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Benefits of potassium intake on metabolic syndrome: The fourth Korean National Health and Nutrition Examination Survey (KNHANES IV)

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ABSTRACT

Objective: Potassium intake may be associated with metabolic syndrome and its components, but there has been little evidence so far. We evaluated the association between the metabolic syndrome and potassium intake in the general population.
Methods: Participants were 7542 adults (≥20 years of age) from the fourth Korean National Health and Nutrition Examination Survey (2007–2009), which is a cross-sectional survey of a nationally representative sample of the Korean population. Data were obtained from standardized questionnaires as well as physical and laboratory examination reports. The 24-h recall method was used for dietary assessment. Metabolic syndrome was defined based on the modified National Cholesterol Education Program-Adult Treatment Panel III criteria. Multivariable logistic regression was performed to estimate the odds of metabolic syndrome and its components across potassium intake quartiles.
Results: After adjusting for various lifestyle and dietary confounders, subjects in the highest quartile of potassium intake had 39% lower odds for metabolic syndrome compared to those in the lowest quartile (adjusted odds ratio [aOR] = 0.61; 95% confidence interval [CI]: 0.42–0.89; p for trend: 0.013). This association was consistent for both sexes. Among the components of metabolic syndrome, potassium intake was inversely related to abdominal obesity and fasting hyperglycemia in multivariate analysis (p for trend = 0.049 and 0.010, respectively).
Conclusion: Our results reveal a significant inverse association between potassium intake and metabolic syndrome in adults. Further studies are required to confirm this association.

1. Introduction

The metabolic syndrome is a combination of several metabolic risk factors such as insulin resistance, central obesity, dyslipidemia, and hypertension [1]. The metabolic syndrome has received increasing attention over the past few years, as its prevalence and socioeconomic burden has increased worldwide, including in South Korea [2]. Therefore, many studies have attempted to identify the causes of metabolic syndrome. Diet constitutes one of the major environmental etiologic factors, and the effects of specific foods, nutrients, or electrolytes on metabolic syndrome have been studied.

Potassium is the most abundant intracellular cationic electrolyte, and is necessary for normal cellular function. It also participates in protein synthesis and carbohydrate metabolism [3,4]. Since potassium is readily excreted in the urine rather than stored in the body, the human body needs a constant intake of potassium in the correct amounts. Nevertheless, average potassium consumption in the US and Korean population in 2009–2010 was approximately only 54% and 58% of the recommended amount, respectively [5–7]. According to some studies, low potassium intake has been associated with high blood pressure [8–11] and the risk of developing diabetes [12,13], although these observations have been considered controversial. Moreover, people who consume more vegetables and fruits, typical examples of potassium-rich food, appear to have a lower risk of metabolic syndrome [14]. Hence, potassium intake may be associated with metabolic syndrome. Nevertheless, there has been little evidence so far that supports this association in the

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general population. Therefore, in the present cross-sectional study, we used data from the fourth Korean National Health and Nutrition Examination Survey 2007–2009 (KNHANES IV) to examine the relationship between potassium intake and metabolic syndrome.

2. Methods

2.1. Subjects

The KNHANES IV (2007–2009) is a nationwide survey representing the non-institutionalized civilian Korean population. It consists of the Health Interview Survey, Health Examination Study, and Nutrition Survey. A stratified, multistage probability sampling design was used, and sampling units were based on geographical area, age, and sex. The details of the KNHANES have been previously described [2]. All subjects provided informed consent prior to inclusion in the study.

Initial candidates for the present study included 14,210 adults (≥20 years old) who had completed: 1) the Nutrition Survey; and 2) physical and laboratory examinations with reports on waist circumference, blood pressure, triglyceride levels, fasting glucose levels, and high-density lipoprotein (HDL) cholesterol levels. Subjects who had fasted for less than 12 h before examination were firstly excluded, since this might have affected accurate evaluation of blood profiles ($n = 3496$). We also excluded participants who answered to the questionnaire that they already had been diagnosed with hypertension, dyslipidemia, diabetes, stroke, congestive heart failure, myocardial infarction, renal failure, liver cirrhosis, or any type of cancer by physicians, because individuals' dietary behavior and health status might have been changed after being diagnosed with such diseases ($n = 2860$). We further excluded subjects who reported implausible total energy intake ($n = 249$; <800 or >4200 kcal/day for men; <600 or >3500 kcal/day for women) and who were taking medications that could affect blood pressure, carbohydrate metabolism, and lipoprotein profiles ($n = 8$). Pregnant women were also excluded because of the physiological changes that occur during pregnancy ($n = 55$). Following these exclusions, we included 7542 subjects (2684 men and 4858 women) in the present analysis.

