Original Article

The Association Between Dietary Intake of Sodium, Potassium, and Na:K Ratio with the Risk of NAFLD: A Case–Control Study Among Iranian Adults

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Abstract

Backgrounds: Dietary sodium (Na) and potassium (K) relationship with chronic disease has drawn more attention recently. Epidemiological studies reported controversial findings about high salt and Na diets with the risk of nonalcoholic fatty liver disease (NAFLD) and studies about the association between K and NAFLD are scare. Present study aimed to examine the associations between dietary intake of Na, K, and Na:K ratio with the risk of NAFLD. **Methods:** We analyzed data from a case–control study of 225 patients with NAFLD cases and 450 controls. Dietary intake of Na and K measured using a validated 168 item food frequency questionnaire. Adjusted logistic regression models were used to report odds ratio (OR) 95% confidence interval (CI) of NAFLD across tertiles of Na, K, and Na:K ratio. **Results:** The mean ± standard deviation of age and body mass index of participants (47% female) were 38.1 ± 8.8 years and 26.8 ± 4.3 Kg/m^2. In the age- and sex-adjusted model, there was any significant association between Na, K, and Na: K ratio with the risk of NAFLD. In the final adjusted model, the OR (95%CI) of the highest vs the lowest tertiles of K, Na, and Na:K was 0.39 (0.19–0.80), 0.71 (0.40–1.25), and 1.10 (0.61–1.97), respectively. **Conclusion:** The present study indicates that higher dietary K was related to lower odds of NAFLD; however, there was no association between dietary Na and Na: K ratio with odds of NAFLD.

Keywords: *Non-alcoholic fatty liver disease, potassium, sodium, sodium: potassium ratio*

Introduction

Nonalcoholic fatty liver disease (NAFLD) is recognized as one of the important public health problem worldwide and its incidence has been increasing rapidly along with the rise in obesity.[1] A meta-analysis involving a total of 85,15431 participants over 18 from 22 countries estimated that the prevalence of NAFLD was about 25%.[2]

NAFLD has been considered as a multi-factorial disease, and several factors are known to contribute to the development of NAFLD, such as genetics and lifestyle (e.g., nutrition and physical activity).[3] Several studies have investigated the relationships between the intake of individual nutrients and the risk of NAFLD. The potential role of zinc, iron, calcium, and vitamins A, B, D, and E deficiencies in NAFLD risk has been discussed in many studies.[4,5]

Among the micronutrients, the roles of sodium (Na) and potassium (K) have been less considered in incidence and development of NAFLD.[6] Salt consumption as main source of Na in diet is higher than recommendations among most countries and community-based nutritional interventions tried to reduce its intake by behavioral change.[7] High consumption of dietary Na is suggested to be related to various metabolic disorders, including type 2 diabetes mellitus, hypertension, and cardiovascular disease.[8] Several evidences have suggested the potential link between Na intake and insulin sensitivity. Both human and animal studies have shown some evidence that high Na intake has an adverse effect on insulin resistance.[9,10] Furthermore, some epidemiological studies have shown higher Na intake was associated with metabolic syndrome and obesity.[11]

For K intake, some clinical evidence has suggested that decreased K intake may contribute to various metabolic disorders, which may consequently increase the risk of NAFLD.[6,12] Some interventional studies

How to cite this article: Salehi-Sahlabadi A, Mirfazli E, Teymori F, Roosta S, Mokari A, Azadi M, *et al*. The association between dietary intake of sodium, potassium, and Na: K ratio with the risk of NAFLD: A case–control study among Iranian adults. Int J Prev Med 2021;12:179.

Access this article online

Website: www.ijpvmjournal.net/www.ijpm.ir

DOI: 10.4103/ijpvm.IJPVM_343_20

Quick Response Code:

also suggested that correcting serum K level contributes an improvement in insulin sensitivity.[13,14]

Given the evidence of links between required levels of Na and K intake and metabolic disorders and insulin resistance, it could be hypothesized that high Na and/or low K intake may be associated with NAFLD risk. Thus, we evaluated the associations between dietary intake of Na, K, and Na: K ratio and the risk of NAFLD in a population-based study.

