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Sex difference in the relationship between 24-h sodium–potassium ratio and prevalence of metabolic syndromes: a cross-sectional study

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Maintaining a balanced ratio between sodium and potassium intake is one of the most important dietary and lifestyle factors in the development of metabolic syndrome (MetS), but available evidence is still limited, particularly when using urine samples to estimate this ratio. We aim to evaluate the associations between the 24-h urinary sodium–potassium ratio (24hUNa/KE) and MetS risk through a large health check-up program in China. This cross-sectional study analyzed health check-up data from 59,292 participants at the Third Xiangya Hospital's Department of Health Management in Changsha, China, from 2018 to 2021. Each participant gave one fasting urine sample to analyze sodium, potassium, and creatinine levels during the check-up. The Kawasaki formula estimated 24-h urinary sodium (24hUNaE) and potassium excretion (24hUKE), with the 24hUNa/KE ratio calculated by dividing 24hUNaE by 24hUKE. The prevalence of MetS was found to be 19.27%. Notably, the overall MetS prevalence was higher in men (28.08%) than in women (7.83%). In women, MetS prevalence increased from 6.35 to 10.30% across the lowest to highest 24hUNa/KE quartiles. A significant increase in MetS prevalence was associated with each standard deviation increase in 24hUNa/KE (adjusted odds ratio [AOR], 1.03; 95% confidence interval [CI] 1.01–1.06), particularly for central obesity (AOR, 1.04; 95% CI 1.02–1.06) and elevated blood pressure (AOR, 1.20; 95% CI 1.17–1.22). In women, a one standard deviation increase in the 24hUNa/KE ratio raised the risk of MetS by 9% (AOR, 1.09; 95% CI 1.05–1.14), but no significant link was found in men. A strong positive link exists between 24hUNa/KE and MetS and its components, especially central obesity and high blood pressure, with a more significant effect in women.

Keywords Metabolic syndromes (MetS), Estimated 24-h urinary sodium–potassium ratio (24hUNa/KE), Restricted cubic splines (RCS)

Metabolic syndrome (MetS) represents a substantial and formidable challenge to public health, profoundly impacting the overall health outcomes on a global scale. This complex and multifaceted pathological condition is characterized by a constellation of risk factors that are associated with cardiovascular diseases (CVD). These risk factors encompass central obesity, elevated blood pressure, glucose intolerance, insulin resistance, and dyslipidemia. Together, these factors create a dangerous combination that significantly increases the risk of developing serious health issues, including CVD, stroke, and diabetes[1–4]. With global aging and lifestyle changes, the prevalence of MetS is rising, especially in developing countries like China, where it surged from 13.7% to 31.1% among individuals aged 20 years and older between 2000 and 2017[5,6], even if there were minor variations in the definition of MetS, which was primarily stemmed from modifications made to the waist circumference cutoff values. The widespread

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prevalence of MetS underscores the urgent need for comprehensive public health strategies aimed at prevention, early detection, and effective management of this condition.

Despite the fact that numerous studies have indicated that individuals could manage and regulate their blood pressure levels by cutting down on their consumption of sodium[7–9]. A previous research has shown that both a diet with deficient sodium and excessively high sodium can be associated with a higher risk of mortality and cardiovascular-related events when compared to a diet that maintains a moderate level of sodium intake[10]. In a large-scale cohort study conducted within a Chinese population, it was observed that the group with the lowest risk of health complications had sodium excretion levels ranging from 3 to 5 g per day[11]. Interestingly, these levels of sodium excretion were found to be above all the current dietary recommendations set forth by the World Health Organization (WHO), which suggests a maximum intake of < 2 g of sodium per day, as well as the Chinese dietary guidelines, which recommend < 2.4 g per day[12–14]. A recent meta-analytic review[15] encompassing seventeen cross-sectional studies investigated the potential correlation between sodium intake and MetS. The findings from our previous study also suggested that individuals with MetS exhibited significantly higher levels of sodium status compared to those with a healthy metabolic profile[16].

The intricate relationship between sodium consumption and cardiovascular risk appears to be modulated by potassium intake, as studies have indicated that elevated potassium levels can temper the blood pressure response (one of the five components of MetS), and diminish the likelihood of stroke resulting from excessive sodium intake. Nevertheless, meta-analyses of limited studies suggest that increased potassium intake does not confer any advantages on blood lipid levels or cardiovascular diseases[17]. A further meta-analysis was conducted to explore the correlation between potassium intake and obesity/MetS, revealing a 20% reduction in MetS risk among individuals with high potassium consumption[18] . However, the assessment of potassium intake was primarily based on self-reported data, including 24-h dietary recall questionnaires, food frequency questionnaires (FFQs), and dietary records[19–24].

Field urine samples are acknowledged as a practical substitute for 24-h urine collection in extensive population-based surveys, providing a more precise indication of sodium and potassium intake than dietary questionnaires. As a result, urine testing was integrated into our health screening program in 2018 to explore the correlation between the MetS prevalence and its components with estimated urine sodium and potassium. In our previous study, we found that high estimated sodium excretion levels may be related to the total prevalence of Metabolic Syndrome (MetS), with a more pronounced association for central obesity and elevated blood pressure levels. We also observed sex differences in the prevalence of MetS[16]. Therefore, our aim is to evaluate the linear associations between the 24-h urinary sodium–potassium ratio estimation (24hUNa/KE) and MetS risk within the same population, and to investigate potential sex differences. Furthermore, recognizing the complexity of biological connections, we have taken an additional step by employing restricted cubic splines (RCS) to delve into possible nonlinear correlations.

Methods

Study subjects

During the period from 2018 to 2021, a total of 68,647 eligible random urine samples were collected from individuals attending routine health check-ups at the Health Management Center, The Third Xiangya Hospital in Changsha, China. The number of participants undergoing physical health assessments was 20,427 in 2018, 19,615 in 2019, 16,307 in 2020, and 12,298 in 2021. Following the verification of repeated measurements using unique Chinese residential ID card numbers, 61,039 participants completed at least one physical examination. Consequently, this cross-sectional study focused on these 61,039 participants to investigate the potential influence of the 24hUNa/KE on the prevalence of MetS. All participants signed informed consent forms, and study protocol and consent forms were approved by the Ethics Committee of the Third Xiangya Hospital (2020-S498).

