

Association Between Dietary Diabetes Risk Reduction Score and Chronic Kidney Disease in Adults: Tehran Lipid and Glucose Study

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Abstract

Aims: To examine the association of dietary diabetes risk reduction score (DDRRS) with chronic kidney disease (CKD) among an Iranian population.

Methods: We followed-up 2076 \geq 20 years old participants of Tehran Lipid and Glucose Study (2006-2008), who were initially free of CKD for 5.98 years. Dietary diabetes risk reduction score was calculated on the basis of scoring eight components using a valid and reliable 168-item food frequency questionnaire. CKD was defined as estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m². A Cox proportional hazard regression model was used to assess association between the quartiles of DDRRS and incidence of CKD.

Results: Mean \pm SD age of the study population (53% women) was 37.6 ± 12.61 years. A total of 357 incident cases of CKD were reported. The median (25-75 interquartile range) of DDRRS was 20 (18-22). After adjustment for age, sex, smoking status, total energy intake, body mass index, hypertension, diabetes, eGFR, and physical activity, individuals in the highest versus lowest quartile of DDRRS were 33% less likely to have CKD (OR: 0.67; 95% CI: 0.48-0.96, P for trend: 0.043).

Conclusion: Our findings suggest an inverse association between DDRRS and risk of incident CKD in the fourth versus first quartile of DDRRS.

Introduction

Chronic kidney disease (CKD), generally diagnosed as glomerular filtration rate (GFR) < 60 ml/min/1.73 m² ascertained by presence of albuminuria, is a global public health problem with severe outcomes such as cardiovascular disease and high mortality rates[1]. Its worldwide prevalence is 10.6% and even higher amongst diabetic individuals[2], while the overall prevalence in Iran is 18.9%, and rising at an alarming rate[3]. Diabetes, aging, high blood pressure, obesity, and poor dietary intakes have been identified as major CKD risk factors [4, 5].

As rigid control of hypertension and diabetes, and lifestyle modifications are essential in the prevention of CKD; addressing dietary issues can be of great importance in this prevention [6]. Since nutrients and foods are never consumed in isolation, and every single component of diet affects other particles synergistically, assessing dietary patterns instead of individual components, by indicators of diet quality, is important [7]. Dietary diabetes risk reduction score (DDRRS) is a priori dietary index, consisting of eight dietary factors previously shown to be predictive of diabetes; these factors include cereal fiber, nuts, coffee, poly unsaturated fatty acids (PUFA) to saturated fatty acids (SFA) ratio, glycemic index (GI), sugar sweetened beverages (SSB), trans fatty acids (TFA), and red and processed meat, introduced in 2015 to evaluate diet quality among women of different ethnic groups in association to incidence of diabetes [8]. The DDRRS has shown protective role against risk of diabetes among women of minority ethnic groups in the United States [8].

Previously, studies have investigated the protective role of particular nutrients and foods, including PUFA, coffee, nuts, total and cereal fiber [9–13] against the risk of incident CKD, and a direct association has been shown between CKD risk and GI, SSBs, and red and processed meat consumption as detrimental components of DDRRS [11, 12, 14]. However, to the best of our knowledge, no study has yet examined the association of DDRRS with the incidence of CKD. Therefore, we aimed to assess the relation between this dietary score and risk of CKD among Iranian adults in a population based cohort study.

Materials And Methods

Subjects

This study was performed within the framework of the Tehran Lipid and Glucose Study (TLGS), a prospective cohort of 15005 urban participants aged ≥ 3 years with the aim of preventing non communicable diseases [15]. The baseline survey was a cross-sectional study conducted from 1999 to 2001, followed by prospective surveys 2 (2002–2005), 3 (2006–2008), 4 (2009–2011) and 5 (2012–2015).

From among 12519 participants who participated in survey 3, 3656 were randomly selected for dietary assessment. For the current study, we selected men and women ≥ 20 years, who accounted for 3029 participants, of whom 2636 were CKD free. We excluded individuals who reported daily energy intakes outside the range of 800–4200 kcal/day ($n = 148$). Furthermore, participants with a history of myocardial infarction ($n = 16$), cerebro-vascular accident ($n = 4$) or cancer ($n = 6$), those having special diets for diabetes ($n = 75$) or hypertension ($n = 87$), and pregnant women ($n = 19$) were excluded. Finally, 2076 participants were followed until survey 5 (response rate: 91%), with a median duration of 5.98 years (Fig. 1).