Methods

Study population

This study was conducted as a case–control study in the Metabolic Liver Disease Research Center affiliated to Isfahan University of Medical Sciences. A total of 225 newly diagnosed patients with NAFLD and 450 control subjects aged 20–60 years were recruited. Informed participant consent was attained prior to study commencement. The protocol for the present study was approved by the research ethics committee at the National Nutrition and Food Technology Research Institute of Shahid Beheshti University of Medical Science.

All participants prepared informed consent preceding the investigation enlistment. A gastroenterologist following the laboratory tests and liver steatosis in the ultrasound analyzed the NAFLD. The control group individuals were coordinated with the patients in terms of their age and sex. Moreover, all controls experienced an ultrasound assessment and no proof of hepatic steatosis was seen among the control group. Exclusion criteria were history of diseases, such as renal and hepatic (Wilson's disease, autoimmune liver disease, virus infection, and alcoholic fatty liver), cardiovascular disease, malignancy, thyroid disorder, autoimmune diseases, and special dietary or physical activity regimens. Participants who did not complete more than 35 items of the food frequency questionnaire and those who have under or over reporting in daily energy intakes (≤800 or ≥4500 kcal/d) were excluded from the study (8 participants) and were replaced by new participants. All patients were administered a questionnaire to capture demographic, medical history, medications, diet, alcohol, smoking, and education.

In this study, according to the following formula, 225 people for case group and 450 for control group were selected.

$$n=\left(\frac{1+\varphi}{\varphi}\right)\frac{\left(z_{1-\frac{\alpha}{2}}+z_{1-\beta}\right)^2}{(\log OR)^2\bar{\pi}(1-\bar{\pi})}=225$$

[Considering that z = 1.96, OR = 1.5, and prevalence (π) = 0.35]. Controls were twice the case group

Dietary assessment

Dietary intake was calculated from 1 year prior to diagnose by interview for case and control, respectively, using a previously validated food frequency questionnaire among Iranian adults.[15] Participants were asked to report their average dietary intake during the previous year by choosing one of the following choices: never or less than once a month, 3–4 times per month, once a week, 2–4 times per week, 5–6 times per week, once daily, 2–3 times per day, 4–5 times per day, and 6 or more times a day. Portion sizes of each food item were converted into grams by using standard Iranian household measures.[16] Nutrients consumption including Na and K were assessed using the Nutrient Composition of Iranian Foods[17] accompanied by USDA Food Composition Data. Then, the frequencies of consumed foods were transformed into daily intakes. The nutrient compositions of all foods were derived by using modified nutritionist IV software.

Assessment of other variables

Information on other variables including age, sex, marital status, socioeconomic status (SES), and smoking status was obtained from a demographic questionnaire. SES score, as an index of SES, was calculated based on three variables including family size (≤4, >4 people), education (academic and non-academic education), and acquisition (house ownership or not). For each of these variables, participants were given a score of 1 (if their family members were ≤4, were academically educated, or owned a house) or given a score of 0 (if their family members were >4, or had non-academic education, or leasehold property). Then, the total SES score was computed by summing up the assigned scores (minimum SES score of 0 to a maximum score of 3). Participants who had a score of 3, 2, and sum of subjects with 1 and 0 were classified as high, moderate, and low SES, respectively. Measuring physical activity of participants was conducted using International Physical Activity Questionnaire (IPAQ) through the face to face interview. All results of the IPAQ were expressed as metabolic equivalents per week (METs/week).