Inclusion criteria

Participants meeting the specified criteria were included in the final analysis: (1) age at 18 years or older; (2) data relevant to the diagnosis of MetS were available, including age, sex, systolic blood pressure (SBP), diastolic blood pressure (DBP), waist circumference (WC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), fasting blood glucose (FBG), and history of hypertension and diabetes mellitus and medication use; (3) urinary sodium, urinary potassium, and creatinine excretions were provided; (4) reasonable blood pressure values were available: excluding SBP < 70 mmHg or SBP > 260 mmHg or DBP < 40 mmHg or DBP > 140 mmHg; (5) lipids had no abnormal values: excluding TG levels < 0.2 mmol/L or TG levels > 30 mmol/L or HDL-C levels < 0.1 mmol/L or HDL-C > 10.0 mmol/L. (6) had plausible 24hUNaE values: those with a 24hUNaE ≥ 12g/day were excluded.

Data collection

We collected information, including age, sex, smoking status, drinking status, and physical activities, via a structured questionnaire. Physical activity was defined as engaging in exercise at least three times per week for a minimum of 30 min per session. Each participant underwent a comprehensive health assessment by a qualified physician, which involved measurements of weight, height, WC, hip circumference (HC), SBP, and DBP. BP measurements were taken bilaterally with an OMRON automatic digital BP monitor (OMRON HBP-9021, OMRON Healthcare, Scarborough, Ontario, Canada), following the Chinese Guidelines for Blood Pressure Measurement. Fasting blood samples were analyzed for levels of FBG, total cholesterol, TG, LDL cholesterol, and HDL cholesterol using a LEADMAN monitoring kit (Beijing LEADMAN Biochemical Co, China). During the health check-up, participants provided a mid-stream urine sample in a labeled container, which was promptly sent to the Clinical Laboratory Department for analysis of urinary sodium, potassium, and creatinine within two hours. Urinary sodium and potassium excretions were measured using the ion-selective electrode method, while

urinary creatinine levels were determined via the dynamic enzyme method (Beijing LEADMAN Biochemical Co, China). Detailed protocols for the physical examination, blood sampling, and data collection have been previously published[16,25–28].

Urinary sodium and potassium estimations

Since 2018, sodium concentrations in fasting urine samples have been measured and converted into estimates of 24hUKE and 24hUNaE using Tanaka's, Kawasaki's, and INTERSALT Eqs.[29]. These methodologies have been scrutinized and validated within a Chinese cohort, which indicated that all approaches tended to underestimate the actual 24hUNaE and 24hUKE[30]. Notably, the Kawasaki formula exhibited the least deviation from the mean values of 24hUNaE and 24hUKE compared to the other two methods[31]. Consequently, we opted to utilize the Kawasaki method for the current study.

The definitions for MetS and its components

MetS was defined according to the criteria set by the Guidelines for the prevention and treatment of dyslipidemia in Chinese adults (revised in 2016)[32]. A MetS diagnosis required meeting at least three of the following five criteria: (1) WC ≥ 80 cm (females) or ≥ 90 cm (males); (2) TG > 1.7 mmol/L or on specific treatment for this lipid abnormality; (3) HDLC < 1.29 mmol/L (females) or < 1.03 mmol/L (males); (4) BP ≥ 130/85 mmHg or treatment for or diagnosis of hypertension; and (5) FBG > 5.6 mmol/L or diagnosis of type 2 diabetes mellitus (DM).

Statistical analyses

Statistical analyses were performed using the Statistical Analysis System (SAS V.9.4 for Windows; SAS Institute Inc., Cary, NC, USA). Continuous variables were expressed as mean ± standard deviation (Mean ± SD) and analyzed using the Kruskal–Wallis tests. Categorical variables were presented as percentages (%) and counts, analyzed using the binary logistic regression model was employed. The odds ratios (OR) and 95% confidence intervals (CI) were calculated for the prevalence of total MetS and its individual components. Firstly, we hypothesized that the associations between 24hUNa/K and total MetS risk and its compenents are linear, so the 24hUNa/K were categorized into sex-specific quartiles, with the lowest quartile acting as the reference category. Secondly, continuous values of 24hUNa/K were utilized as supplementary independent variables, divided by sex-specific standard deviations. Multivariate logistic regression models evaluated the relationships while controlling for confounding factors, including age (continuous variable), sex (male vs. female), current drinking status (current vs. never/ever drinkers), current smoking status (current vs. never/ever smokers), and physical activity (yes vs. no). The selection of covariates was based on the following two criteria: first, the corresponding regression coefficients of the variable changed by more than 10% when the variable was removed from the overall model, and second, the significance level of the regression coefficients of the variable needed to meet the statistical criterion of $p < 0.10$. Additionally, recognizing the complexity of biological relationships, we explored potential nonlinear associations of 24hUNa/K with total MetS and its components using restricted cubic splines (RCS), with three nodes positioned at the 10th, 50th, and 90th percentiles of 24hUNa/KE. The threshold level of the 24hUNa/K Ratio was determined using a recursive method, selecting the turning point within a predefined interval that maximized the model's likelihood. The fit of the piecewise logistic regression model was compared to a single-segment logistic regression model using a log-likelihood ratio test.

Results

General characteristic of enrolled participants

Between 2018 and 2021, a total of 61,071 participants provided urine samples during their medical examinations at the Health Management Center. Among these, 213 individuals were excluded due to being under 18 years of age. From the remaining 60,858 participants, additional exclusions were made: 1,347 lacked primary physical measurement data, 110 were missing lipid data, 62 had incomplete blood glucose data, 8 lacked blood pressure data, 30 were identified as outliers for 24hUNaE, and 9 had abnormal lipid and/or blood pressure readings. Ultimately, 59,292 participants were included in the analysis, as depicted in Fig. 1.

Finally, this study analyzed 33,506 men and 25,786 women, with mean ages of 44.97 and 42.74 years, respectively. The prevalence of MetS was notably higher in men than in women (28.08% versus 7.83%). Among men, the prevalence of MetS remained relatively constant across the quartiles of 24hUNa/KE, whereas in women, it exhibited a significant increase from 6.35% to 10.30%. The majority of the study population's characteristics were found to be statistically significant ($P < 0.05$) across all quartiles of 24hUNa/KE for both genders, as outlined in Table 1. Further results in younger than 60 years and older than 60 years were presented in Appendix S1. Total MetS prevalence was increased from the lowest quartiles to the highest quartiles of 24hUNa/KE only among those aged < 60 years ($P < 0.01$), while a slight U-shaped association were shown among those aged at 60 years or older ($P = 0.41$).