Measurements

Dietary assessment

A valid and reliable semi-quantitative 168-item food frequency questionnaire (FFQ) was used to assess dietary intakes during the year preceding enrollment [16, 17]. During a face-to-face interview, participants' intake frequency for each food item during the previous year on a daily, weekly, or monthly basis was collected by trained and experienced dietitians. The FFQ contained usual foods with standard portion sizes commonly consumed by Iranians and their frequency of consumption on a daily, weekly or monthly basis. Portion sizes of consumed foods were then converted to grams using household measures. As the Iranian Food Composition Table (FCT) is incomplete, the United States Department of Agriculture (USDA) FCT was referred to measure nutrients. For national foods not listed in the USDA FCT, the Iranian FCT was alternatively used.

Dietary diabetes risk reduction score was calculated according to the study by Rhee et al. [8] using eight components. For the components assumed to be beneficial e.g. cereal fiber, nuts, coffee, and PUFA to SFA ratio, we assigned a score of 1 to 4 based on the participant's quartile of intake in ascending order. On the contrary, for the detrimental components, including GI, TFA, SSBs, and red and processed meats, a score of 1 to 4 was assigned according to quartile of intake in descending order. The DDRRS was calculated as the sum of these values and ranged between 8 and 32, with higher scores indicating a healthier overall diet.

Covariates assessments

Participants were interviewed by qualified interviewers using pretested questionnaires, to collect data on socio-demographics, medical history, medication use, and smoking habits in the third survey of the TLGS.

Physical activity during the preceding year was determined using modifiable activity questionnaire (MAQ) and calculating metabolic equivalent task (MET) hours per week. The reliability and validity for the Persian translated MAQ has been confirmed previously [18]. The MET value of the activity was multiplied by each of the activities duration and all MET-hour products were summed to reach an estimate of daily physical activity, indicating energy expenditure per kilogram of body weight during an average day.

Weight was measured in light clothing with the precision of 0.1 kg on a SECA digital weighing scale (Seca 707; Seca Corporation; range 0.1–150 kg). Height was also recorded without shoes with 0.1 cm precision. Body mass index (BMI) was then calculated by dividing weight (kg) by square of height (m²). Blood pressure was also measured by means of a standardized mercury sphygmomanometer on the right arm while sitting, after a 15-min rest in the supine position. The onset of tapping Korotkoff sound marked the systolic blood pressure (SBP), while the disappearance of Korotkoff sound marked the diastolic blood pressure (DBP). It was measured twice and the mean of the two measurements was considered as the participant's blood pressure.

A 12–14 h overnight fasting blood sample was drawn from each subject for biochemical measurements. Fasting plasma glucose (FPG) and 2-h plasma glucose (equivalent to 75 g anhydrous glucose; Cerestar EP) were measured by enzymatic colorimetric method using glucose oxidase technique utilizing glucose kits (Pars Azmoon, Tehran, Iran). Both inter- and intra-assay coefficients of variation were 2.2% for FPG. Serum creatinine was assessed by standard colorimetric Jaffe_Kinetic reaction method both at baseline and after 5.98 years of follow-up. Both intra- and inter-assay coefficients of variation were < 3.1%.

Definitions

Hypertension was defined as SBP/DBP \geq 140/90 mmHg in participants younger than 60 years and SBP/DBP \geq 150/90 mmHg in those aged 60 years or above, or current therapy for a definite diagnosis of hypertension in participants 60 years or older, according to JNC 8 hypertension guidelines [19]. Diabetes was determined according to the American Diabetes Association criteria as fasting plasma glucose \geq 126 mg/dl or 2-h post 75 g glucose load \geq 200 mg/dl or current therapy for a definite diagnosis of

diabetes [20]. We used the Modification of Diet in Renal Disease equation formula to express GFR in mL/min/1.73 m² of body surface area [21].

$$\text{eGFR} = 186 \times (\text{Serum creatinine})^{-1.154} \times (\text{Age})^{-0.203} \times (0.742 \text{ if female}) \times (1.210 \text{ if African-American}).$$

Participants were then categorized based on their eGFR levels according to the national kidney foundation guidelines [22]: eGFR \geq 60 mL/min/1.73 m² as not having CKD and eGFR < 60 mL/min/1.73 m² as having CKD.

