fresh and dried fruits, low-fat dairy, nuts and seeds, was an independent predictor of the incidence of insulin resistance. Other major dietary patterns, Western and traditional, had no statistically significant association with insulin resistance. Interaction effects of dietary pattern scores also indicated that highest adherence to Western pattern along with lowest score of healthy dietary pattern increased the risk of IR, whereas highest adherence to healthy pattern could modify the unfavorable effects of the Western pattern; overall, the lowest OR of IR was observed in subjects with higher healthy scores along with lower Western scores. Combination of healthy and traditional dietary pattern scores showed that subjects who had the highest score of healthy pattern along with the lowest score of traditional pattern had also lowest OR of IR. In the presence of all dietary pattern scores in the logistic regression model, a 45% reduced risk of IR was observed per 1 unit increase in healthy dietary pattern score.

An overview of previous studies revealed relatively similar findings regarding dietary patterns, type 2 diabetes and metabolic syndrome; however limited data are specifically available for insulin resistance indices. A follow-up of 42,504 men, aged 40-75 years, indicated that the prudent dietary pattern score (characterized by higher consumption of vegetables, fruit, fish, poultry, whole grains) was associated with a modestly lower risk of type 2 diabetes (RR=0.84, 95% CI=0.70-1.00) while the Western pattern score (characterized by higher load of red meat and processed meats, french fries, high-fat dairy products, refined grains, sweets and desserts) increased risk of type 2 diabetes (RR=1.59, 95% CI=1.32-1.93).[9] Similarly, a prospective investigation on the associations between major dietary patterns and risk of type 2 diabetes in women showed an increased risk for diabetes (OR=1.49, 95% CI= 1.26-1.76) in women who had higher scores in the Western pattern.[10] A 15-year follow-up of adult men and women who participated in the China Health and Nutrition Survey indicated that long-term adherence to healthier dietary patterns was associated to lower hemoglobin A_1c (-1.64, 95% CI=-3.17, -0.11), lower HOMA-IR (6.47%, 95% CI=-17.37, 4.42) and a reduced risk of type 2 diabetes (OR=0.86, 95% CI=0.44, 1.67).[8] Another study on 5390 men and women, aged 45-84 years, free of prevalent diabetes and clinical cardiovascular disease, showed that higher scores in Mediterranean dietary pattern, characterized by a high consumption of whole grains, olive oil, legumes, vegetables, fruits, cereals, moderate to high consumption of fish, moderate to low consumption of meat and processed meats, milk and dairy products, were associated with a lower levels of serum glucose and insulin at baseline; after a 6-year follow-up there was no significant association between Mediterranean dietary pattern and the incidence of type 2 diabetes.[23] A nested case-control study on adults also showed no significant association between Western or healthy dietary patterns with the risk of type 2 diabetes, but a higher score for the traditional dietary pattern, included higher load of whole grains, legumes, egg and red meat, was found to be related with an 18% lower risk (OR per 1 SD score=0.82, 95% CI 0.67–0.99).[24]

Adherence to the dietary approaches to stop hypertension (DASH) diet, a diet rich in fiber, fruits, vegetables, and low-fat dairy products, was also accompanied with improvement of insulin sensitivity.[25] In one cross-sectional investigation conducted among a Japanese population, the bread and dairy products pattern and the prudent dietary pattern, characterized by high load of vegetables and fruits, were inversely correlated to a lower prevalence dysglycemia while the high fat/Western, characterized by high load of fried foods, fried dishes and meat was positively correlated with HOMA-IR.[6] Another cross-sectional study of Asian adult women showed that higher scores of the healthy dietary pattern were accompanied with a reduced risk of metabolic syndrome (OR=0.61, 95% CI=0.30-0.79) and insulin resistance (OR=0.51, 0.24-0.88),whereas higher scores of the Western and traditional dietary patterns were associated with a relatively great risk for metabolic syndrome and abnormal glucose homeostasis.[26]