Statistical analysis

Statistical analysis was conducted using Statistical Package Software for Social Science, version 21 (SPSS Inc., Chicago, IL, USA). The Kolmogorov–Smirnov's test and histogram chart was used for testing the normality of the data baseline covariates were expressed as mean ± standard deviation or median (25–75 interquartile range) for continues variables, and number and percentages for categorical variables. Chi-square and linear regression were used to test the trend of categorical and continues variables across tertiles of dietary Na and K (as median value in each tertile), respectively. Dietary Na and K per 1000 Kcal of energy intake were calculated for all participants. Then Na, K, and Na:K ratios were categorized into tertiles based on

their intakes among controls. Logistic regression was used for assessing the association between dietary Na, K, and Na: K ratio, with the risk of NAFLD. Analysis was adjusted for potential confounders including age and sex, body mass index (BMI), physical activity, smoking, SES, dietary intake of energy, calcium, fiber, K (only for analyses of Na intake), and Na (only for analyses of K intake). The odds ratio (OR) with 95% confidence interval (CI) of NAFLD across tertiles of dietary Na, K, Na: K ratio, and K: Na ratio were reported and *P* values <0.05 were considered as statistically significant.

Result

The mean ± standard deviation of age and BMI of the entire participants (47% female) were 38.1 ± 8.8 years and 26.8 ± 4.3 Kg/m^2. There were no significant differences between cases and controls in age and gender distribution; however, NAFLD patients had higher BMI compared with controls.

Table 1 demonstrates the distribution of the baseline covariates by tertiles of dietary Na and K intake. Across tertiles of dietary Na, intake of fat, Na, and Na: K ratio increased ($P < 0.05$); however, the male percent and dietary intakes of carbohydrate, refined grain, high fat dairy, and fruits decreased ($P < 0.05$). There was no significant difference across tertiles of Na intake for other covariates (all *P* for trend >0.05). Also, across tertiles of dietary K, participants age, and dietary intake of protein, calcium, potassium, fruit, vegetable, and nut and legume increased ($P < 0.05$), whereas the male percent and dietary intake of fat, Na: K ratio, and refined grain decreased ($P < 0.05$). For other variables observed no significant difference across tertiles of dietary K (all *P* for trend >0.05).

The association between dietary Na, K, and Na:K ratio with the risk of NAFLD presented in Table 2. In the age- and sex-adjusted model, there was any statistically significant association between these variables with the risk of NAFLD. Although, in the final adjusted model, there were no significant association for the risk of NAFLD between the highest vs the lowest tertiles of Na intake (OR and 95% CI = 0.71 (0.40–1.25)), and Na: K ratio (OR and 95% CI = 1.10 (0.61–1.97)); the highest vs the lowest tertiles of K intake showed lower risk of NAFLD (OR and 95% CI = 0.39 (0.19–0.80), *P* for trend = 0.010).

Discussion

In the present case–control study, we assessed the association between dietary intake of the Na, K, and Na: K ratio with the risk of NAFLD among Iranian adults. Our finding indicates that high intake of K was related to the decreased odds of NAFLD. However, the high dietary intake of Na, and Na: K ratio showed no significant association with the odds of NAFLD.

The mean intake of Na in our study was 4373 mg/d, and 90.7% of participants had a higher intake than 2000 mg as the recommended intake by the World Health Organization's (WHO). This high prevalence of high Na intake makes difficult the comparison of participants who had high compared with normal Na intake. It may contribute to this non-significant result about the relation between Na intake and NAFLD. In a cross-sectional study among Korean adults, 79.4% of the population had a higher Na intake than the WHO recommendations and high-salt foods as a part of a traditional dietary pattern had direct association with NAFLD in men, not in women.[18] However, in a study among Chinese population high-salt dietary pattern showed no significant association with NAFLD.[19] Another Korean cross-sectional study showed that higher Na intake was associated with higher odds of NAFLD among men, not in women[6]; in their study, median intake of Na was almost half of our Na intake, and also the highest vs lowest quintiles of dietary Na (3485 vs 1219) was too different which was more comparable than present study. Differences in study design, ethnic, habitual dietary intake, and methods of dietary assessment may be more additional justifications for inconsistent results between studies.