The association between 24hUNa/KE and MetS

Table 2 displays the odds ratios along with their 95% CIs for the quartiles of total MetS and its components in relation to 24hUNa/KE, both before and after adjustment for potential confounders. A favorable inclination was noted for overall MetS in connection with the 24hUNa/KE (highest quartile compared to lowest quartile: adjusted odds ratio [AOR] 1.10, 95% CI 1.02–1.18, P value for linear < 0.01). Furthermore, a 3% rise in MetS prevalence was uncovered per standard deviation augmentation of 24hUNa/KE (AOR, 1.03; 95% CI 1.01–1.06). Central obesity demonstrated a favorable trend (highest quartile versus lowest quartile: AOR, 1.12; 95% CI 1.06–1.19; $P_{linear} < 0.01$), similarly as elevated blood pressure (highest quartile versus lowest quartile: AOR, 1.82; 95% CI 1.72–1.92; $P_{linear} < 0.01$) exhibiting a favorable trend. On the contrary, decreased LDL-C showed an inverse relationship (highest quartile versus lowest quartile: AOR, 0.85; 95% CI 0.77–0.94; $P_{linear} < 0.01$). A non-

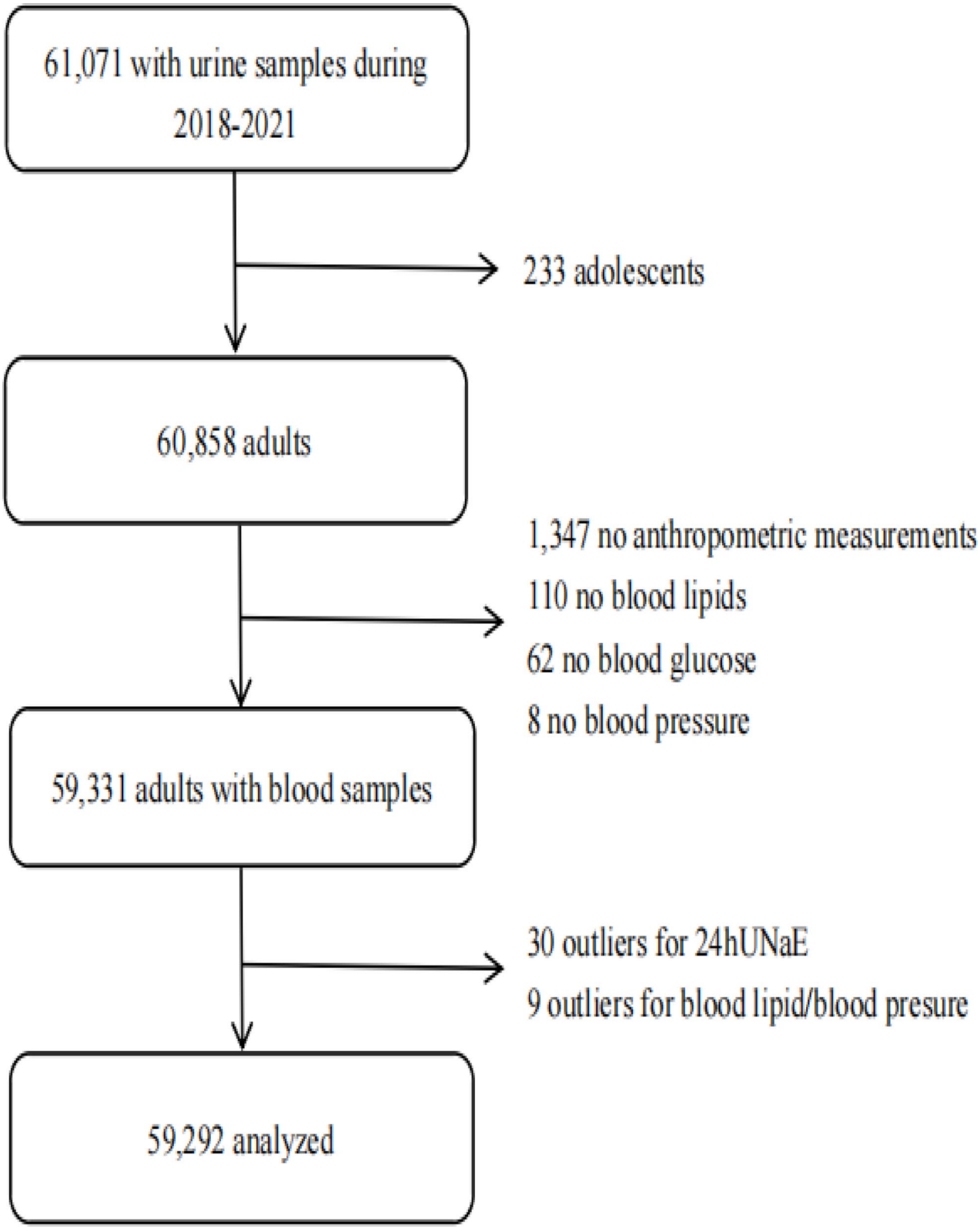

Fig. 1. The flow chart of participant selection.

linear U-shaped correlation was discerned for elevated FBG levels (highest quartile versus lowest quartile: AOR, 0.89; 95% CI 0.83–0.95; $P_{\text{linear}} < 0.01$), whereas no notable correlation was found for elevated TG (highest quartile versus lowest quartile: AOR, 0.97; 95% CI 0.92–1.02; $P_{linear} = 0.29$). Additionally, restricted cubic spline analyses were performed utilizing three knots and controlling for age, sex, current smoking status, current alcohol