2.2. Demographic and socioeconomic factors

Trained interviewers collected data on the demographic factors and health behaviors of participants via personal interviews. The demographic variables were age (20–29, 30–39, 40–49, 50–59, 60–69, and ≥70 years), sex, highest educational level achieved (elementary school education or less, middle or high school education, and college education or more), and monthly household income (quartiles of equivalized household income). The equivalized household income was calculated as the total monthly household income divided by the square root of the total number of household members. The health behavioral variables included smoking (never smoker, past smoker, or current smoker), alcohol consumption (gram alcohol/day), and physical activity (low, moderate, or high). Participants were to choose whether they were never, past, or current smoker at that time. The average amount and number of consumed alcoholic beverages was assessed by self-reported questionnaire, and then converted into the amount of pure alcohol (in gram) consumed per day [15]. Physical activity was quantified as metabolic equivalent of task minutes per week (MET-minutes per week), which was calculated using the scoring protocol of the Korean version of the International Physical Activity Questionnaire (IPAQ) short form [16]. Accordingly, physical activity levels were then classified as low (<600 MET-min per week), moderate (≥600 to <3000 MET-min per week), or high (≥3000 MET-minutes per week).

2.3. Assessment of dietary intake

Dietary intakes were assessed by a single 24-h recall based on a weekday's food consumption, in which all food content and consumed amounts during the last 24 h were obtained from the participants. Based on these data, consumed nutrients and electrolytes were calculated using the food composition table which was made and validated by Rural Development Administration. Dietary variables used in this study included total energy (kcal/day), carbohydrate (%energy), total fat (%energy), protein (%energy), fiber (g/1000 kcal), vitamin C (mg/1000 kcal), sodium (mg/1000 kcal), and potassium (mg/1000 kcal) intakes. Daily potassium intake was divided into quartiles for data analysis. Additionally, we calculated the ratio of dietary sodium to potassium (Na:K ratio) and divided the ratio values into quartiles to assess its effect on metabolic syndrome and its components.

A 24-h recall is one of the few practically available methods to assess dietary intakes in a large population study due to its methodological convenience and feasibility, which has been used in previous population-based studies [17,18]. The validity of 24-h recall method was reported to be satisfactory on the group level [19]. Estimated potassium intake from a single 24-h recall was known to be highly correlated with pooled long term intake ($r = 0.70$) [20], and significantly correlated with 24-h urinary collection [21,22], including in Korean population [23]. Therefore, although it may not be accurate to characterize individual intake, a single 24-h recall can provide a good estimates of group intakes for potassium [21].

2.4. Anthropometric measures

Body weight and height were obtained using standard protocols to the nearest 0.1 kg and 0.1 cm, respectively. Waist circumference was measured at the narrowest point between the lower borders of the rib cage and the uppermost borders of the iliac crest at the end of normal expiration. Well-trained observers manually measured blood pressure with a mercury sphygmomanometer (Baumanometer; Baum, Copiague, NY).

2.5. Laboratory evaluation

Antecubital vein blood samples were drawn and immediately centrifuged. These blood samples were used to evaluate total cholesterol, HDL cholesterol, triglyceride, and glucose levels. Fasting (fasting time ≥ 12 h) plasma lipids and glucose concentrations were measured enzymatically using ADVIA 1650 (Siemens, USA; February 2007–2008) and Hitachi Automatic Analyzer 7600 (Hitachi, Japan; February 2008–2009) systems.

2.6. Definition of metabolic syndrome

We used the modified National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III) criteria to define metabolic syndrome [1] as the presence of 3 or more of the following components: 1) waist circumference ≥90 cm in men and ≥85 cm in women, adopted from the Korean Society for the Study of Obesity criteria [24]; 2) triglyceride level ≥150 mg/dL; 3) HDL cholesterol level <40 mg/dL in men and <50 mg/dL in women; 4) blood pressure ≥130/85 mmHg; and 5) fasting glucose level ≥100 mg/dL.

2.7. Statistical analysis

The participants' characteristics were compared according to the potassium intake (mg/1000 kcal) quartiles. Data were presented as mean ± SE or as proportion (% and SE), and were age-adjusted using the direct method with the total population selected as the standard population. Age-adjusted means were compared using multivariable linear regression analysis.