Favorable effects of healthy dietary patterns such as the Mediterranean pattern, DASH diet, and the Nordic diet on metabolic disorders have been mainly attributed to higher content of fruits, vegetables, whole grains, dairy and dairy components, calcium, vitamin D, and whey protein, as well as monounsaturated fatty acids, and omega-3fattyacids.[27] Effects of dietary patterns on insulin homeostasis may also be mediated through different glucose and insulin responses following meal patterns containing various amounts and types of carbohydrates.[28] Considering recent findings that highlight the critical role of dietary fatty acids on insulin homeostasis during postprandial periods,[29] different fatty acid profiles of various dietary patterns may be partly responsible for diet-induced insulin resistance. Some other different modalities such as glycemic index and glycemic load, fiber, phytochemicals and antioxidant content may also explain different metabolic outcomes of dietary patterns. Several mechanisms have been suggested to link the effects of diet on insulin resistance; there is evidence showing that the effects of diet on leptin and adiponectin, and other cytokines such as tumor necrosis factor-α and interleukin-6could modulate insulin sensitivity.[30,31] Chemerin, a recently discovered chemokine regulates glucose uptake in both adipose tissue and skeletal muscle, was also found to be mediate the effect of diet on insulin homeostasis and HOMA-IR.[32] Considering the pathological effect of oxidative stress and inflammatory pathways on β-cell function and insulin signaling, and the determinant role of diet on endogenous

oxidative/anti-oxidative balance and systemic sub-clinical inflammation, it is possible that the indirect effect of dietary patterns on the development of insulin resistance may be linked to these mechanisms.[33-35]

The strengths of this study were a prospective setting and use of a validated FFQ to assess regular dietary intakes and to determine major dietary patterns. Some limitations of our study need to be considered in the interpretation of findings. The dietary patterns were assessed using food intake data only, whereas eating behaviors such as meal and snacking patterns may provide more accurate information regarding regular dietary patterns. Use of factor analysis for dietary data reduction has also been criticized due to its subjectivity in nature and limited generalizability to other populations. Moreover, lack of data on postprandial levels of glucose and insulin to calculate the disposition index and accurately justify homeostasis models of insulin, was another limitation of this study.

In conclusion, the findings of the present study demonstrated that a dietary pattern with a higher amounts of vegetable oils, fresh and dried fruits, nuts and seeds, as well as low-fat dairy products, was associated with a lower incidence rate of insulin resistance, findings that indicate the protective effects of plant-based and low-fat foods against the development of insulin resistance and its consequential metabolic disorders.

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AUTHOR DISCLOSURES

The authors declare no conflict of interest.

