Original Article

Consumption of fruits, vegetables, and nuts influences the association between serum uric acid and hypertension in Korean adults: A nationwide survey (KNHANES 2016–2021)

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Background and Objectives: This study investigated the influence of dietary intake on the relationship between serum uric acid (SUA) levels and the risk of hypertension. **Methods and Study Design:** Data from the 7th and 8th Korean National Health and Nutrition Examination Survey, a nationally representative survey, were analyzed. A total of 19,140 adults aged 19–64 years were included. Dietary intake was assessed using a 24–hour recall method. SUA levels were measured using a Hitachi Automatic Analyzer 7600–210. Hypertension was defined as a systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or the use of antihypertensive medication. Statistical analyses included generalized linear regression, Cochran–Mantel–Haenszel analysis, and multivariate logistic regression. **Results:** The highest SUA quartile (Q4) had an odds ratio (OR) of 1.68 (95% CI: 1.41–1.99) for hypertension compared to the lowest quartile (Q1). Additionally, the association between elevated SUA levels and increased hypertension risk was stronger in participants who consumed fewer fruits (OR: 1.79, 95% CI: 1.44–2.21), vegetables (1.80, 1.43–2.25), nuts (1.87, 1.51–2.33), and milk (2.20, 1.73–2.79). The ORs (95 % CI) for the highest SUA quartile on hypertension were 1.68 (1.39–2.04), 1.35 (1.13–1.62), and 1.89 (1.57–2.27) in those consuming more seafood, meat, and alcohol, respectively. **Conclusions:** Reduced intake of fruits, vegetables, nuts, and milk, and increased consumption of seafood and alcohol, exacerbates the positive association between SUA levels and the risk of hypertension. These findings suggest dietary modifications as a potential strategy for hypertension prevention and management.

Key Words: blood pressure, food intake, hypertension, hyperuricemia, uric acid

INTRODUCTION

The increasing prevalence of hypertension worldwide is a leading cause of cardiovascular disease (CVD).[1,2] Given the close association between hypertension and CVD and the fact that the prevalence of hypertension has not decreased in recent decades, CVD continues to account for approximately 33% of global deaths.[2] Therefore, lowering blood pressure in patients with hypertension may prevent or alleviate the associated complications and mortality.[3,4]

Uric acid (UA) is the final product of purine degradation. These purines can be derived from two main sources: endogenous nucleoproteins and exogenous (dietary) precursor proteins in the liver. Increased UA generation, decreased renal UA excretion, and reduced intestinal secretion contribute to an imbalance in UA levels, leading to increased serum uric acid (SUA) levels, i.e., hyperuricemia.[5] Hyperuricemia has been implicated as a potential risk factor and mediator of gout, metabolic syndrome, renal dysfunction, and CVD.[6-8] The potential role of increased SUA levels in the pathogenesis of hypertension was reported over a century ago.[9] Emerging evidence suggests that SUA-mediated kidney afferent arteriolopathy, arterial stiffness, renin-angiotensin-aldosterone system (RAAS) activation, oxidative stress, inflammation, and endothelial dysfunction may be molecular mechanisms involved in the development of hypertension.[10-12] Two systematic reviews and meta-analyses have suggested that hyperuricemia is positively associated with an increased risk of hypertension.[13-14] However, a clear consensus is lacking on the exact effect of SUA on hypertension risk from several epidemiological and clinical studies.[6,7,15,16]

Accumulating evidence indicates a close association between SUA levels and dietary components. The con-

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sumption of fruits, vegetables, legumes, whole grains, dairy products, calcium, and vitamin C is inversely associated with SUA levels, whereas the intake of meat, seafood, fructose-rich beverages, and alcohol is positively associated with increased SUA concentrations.[17-19] A negative relationship between the Dietary Approaches to Stop Hypertension (DASH) diet and SUA levels has been reported, implying the potential SUA-lowering effects of dietary approaches on individuals with hypertension and hyperuricemia.[20,21] Accordingly, dietary intake is believed to affect SUA levels and blood pressure. However, no studies have investigated whether dietary intake influences the relationship between SUA and hypertension. Therefore, there is a need to explore this potential association.

This study investigated the impact of dietary intake on the relationship between SUA levels and hypertension prevalence among Korean adults aged 19–64 years using data from the Korea National Health and Nutrition Examination Survey (KNHANES).

METHODS

Study population

The study population was drawn from the KNHANES using a stratified multistage probability cluster design. This design involved dividing the population into several strata and then randomly selecting clusters within each stratum. This method ensures that the sample is nationally representative and accounts for various demographic factors, enhancing the reliability and generalizability of the findings. Cross-sectional data from 2016 to 2021 were obtained from the Korea Disease Control and Prevention Agency (KDCA). The survey comprised three sections: a health questionnaire, examinations, and nutritional assessment. More information on the survey is available on the website http://knhanes.cdc.go.kr.[22] Among the 46,828 participants in the 2016–2021 KNHANES, 14,848 individuals who were <19 years old and >65 years old, 240 who were pregnant or lactating, and 6,194 who reported low or high energy intake (<500 or >5000 kcal/day) were excluded. Among the 25,546 individuals, 2,436 with missing SUA data, 165 with hypertension, and 3,805 with type 2 diabetes, CVD, or renal disease were also excluded. The final sample size was 19,140 participants (7,885 male and 11,255 female). This study was performed in accordance with the Declaration of Helsinki of 1995 (revised in 2000) and approved by the Institutional Review Board (IRB) of the KDCA (IRB No: 2018-01-03-P-A and 2018-01-03-C-A). The requirement for informed consent was waived because of the use of anonymous and identified information.