Multivariable logistic regression analysis was performed to estimate the odds ratio and 95% confidence interval (CI) of metabolic syndrome and its individual components according to the potassium intake quartiles, using the lowest quartile as reference. We used the multivariate nutrient density model [25] to account for the effects of total energy intake on metabolic syndrome. Odds ratios were initially calculated following adjustment for age (categorical), sex, and BMI (continuous) in model 1. In model 2, categorical variables that were further adjusted for included education, monthly household income, physical activity, as well as smoking status, and alcohol consumption was also adjusted as a continuous variable. Dietary factors such as total energy, carbohydrate, total fat, fiber, vitamin C, and sodium intakes were additionally adjusted for as continuous variables in model 3. To assess the possible effect of the Na:K ratio, we also calculated the odds ratios of metabolic syndrome and its components across the potassium intake quartiles after additionally adjusting for the Na:K ratio in model 3.

Reported probability values were 2-sided and a $p < 0.05$ was considered statistically significant. All statistical analyses were performed using STATA 12.1 (Stata Corp., College Station, TX, USA) with "svy" commands to account for complex sampling design, and included sampling weights, which enabled the results to represent the entire national adult population.

3. Results

3.1. Characteristics of the study population

The age-adjusted prevalence rate of metabolic syndrome was 18.1 ± 0.6% of the weighted total population, where 20.6 ± 1.0% of men and 16.3 ± 1.0% of women had metabolic syndrome. Participants with metabolic syndrome had significantly lower potassium intake than those without metabolic syndrome (age-adjusted means, 1616.2 ± 11.1 vs. 1586.2 ± 28.4 mg/1000 kcal, respectively). Demographic and socioeconomic characteristics of participants and their dietary intakes based on their potassium intake quartiles are presented in Table 1.

3.2. Odds ratios of metabolic syndrome and its components

Potassium intake was inversely associated with metabolic syndrome, as demonstrated by the results presented in Table 2. Prevalence of metabolic syndrome decreased with higher quartiles of potassium intake, and subjects in the highest potassium intake quartile had 39% lower odds of having metabolic syndrome compared to those in the lowest quartile after adjusting for various potential confounders in model 3 (adjusted odds ratio [aOR] = 0.61; 95% CI: 0.42–0.89; $p = 0.010$; p for trend = 0.013). Furthermore, potassium intake was inversely related to individual components of the metabolic syndrome, especially abdominal

Table 1
Characterization of participants according to potassium intake quartiles.

	Potassium intake			
	Quartile 1	Quartile 2	Quartile 3	Quartile 4
Median potassium intake[a] (range, mg/1000 kcal)	1055.1 (<1237.6)	1387.7 (1237.6–1533.9)	1693.6 (1534.0–1904.8)	2229.2 (≥1904.9)
Age[a] (years)	47.5 ± 0.4	44.6 ± 0.3	45.8 ± 0.3	47.0 ± 0.3
BMI (kg/m^2)	23.2 ± 0.1	23.2 ± 0.1	23.2 ± 0.1	23.5 ± 0.1
Education (%)				
≤Elementary	27.5 ± 1.1	22.4 ± 1.0	21.9 ± 1.1	20.6 ± 1.0
Middle/High	49.3 ± 1.5	48.9 ± 1.6	48.5 ± 1.4	49.1 ± 1.5
≥College	23.2 ± 1.3	28.8 ± 1.4	29.7 ± 1.3	30.4 ± 1.4
Household income[b] (%)				
Quartile 1 (low)	21.9 ± 1.2	17.6 ± 1.2	14.7 ± 0.9	16.5 ± 1.2
Quartile 2	28.6 ± 1.4	23.9 ± 1.5	25.5 ± 1.5	23.0 ± 1.4
Quartile 3	26.8 ± 1.4	28.0 ± 1.3	29.1 ± 1.5	29.9 ± 1.6
Quartile 4 (high)	22.7 ± 1.4	30.5 ± 1.8	30.8 ± 1.8	30.6 ± 1.6
Physical activity[c]				
Low	27.8 ± 1.3	27.9 ± 1.2	26.5 ± 1.4	25.0 ± 0.8
Moderate	40.3 ± 1.5	38.0 ± 1.4	41.8 ± 1.5	40.6 ± 0.8
High	31.9 ± 1.4	34.1 ± 1.5	31.6 ± 1.5	34.4 ± 0.8
Smoker (%)				
Never	54.2 ± 1.5	55.3 ± 1.5	59.2 ± 1.5	67.4 ± 1.4
Past	18.3 ± 1.3	20.8 ± 1.2	21.9 ± 1.3	16.9 ± 1.2
Current	27.5 ± 1.4	24.0 ± 1.4	18.9 ± 1.2	15.7 ± 1.1
Alcohol consumption (g/day)	9.8 ± 0.5	7.4 ± 0.4	6.0 ± 0.4	5.3 ± 0.3
Daily dietary intakes				
Total energy (kcal/day)	1879.6 ± 21.8	1846.0 ± 18.1	1779.9 ± 18.5	1667.4 ± 20.5
Carbohydrate (%energy)	65.0 ± 0.4	66.7 ± 0.4	68.4 ± 0.3	71.3 ± 0.4
Total fat (%energy)	7.3 ± 0.1	7.6 ± 0.1	7.4 ± 0.1	6.9 ± 0.1
Protein (%energy)	12.3 ± 0.1	13.9 ± 0.1	15.0 ± 0.1	15.9 ± 0.1
Fiber (g/1000 kcal)	2.7 ± 0.0	3.4 ± 0.0	4.1 ± 0.0	6.1 ± 0.1
Vitamin C (mg/1000 kcal)	27.7 ± 0.6	45.5 ± 0.8	61.1 ± 1.2	98.0 ± 2.6
Sodium (mg/1000 kcal)	2047.3 ± 29.8	2511.0 ± 33.5	2777.4 ± 34.1	3205.4 ± 47.1
Na:K ratio[d]	2.0 ± 0.0	1.8 ± 0.0	1.6 ± 0.0	1.4 ± 0.0