Statistical analysis

Data were analyzed using the Statistical Package for Social Sciences program (SPSS) (version 15.0; SPSS Inc., Chicago, IL, USA) and Software for Statistics and Data Science (STATA) software package. P-values < 0.05 were assumed statistically significant. The Kolmogorov–Smirnov test of normality and histogram chart were used to assess normality. DDRRS was categorized into quartile cutoff points of < 18, 18–20, 21–22 and > 22. Continuous variables were reported as mean \pm SD and categorical variables as percentages. For the continuous variables, age adjusted mean values were calculated using analysis of covariance (ANCOVA) while generalized linear models were used for the age adjusted percentages of categorical variables. Tests of trend across quartiles of DDRRS (as median values in each quartile) were conducted using linear regression test. Median (25–75 interquartile range) follow-up time was 5.98 years (25–75 interquartile range: 5.5–6.5; Fig. 1). Cox proportional hazard regression models were used to assess the hazard ratios (HRs) and 95% confidence interval (CI) of CKD across quartiles of DDRRS. Age, sex, smoking status, total energy intake, BMI, hypertension, diabetes, eGFR, and physical activity were regarded as confounders. To calculate the trend of HR across increasing quartiles of DDRRS, we considered the quartile categories as continuous variables.

Results

The mean \pm SD age of the study population (53% women) was 37.6 \pm 12.61 years. The median (25–75 interquartile range) of DDRRS for the total population was 20 (18–22), and the incidence rate of CKD outcomes was 32/1000 during 5.98-year of follow-up. General characteristics of study participants are presented in Table 1. No significant differences were found by means of, sex, BMI, smoking status, physical activity, diabetes, hypertension, antihypertensive medication, serum creatinine, and eGFR across quartiles of DDRRS (Table 1).

Table 1

Age adjusted general characteristics of study participants according to the quartiles of dietary diabetes risk reduction score

	dietary diabetes risk reduction score quartiles				P for trend
	Q1	Q2	Q3	Q4	
Age (years)	37.18 ± 12.75	36.78 ± 12.75	36.71 ± 12.30	39.25 ± 11.85	0.307
Women (%)	50.8	55.4	52.0	53.8	0.632
Body mass index (kg/m ²)	26.60 ± 8.20	26.62 ± 8.66	26.39 ± 9.56	27.27 ± 10.93	0.316
Current smoker (%)	25	23	21	24	0.556
Physical activity (MET h/week)	63.86 ± 50.72	57.46 ± 44.15	63.27 ± 51.87	56.59 ± 50.49	0.142
Diabetes (%)	2	2	2	4	0.066
Hypertension (%)	6	6	5	9	0.197
Antihypertensive drug (%)	0.7	0.6	0.6	1.2	0.100
Serum creatinine (mg/dL)	1.03 ± 0.46	1.02 ± 0.46	1.03 ± 0.46	1.02 ± 0.46	0.517
eGFR (mL/min/1.73 m ²)	75.93 ± 4.88	76.19 ± 16.40	75.89 ± 18.22	75.44 ± 20.50	0.172
Data are represented as age-adjusted mean ± SD for continuous variables and percent for categorical variables.					

Dietary intakes of participants are presented in Table 2. Total energy and protein intakes were not significantly different across quartiles of DDRRS. However, participants in the highest quartile of DDRRS had a lower intake of animal protein, total fat, and saturated fat and higher intake of plant protein, total carbohydrates, sugar, fiber, vitamin C, potassium, and magnesium compared with those in the lowest quartile ($P < 0.05$).

Table 2

Age adjusted dietary intakes of study participants according to the quartiles of dietary diabetes risk reduction score

	dietary diabetes risk reduction score quartiles				P for trend
	Q1	Q2	Q3	Q4	
Dietary diabetes risk reduction score (DDRRS)	16.51 ± 1.82	19.47 ± 2.28	21.45 ± 2.28	24.33 ± 2.73	
DDRRS components					
Cereal fiber (g)	16.34 ± 32.35	18.98 ± 34.63	23.76 ± 37.36	26.08 ± 43.28	0.022
Coffee (cup)	0.03 ± 0.46	0.04 ± 0.46	0.07 ± 0.46	0.12 ± 0.46	0.093
PUFA/SFA *	0.61 ± 0.46	0.65 ± 0.46	0.68 ± 0.46	0.73 ± 0.46	0.001
Nuts (serving)	0.03 ± 0.004	0.06 ± 0.004	0.08 ± 0.004	0.11 ± 0.005	0.011
red and processed meat (serving)	0.64	0.55	0.49	0.40	0.039
Glycemic index	65.21 ± 13.21	62.53 ± 14.12	60.71 ± 15.03	57.17 ± 17.31	0.002
Sugar sweetened beverages (serving)	0.20 ± 0.46	0.12 ± 0.46	0.10 ± 0.46	0.06 ± 0.46	0.076
Trans fatty acids (% energy)	0.21 ± 0.23	0.19 ± 0.23	0.16 ± 0.23	0.14 ± 0.27	0.001
Other nutritional factors					
Total energy intake (kcal/day)	2206 ± 1275	2258 ± 1367	2351 ± 1485	2351 ± 1699	0.142
Protein (% energy)	13.6 ± 4.10	13.57 ± 4.56	13.62 ± 5.01	13.92 ± 5.92	0.305
Animal protein (% energy)	2.10 ± 0.03	1.93 ± 0.03	1.80 ± 0.03	1.68 ± 0.04	0.006
Plant protein (% energy)	11.50 ± 2.50	11.64 ± 2.64	11.82 ± 2.90	12.24 ± 3.43	0.013
Carbohydrate (% energy)	55.52 ± 12.30	56.97 ± 13.67	58.47 ± 14.58	60.01 ± 16.86	0.007