Measurement of uric acid

Blood samples were collected after an overnight fast. Biochemical markers, including SUA, blood urea nitrogen, creatinine, total cholesterol, triglycerides, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and fasting serum glucose levels were measured on the same day. Blood samples were analyzed using a Hitachi Automatic Analyzer 7600–210 (Hitachi, Tokyo, Japan).

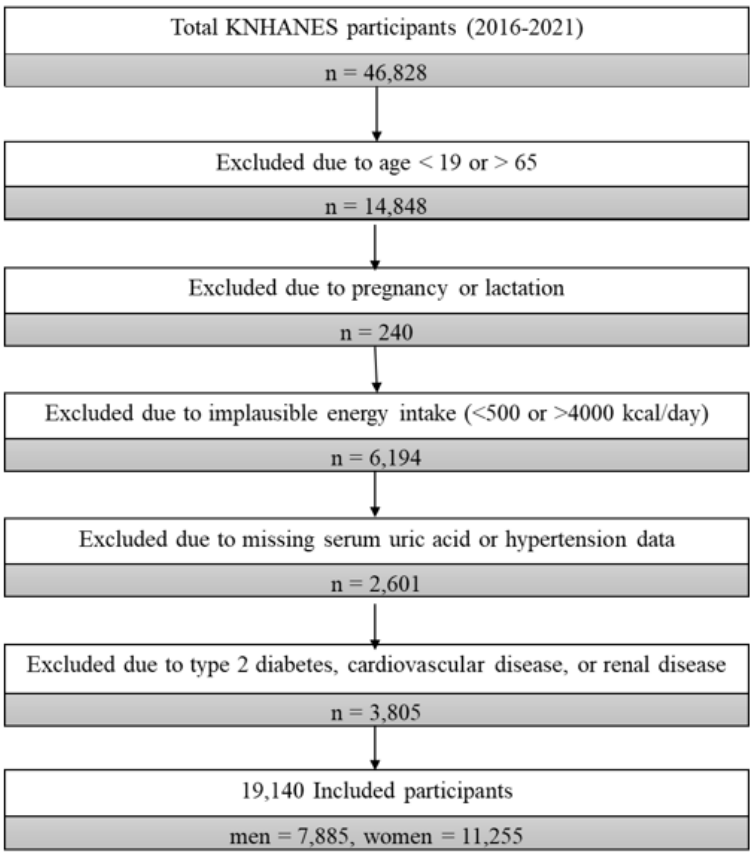

Figure 1. Participants flow diagram

Assessment of dietary intake

Dietary intake was assessed by trained nutritionists using 24-hour single recall, in which trained dietitians collected detailed information about all foods and drinks consumed by participants over the past 24h. Although this method is useful for capturing detailed dietary data, it may introduce recall bias, as participants may not accurately remember or report their intake. This potential limitation could affect the study's findings by either underestimating or overestimating actual intake. However, in the 2009 KNHANES, only slight changes were observed when a single 24h dietary recall was compared with data obtained over 2–10 days.[23] Calculations of calories and nutrient intake in this study were based on the Food Composition Table published by the Korean Rural Development Administration (RDA).[24-26] Daily nutrient and food intake were analyzed as intake per 1,000 kcal of calories to exclude the effect of the difference in calories intake on the intake of each nutrient and food as much as possible. Individuals were then classified into high- and low-consumption groups according to the median intake of each food item.

Blood pressure measurement and hypertension definition

Participants were rested for over 5 min in a sitting position before blood pressure was measured three times on the right arm using a mercury sphygmomanometer (Baumanometer Wall Unit 33(0850), W.A. Baum Co. Inc., Copiague, NY, USA). The average of the second and third blood pressure measurements was estimated and used in this study. Hypertension was defined as a mean systolic blood pressure (SBP) ≥ 140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg or the use of antihypertensive medications, according to the criteria of the Seventh Report of the Joint National Committee (JNC VII) criteria.[27]

General characteristics, anthropometric measurements, and biochemical variables

Health questionnaires and examinations were used to collect information on demographic and socioeconomic characteristics including age, sex, income, education, alcohol consumption, aerobic exercise, resistance exercise, and smoking status. Household monthly income was categorized into two groups: <50th percentile or ≥50th percentile. Education was classified <high school and ≥high school. Alcohol intake was categorized into two groups: current drinkers and nondrinkers. Data were divided into yes or no for both aerobic and resistance exercise. Additionally, anthropometric measurements of height and weight were performed by trained medical staff following standard procedures for health examinations. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Height and weight were measured to the nearest 0.1 cm and 0.1 kg using a stadiometer and an electronic balance, respectively.