Data represent age-adjusted mean or prevalence (%) ± SE, except for age and potassium intake.
[a] Non-adjusted values.
[b] Monthly equivalized household income.
[c] Defined as low (<600 MET-minutes per week), moderate (≥600 to <3000 MET-minutes per week), and high (≥3000 MET-minutes per week) levels of physical activity.
[d] Ratio of dietary sodium to potassium.

Table 2
Multivariate odds ratio for metabolic syndrome and its components according to potassium intake quartiles.

	Potassium intake				
	Quartile 1	Quartile 2	Quartile 3	Quartile 4	*p* for trend
Metabolic syndrome[a]					
No. of events (%)	369 (16.0)	310 (16.5)	297 (15.8)	293 (15.5)	–
Model 1[b]	1.00	0.82 (0.64–1.06)	0.81 (0.62–1.04)	0.70 (0.54–0.90)	0.009
Model 2[c]	1.00	0.79 (0.61–1.04)	0.77 (0.59–1.00)	0.68 (0.53–0.86)	0.002
Model 3[d]	1.00	0.78 (0.58–1.04)	0.73 (0.53–1.00)	0.61 (0.42–0.89)	0.013
Abdominal obesity[a]					
No. of events (%)	400 (21.2)	386 (20.5)	381 (20.2)	401 (21.3)	–
Model 1[b]	1.00	1.04 (0.79–1.38)	0.89 (0.64–1.23)	0.90 (0.66–1.22)	0.337
Model 2[c]	1.00	1.09 (0.79–1.48)	0.88 (0.64–1.22)	0.70 (0.51–0.96)	0.011
Model 3[d]	1.00	1.19 (0.85–1.68)	0.91 (0.62–1.34)	0.67 (0.43–1.07)	0.049
High blood pressure[a]					
No. of events (%)	455 (24.1)	381 (20.2)	382 (20.3)	349 (18.5)	–
Model 1[b]	1.00	0.89 (0.71–1.11)	0.90 (0.72–1.13)	0.79 (0.63–0.98)	0.048
Model 2[c]	1.00	0.92 (0.71–1.18)	0.91 (0.71–1.18)	0.84 (0.60–1.17)	0.325
Model 3[d]	1.00	0.92 (0.71–1.18)	0.92 (0.70–1.20)	0.85 (0.59–1.22)	0.396
Fasting hyperglycemia[a]					
No. of events (%)	374 (19.8)	334 (17.7)	315 (16.7)	359 (19.1)	–
Model 1[b]	1.00	0.89 (0.72–1.09)	0.81 (0.65–1.00)	0.91 (0.74–1.11)	0.238
Model 2[c]	1.00	0.85 (0.67–1.08)	0.76 (0.60–0.96)	0.86 (0.68–1.08)	0.131
Model 3[d]	1.00	0.82 (0.64–1.06)	0.68 (0.52–0.89)	0.68 (0.50–0.94)	0.010
Hypertriglyceridemia[a]					
No. of events (%)	490 (26.0)	411 (21.8)	397 (21.1)	413 (21.9)	–
Model 1[b]	1.00	0.76 (0.63–0.92)	0.79 (0.65–0.95)	0.83 (0.68–1.01)	0.098
Model 2[c]	1.00	0.78 (0.63–0.97)	0.84 (0.67–1.04)	0.96 (0.78–1.17)	0.861
Model 3[d]	1.00	0.75 (0.59–0.94)	0.79 (0.61–1.01)	0.88 (0.66–1.18)	0.403
Low HDL[a]					
No. of events (%)	812 (43.1)	834 (44.2)	876 (46.5)	890 (47.2)	–
Model 1[b]	1.00	1.14 (0.95–1.36)	1.21 (1.02–1.45)	1.09 (0.92–1.30)	0.218
Model 2[c]	1.00	1.03 (0.85–1.24)	1.08 (0.89–1.30)	1.04 (0.89–1.25)	0.579
Model 3[d]	1.00	1.03 (0.84–1.26)	1.05 (0.85–1.30)	0.96 (0.75–1.24)	0.886