Another cross-sectional study among Korean adults observed that high Na intake, estimated by spot analysis of 24-h urinary Na excretion, was related with high odds of surrogate indicators of NAFLD.[20] Regarding the considerable differences of the methods of calculating Na intake, NAFLD definition, and adjusted variables between our studies it so difficult to compare the results.

In the present study, despite the adjusting energy intake and main nutrients which may affect the relation between Na and NAFLD, regarding that across increasing Na intake the consumption of dietary fat and refined grain increased; however, carbohydrate intake, high fat dairy, and fruit decreased, the overall interaction of beneficial and detrimental components of diet may played a role for disappearing eventually association between Na intake and odds of NAFLD.

In the Korean cross-sectional study higher Na: K ratio was related with higher prevalence ratio of NAFLD in the separate analysis in men and women. However, in our case–control study Na: K ratio was not significantly associated with the odds of NAFLD. The mean intake of both Na and K in the present study was almost two times higher than Korean study and regarding the sample size limitation we do not separate analysis by sex**.** These reasons beside above-mentioned differences between studies may justify the inconsistent finding. Some biological mechanisms such as the effects of Na intake on increasing leptin levels,[21] adipocyte hypertrophy, fatness, and development of obesity has been reported,[22-24] however, this case–control study observed no association between Na intake of the odds of NAFLD.

Table 1: Characteristics and dietary intakes across tertiles of sodium and potassium intake among the study population*

	Tertiles of dietary sodium intake			*P*-trend	Tertiles of dietary Potassium intake			*P*-trend
	T1 (*n*=230)	T2 (*n*=225)	T3 (*n*=220)		T1 (*n*=239)	T2 (*n*=222)	T3 (*n*=214)	
Age (year)	38.6±9.7	38.5±9.3	37.3±8.1	0.074	36.8±8.6	37.9±8.2	39.7±9.4	0.001
Male, *n* (%)	142 (61.7)	115 (51.1)	101 (45.9)	0.003	136 (56.9)	124 (55.9)	98 (45.8)	0.036
BMI (Kg/m^2)	26.7±4.2	26.7±4.1	27.0±4.5	0.376	26.6±4.2	26.8±4.0	27.0±4.6	0.287
Smoking, *n* (%)	12 (5.2)	6 (2.7)	10 (4.5)	0.370	6 (2.5)	12 (5.4)	10 (4.7)	0.267
Physical activity (MET/min/week)	1418±800	1425±894	1455±948	0.642	1432±894	1464±800	1401±945	0.690
Socioeconomic status, *n* (%)				0.805				0.168
Low	80 (34.8)	74 (32.9)	69 (31.4)		82 (34.3)	74 (33.3)	67 (31.3)	
Middle	100 (43.5)	103 (48.0)	107 (48.6)		101 (42.3)	97 (43.7)	112 (52.3)	
High	50 (21.7)	48 (21.3)	44 (20.0)		56 (23.4)	51 (23.0)	35 (16.4)	
Dietary intake								
Macro and micronutrients								
Energy intake (Kcal/d)	2349±585	2227±641	2245±689	0.220	2271±709	2326±611	2224±585	0.412
Carbohydrate (% of energy)	56.4±7.2	56.6±6.1	54.8±7.1	0.003	55.9±7.7	55.2±6.5	56.5±5.9	0.279
Protein (% of energy)	13.1±2.1	13.4±2.3	13.2±2.5	0.836	12.2±1.9	13.4±2.0	14.1±2.4	<0.001
Fat (% of energy)	30.5±7.1	30.0±6.1	32.0±7.1	0.004	31.8±8.0	31.2±6.4	29.2±5.3	<0.001
Fiber (g/1000 Kcal)	14.5±5.5	17.7±7.6	16.3±7.9	0.170	16.4±9.3	15.2±5.7	16.8±5.2	0.500
Calcium (mg/1000 Kcal)	528±152	544±160	521±156	0.345	435±116	526±114	643±160	<0.001
Sodium (mg/1000 Kcal)	1087±171	1567±155	3469±2140	<0.001	2162±2057	1963±1120	1931±1423	0.130
Potassium (mg/1000 Kcal)	1619±377	1570±355	1548±377	0.071	1203±163	1571±85	2009±231	<0.001
Sodium to potassium ratio	0.70±0.19	1.05±0.30	2.42±1.91	<0.001	1.87±1.91	1.25±0.72	0.97±0.75	<0.001
Food groups								
Whole grains (g/day)	56.3 (23.6-108.2)	59.5 (29.0-127.0)	56.6 (27.3-117.0)	0.169	53.1 (22.2-122.3)	56.2 (26.5-106.9)	64.8 (32.0-112.8)	0.192
Refined grains (g/day)	361±193	331±146	302±158	<0.001	412±191	335±143	241±113	<0.001
High fat dairy products (g/d)	181±146	147±131	142±128	0.013	154±137	172±142	145±127	0.427
Fruits (g/day)	363±244	306±207	293±224	0.006	196±151	344±219	438±239	<0.001
Vegetables (g/day)	298±149	272±130	297±156	0.589	223±119	292±125	360±158	<0.001
Nuts and legume (g/d)	21.2±18.8	21.5±19.7	22.0±19.9	0.644	17.1±15.9	23.9±19.3	24.1±22.2	<0.001
Red and process meat (g/day)	23.4±17.7	20.9±15.5	24.8±20.4	0.152	22.0±16.6	25.1±19.0	21.9±18.3	0.880