Statistical analysis

Data were analyzed using SAS v9.4 (SAS Institute Inc., Cary, NC, USA). Participants were stratified into quartiles based on their SUA levels. Generalized linear regression and Cochran–Mantel–Haenszel analysis were used to explore the association between daily dietary intake, SUA concentrations and linear trends according to SUA quartiles. Data are presented as mean ± standard error for continuous variables and as frequency (percentage) for categorical data. To demonstrate the accuracy of the estimated regression coefficients, we have presented the standard error. The standard error shows how narrowly the coefficients are distributed, thus helping to assess the reliability of the coefficient estimates. Odds ratio (OR) and 95% confidence interval (CI) were estimated using multivariate logistic regression analysis with adjustments of age (years), sex (men, women), BMI (kg/m^2), education level (<high school, ≥high), monthly income status (< 50th, ≥50th), alcohol intake (yes, no), smoking status (yes, no), aerobic exercise (yes, no), and energy intake (kcal/day). Adjusting for these potential confounders ensures that the observed associations reflect true relationships rather than being influenced by these variables. This comprehensive adjustment increases the validity of our findings, highlighting the importance of SUA levels and dietary intake in hypertension risk. It also suggests that interventions aimed at modifying these confounders could effectively manage hypertension risk in the population. Significance was defined as $p < 0.05$.

RESULTS

A total of 19,140 adults (men: 7,885, women: 11,255) were included in this study. Table 1 presents the characteristics of the study participants across the SUA quartiles. The average SUA levels exhibited an increasing trend across the SUA quartiles. Participants with higher SUA levels were more likely to be younger and male. Quartile 4 (highest SUA levels) was characterized by the highest BMI, elevated SBP and DBP, increased concentrations of total cholesterol, triglycerides, LDL cholesterol, and high-sensitivity C-reactive protein (hs-CRP), and an elevated atherogenic index; however, it had the lowest HDL cholesterol. Participants with higher SUA levels were more likely to be current drinkers and smokers and had higher education levels.

Table 2 shows the dietary intake information, including daily nutrient intake and food categories of the study participants, classified by their SUA levels. SUA concentrations were positively associated with the daily intake of protein, fat, saturated fatty acids, monounsaturated fatty acids, sodium, and niacin and inversely associated with carbohydrate and folate intake. Additionally, participants with higher SUA levels consumed more meat and alcohol. Table 3 shows the results of the multivariate analysis used to assess the association between SUA quartiles and hypertension risk. As SUA levels increased, the prevalence of hypertension also increased. Participants with high SUA levels had significantly higher odds of developing hypertension compared to those with low SUA levels. After adjusting for age, sex, BMI, education level, income status, alcohol intake, smoking, exercise, and energy intake, the ORs were 1.22 (95% CI: 1.04–1.43) and 1.68 (95% CI: 1.41–1.99) in Q3 and Q4, respectively, compared with that in Q1 (p-trend < 0.001).

Table 1. General characteristics of the participants according to serum uric acid quartiles[†]

	Q 1 (n = 4,606) <4.0 mg/dL	Q 2 (n = 4,714) 4.1-4.8 mg/dL	Q 3 (n = 4,978) 4.9-5.9 mg/dL	Q 4 (n = 4,842) >6.0 mg/dL	*p*-trend[‡]
Age (years)	45.0 ± 0.17	44.7 ± 0.18	43.9 ± 0.18	41.5 ± 0.18	<0.001
Sex (men, %)	395 (8.6)	857 (18.2)	2456 (49.4)	4174 (86.3)	<0.001
Body mass index (kg/m^2)	22.3 ± 0.05	23.0 ± 0.05	24.0 ± 0.05	25.5 ± 0.05	<0.001
SBP (mmHg)	112 ± 0.21	113 ± 0.21	116 ± 0.21	120 ± 0.20	<0.001
DBP (mmHg)	72.8 ± 0.13	74.3 ± 0.14	76.5 ± 0.14	79.6 ± 0.15	<0.001
Glucose (mg/dL)	93.3 ± 0.14	94.1 ± 0.17	95.5 ± 0.15	97.1 ± 0.16	<0.001
HbA1c (%)	5.47 ± 0.01	5.50 ± 0.01	5.53 ± 0.01	5.54 ± 0.01	<0.001
Total cholesterol (mg/dL)	191 ± 0.50	195 ± 0.51	196 ± 0.52	200 ± 0.53	<0.001
Triglycerides (mg/dL)	96.8 ± 0.97	107 ± 1.14	128 ± 1.36	175 ± 2.08	<0.001
LDL cholesterol (mg/dL)	114 ± 0.44	118 ± 0.46	119 ± 0.47	121 ± 0.49	<0.001
HDL cholesterol (mg/dL)	57.5 ± 0.19	55.8 ± 0.19	52.1 ± 0.18	47.6 ± 0.16	<0.001
Atherogenic index	2.4 ± 0.01	2.7 ± 0.02	2.9 ± 0.02	3.4 ± 0.02	<0.001
Hs-CRP (mg/L)	0.86 ± 0.03	0.98 ± 0.03	1.10 ± 0.04	1.29 ± 0.04	<0.001
BUN (mg/dL)	13.2 ± 0.06	13.6 ± 0.06	14.2 ± 0.05	14.8 ± 0.06	<0.001
Creatinine (mg/dL)	0.68 ± 0.002	0.73 ± 0.002	0.81 ± 0.002	0.93 ± 0.003	<0.001
Serum uric acid (mg/dL)	3.49 ± 0.007	4.45 ± 0.003	5.36 ± 0.004	6.97 ± 0.012	<0.001
Current drinker (%)	3334 (72.3)	3555 (75.2)	4018 (81.9)	4204 (87.4)	<0.001
Current smoker (%)	322 (7.0)	561 (12.0)	1028 (20.8)	1579 (32.8)	0.001
Aerobic exercise (%)	1977 (44.7)	2070 (45.7)	2343 (48.8)	2366 (51.2)	0.569
Education (≥ high school, %)	3743 (84.6)	3864 (85.3)	4212 (87.7)	4203 (90.8)	0.010
Monthly income (≥ 50th, %)	3133 (68.2)	3171 (67.5)	3429 (69.0)	3377 (69.9)	0.632