Data are represented as age-adjusted mean ± SD for continuous variables or percent for categorical variables.

* Poly unsaturated fatty acids (PUFA) to saturated fatty acids (SFA) ratio

	dietary diabetes risk reduction score quartiles				P for trend
Total sugar (% energy)	19.93 ± 10.02	20.71 ± 10.94	21.44 ± 11.85	22.75 ± 13.21	0.015
Dietary fiber (g/1000 kcal)	13.90 ± 11.39	15.82 ± 13.67	18.15 ± 13.21	19.34 ± 15.0	0.007
Total fat (% energy)	32.70 ± 12.30	31.94 ± 13.21	30.66 ± 14.12	29.42 ± 16.40	0.003
Saturated fat (% energy)	11.37 ± 9.11	11.12 ± 10.02	10.08 ± 10.48	9.31 ± 12.30	0.007
Vitamin C (mg/1000 kcal)	53.92 ± 61.05	62.56 ± 65.16	66.73 ± 71.08	69.99 ± 81.10	0.046
Sodium (mg/1000 kcal)	2078 ± 2688	2024 ± 2916	1997 ± 3144	2011 ± 3599	0.143
Potassium (mg/1000 kcal)	1546 ± 756	1636 ± 811	1679 ± 879	1770 ± 1007	0.002
Magnesium (mg/1000 kcal)	151.5 ± 55.13	161.4 ± 59.23	168.8 ± 64.24	188.9 ± 73.36	0.016
Data are represented as age-adjusted mean ± SD for continuous variables or percent for categorical variables.					
* Poly unsaturated fatty acids (PUFA) to saturated fatty acids (SFA) ratio					

The association between quartiles of DDRRS and risk of incident CKD is presented in Table 3. No significant association was established between the quartiles of DDRRS and CKD risk in the crude model. However, after adjusting for age, sex, smoking, total energy intake, BMI, hypertension, diabetes, eGFR, and physical activity, the HR for participants in the highest, compared with the lowest quartile of DDRRS was 0.67 (95% CI: 0.47–0.96, P for trend = 0.043).

Table 3

HRs (95% CI) of chronic kidney disease risk according to quartile of dietary diabetes risk reduction score components across participants of the TLGS

	dietary diabetes risk reduction score quartiles				P _{trend} *
	Q1	Q2	Q3	Q4	
Range of DDRRS	(10–18)	(19–20)	(21–22)	(23–30)	
Case/Total	113/651	100/571	85/485	59/369	
Model 1§	Ref	1.03 (0.78–1.34)	1.01 (0.76–1.34)	0.93 (0.68–1.27)	0.676
Model 2‡	Ref	0.92 (0.68–1.24)	0.95 (0.70–1.29)	0.67 (0.48–0.96)	0.043
* P _{trend} across quartiles was calculated by exposure modeled as a continuous variable					

§ The crude model.

‡ The multivariate model, adjusted for age, sex, smoking status, total energy intake, BMI, hypertension, diabetes, eGFR, and physical activity

Discussion

In this prospective cohort study, higher DDRRS was inversely associated with CKD, independent of age, sex, smoking, total energy intake, BMI, hypertension, diabetes, eGFR, and physical activity, after 5.98 years of follow-up.