HDL: high-density lipoprotein.
[a] Metabolic syndrome was defined as ≥3 components of the following: 1) abdominal obesity (waist ≥90 cm in men, ≥85 cm in women); 2) hypertriglyceridemia (≥150 mg/dL); 3) low HDL (<40 mg/dL in men, <50 mg/dL in women); 4) high blood pressure (≥130/85 mmHg); and 5) fasting hyperglycemia (≥100 mg/dL).
[b] Model 1: Adjusted for age (20–29, 30–39, 40–49, 50–59, 60–69, ≥70 years), sex, BMI (continuous).
[c] Model 2: Adjusted for education (elementary school or less, middle or high school, college or more), income (quartiles of equivalized household income), physical activity (low, moderate, high), smoking status (never smoker, past smoker, current smoker), and alcohol consumption (g/day) in addition to model 1.
[d] Model 3: Adjusted for total energy (kcal/day), carbohydrate (%energy), total fat (%energy), fiber (g/1000 kcal), vitamin C (mg/1000 kcal), and sodium (mg/1000 kcal) intakes as continuous variables in addition to model 2.

obesity, high blood pressure, and fasting hyperglycemia. Subjects who ingested more potassium had a lower odds ratio for abdominal obesity after adjusting for various factors. This trend was less prominent, though still significant, in model 3. The inverse association between high blood pressure and potassium intake was attenuated after adjusting for various lifestyle and dietary factors. Among those factors, dietary fiber affected the association the most. The inverse relationship between fasting hyperglycemia and potassium intake was significant after adjusting for all lifestyle and dietary factors.

The inverse trends in the odds ratios of metabolic syndrome remained significant (p for trend = 0.006) after additionally adjusting for the dietary Na:K ratio. The Na:K ratio did not significantly affect the odds ratios of metabolic syndrome ($p = 0.647$) and its components ($p > 0.1$) (data not shown).

Sex-specific analysis revealed that the inverse relationship between potassium intake and metabolic syndrome was consistent for both sexes (p for trend = 0.043 for men, and 0.008 for women, according to model 3). Moreover, the effect of the interaction of potassium intake with sex on the association with metabolic syndrome was not statistically significant (p for interaction > 0.1).

Finally, because of concerns about the validity of a single 24-h recall for assessment of individuals' habitual diets, we repeated above analyses after excluding participants who responded that they had meals more (or less) than usual during the last 24 h. All findings were maintained in the remaining participants ($n = 5765$) with small changes in detailed values (Supplementary Table 1).

4. Discussion

Our study suggests an inverse relationship between potassium intake and metabolic syndrome and its components in adults. This association was independent of other possible confounding factors and was consistent for both sexes.