BUN, blood urea nitrogen; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; Hs-CRP, high sensitivity C-reactive protein; LDL, low-density lipoprotein; SBP; systolic blood pressure.
[†]Values were expressed as means ± standard error or number (%).
[‡]*p*-trend obtained in general linear model analysis and Cochran-Mantel-Haenszel analysis with adjustment for age and sex.

Table 2. Daily dietary intake of the participants according to serum uric acid quartiles[†]

	Q 1 (n = 4,606) <4.0 mg/dL	Q 2 (n = 4,714) 4.1-4.8 mg/dL	Q 3 (n = 4,978) 4.9-5.9 mg/dL	Q 4 (n = 4,842) >6.0 mg/dL	*p*-trend[‡]
Energy (kcal)	1745 ± 9.78	1817 ± 10.6	2007 ± 11.6	2249 ± 12.5	0.230
Carbohydrate (g)	61.9 ± 0.18	60.8 ± 0.18	59.4 ± 0.19	56.6 ± 0.20	<0.001
Protein (g)	14.7 ± 0.06	14.8 ± 0.06	14.9 ± 0.06	15.1 ± 0.07	0.015
Fat (g)	21.3 ± 0.14	21.7 ± 0.13	21.8 ± 0.13	21.8 ± 0.14	0.001
SFA	7.65 ± 0.06	7.77 ± 0.06	7.82 ± 0.06	7.78 ± 0.06	0.036
MUFA	7.50 ± 0.06	7.66 ± 0.06	7.77 ± 0.06	7.89 ± 0.06	0.001
PUFA	6.18 ± 0.05	6.27 ± 0.05	6.21 ± 0.05	6.21 ± 0.05	0.071
Calcium (mg)	291 ± 2.23	288 ± 2.17	271 ± 1.96	253 ± 1.88	0.061
Phosphorus (mg)	571 ± 2.12	570 ± 2.14	557 ± 2.02	543 ± 2.11	0.871
Iron (mg)	6.2 ± 0.04	6.1 ± 0.04	5.9 ± 0.04	5.7 ± 0.04	0.090
Sodium (mg)	1750 ± 11.7	1763 ± 12.7	1763 ± 11.3	1769 ± 10.9	0.023
Potassium (mg)	1559 ± 7.90	1547 ± 7.75	1479 ± 7.22	1379 ± 6.97	0.871
Vitamin A (μgRE)	347 ± 5.94	335 ± 5.54	321 ± 5.25	293 ± 4.70	0.932
Carotene (μg)	1647 ± 22.5	1621 ± 23.3	1508 ± 20.6	1364 ± 19.2	0.779
Retinol (μg)	84.5 ± 2.53	85.0 ± 2.24	80.3 ± 2.02	76.5 ± 2.16	0.792
Vitamin B1 (mg)	0.67 ± 0.004	0.67 ± 0.004	0.67 ± 0.004	0.66 ± 0.004	0.228
Vitamin B2 (mg)	0.88 ± 0.005	0.87 ± 0.005	0.85 ± 0.005	0.84 ± 0.005	0.799
Niacin (mg)	6.91 ± 0.04	6.98 ± 0.04	7.04 ± 0.04	6.99 ± 0.05	0.006
Folate (μgDFE)	182 ± 1.14	179 ± 1.14	170 ± 1.08	157 ± 1.05	0.017
Vitamin C (mg)	39.9 ± 0.73	38.5 ± 0.59	34.4 ± 0.59	31.7 ± 0.79	0.133
Fruits (g)	107.0 ± 2.10	101.5 ± 2.01	87.1 ± 1.84	63.8 ± 1.62	0.563
Vegetables (g)	165.0 ± 1.62	166.0 ± 1.76	157.5 ± 1.52	149.4 ± 1.45	0.271
Nuts (g)	4.0 ± 0.21	4.3 ± 0.27	3.6 ± 0.21	2.7 ± 0.16	0.468
Milk (g)	55.4 ± 1.30	54.7 ± 1.27	46.2 ± 1.10	39.5 ± 1.10	0.839
Seafood (g)	17.8 ± 0.85	15.4 ± 0.73	14.4 ± 0.65	13.1 ± 0.74	0.061
Meat (g)	53.6 ± 0.95	56.3 ± 0.92	60.5 ± 0.91	69.0 ± 1.00	0.008
Alcohol drink (g)	34.6 ± 1.71	39.3 ± 1.73	50.0 ± 1.80	71.0 ± 2.15	<0.001

SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid; BUN, blood urea nitrogen; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; Hs-CRP, high sensitivity C-reactive protein; LDL, low-density lipoprotein; SBP; systolic blood pressure.
[†]Values were expressed as means ± standard error. Nutrient and food intake was represented as grams per 1000 kcal.
[‡]*p*-trend obtained in general linear model analysis with adjustment for age, sex, body mass index, alcohol intake, smoking, education level, income status, and exercise.