Mean ± SD or % (n)	Total	Quarter of 24-h urine sodium-to-potassium ratio				
		Q1 (< 1.64)	Q2 (1.64–1.99)	Q3 (1.99–2.38)	Q4 (> 2.38)	P_{value} [1]
Males (sample size)	33,506	8,047	8,214	8,404	8,841	
Age (years)	44.97 ± 11.86	44.62 ± 11.83	44.73 ± 11.57	44.96 ± 11.74	45.53 ± 12.24	< 0.01
Height (cm)	168.38 ± 6.18	169.27 ± 6.11	168.62 ± 6.12	168.14 ± 6.11	167.58 ± 6.24	< 0.01
Weight (kg)	71.61 ± 10.74	72.00 ± 10.91	71.68 ± 10.52	71.58 ± 10.64	71.21 ± 10.87	< 0.01
BMI (kg/m2)	25.22 ± 3.24	25.09 ± 3.29	25.18 ± 3.20	25.28 ± 3.20	25.31 ± 3.25	< 0.01
WC (cm)	87.33 ± 8.94	87.22 ± 9.09	87.32 ± 8.84	87.36 ± 8.86	87.40 ± 8.99	0.63
HC (cm)	96.07 ± 5.85	96.08 ± 5.90	96.11 ± 5.72	96.12 ± 5.80	95.97 ± 5.96	0.32
WHR	0.91 ± 0.06	0.91 ± 0.06	0.91 ± 0.06	0.91 ± 0.06	0.91 ± 0.06	< 0.01
SBP (mmHg)	125.79 ± 14.74	122.86 ± 13.93	124.72 ± 14.20	126.56 ± 14.69	128.72 ± 15.36	< 0.01
DBP (mmHg)	78.28 ± 10.91	76.71 ± 10.58	77.81 ± 10.68	78.57 ± 10.90	79.87 ± 11.19	< 0.01
FBG (mmol/L)	5.76 ± 1.58	5.87 ± 1.89	5.76 ± 1.59	5.71 ± 1.45	5.70 ± 1.34	< 0.01
TC (mmol/L)	5.08 ± 0.99	5.10 ± 1.06	5.09 ± 0.97	5.08 ± 0.97	5.07 ± 0.96	0.14
TG (mmol/L)	1.70 (1.14–2.62)	1.75(1.19–2.70)	1.72 (1.17–2.63)	1.69 (1.13–2.63)	1.65 (1.10–2.54)	< 0.01
HDL-C (mmol/L)	1.21 ± 0.25	1.20 ± 0.25	1.21 ± 0.25	1.22 ± 0.25	1.23 ± 0.26	< 0.01
LDL-C (mmol/L)	2.85 ± 0.83	2.86 ± 0.85	2.86 ± 0.83	2.85 ± 0.83	2.84 ± 0.82	0.36
24hUCrE (g/day)	1.69 ± 0.27	1.70 ± 0.27	1.69 ± 0.26	1.68 ± 0.27	1.66 ± 0.28	< 0.01
24hUNaE (g/day)	4.31 ± 1.19	3.14 ± 0.78	4.05 ± 0.76	4.62 ± 0.85	5.33 ± 1.08	< 0.01
24hUKE (g/day)	2.14 ± 0.44	2.30 ± 0.45	2.22 ± 0.41	2.13 ± 0.38	1.91 ± 0.41	< 0.01
Salt intake (g/day)	8.20 ± 1.82	6.42 ± 1.32	7.84 ± 1.19	8.68 ± 1.30	9.70 ± 1.59	< 0.01
24hUNa/KE	2.08 ± 0.68	1.36 ± 0.22	1.82 ± 0.10	2.17 ± 0.11	2.87 ± 0.71	< 0.01
Current smoking	42.40 (13,011)	48.41 (3,531)	41.98 (3,158)	41.24 (3,190)	38.51 (3,132)	< 0.01
Current drinking	49.42 (15,166)	49.01 (3,575)	49.30 (3,708)	49.86 (3,857)	49.50 (4,026)	0.76
Physical activities	62.07 (19,046)	62.96 (4,593)	63.79 (4,798)	62.09 (4,803)	59.65 (4,852)	< 0.01
Central obesity	39.91 (13,373)	39.77 (3,200)	40.42 (3,320)	39.92 (3,355)	39.57 (3,498)	0.71
Low HDL-C	18.83 (6,308)	20.78 (1,672)	19.03 (1,563)	18.31 (1,539)	17.35 (1,534)	< 0.01
Elevated TG	50.24 (16,835)	52.23 (4,203)	51.19 (4,205)	49.71 (4,178)	48.06 (4,249)	0.45
Elevated BP	43.94 (14,721)	37.34 (3,005)	41.51 (3,410)	45.30 (3,807)	50.89 (4,499)	< 0.01
Elevated FBG	18.79 (6,296)	20.42 (1,643)	18.64 (1,531)	17.84 (1,499)	18.36 (1,623)	< 0.01
Total MetS	28.08 (9,408)	28.12 (2,263)	28.00 (2,300)	27.76 (2,333)	28.41 (2,512)	0.82
Females (sample size)	25,786	6,776	6,608	6,420	5,982	
Age (years)	44.68 ± 11.96	42.74 ± 11.97	44.56 ± 11.73	45.70 ± 11.75	45.89 ± 12.15	< 0.01
Height (cm)	157.03 ± 5.57	157.83 ± 5.48	157.19 ± 5.43	156.72 ± 5.53	156.29 ± 5.75	< 0.01
Weight (kg)	56.24 ± 7.83	55.82 ± 7.80	56.22 ± 7.63	56.33 ± 7.74	56.66 ± 8.14	< 0.01
BMI (kg/m2)	22.82 ± 3.03	22.41 ± 2.97	22.76 ± 2.91	22.94 ± 3.01	23.21 ± 3.19	< 0.01
WC (cm)	76.06 ± 8.54	74.84 ± 8.40	75.81 ± 8.36	76.52 ± 8.45	77.24 ± 8.79	< 0.01
HC (cm)	91.72 ± 5.49	91.45 ± 4.45	91.64 ± 5.37	91.79 ± 5.49	92.02 ± 5.65	< 0.01
WHR	0.83 ± 0.07	0.82 ± 0.06	0.83 ± 0.07	0.83 ± 0.07	0.84 ± 0.07	< 0.01
SBP (mmHg)	118.42 ± 16.82	114.67 ± 15.13	117.30 ± 15.76	119.89 ± 17.26	122.33 ± 18.18	< 0.01
DBP (mmHg)	71.16 ± 10.61	69.71 ± 10.00	70.47 ± 10.01	71.74 ± 10.88	72.94 ± 11.30	< 0.01
FBG (mmol/L)	5.40 ± 1.05	5.37 ± 1.12	5.40 ± 1.08	5.42 ± 1.02	5.42 ± 0.97	< 0.01
TC (mmol/L)	4.99 ± 0.97	4.93 ± 0.96	4.98 ± 0.96	5.04 ± 0.98	5.01 ± 0.98	< 0.01
TG (mmol/L)	1.07 (0.76, 1.56)	1.03 (0.56, 1.50)	1.07 (0.76, 1.54)	1.09 (0.77, 1.58)	1.09 (0.77, 1.63)	< 0.01
HDL-C (mmol/L)	1.47 ± 0.29	1.46 ± 0.29	1.47 ± 0.29	1.47 ± 0.29	1.47 ± 0.30	0.26
LDL-C (mmol/L)	2.89 ± 0.81	2.86 ± 0.81	2.89 ± 0.80	2.93 ± 0.82	2.89 ± 0.82	< 0.01
24hUCrE (g/day)	1.00 ± 0.10	1.01 ± 0.10	1.00 ± 0.10	0.99 ± 0.10	0.99 ± 0.10	< 0.01
24hUNaE (g/day)	3.81 ± 1.09	2.78 ± 0.70	3.64 ± 0.70	4.16 ± 0.78	4.82 ± 1.00	< 0.01
24hUKE (g/day)	1.92 ± 0.39	2.03 ± 0.39	2.00 ± 0.37	1.92 ± 0.35	1.72 ± 0.39	< 0.01
Salt intake (g/day)	8.00 ± 1.91	6.26 ± 1.40	7.75 ± 1.37	8.58 ± 1.37	9.61 ± 1.72	< 0.01
UNaE/UKE	2.04 ± 0.70	1.36 ± 0.22	1.82 ± 0.10	2.17 ± 0.11	2.90 ± 0.82	< 0.01
Current smoking	1.86 (437)	2.97 (165)	1.97 (117)	1.45 (85)	1.29 (70)	< 0.01
Current drinking	8.58 (2,016)	9.44 (584)	8.73 (526)	7.94 (464)	8.13 (442)	< 0.01
Physical activities	58.73 (13,797)	59.71 (3,694)	61.22 (3,687)	58.55 (3,422)	55.06 (2,994)	< 0.01
Central obesity	15.77 (4,067)	12.60 (854)	14.32 (946)	16.96 (1,089)	16.69 (1,178)	< 0.01
Low HDL-C	3.31 (854)	3.66 (248)	2.83 (187)	3.24 (208)	3.53 (211)	0.04
Elevated TG	20.69 (5,336)	18.99 (1,287)	19.70 (1,320)	21.56 (1,384)	22.79 (1,363)	< 0.01
Continued						