Although no study has yet investigated the association between DDRRS and CKD, dietary patterns with similar beneficial components as DDRRS have shown an inverse relation with CKD [23, 24]. One of such patterns is Dietary Approaches to Stop Hypertension (DASH) style diet, including high intake of whole grains, nuts and legumes and low intake of red and processed meat and sweetened beverages, similar to DDRRS. Previously, it has been reported that DASH diet can reduce CKD risk by 59% [23]. Mediterranean dietary pattern is another example, including high intake of fruits and nuts, vegetables, cereals, legumes, fish, monounsaturated to saturated fatty acid ratio, and low intake of meat and dairy products; this dietary pattern was associated with 47% decrease in CKD incidence [24]. Furthermore, subjects in the lower quartile of DDRRS in our study presented a more western like dietary pattern, which adversely affected the risk of CKD by being abundant in refined grains, sugary drinks, saturated and trans fat, but poor in whole grains and PUFA, according to previous studies [25].

Several components of DDRRS have been individually associated with kidney function [9–12, 14, 26, 27]. Gopinath et al. reported that a high GI intake increased the likelihood of having eGFR < 60 mL/min/1.73 m² by 55%, while the highest dietary cereal fiber intake was associated with a 50% lower CKD risk [12]. Consumption of sugar sweetened beverages was another component of DDRRS, which has been proved to increase risk of CKD [14, 26]. Findings of the current study demonstrated that higher

adherence to DDRRS was accompanied by higher total carbohydrate and fiber but lower sugar consumption. According to a recent study, low-carbohydrate, high-protein diet can lead to higher CKD risk [28]. The detrimental effect of such a diet on kidney has been partly explained by the lower intake of dietary fiber. Studies have shown that dietary fiber intake can reduce the risk of CKD and enhance kidney function [13, 29–31].

The beneficial effect of DDRRS could be partly attributed to the inclusion of nuts as positive, and red and processed meat as negative components. Our findings also indicate that animal protein consumption declined and plant protein consumption increased along with higher adherence to DDRRS. Red and processed meat intake has been directly associated with risk of hypertension [32] and CKD [11]. However, intake of nuts had a protective impact on CKD risk [11]. These associations could be explained through various mechanisms, one of which is the difference in metabolism of protein sources. Cooked meat contains a high amount of Maillard Reaction Products (MRPs). MRPs increase oxidative stress and inflammation through various chemical reactions, which in turn can lead to the development of hypertension and kidney dysfunction[33]. Plant sources of protein such as nuts, legumes and whole grain result in to less dietary acid load compared with animal proteins like red and processed meat [34].

The protective role of PUFA to SFA ratio in DDRRS has been backed up by our previous study, showing a 27% increase in CKD risk for participants in the highest versus lowest quartile of PUFA [9]. Furthermore, another study on the role of fatty acids on kidney dysfunction reported a direct association between saturated fatty acids and albuminuria and CKD [27]. However, they found no significant association with TFA [27].

Coffee consumption is another component of DDRRS, considered to be beneficial. A recent study on coffee consumption and incident kidney disease demonstrated that each additional cup of coffee per day is associated with 3% decrease in CKD risk [10]. This may be due to antioxidants which protect the glomerular endothelium from oxidative stress and systemic inflammation[35], or caffeine itself, by increasing eGFR and renal blood flow[36].

Diet of participants in higher quartiles of DDRRS was richer in potassium, magnesium, and vitamin C, micronutrients previously shown to prevent CKD incident [37]. These protective effects may be due to vitamin C acting as an antioxidant [38], and magnesium and potassium lower the renal acid load [34]. High magnesium intake may reflect high plant protein consumption[34], which in turn lowers the amount of fibroblast growth factor 23 and increases bicarbonate levels; thus, protecting against CKD [39]. Furthermore, low serum magnesium concentrations have been suggested to promote endothelial dysfunction by stimulating inflammatory and pro-atherogenic cytokines which can lead to kidney dysfunction [40]. So, these might also explain why higher adherence to DDRRS was associated with a lower risk of CKD in our study.

To the best of our knowledge, this study was the first to investigate the relation between dietary diabetes risk reduction score and incident CKD. A noteworthy strength of our study was its prospective design in a large sized, population based cohort. Additionally, using a valid and reliable FFQ and physical activity

questionnaire, we were able to capture habitual dietary intake. We recognize the inherent limitations of our research too, first of which was creatinine measurements which were not repeated within three months to confirm a chronic reduction in eGFR. Secondly, there were missing data on the proteinuria of participants, so we could not consider it in the CKD definition. There was also the risk of some unknown or unmeasured confounders which we might have failed to take into account.

Conclusions

Overall, we observed that a healthy diet based on DDRRS, previously shown to protect against diabetes, can be preventive of CKD as well. This is an important finding, since it can help to define a dietary pattern which is easily adhered by the public to prevent the growing poor health outcomes such as diabetes and chronic kidney disease.