Table 3. Prevalence and odds ratio (95% confidence interval) of serum uric acid quartiles on hypertension

	Prevalence (%)†	Unadjusted OR (95% CI)‡	Adjusted OR (95% CI)§
Serum uric acid quartiles			
Q 1 (n = 4,606)	332 (7.2)	1.00 (reference)	1.00 (reference)
Q 2 (n = 4,714)	423 (9.0)	1.27 (1.09 - 1.47)	1.12 (0.95 - 1.31)
Q 3 (n = 4,978)	588 (11.8)	1.72 (1.50 - 1.99)	1.22 (1.04 - 1.43)
Q 4 (n = 4,842)	845 (17.5)	2.72 (2.38 - 3.11)	1.68 (1.41 - 1.99)
p-trend		<0.001	<0.001
Hyperuricemia			
No (n = 16,638)	1,699 (10.2)	1.00 (reference)	1.00 (reference)
Yes (n = 2,502)	489 (19.5)	2.14 (1.91 - 2.39)	1.54 (1.35 - 1.74)
p-value		<0.001	<0.001

CI, confidence interval; OR, odd ratio.
†Prevalence was conducted by Cochran-Mantel-Haenszel analysis with adjustment for age and sex.
‡Odd ratios and 95% CIs were conducted by general linear model analysis.
§Adjusted ORs (95% CIs) was obtained with adjustment for age, sex, body mass index, education level, income status, alcohol intake, smoking, exercise, and energy intake.

Table 4 illustrates the association between SUA levels and the risk of hypertension according to food intake. The stratified analysis demonstrated that low intake of fruits (OR: 1.79, 95% CI: 1.44–2.21, *p*-trend < 0.001), vegetables (OR: 1.80, 95% CI: 1.43–2.25, *p*-trend < 0.001), nuts (OR: 1.87, 95% CI: 1.51–2.33, *p*-trend < 0.001), and milk (OR: 2.20, 95% CI: 1.73–2.79, *p*-trend < 0.001) had a stronger association with SUA levels and hypertension risk compared with high intake of these foods. Additionally, the ORs of Q4 in SUA levels on hypertension were 1.68 (95% CI: 1.39–2.04, *p*-trend < 0.001), 1.35 (95% CI: 1.13–1.62, *p*-trend < 0.001), and 1.89 (95% CI: 1.57–2.27, *p*-trend < 0.001) in participants consuming more seafood, meat, and alcohol, respectively.

DISCUSSION

This study investigated the effect of daily food intake on the relationship between SUA levels and hypertension in Korean adults using data from the KNHANES. An increasing trend in SBP, DBP, and hs-CRP levels was observed in participants with elevated SUA levels in the quartiles. This positive association between SUA levels and odds of hypertension persisted even after adjustment. The odds of hypertension were significantly higher in participants in the highest SUA quartile who consumed fewer fruits, vegetables, nuts, and milk and more seafood and alcohol daily than in participants who did not.

Accumulating evidence from epidemiological studies has shown a strong association between SUA levels and hypertension. After dividing the SUA results into quartiles and adjusting for multiple confounders, the OR in the highest quartile was 1.90 (1.55–2.33) compared with the lowest SUA quartile in a United States (US) national cross-sectional study.[28] Using a nationwide representative data from China Adult Chronic Disease and Nutrition Surveillance, the ORs of hypertension in Han and Yugur participants with the highest SUA in the quintile were 3.16 (2.26–4.43) and 2.37 (1.46–3.89), respectively.[29] Ali et al. also reported that higher SUA concentrations were associated with increased SBP and DBP in the quartiles of Bangladeshi adults.[30] In addition, two meta-analyses revealed a close relationship between an increased risk of hypertension and hyperuricemia.[13,14] Similar to previous rural, community-based prospective cohort studies,[31-33] respective cross-sectional studies,[34] and nationwide-population-based studies,[35,36] elevated SUA levels in Korean adults in the current study were strongly linked to higher hypertension events.

A positive association between SUA levels and the prevalence of hypertension has not been consistently reported, owing to differences in baseline characteristics, such as age and sex. A previous meta-analysis indicated that the risk of hyperuricemia-associated hypertension may be relatively high in younger and female participants.[13] In the general adult population in the US, a lower risk of hypertension was observed in older age groups than in younger age groups, with differences between men and women.[28] According to a community-based prospective study in Korea, the highest risk of hypertension was observed in women aged 40–49 years with the highest SUA levels (OR: 1.44, 95% CI: 1.15–1.81).[33] A strong association between SUA levels and hypertension has been reported in Bangladeshi female adults.[30] These results indicate that women with high SUA concentrations may be more susceptible to hypertension than men.[36] Inconsistent with the above, another study found that younger participants, both male and female, had a higher risk of SUA-related hypertension than older adults in Korea.[32] Additionally, a positive relationship between the blood pressure and SUA quartiles was observed in both male and female residents of the Japanese community.[37] In the present study, participants with the highest SUA levels were more likely to be younger and male. Considering that older individuals and men have more risk factors for hypertension, further longitudinal studies are needed to determine whether changes in SUA levels with age or sex may cause hypertension or serve as an early physiological marker of hypertension in the Korean population.