Mean ± SD or % (n)	Total	Quarter of 24-h urine sodium-to-potassium ratio				
		Q1 (< 1.64)	Q2 (1.64–1.99)	Q3 (1.99–2.38)	Q4 (> 2.38)	P_{value} [1]
Elevated BP	26.14 (6,741)	19.07 (1,292)	23.12 (1,528)	29.16 (1,872)	34.25 (2,049)	< 0.01
Elevated FBG	9.97 (2,570)	8.85 (600)	9.22 (609)	10.51 (675)	11.47 (686)	< 0.01
Total MetS	7.83 (2,020)	6.35 (430)	6.70 (443)	8.27 (531)	10.30 (616)	< 0.01

Table 1. General characteristics of participants with physical examinations during 2018–2021. SD, standard deviance; MetS, metabolic syndromes; BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist to hip ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood glucose; TC, total cholesterol; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; 24hUCrE, 24-h urinary creatinine excretion (estimated); 24hUNaE, 24-h urinary sodium excretion (estimated); 24hUKE, 24-h urinary potassium excretion (estimated); 24hUNa/KE, 24-h urine sodium-to-potassium ratio (estimated); [1] P value was obtained by two-sample non-parameter Wilcoxon tests for continuous variables and the binary logistic regression model for categorical variables.

consumption, and physical activity levels (as depicted in Fig. 2). However, a non-linear hypothesis was not established for the associations of 24hUNa/KE with total MetS prevalence and its five components. Meanwhile, we performed segmentation tests for 24hUNa/KE with lower HDL-C levels and elevated FBG levels and the results showed that they all had an effect in the low exposure segment and no effect in the higher exposure segment.

Subgroup analyses

Subgroup analyses in Table 2 indicated that increased blood pressure exhibited a robust positive link with 24hUNa/KE in both genders. In women, reduced HDL-C levels failed to demonstrate statistical significance, whereas in men, lower HDL-C levels were inversely associated (AOR, 0.86; 95% CI 0.79–0.93; $P_{trend} < 0.01$). In women, a notable positive link was observed for central obesity (AOR, 1.42; 95% CI 1.28–1.58; $P_{trend} < 0.01$), whereas in men, this association was insignificant. Among men, higher TG levels demonstrated a negative correlation (AOR, 0.88; 95% CI 0.83–0.94; $P_{trend} < 0.01$), and elevated FBG levels also showed an inverse relationship in men (AOR, 0.80; 95% CI 0.74–0.87; $P_{trend} < 0.01$). The nonlinear assumption is rejected in the RCS analyses, both for males (Fig. 3) and females (Fig. 4).

Age subgroup analyses revealed a significant positive link between elevated BP and central obesity in individuals under and over 60. In those under 60, central obesity was associated with increased BP (AOR, 1.19; 95% CI 1.12–1.26; $P_{trend} < 0.01$), but this was not observed in older adults. Low HDL-C negatively correlated with 24hUNa/KE in both groups (< 60 years: AOR, 0.89; 95% CI 0.82–0.96; $P_{trend} < 0.01$; > 60 years: AOR, 0.75; 95% CI 0.57–0.97; $P_{trend} = 0.04$). Elevated BP and 24hUNa/KE were positively correlated in both age groups (< 60 years: AOR, 1.87; 95% CI 1.77–1.98; $P_{trend} < 0.01$; > 60 years: AOR, 1.69; 95% CI 1.44–1.97; $P_{trend} < 0.01$). MetS was positively correlated in those under 60 (AOR, 1.17; 95% CI 1.09–1.25; $P_{trend} < 0.01$), but not in older adults, where it was negatively associated (AOR, 0.79; 95% CI 0.68–0.93; $P_{trend} < 0.01$). As show in Appendix S2.