Abbreviations

DDRRS: dietary diabetes risk reduction score

CKD: chronic kidney disease

PUFA: poly unsaturated fatty acids

SFA saturated fatty acids

GI: glycemic index

SSB: sugar sweetened beverages

TFA: trans fatty acids

TLGS: Tehran Lipid and Glucose Study

FFQ: food frequency questionnaire

USDA: United States Department of Agriculture

FCT: food composition table

MAQ: modifiable activity questionnaire

MET: metabolic equivalent task

BMI: Body mass index

SBP: systolic blood pressure

DBP: diastolic blood pressure

FPG: Fasting plasma glucose

MDRD: Diet in Renal Disease

eGFR: estimated glomerular filtration rate

SPSS: Statistical Package for Social Sciences program

STATA: Software for Statistics and Data Science

ANCOVA: analysis of covariance

HR: hazard ratios

DASH: Dietary Approaches to Stop Hypertension

Declarations

Ethics approval: The study protocol was approved by the ethics committee of the Research Institute for Endocrine Sciences, affiliated to Shahid Beheshti University of Medical Sciences, Tehran, Iran. A written informed consent was obtained from all subjects.

Consent for publication: Not applicable

Availability of data and materials: The datasets analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests

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Author's Contributions

P.M, M.R, and H.F contributed in conception, design, and statistical analysis. Zh.T and H.F contributed in data collection and manuscript drafting. G.A, and F.A supervised the study. All authors approved the final version of the manuscript.

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References

1. Levey AS, Eckardt KU, Tsukamoto Y, Levin A, Coresh J, Rossert J, et al. Definition and classification of chronic kidney disease: a position statement from Kidney Disease: Improving Global Outcomes (KDIGO). *Kidney Int.* 2005;67:2089–100.
2. Anothaisintawee T, Rattanasiri S, Ingsathit A, Attia J, Thakkinstian A. Prevalence of chronic kidney disease: a systematic review and meta-analysis. *Clin Nephrol.* 2009;71:244–54.
3. Hosseinpanah F, Kasraei F, Nassiri AA, Azizi F. High prevalence of chronic kidney disease in Iran: a large population-based study. *BMC Public Health.* 2009;9:44.
4. Tohidi M, Hasheminia M, Mohebi R, Khalili D, Hosseinpanah F, Yazdani B, et al. Incidence of chronic kidney disease and its risk factors, results of over 10 year follow up in an Iranian cohort. *PLoS One.* 2012;7:e45304.
5. Kazancioglu R. Risk factors for chronic kidney disease: an update. *Kidney Int Suppl* (2011) 2013;3:368 – 71.
6. Bello AK, Nwankwo E, El Nahas AM. Prevention of chronic kidney disease: a global challenge. *Kidney Int Suppl* 2005:S11-7.
7. Slattery ML. Defining dietary consumption: is the sum greater than its parts? *Am J Clin Nutr.* 2008;88:14–5.
8. Rhee JJ, Mattei J, Hughes MD, Hu FB, Willett WC. Dietary diabetes risk reduction score, race and ethnicity, and risk of type 2 diabetes in women. *Diabetes Care.* 2015;38:596–603.
9. Yuzbashian E, Asghari G, Mirmiran P, Hosseini FS, Azizi F. Associations of dietary macronutrients with glomerular filtration rate and kidney dysfunction: Tehran lipid and glucose study. *J Nephrol.* 2015;28:173–80.
10. Hu EA, Selvin E, Grams ME, Steffen LM, Coresh J, Rebholz CM. Coffee Consumption and Incident Kidney Disease: Results From the Atherosclerosis Risk in Communities (ARIC) Study. *Am J Kidney Dis.* 2018;72:214–22.
11. Haring B, Selvin E, Liang M, Coresh J, Grams ME, Petruski-Ivleva N, et al. Dietary Protein Sources and Risk for Incident Chronic Kidney Disease: Results From the Atherosclerosis Risk in Communities (ARIC) Study. *J Ren Nutr.* 2017;27:233–42.
12. Gopinath B, Harris DC, Flood VM, Burlutsky G, Brand-Miller J, Mitchell P. Carbohydrate nutrition is associated with the 5-year incidence of chronic kidney disease. *J Nutr.* 2011;141:433–9.
13. Mirmiran P, Yuzbashian E, Asghari G, Sarverzadeh S, Azizi F. Dietary fibre intake in relation to the risk of incident chronic kidney disease. *Br J Nutr.* 2018;119:479–85.