Several intervention studies on the influence of diet on SUA concentrations have shown a lowering effect with the consumption of fruits, vegetables, legumes, dairy products, whole grains, calcium, and vitamin C, and an increasing effect with the consumption of meat, seafood, fructose-rich beverages, and alcohol.[17-19] A strong association between hyperuricemia and higher alcohol, meat, and seafood consumption was observed in 856 Chinese women

Table 4. Prevalence and Odds ratio (95% confidence interval) of serum uric acid quartiles on hypertension according to food intake

	Q 1 (n = 4,606) <4.0 mg/dL	Q 2 (n = 4,714) 4.1-4.8 mg/dL	Q 3 (n = 4,978) 4.9-5.9 mg/dL	Q 4 (n = 4,842) >6.0 mg/dL	*p*-trend
Fruits					
High (≥62.6g)					
N (%)	171 (8.1)	229 (10.2)	341 (13.4)	482 (18.1)	
OR (95% CI)†	1.00 (reference)	1.09 (0.89 - 1.33)	1.11 (0.91 - 1.37)	1.38 (1.11 - 1.72)	<0.001
Low (<62.6g)					
N (%)	161 (6.5)	194 (7.9)	247 (10.1)	363 (16.7)	
OR (95% CI)†	0.88 (0.69 - 1.12)	1.03 (0.83 - 1.30)	1.22 (0.99 - 1.51)	1.79 (1.44 - 2.21)	<0.001
Vegetables					
High (≥253.1g)					
N (%)	200 (7.7)	249 (9.7)	303 (12.2)	319 (16.7)	
OR (95% CI)†	1.00 (reference)	1.13 (0.91 - 1.41)	1.24 (1.00 - 1.53)	1.62 (1.30 - 2.01)	<0.001
Low (<253.1g)					
N (%)	132 (6.6)	174 (8.1)	285 (11.5)	526 (17.9)	
OR (95% CI)†	1.02 (0.81 - 1.29)	1.13 (0.90 - 1.41)	1.22 (0.98 - 1.52)	1.80 (1.43 - 2.25)	<0.001
Nuts					
High (≥0.6g)					
N (%)	181 (7.7)	212 (8.9)	299 (12.1)	402 (17.2)	<0.001
OR (95% CI)†	1.00 (reference)	1.04 (0.84 - 1.30)	1.17 (0.95 - 1.45)	1.53 (1.23 - 1.91)	
Low (<0.6g)					
N (%)	151 (6.7)	211 (9.1)	289 (11.6)	443 (17.7)	<0.001
OR (95% CI)†	1.03 (0.81 - 1.30)	1.24 (1.00 - 1.55)	1.30 (1.05 - 1.61)	1.87 (1.51 - 2.33)	
Milk					
High (≥0.05g)					
N (%)	122 (5.5)	157 (7.0)	224 (10.1)	299 (15.1)	<0.001
OR (95% CI)†	1.00 (reference)	1.17 (0.91 - 1.51)	1.39 (1.09 - 1.77)	1.84 (1.43 - 2.37)	
Low (<0.05g)					
N (%)	210 (8.8)	266 (10.7)	364 (13.2)	546 (19.0)	<0.001
OR (95% CI)†	1.39 (1.09 - 1.77)	1.51 (1.19 - 1.90)	1.56 (1.24 - 1.97)	2.20 (1.73 - 2.79)	
Seafood					
Low (<1.4g)					
N (%)	147 (6.5)	224 (9.4)	276 (11.2)	395 (16.1)	<0.001
OR (95% CI)†	1.00 (reference)	1.20 (1.00 - 1.45)	1.17 (0.97 - 1.42)	1.54 (1.27 - 1.87)	
High (≥1.4g)					
N (%)	185 (8.0)	199 (8.5)	312 (12.4)	450 (18.8)	<0.001
OR (95% CI)†	1.14 (0.95 - 1.38)	1.04 (0.87 - 1.26)	1.34 (1.12 - 1.60)	1.68 (1.39 - 2.04)	
Meat					
Low (<76.0g)					
N (%)	228 (8.4)	245 (9.6)	305 (12.7)	370 (19.3)	<0.001
OR (95% CI)†	1.00 (reference)	1.01 (0.86 - 1.18)	1.10 (0.94 - 1.30)	1.50 (1.26 - 1.80)	
High (≥76.0g)					
N (%)	104 (5.5)	178 (8.2)	283 (11.0)	475 (16.3)	<0.001
OR (95% CI)†	0.86 (0.71 - 0.06)	0.96 (0.80 - 1.16)	1.12 (0.94 - 1.33)	1.35 (1.13 - 1.62)	
Alcohol					
Low (<0.001g)					
N (%)	267 (7.4)	303 (8.4)	406 (11.2)	507 (15.9)	
OR (95% CI)†	1.00 (reference)	0.98 (0.85 - 1.13)	1.06 (0.91 - 1.22)	1.32 (1.12 - 1.55)	<0.001
High (≥0.001g)					
N (%)	65 (6.7)	120 (10.9)	182 (13.3)	338 (20.6)	
OR (95% CI)†	0.88 (0.69 - 1.12)	1.16 (0.95 - 1.43)	1.45 (1.20 - 1.75)	1.89 (1.57 - 2.27)	<0.001