*Data are presented as mean±standard deviation or median (25-75 interquartile range) for continues variables, and number and percentages for categorical variables.

Table 2: Odds ratios (ORs) and 95% confidence intervals (CIs) for NAFLD based on tertiles dietary sodium, potassium, Na: K ratio

	Tertiles of dietary intake			*P* for trend
	T1	T2	T3	
Sodium				
Median intake	2453	3459	7345	
NAFLD/control	80/150	75/150	70/150	
Model 1*	1.00 (Ref)	0.95 (0.64-1.40)	0.90 (0.60-1.34)	0.647
Model 2†	1.00 (Ref)	0.87 (0.49-1.53)	0.71 (0.40-1.25)	0.246
Potassium				
Median intake	2707	3564	4353	
NAFLD/control	89/150	72/150	64/150	
Model 1*	1.00 (Ref)	0.79 (0.54-1.17)	0.70 (0.47-1.05)	0.086
Model 2†	1.00 (Ref)	0.52 (0.29-0.93)	0.39 (0.19-0.80)	0.010
Sodium to potassium ratio				
Median ratio	0.67	1.02	1.92	
NAFLD/control	68/148	79/149	78/153	
Model 1*	1.00 (Ref)	1.18 (0.79-1.76)	1.14 (0.77-1.71)	0.655
Model 2†	1.00 (Ref)	1.16 (0.65-2.06)	1.10 (0.61-1.97)	0.871

*Model 1: adjusted for age and sex. †Model 2: Additionally adjusted for BMI, physical activity, smoking, SES, dietary intake of energy, calcium, fiber, potassium (only for analyses of sodium intake), and sodium (only for analyses of potassium intake)

Studies about the dietary K intake and NAFLD relationship are scare;[6] however, there were several studies which revealed the beneficial relationship between dietary k and chronic disease, such as obesity, metabolic syndrome, and cardiovascular disease.[25-27] In a meta-analysis of the epidemiological studies, higher K intake was associated with lower odds of metabolic syndrome and a nonlinear analysis indicated a protective effect of adequate K intake on obesity and metabolic syndrome.[26] Furthermore, in a recent study among US Hispanic/Latino adults, K intake was associated with lower BMI and smaller waist circumferences.[27] Shin *et al.* indicated that among the components of metabolic syndrome, K intake was inversely related to abdominal obesity and fasting hyperglycemia[28]; these two components are vigorous risk factors of NAFLD.[29,30] Previous Korean cross-sectional study showed no inverse association for energy-adjusted K intake.[6] It is important to be considered that in this study, K intake among all categories was insufficient (lower than 3500 mg/d) among men and women and almost was two times lower than K intake in the present study.