14. Yuzbashian E, Asghari G, Mirmiran P, Zadeh-Vakili A, Azizi F. Sugar-sweetened beverage consumption and risk of incident chronic kidney disease: Tehran lipid and glucose study. *Nephrology (Carlton)*. 2016;21:608–16.
15. Azizi F, Ghanbarian A, Momenan AA, Hadaegh F, Mirmiran P, Hedayati M, et al. Prevention of non-communicable disease in a population in nutrition transition: Tehran Lipid and Glucose Study phase II. *Trials*. 2009;10:5.
16. Asghari G, Rezazadeh A, Hosseini-Esfahani F, Mehrabi Y, Mirmiran P, Azizi F. Reliability, comparative validity and stability of dietary patterns derived from an FFQ in the Tehran Lipid and Glucose Study. *Br J Nutr*. 2012;108:1109–17.
17. Esfahani FH, Asghari G, Mirmiran P, Azizi F. Reproducibility and relative validity of food group intake in a food frequency questionnaire developed for the Tehran Lipid and Glucose Study. *J Epidemiol*. 2010;20:150–8.
18. Momenan AA, Delshad M, Sarbazi N, Rezaei Ghaleh N, Ghanbarian A, Azizi F. Reliability and validity of the Modifiable Activity Questionnaire (MAQ) in an Iranian urban adult population. *Arch Iran Med*. 2012;15:279–82.
19. Page MR. The JNC 8 hypertension guidelines: an in-depth guide. *Am J Manag Care*. 2014;20:E8.
20. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. 2014;37(Suppl 1):81–90.
21. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med*. 1999;130:461–70.
22. K/DOQI clinical. practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Am J Kidney Dis*. 2002;39:1–266.
23. Asghari G, Yuzbashian E, Mirmiran P, Azizi F. The association between Dietary Approaches to Stop Hypertension and incidence of chronic kidney disease in adults: the Tehran Lipid and Glucose Study. *Nephrol Dial Transplant*. 2017;32:ii224–30.
24. Asghari G, Farhadnejad H, Mirmiran P, Dizavi A, Yuzbashian E, Azizi F. Adherence to the Mediterranean diet is associated with reduced risk of incident chronic kidney diseases among Tehranian adults. *Hypertens Res*. 2017;40:96–102.
25. Odermatt A. The Western-style diet: a major risk factor for impaired kidney function and chronic kidney disease. *Am J Physiol Renal Physiol*. 2011;301:F919-31.
26. Rebholz CM, Young BA, Katz R, Tucker KL, Carithers TC, Norwood AF, et al. Patterns of Beverages Consumed and Risk of Incident Kidney Disease. *Clin J Am Soc Nephrol*. 2019;14:49–56.
27. Lin J, Judd S, Le A, Ard J, Newsome BB, Howard G, et al. Associations of dietary fat with albuminuria and kidney dysfunction. *Am J Clin Nutr*. 2010;92:897–904.
28. Farhadnejad H, Asghari G, Emamat H, Mirmiran P, Azizi F. Low-Carbohydrate High-Protein Diet is Associated With Increased Risk of Incident Chronic Kidney Diseases Among Tehranian Adults. *J Ren Nutr* 2018.