In addition, We were concerned that diabetes and dyslipidemia associated with metabolic syndrome might have confounded the results. In the dyslipidaemic population, 24hUNa/KE was linked to higher blood pressure (AOR 1.73; 95% CI 1.59–1.88; $P_{trend} < 0.01$), lower FBG (AOR 0.87; 95% CI 0.79–0.96; $P_{trend} < 0.01$), and increased MetS (AOR 1.16; 95% CI 1.07–1.27; $P_{trend} < 0.01$). In the diabetic population, it was associated with elevated blood pressure (AOR 1.99; 95% CI 1.64–2.43; $P_{trend} < 0.01$) and MetS (AOR 1.28; 95% CI 1.04–1.56; $P_{trend} < 0.01$). Further analyses were performed and the results are presented in Appendix S3.

Discussions

This research examined the link between the 24hUNa/KE and MetS, particularly emphasizing gender disparities. These results indicated a notably higher prevalence of MetS in males (28.08%) in contrast to females (7.83%). It is worth noting that, as 24hUNa/KE levels rose, the prevalence of MetS stabilized in males but showed a marked increase in females, having a notably strong link to central obesity and elevated blood pressure. Upon accounting for possible confounding factors, the correlation between 24hUNa/KE and MetS risk stayed statistically significant.

Our research has furnished additional evidence supporting the connection between MetS and the interplay between sodium and potassium intake. It uncovered substantial positive linear correlations between 24hUNa/KE and MetS, both prior to and following adjustments for confounding variables, which are in line with recent studies[33–35]. Conversely, several studies indicated no notable association between the ratios of urinary sodium to potassium and MetS or its constituent components[36,37]. Furthermore, we employed RCS modeling to investigate potential nonlinear connections between 24hUNa/KE and MetS risk, but there is no evidence of a nonlinear relationships and linear associations were established except for Elevated TG. It demonstrated a favorable direct correlation with central obesity and elevated blood pressure, yet a negative correlation with decreased HDL-C and elevated blood glucose. Therefore, the positive correlation that exists between MetS and 24hUNa/KE is primarily attributable to the impacts of increased blood pressure levels and the presence of abdominal obesity. This finding is supported by previous researches, which has also identified these factors as significant contributors to the association between MetS and the 24hUNa/KE[35,38–40], including a Chinese study[35]. On the one hand, it may be due to the fact that excess sodium leads to an increase in intracellular calcium ion concentration in