CI, confidence interval; OR, odd ratio

†OR and 95% CIs were conducted by general linear model analysis with adjustment for age, sex, body mass index, education level, income status, alcohol intake, smoking, exercise, and energy intake.

aged 60–102 years.[38] In addition, an inverse association between dairy consumption and SUA levels has been reported in a study that used a representative sample of women and men in the US.[19] In the current study, participants with higher SUA levels tend to eat less fruit, nuts, and milk, and seafood, although this was not significant. However, participants in the highest SUA quartile consumed more meat and alcohol than those in the lowest quartile.

Researchers conducted an intervention study using the DASH diet to determine the effect of food intake on the relationship between SUA levels and hypertension risk. DASH is a healthy eating plan designed for individuals with high blood pressure, focusing on nutrients such as calcium, fiber, magnesium, and potassium found in fruits, vegetables, legumes, dairy products, and whole grains. Participants with higher DASH scores tend to have lower SUA levels and a lower risk of hypertension compared to

those with lower DASH scores among 66,427 Chinese adults.[39] In an ancillary study of the DASH-sodium trial, a 30-day DASH diet intervention reduced SUA concentrations in 103 participants with prehypertension and stage I hypertension.[40] In the present study, SUA levels were negatively associated with niacin and folate intake and positively associated with protein, fat, and sodium intake. Furthermore, an increased risk of hypertension was observed in participants with a lower intake of fruits, vegetables, nuts, and milk and higher levels of SUA after adjusting for potential confounders. Participants who consumed more seafood and alcohol and had higher SUA levels had a higher risk of hypertension. Overall, our findings demonstrate the impact of food intake and SUA levels on the risk of hypertension.

Several mechanisms underlying the association between SUA and hypertension have been proposed based on *in vivo* and *in vitro* studies. In rats with mild hyperuricemia, hypertension and renal fibrosis are partially induced by RAAS activation and reduced nitric oxide synthase, a marker of oxidative stress.[41] In human vascular smooth muscle cells and HepG2 cells, UA upregulated expression and secretion of the inflammatory monocyte chemoattractant protein-1 and stimulated the activation of nuclear factor-κB.[42-44] A cross-sectional study of 1,253 men and 1,478 women showed that high SUA levels were closely associated with the circulating inflammatory component, hs-CRP, which contributes to the development of cardiovascular and metabolic diseases.[44] In this study, participants with higher SUA levels exhibited higher hs-CRP concentrations than those with lower levels. Therefore, a potential explanation for the relationship between SUA levels and hypertension risk may be UA-induced inflammation.

The mechanisms underlying the influence of daily food intake on the association between elevated SUA levels and the risk of hypertension have not been fully elucidated. As mentioned earlier, low-grade inflammation may be the mechanism underlying the relationship between SUA levels and hypertension. Increased vegetable and fruit intake decrease the circulating levels of hs-CRP and IL-6, whereas increased meat and alcohol consumption increases the levels of inflammatory biomarkers and stress.[45-47] Dietary components play a crucial role in SUA metabolism and blood pressure regulation through various biological mechanisms.[17] Fruits and vegetables are rich in antioxidants, vitamins, and minerals, which help reduce oxidative stress and inflammation, potentially lowering SUA levels and improving endothelial function. Dairy products provide calcium, potassium, and magnesium, which are essential for vascular health and can modulate blood pressure. Conversely, high meat consumption, particularly red and processed meats, can lead to increased purine intake, subsequently raising SUA levels and promoting hyperuricemia. Alcohol, especially beer, also contains high purine content and contributes to elevated SUA levels and increased blood pressure through its effects on liver metabolism and diuretic properties. Seafood, although generally healthy, can also be a source of purines, which need to be balanced within the diet. Understanding these dietary influences can aid in developing targeted nutritional interventions to manage SUA levels and prevent hypertension.

To our knowledge, this is the first study to demonstrate the influence of daily food intake on SUA concentrations and the risk of hypertension in a Korean adult population based on a nationally representative sample. However, this study has some limitations. First, the cross-sectional design did not allow us to study the causal relationships among food intake, SUA levels, and hypertension. Cross-sectional studies capture a snapshot of a population at a single point in time, which limits our ability to establish temporal relationships between variables. Consequently, while we can observe associations between SUA levels, dietary intake, and hypertension, we cannot infer causality. Further longitudinal studies are required to confirm this causal relationship in the Korean adult population. Second, daily food intake was evaluated using a single 24h dietary recall, which may not accurately reflect long-term or usual dietary intakes. Moreover, the potential effect of food intake may be underestimated or overestimated.