29. Diaz-Lopez A, Bullo M, Basora J, Martinez-Gonzalez MA, Guasch-Ferre M, Estruch R, et al. Cross-sectional associations between macronutrient intake and chronic kidney disease in a population at high cardiovascular risk. *Clin Nutr.* 2013;32:606–12.
30. Xu H, Huang X, Riserus U, Krishnamurthy VM, Cederholm T, Arnlov J, et al. Dietary fiber, kidney function, inflammation, and mortality risk. *Clin J Am Soc Nephrol.* 2014;9:2104–10.
31. Metcalf PA, Baker JR, Scragg RK, Dryson E, Scott AJ, Wild CJ. Dietary nutrient intakes and slight albuminuria in people at least 40 years old. *Clin Chem.* 1993;39:2191–8.
32. Borgi L, Curhan GC, Willett WC, Hu FB, Satija A, Forman JP. Long-term intake of animal flesh and risk of developing hypertension in three prospective cohort studies. *J Hypertens.* 2015;33:2231–8.
33. Tessier FJ, Birlouez-Aragon I. Health effects of dietary Maillard reaction products: the results of ICARE and other studies. *Amino Acids.* 2012;42:1119–31.
34. Rebholz CM, Coresh J, Grams ME, Steffen LM, Anderson CA, Appel LJ, et al. Dietary Acid Load and Incident Chronic Kidney Disease: Results from the ARIC Study. *Am J Nephrol.* 2015;42:427–35.
35. Wijarnpreecha K, Thongprayoon C, Thamcharoen N, Panjawatanan P, Cheungpasitporn W. Association of coffee consumption and chronic kidney disease: A meta-analysis. *Int J Clin Pract* 2017;71.
36. Choi HK, Curhan G. Coffee, tea, and caffeine consumption and serum uric acid level: the third national health and nutrition examination survey. *Arthritis Rheum.* 2007;57:816–21.
37. Farhadnejad H, Asghari G, Mirmiran P, Yuzbashian E, Azizi F. Micronutrient Intakes and Incidence of Chronic Kidney Disease in Adults: Tehran Lipid and Glucose Study. *Nutrients.* 2016;8:217.
38. Asghari G, Yuzbashian E, Shahemi S, Gaeini Z, Mirmiran P, Azizi F. Dietary total antioxidant capacity and incidence of chronic kidney disease in subjects with dysglycemia: Tehran Lipid and Glucose Study. *Eur J Nutr.* 2018;57:2377–85.
39. Scialla JJ, Appel LJ, Wolf M, Yang W, Zhang X, Sozio SM, et al. Plant protein intake is associated with fibroblast growth factor 23 and serum bicarbonate levels in patients with chronic kidney disease: the Chronic Renal Insufficiency Cohort study. *J Ren Nutr.* 2012;22:379–88. .e1.
40. Ferre S, Baldoli E, Leidi M, Maier JA. Magnesium deficiency promotes a pro-atherogenic phenotype in cultured human endothelial cells via activation of NFκB. *Biochim Biophys Acta.* 2010;1802:952–8.
41. Examined participants. in the third survey(n=12519)Survey 3 of TLGS(2006-2008)s.

Figures

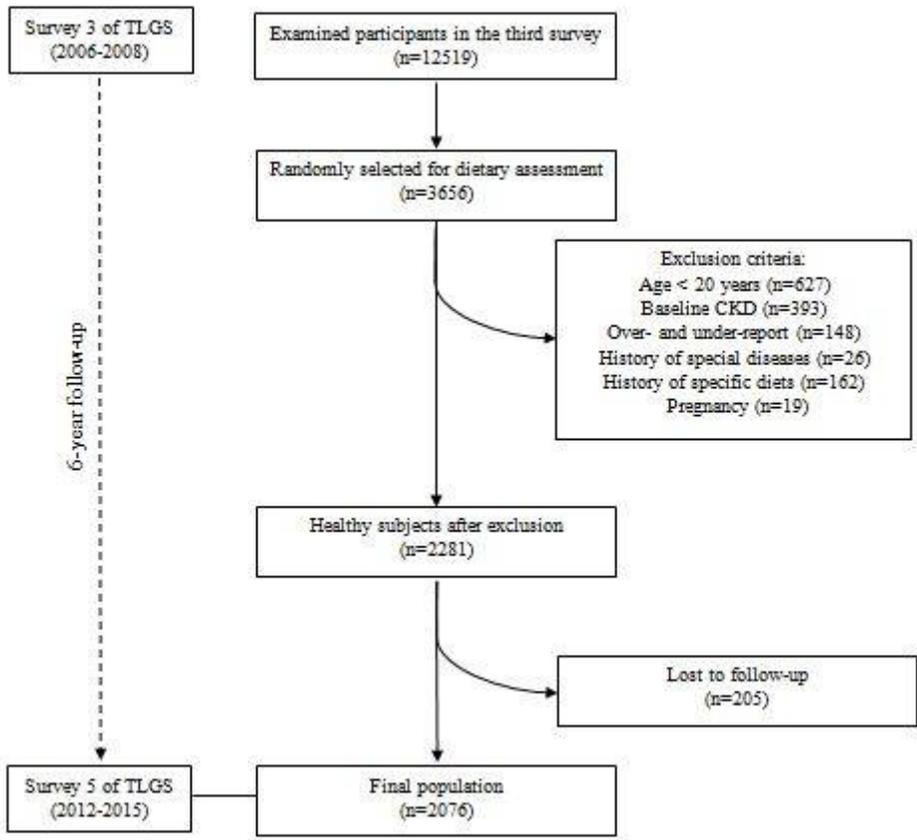


Figure 1

Flow chart of the Tehran Lipid and Glucose Study (TLGS) participants.