REFERENCES

1. Meier JJ, Bonadonna RC. Role of reduced beta-cell mass versus impaired beta-cell function in the pathogenesis of type 2 diabetes. Diabetes Care. 2013:36(Suppl 2);113S-9S. doi: 10. 2337/dcS13-2008.
2. Paneni F, Costantino S, Cosentino F. Insulin resistance, diabetes, and cardiovascular risk. Curr Atheroscler Rep. 2014;16:419. doi: 10.1007/s11883-014-0419-z.
3. Isharwal S, Misra A, Wasir JS, Nigam P. Diet & insulin resistance: a review & Asian Indian perspective. Indian J Med Res. 2009;129:485-99.
4. Mahalle N, Kulkarni MV, Naik SS, Garg MK. Association of dietary factors with insulin resistance and inflammatory markers in subjects with diabetes mellitus and coronary artery disease in Indian population. J Diabetes Complications. 2014;28:536-41. doi: 10.1016/j.jdiacomp. 2012.09.008.
5. Wang B, Liu K, Mi M, Wang J. Effect of fruit juice on glucose control and insulin sensitivity in adults: a meta-analysis of 12 randomized controlled trials. PLoS One. 2014; 9:e95323. doi: 10.1371/journal.pone.0095323.
6. Arisawa K, Uemura H, Yamaguchi M, Nakamoto M, Hiyoshi M, Sawachika F, Katsuura-Kamano S. Associations of dietary patterns with metabolic syndrome and insulin resistance: a cross-sectional study in a Japanese population. J Med Invest. 2014;61:333-44. doi: 10.2152/jmi.61.333.
7. Canete R, Gil-Campos M, Aguilera CM, Gil A. Development of insulin resistance and its relation to diet in the obese child. Eur J Nutr. 2007;46:181-7. doi: 10.1007/s00394-007-0648-9 .
8. Batis C, Mendez MA, Sotres-Alvarez D, Gordon-Larsen P, Popkin B. Dietary pattern trajectories during 15 years of follow-up and HbA1c, insulin resistance and diabetes prevalence among Chinese adults. J Epidemiol Community Health. 2014;68:773-9. doi: 10.1136/jech-2013-203560.
9. van Dam RM, Rimm EB, Willett WC, Stampfer MJ, Hu FB. Dietary patterns and risk for type 2 diabetes mellitus in U.S. men. Ann Intern Med. 2002;136:201-9. doi: 10.7326/0003-4819-136-3-200202050-00008.
10. Fung TT, Schulze M, Manson JE, Willett WC, Hu FB. Dietary patterns, meat intake, and the risk of type 2 diabetes in women. Arch Intern Med. 2004;164:2235-40. doi: 10. 1001/archinte.164.20.2235.
11. Panagiotakos DB, Pitsavos C, Skoumas Y, Stefanadis C. The association between food patterns and the metabolic syndrome using principal components analysis: the ATTICA study. J Am Diet Assoc. 2007;107:979-87. doi: 10. 1016/j.jada.2007.03.006.
12. Kastorini C-M, Milionis HJ, Esposito K, Giugliano D, Goudevenos JA, Panagiotakos DB. The effect of Mediterranean diet on metabolic syndrome and its components: a meta-analysis of 50 studies and 534,906 individuals. J Am Coll Cardiol. 2011;57:1299-313. doi: 10. 1016/j.jacc.2010.09.073.
13. Suliga E, Koziel D, Ciesla E, Gluszek S. Association between dietary patterns and metabolic syndrome in individuals with normal weight: a cross-sectional study. Nutr J. 2015:14;55. doi: 10.1186/s12937-015-0045-9.
14. Imamura F, Lichtenstein AH, Dallal GE, Meigs JB, Jacques PF. Generalizability of dietary patterns associated with incidence of type 2 diabetes mellitus. Am J Clin Nutr. 2009; 90:1075-83. doi: 10.3945/ajcn.2009.28009.
15. Azizi F, Rahmani M, Emami H, Mirmiran P, Hajipour R, Madjid M et al. Cardiovascular risk factors in an Iranian urban population: Tehran lipid and glucose study (phase 1). Soz Praventivmed. 2002;47:408-26.
16. Kriska AM, Knowler WC, LaPorte RE, Drash AL, Wing RR, Blair SN, Bennett PH, Kuller LH. Development of questionnaire to examine relationship of physical activity and diabetes in Pima Indians. Diabetes Care. 1990;13:401-11. doi: 10.2337/diacare.13.4.401.
17. Borai A, Livingstone C, Kaddam I, Ferns G. Selection of the appropriate method for the assessment of insulin resistance. BMC Med Res Methodol. 2011;11:158. doi: 10. 1186/1471-2288-11-158.
18. Muniyappa R, Lee S, Chen H, Quon MJ. Current approaches for assessing insulin sensitivity and resistance in vivo: advantages, limitations, and appropriate usage. Am J Physiol Endocrinol Metab. 2008;294:E15-26. doi: 10.1152/ajpendo.00645.2007.
19. Asghar Ghasemi MT, Arash Derakhshan, Mitra Hasheminia, Fereidoun Azizi, Farzad Hadaegh. Cut-off points of homeostasis model assessment of insulin resistance, beta-cell function, and fasting serum insulin to identify future type 2 diabetes: Tehran Lipid and Glucose Study. Acta Diabetol. 2015;52:905-15. doi: 10.1007/s00592-015-0730-3.
20. Enzevaei A, Salehpour S, Tohidi M, Saharkhiz N. Subclinical hypothyroidism and insulin resistance in polycystic ovary syndrome: is there a relationship? Iran J Reprod Med. 2014;7:481-6.