The current study demonstrated a positive association between SUA levels and the risk of hypertension among Korean adults. Participants who consumed fewer fruits, vegetables, nuts, and milk and more seafood and alcohol showed a stronger association between SUA concentrations and hypertension risk. These findings may be useful in developing dietary guidelines for preventing and controlling hypertension. However, further longitudinal studies are needed to confirm causality and better understand the long-term impact of dietary patterns on SUA levels and hypertension risk.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Supplementary Tables

Supplementary Table 1. General characteristics of the participants according to serum uric acid quartiles†

	Without hypertension (n = 16,638)	With hypertension (n = 2,502)	*p*-value‡
Age (years)	44.2 ± 0.10	41.1 ± 0.25	<0.001
Sex (men, %)	6,049 (36.4)	1,836 (73.4)	<0.001
Body mass index (kg/m^2)	23.4 ± 0.03	26.1 ± 0.08	<0.001
SBP (mmHg)	114 ± 0.11	120 ± 0.29	<0.001
DBP (mmHg)	75.2 ± 0.07	80.1 ± 0.21	<0.001
Glucose (mg/dL)	94.7 ± 0.08	97.2 ± 0.22	<0.001
HbA1c (%)	5.50 ± 0.003	5.56 ± 0.01	<0.001
Total cholesterol (mg/dL)	194 ± 0.28	204 ± 0.76	<0.001
Triglycerides (mg/dL)	118 ± 0.72	185 ± 3.12	<0.001
LDL cholesterol (mg/dL)	117 ± 0.25	123 ± 0.71	<0.001
HDL cholesterol (mg/dL)	54.1 ± 0.10	47.3 ± 0.23	<0.001
Atherogenic index	2.8 ± 0.01	3.5 ± 0.03	<0.001
Hs-CRP (mg/L)	1.00 ± 0.02	1.44 ± 0.06	<0.001
BUN (mg/dL)	13.8 ± 0.03	14.8 ± 0.08	<0.001
Creatinine (mg/dL)	0.77 ± 0.001	0.92 ± 0.005	<0.001
Serum uric acid (mg/dL)	4.74 ± 0.01	7.48 ± 0.02	<0.001
Current drinker (%)	12,980 (78.4)	2,131 (85.8)	<0.001
Current smoker (%)	2,742 (16.6)	748 (30.1)	<0.001
Aerobic exercise (%)	8,455 (52.9)	1,169 (49.1)	0.001
Education (≥ high school, %)	13,874 (86.7)	2,148 (90.0)	<0.001
Monthly income (≥ 50th, %)	11,410 (68.7)	1,700 (68.1)	0.532

BUN, blood urea nitrogen; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein; Hs-CRP, high sensitivity C-reactive protein; LDL, low-density lipoprotein; SBP; systolic blood pressure.
†Values were expressed as means ± standard error or number (%).
‡*p*-value obtained in general linear model analysis and Cochran-Mantel-Haenszel analysis with adjustment for age and sex.

Supplementary Table 2. Daily dietary intake of the participants according to serum uric acid quartiles[†]

	Without hypertension (n = 16,638)	With hypertension (n = 2,502)	*p*-value[‡]
Energy (kcal)	1931 ± 6.08	2139 ± 17.3	0.004
Carbohydrate (g)	150 ± 0.25	141 ± 0.71	<0.001
Protein (g)	37.1 ± 0.08	37.8 ± 0.24	0.261
Fat (g)	24.0 ± 0.08	24.7 ± 0.22	0.067
SFA	7.73 ± 0.03	7.92 ± 0.09	0.318
MUFA	7.66 ± 0.03	8.03 ± 0.09	0.044
PUFA	6.21 ± 0.02	6.29 ± 0.07	0.301
Calcium (mg)	279 ± 1.12	254 ± 2.66	0.063
Phosphorus (mg)	562 ± 1.12	544 ± 3.03	0.514
Iron (mg)	6.0 ± 0.02	5.7 ± 0.06	0.144
Sodium (mg)	1763 ± 6.29	1752 ± 15.3	0.040
Potassium (mg)	1505 ± 4.04	1387 ± 10.0	0.233
Vitamin A (μgRE)	327 ± 2.90	303 ± 7.48	0.691
Carotene (μg)	1553 ± 11.6	1396 ± 27.7	0.812
Vitamin B1 (mg)	0.67 ± 0.002	0.66 ± 0.006	0.018
Vitamin B2 (mg)	0.86 ± 0.003	0.84 ± 0.007	0.610
Niacin (mg)	6.98 ± 0.02	7.01 ± 0.07	0.675
Folate (μgDFE)	174 ± 0.60	156 ± 1.46	0.002
Vitamin C (mg)	36.7 ± 0.35	31.7 ± 1.17	0.299
Fruits (g)	93.0 ± 1.04	66.5 ± 2.37	0.804
Vegetables (g)	161 ± 0.86	150 ± 2.06	0.627
Nuts (g)	3.8 ± 0.12	2.6 ± 0.22	0.375
Milk (g)	49.8 ± 0.64	42.4 ± 1.64	0.696
Seafood (g)	15.6 ± 0.40	12.2 ± 0.99	0.303
Meat (g)	58.5 ± 0.50	69.8 ± 1.42	0.014
Alcohol drink (g)	46.2 ± 0.97	67.6 ± 3.05	0.002

SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid

[†]Values were expressed as means ± standard error. Nutrient and food intake was represented as grams per 1000 kcal.

[‡]*p*-value obtained in general linear model analysis with adjustment for age, sex, body mass index, alcohol intake, smoking, education level, income status, and exercise.