MetS and its components	Prevalence %	Unadjusted odds ratios (95% confidence intervals) for 24-h urine sodium-to-potassium ratio						
		Q1(< 1.64)	Q2(1.64–1.99)	Q3(1.99–2.38)	Q4(> 2.38)	P for trend	Per SD increase	P_{value}
Overall		14,823	14,822	14,824	14,823		59,292	
Unadjusted models								
Central obesity	29.41	1.00	1.07 (1.02, 1.13)	1.14 (1.08, 1.20)	1.22 (1.16, 1.29)	< 0.01	1.07 (1.05, 1.09)	< 0.01
Elevated TG	37.39	1.00	1.01 (0.96, 1.05)	1.02 (0.97, 1.07)	1.04 (0.99, 1.09)	0.11	1.01 (0.99, 1.02)	0.45
Low HDL-C	12.08	1.00	0.90 (0.84, 0.96)	0.90 (0.84, 0.96)	0.90 (0.84, 0.96)	< 0.01	0.97 (0.95, 0.99)	< 0.01
Elevated BP	36.20	1.00	1.22 (1.17, 1.29)	1.52 (1.45, 1.60)	1.94 (1.85, 2.03)	< 0.01	1.22 (1.20, 1.24)	< 0.01
Elevated FBG	14.95	1.00	0.95 (0.89, 1.01)	0.96 (0.90, 1.03)	1.04 (0.97, 1.10)	0.23	1.01 (0.99, 1.03)	0.03
Total MetS	19.27	1.00	1.02 (0.96, 1.09)	1.08 (1.02, 1.14)	1.21 (1.14, 1.28)	< 0.01	1.06 (1.04, 1.08)	< 0.01
Adjusted models[1]								
Central obesity	29.41	1.00	1.06 (1.00, 1.12)	1.10 (1.04, 1.16)	1.12 (1.06, 1.19)	< 0.01	1.04 (1.02, 1.06)	< 0.01
Elevated TG	37.39	1.00	0.99 (0.94, 1.05)	1.00 (0.94, 1.05)	0.97 (0.92, 1.02)	0.30	0.99 (0.97, 1.01)	0.70
Low HDL-C	12.08	1.00	0.89 (0.81, 0.99)	0.85 (0.77, 0.94)	0.85 (0.77, 0.94)	< 0.01	0.97 (0.95, 1.00)	< 0.01
Elevated BP	36.20	1.00	1.18 (1.12, 1.25)	1.45 (1.37, 1.53)	1.82 (1.72, 1.92)	< 0.01	1.20 (1.17, 1.22)	< 0.01
Elevated FBG	14.95	1.00	0.90 (0.84, 0.96)	0.88 (0.82, 0.94)	0.89 (0.83, 0.95)	< 0.01	0.96 (0.93, 0.98)	< 0.01
Total MetS	19.27	1.00	1.02 (0.95, 1.09)	1.05 (0.98, 1.12)	1.10 (1.03, 1.18)	< 0.01	1.03 (1.01, 1.06)	0.02
Females		6,776	6,608	6,420	5,982		25,786	
Unadjusted models								
Central obesity	15.77	1.00	1.16 (1.05, 1.28)	1.42 (1.29, 1.56)	1.70 (1.55, 1.87)	< 0.01	1.15 (1.12, 1.18)	< 0.01
Elevated TG	20.69	1.00	1.05 (0.96, 1.14)	1.17 (1.08, 1.28)	1.26 (1.16, 1.37)	< 0.01	1.07 (1.04, 1.10)	< 0.01
Low HDL-C	3.31	1.00	0.77 (0.63, 0.93)	0.88 (0.73, 1.06)	0.96 (0.80, 1.16)	0.94	1.01 (0.94, 1.08)	0.04
Elevated BP	26.14	1.00	1.28 (1.18, 1.39)	1.75 (1.61, 1.90)	2.21 (2.04, 2.40)	< 0.01	1.24 (1.21, 1.27)	< 0.01
Elevated FBG	9.97	1.00	1.05 (0.93, 1.18)	1.21 (1.08, 1.36)	1.33 (1.19, 1.50)	< 0.01	1.09 (1.05, 1.13)	< 0.01
Total MetS	7.83	1.00	1.06 (0.92, 1.22)	1.33 (1.17, 1.52)	1.69 (1.49, 1.93)	< 0.01	1.15 (1.10, 1.19)	< 0.01
Adjusted models[2]								
Central obesity	15.77	1.00	1.06 (0.95, 1.18)	1.20 (1.08, 1.34)	1.42 (1.28, 1.58)	< 0.01	1.11 (1.07, 1.14)	< 0.01
Elevated TG	20.69	1.00	0.95 (0.87, 1.05)	1.02 (1.93, 1.12)	1.09 (0.99, 1.19)	0.06	1.02 (0.99, 1.05)	0.07
Low HDL-C	3.31	1.00	0.78 (0.64, 0.96)	0.89 (0.73, 1.09)	0.96 (0.78, 1.17)	0.88	1.01 (0.94, 1.08)	0.10
Elevated BP	26.14	1.00	1.13 (1.03, 1.25)	1.47 (1.34, 1.62)	1.96 (1.78, 2.16)	< 0.01	1.19 (1.15, 1.23)	< 0.01
Elevated FBG	9.97	1.00	0.94 (0.83, 1.07)	0.98 (0.86, 1.11)	1.08 (0.95, 1.23)	0.18	1.05 (1.01, 1.09)	0.18
Total MetS	7.83	1.00	0.95 (0.81, 1.10)	1.07 (0.93, 1.24)	1.39 (1.21, 1.61)	< 0.01	1.09 (1.05, 1.14)	< 0.01
Males		8,047	8,214	8,404	8,841		33,506	
Unadjusted models								
Central obesity	39.91	1.00	1.03 (0.97, 1.09)	1.01 (0.95, 1.07)	0.99 (0.93, 1.06)	0.63	1.01 (0.98, 1.03)	0.71
Elevated TG	50.24	1.00	0.96 (0.90, 1.02)	0.90 (0.85, 0.96)	0.85 (0.80, 0.90)	< 0.01	0.95 (0.93, 0.97)	< 0.01
Low HDL-C	18.83	1.00	0.90 (0.83, 0.97)	0.86 (0.79, 0.92)	0.80 (0.74, 0.87)	< 0.01	0.94 (0.91, 0.97)	< 0.01
Elevated BP	43.94	1.00	1.19 (1.12, 1.27)	1.39 (1.31, 1.48)	1.74 (1.64, 1.85)	< 0.01	1.20 (1.17, 1.22)	< 0.01
Elevated FBG	18.79	1.00	0.89 (0.83, 0.97)	0.85 (0.78, 0.92)	0.88 (0.81, 0.95)	< 0.01	0.95 (0.93, 0.98)	< 0.01
Total MetS	28.08	1.00	0.99 (0.93, 1.07)	0.98 (0.92, 1.05)	1.02 (0.95, 1.09)	0.76	1.01 (0.99, 1.04)	0.82
Adjusted models[2]								
Central obesity	39.91	1.00	1.04 (0.97, 1.11)	1.02 (0.96, 1.09)	1.00 (0.93, 1.06)	0.76	1.00 (0.98, 1.03)	0.52
Elevated TG	50.24	1.00	0.98 (0.92, 1.05)	0.93 (0.87, 0.99)	0.88 (0.83, 0.94)	< 0.01	0.96 (0.94, 0.98)	< 0.01
Low HDL-C	18.83	1.00	0.94 (0.86, 1.02)	0.91 (0.84, 0.99)	0.86 (0.79, 0.93)	< 0.01	0.97 (0.94, 1.00)	< 0.01
Elevated BP	43.94	1.00	1.18 (1.10, 1.27)	1.38 (1.29, 1.48)	1.69 (1.58, 1.81)	< 0.01	1.19 (1.16, 1.22)	< 0.01
Elevated FBG	18.79	1.00	0.87 (0.80, 0.95)	0.82 (0.76, 0.90)	0.80 (0.74, 0.87)	< 0.01	0.92 (0.90, 0.95)	< 0.01
Total MetS	28.08	1.00	1.02 (0.95, 1.10)	1.01 (0.94, 1.09)	1.02 (0.95, 1.09)	0.74	1.01 (0.98, 1.04)	0.96

Table 2. The associations of MetS and its components with 24-h urine sodium-to-potassium ratio. MetS, metabolic syndromes; SD, standard deviance; OR, odds ratio; CI confidence interval; Q, quartile; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; BP, blood pressure; FBG, fasting blood glucose; 24hUNa/K, 24-h urine sodium-to-potassium ratio (estimated); [1]Adjusted for age, sex, current drinkers, current smokers, physical activities; [2]Adjusted for age, current drinkers, current smokers, physical activities.

vascular smooth muscle cells, which promotes vasoconstriction and collagen deposition and increases arterial stiffness, leading to increased blood pressure[41]. It may also be due to the fact that sodium retention stimulates aldosterone secretion, which further promotes sodium reabsorption and potassium excretion, creating a vicious cycle and leading to increased blood pressure[42]. Studies in the Chinese population have shown that for every 1