The mean intake of K among our participants was 3578 mg/d and 51.1% of population had lower intake than 3500 mg/d, which recognized as insufficient K intake by the WHO. Our data provided the ability to compare the participants who were in the different levels of K intake and help for better interpretation of results. We observed that participants in the highest tertile of K (median intake = 4353) compared to the lowest (median intake = 2707) had 61% lower odds of NAFLD. In our study, across tertiles of K intake the overall diet characteristics of participants showed to be more desirable dietary pattern, as the consumption of beneficial component, such as fruit, vegetable, nut, and grain increased and refined grains decreased; in addition, dietary fat decreased and it replaced with protein intake which it considered to be a good replacement for protection against fatty liver. So, it seems that a high K diet bring along with more healthy diet.

Tal *et al*. indicated that increasing K intake has the strongest correlation with weight loss and BMI reduction among dietary components in a weight-loss-oriented multidisciplinary intervention in obese adults. In that study like the present study increasing K intake was related with higher protein intake which may help to weight reduction through the elevating dietary thermogenesis, satiety, and fat loss which previously indicated following adherence to a high protein diets.[31]

The mechanisms through which higher dietary K may facilitate weight loss and fatty liver reduction remain unclear. Previously has been observed that potassium depletion leads to changes in glucose homeostasis and appears to be associated with abdominal obesity in patients with diuretic therapy.[32] Because of the fundamental role of K in insulin secretion and carbohydrate metabolism,[33,34] higher K intake may play a effective role in improving insulin secretion and impaired fasting glucose[35] also proposed that K intake may effects on body weight by reducing inflammation, improvement in insulin sensitivity,[36] and beneficial effects on fat deposition/mobilization or energy balance.[37]

Strength and limitation

The present study has several strong points. It is the first study in Iran that investigated the association between dietary Na, K, Na: K ratio, and risk of NAFLD. Dietary intakes collected by trained dieticians in a face to face interview decrease the chance of occurring measurement bias. Furthermore, to minimize the selection bias, individuals in the control group were chose only from individuals with conditions not related to diet or other major risk factors of NAFLD. However, this study has some limitations. First, although we used a validated FFQ for dietary assessment, some degree of measurement error, and misclassification must be considered. Second, like other case–control studies, disease-specific recall bias and selection bias may exist; e.g., in case–control studies, there is the possibility that cases may recall their diets differently after a disease diagnosis.[38,39] However, in order to reduce the possibility of recall bias, only newly incident cases were enrolled as participants in our study. However, we cannot eliminate it entirely. Third, although we adjusted a wide variety of variables in our analyses, the confounding effect of some unknown and unmeasured residual confounders may have occurred.

Conclusion

O finding showed that higher intake of K was related to the decreased odds of NAFLD; however, higher dietary intake of Na and Na: K ratio was not significantly associated with the odds of NAFLD. We suggest that more prospective studies are required to investigate the relationship between Na and K intake from a usual diet with the risk of NAFLD.

Authors' contributions

A.S., F.T., and A.M. conceptualized and designed the study. S.R., E.M., M.J, and M.A. participated in the data acquisition and literature review of the articles. F.T. and A.H. analyzed and interpreted the data. A.S., E.M., S.R., M.J., A.M., and M.A. drafted the initial manuscript. A.H. and F.T. supervised the project and all authors approved the final version of the manuscript as submitted.

Acknowledgment

This study is related to the project NO. 1398/10554 from Student Research Committee, Shahid Beheshti University of Medical Sciences (SBMU), Tehran, Iran. The authors

also appreciate the Student Research Committee and Research & Technology Chancellor in SBMU for their financial support of this study.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Received: 04 Jul 20 **Accepted:** 22 Sep 20
Published: 30 Dec 21

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