Several studies have showed that blood pressure is inversely correlated with dietary potassium intake, as assessed using dietary surveys or 24-h urinary potassium excretion [9–11], although this observation has been considered controversial and remains open to dispute [26,27]. Our study demonstrated that higher levels of potassium intake were associated with lower blood pressure levels, but the inverse association was attenuated after adjusting for major lifestyle and dietary factors. Therefore, high potassium intake levels by themselves were insufficient to explain the lower blood pressure levels. Although the exact mechanism is unclear, it has been suggested that higher potassium intake levels attenuate salt sensitivity and the pressor effects of salt [28]. Thus, some studies have investigated the biological interaction of sodium and potassium in particular, and have reported that the Na:K ratio is more closely related to blood pressure than potassium itself [9,10]. In contrast, we found no significant association between the Na:K ratio and high blood pressure or metabolic syndrome.

Potassium also plays a critical role in insulin secretion from pancreatic beta cells [29] and in carbohydrate metabolism, particularly in the conversion of glucose to glycogen [3,30]. Therefore, hypokalemia may lead to impaired insulin secretion as well as

impaired glucose tolerance. Two recent studies have reported that a low level of serum potassium is an independent predictor of incident diabetes [31,32]. In contrast to the serum potassium level, however, the association between dietary potassium intake level and the risk of diabetes has been controversial [12,31]. According to our results after multivariable adjustment, individuals with higher dietary potassium intake levels were less likely to have fasting hyperglycemia. Since diabetic patients were excluded from our study, we could not directly evaluate the association between potassium intake and diabetes. Nevertheless, our results suggested possible beneficial contribution of dietary potassium intake on fasting hyperglycemia (or impaired fasting glucose), a pre-diabetic status which is closely related to the future development of diabetes [33]. Interestingly, this inverse relationship was not significant before adjusting for dietary factors (model 1 and model 2). Once we further adjusted for dietary factors in model 3, however, the inverse association became statistically significant. This can be explained by possible contribution of diets on fasting glucose level [34]. Among several dietary factors, total energy intake was the most significant negative confounding factor for the association between potassium intake and fasting hyperglycemia (data not shown). More studies are needed to confirm this result and to examine the underlying mechanism.

Until recently, no studies had described the association between potassium intake and abdominal obesity. Our results suggest that potassium intake is inversely related to abdominal obesity. Given the well-documented beneficial association between obesity and the intake of fruits and vegetables [35], which are the major food sources of potassium, future studies could consider potassium one of the possible dietary components affecting abdominal obesity.

The main sources of dietary potassium intake are fruits and vegetables which were shown to be beneficial to metabolic syndrome [14]. However, we could not consider total intakes of fruits/vegetables in the present study, since publicly available KNHANES IV does not provide such data. Future studies can focus on the association between metabolic syndrome and dietary fruits/vegetables as well as other elements abundant in fruits/vegetables.

4.1. Limitations and strengths

This study has several limitations. First, as with other nutritional epidemiology studies, a single 24-h dietary recall is not optimal for assessing individual' long-term, habitual diet, and may not be accurate in assessment of exact dietary intake due to fault of memory and errors in estimating portion size. However, adults are likely to have a stable nutrient intake over time [36,37], and potassium intake assessed by a single 24-h recall was significantly correlated with pooled long term intake [20] as well as 24-h urinary potassium excretion [21–23]. In addition, all findings were maintained even if we restricted the analyses to the participants who answered that they had meals as usual during the last 24 h, in which the results of 24-h recall could better represent the participants' usual diets (see Supplementary material). Further studies with multiple 24-h dietary recalls or 24-h urinary excretion are needed to confirm this association. Second, the cross-sectional nature of this study prevented us from determining an exact cause-and-effect relationship. Nevertheless, to overcome the issue of reverse causality in such a cross-sectional study design, we excluded subjects with chronic diseases who might have altered their eating behaviors after diagnosis. In spite of these limitations, this study is important since it is the first to examine the relationship between potassium intake and metabolic syndrome and its components in adults representative of the whole nation after adjusting for several confounders.

5. Conclusion

In conclusion, our study found a significant inverse association between potassium intake and metabolic syndrome in the general adult population. Further studies are required to confirm this association.

Author contributions

Dr. Shin had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. *Study concept and design:* Shin. *Acquisition of data:* Shin and Park. *Analysis and interpretation of data:* Shin, Joh, Kim, and Park. *Drafting of the manuscript:* Shin. *Critical revision of the manuscript for important intellectual content:* Shin, Joh, Kim, and Park. *Statistical analysis:* Shin and Kim. *Study supervision:* Park. All authors gave final approval of the version to be published.

Conflicts and interest of disclosures

None reported.

Disclosure

We have no conflicts to disclosure.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.atherosclerosis.2013.06.025.

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