21. Mirmiran P, Esfahani FH, Mehrabi Y, Hedayati M, Azizi F. Reliability and relative validity of an FFQ for nutrients in the Tehran lipid and glucose study. Public Health Nutr. 2010;13:654-62. doi: 10.1017/S1368980009991698.
22. Hosseini-Esfahani F, Jessri M, Mirmiran P, Bastan S, Azizi F. Adherence to dietary recommendations and risk of metabolic syndrome: Tehran Lipid and Glucose Study. Metabolism. 2010;59:1833-42. doi: 10.1016/j.metabol.2010.06.013.
23. Abiemo EE, Alonso A, Nettleton JA, Steffen LM, Bertoni AG, Jain A, Lutsey PL. Relationships of the Mediterranean dietary pattern with insulin resistance and diabetes incidence in the Multi-Ethnic Study of Atherosclerosis (MESA). Br J Nutr. 2013;109:1490-7. doi: 10.1017/S0007114512003339.
24. Moslehi N, Hosseini-Esfahani F, Hosseinpanah F, Mirmiran, P, Azizi F. Patterns of food consumption and risk of type 2 diabetes in an Iranian population: a nested case–control study. Nutrition & Dietetics. 2016;73:169-76. doi: 10.1111/1747-0080.12189.
25. Hinderliter AL, Babyak MA, Sherwood A, Blumenthal JA. The DASH diet and insulin sensitivity. Curr Hypertens Rep. 2011;13:67-73. doi: 10.1007/s11906-010-0168-5.
26. Esmaillzadeh A, Kimiagar M, Mehrabi Y, Azadbakht L, Hu FB, Willett WC. Dietary patterns, insulin resistance, and prevalence of the metabolic syndrome in women. Am J Clin Nutr. 2007;3:910-8.
27. Calton EK, James AP, Pannu PK, Soares MJ. Certain dietary patterns are beneficial for the metabolic syndrome: reviewing the evidence. Nutr Res. 2014;34:559-68. doi: 10.1016/j.nutres.2014.06.012.
28. Song SJ, Lee JE, Paik H-Y, Park MS, Song YJ. Dietary patterns based on carbohydrate nutrition are associated with the risk for diabetes and dyslipidemia. Nutr Res Pract. 2012;6:349-56. doi: 10.4162/nrp.2012.6.4.349.
29. Lopez S, Bermudez B, Abia R, Muriana FJ. The influence of major dietary fatty acids on insulin secretion and action. Curr Opin Lipidol. 2010;21:15-20. doi: 10.1097/MOL.0b013e3283346d39.
30. Stefanyk LE, Dyck DJ. The interaction between adipokines, diet and exercise on muscle insulin sensitivity. Curr Opin Clin Nutr Metab Care. 2010;13:255-9. doi: 10.1097/MCO.0b013e328338236e.
31. Morrison CD, Huypens P, Stewart LK, Gettys TW. Implications of crosstalk between leptin and insulin signaling during the development of diet-induced obesity. Biochim Biophys Acta. 2009;1792:409-16. doi: 10.1016/j.bbadis.2008.09.005.
32. Lloyd JW, Zerfass KM, Heckstall EM, Evans KA. Diet-induced increases in chemerin are attenuated by exercise and mediate the effect of diet on insulin and HOMA-IR. Ther Adv Endocrinol Metab. 2015;5:189-98. doi: 10.1177/2042018815589088.
33. Miller ER, 3rd, Erlinger TP, Sacks FM, Svetkey LP, Charleston J, Lin PH, Appel LJ. A dietary pattern that lowers oxidative stress increases antibodies to oxidized LDL: results from a randomized controlled feeding study. Atherosclerosis. 2005;183:175-82. doi: 10.1016/j.atherosclerosis.2005.04.001.
34. McGeoghegan L, Muirhead C, Almoosawi S. Association between dietary patterns and biomarkers of inflammation and antioxidant status in British adults: results from the National Diet and Nutrition Survey rolling programme Years 1–3. Proceedings of the Nutrition Society. 2015;74:E61. doi: 10.1017/S0029665115000762.
35. Xie Z, Lin H, Fang R, Shen W, Li S, Chen B. Effects of a fruit-vegetable dietary pattern on oxidative stress and genetic damage in coke oven workers: a cross-sectional study. Environ Health. 2015;14:40. doi: 10.1186/s12940-015-0028-5.