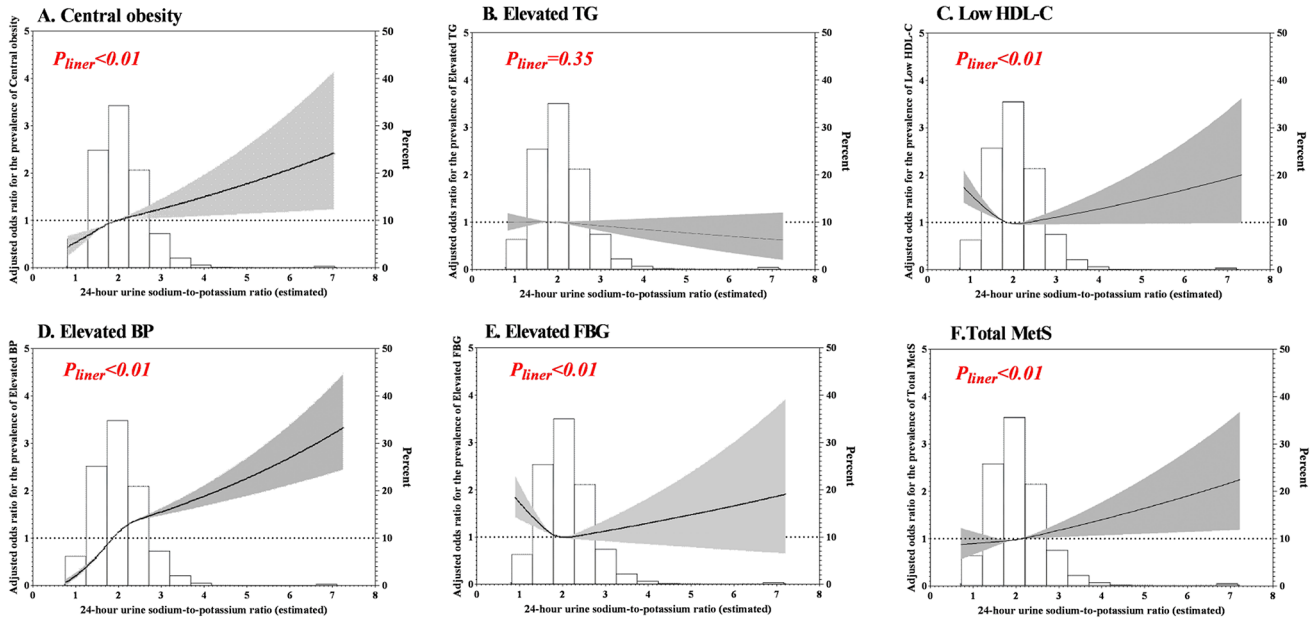

Fig. 2. Adjusted associations of 24-h urine sodium-to-potassium ratio (estimated) with total MetS and its components using RCS, (**A**) Central obesity; (**B**) Elevated TG; (**C**) Low HDL-C; (**D**) Elevated BP; (**E**) Elevated FBG; (**F**) Total MetS.

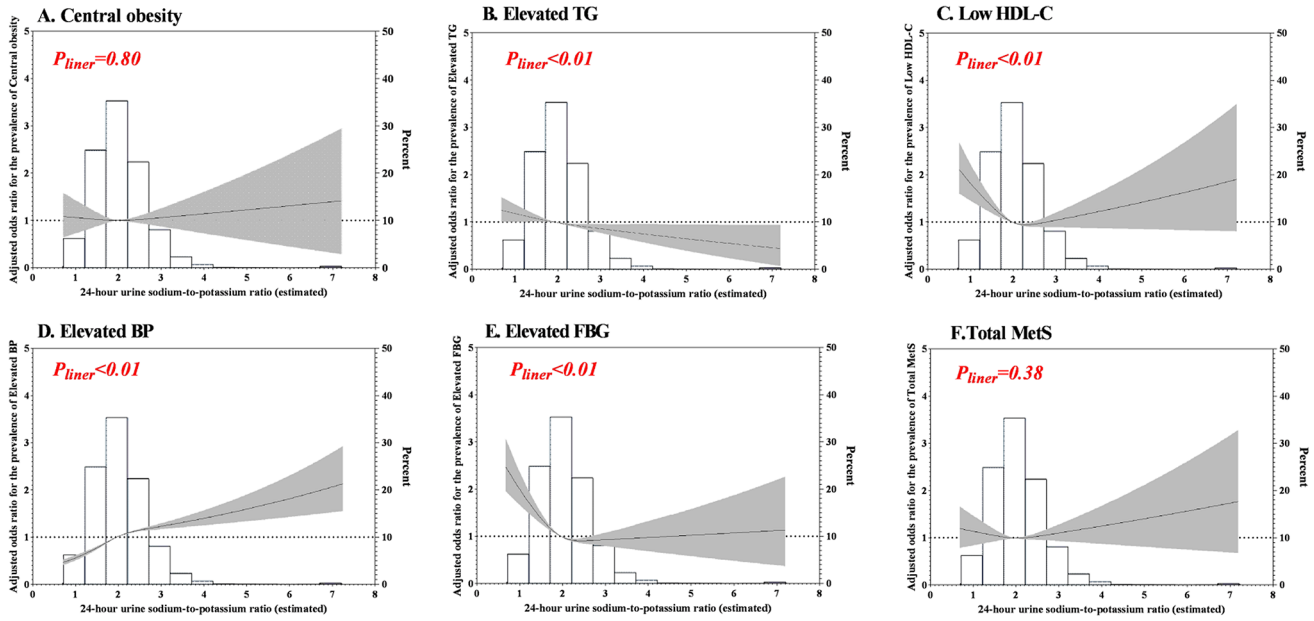

Fig. 3. Adjusted associations of 24-h urine sodium-to-potassium ratio (estimated) with total MetS and its components using RCS in males (**A**) Central obesity; (**B**) Elevated TG; (**C**) Low HDL-C; (**D**) Elevated BP; (**E**) Elevated FBG; (**F**) Total MetS.

unit increase in 24-h UNa/K, systolic and diastolic blood pressure increased by 1.21 mmHg and 0.44 mmHg, respectively[43]. On the other hand, sodium promotes preadipocyte differentiation and adipocyte hypertrophy by activating the salt-receptor (MR), thereby increasing visceral fat deposition[44]. A study in Shandong Province, China, found that the risk of central obesity was increased by 47% in the group with the highest urinary sodium, and that urinary sodium was positively correlated with waist circumference and waist-to-height ratio[45]. Even though the World Health Organization suggests that daily sodium consumption should be kept below 2 g and potassium intake should exceed 3.5 g, it is also crucial for maintaining good health to keep a balanced ratio between the intake of sodium and potassium. Furthermore, individual variability in sodium and potassium handling may also play a role in the observed associations. Genetic differences in renal sodium and potassium

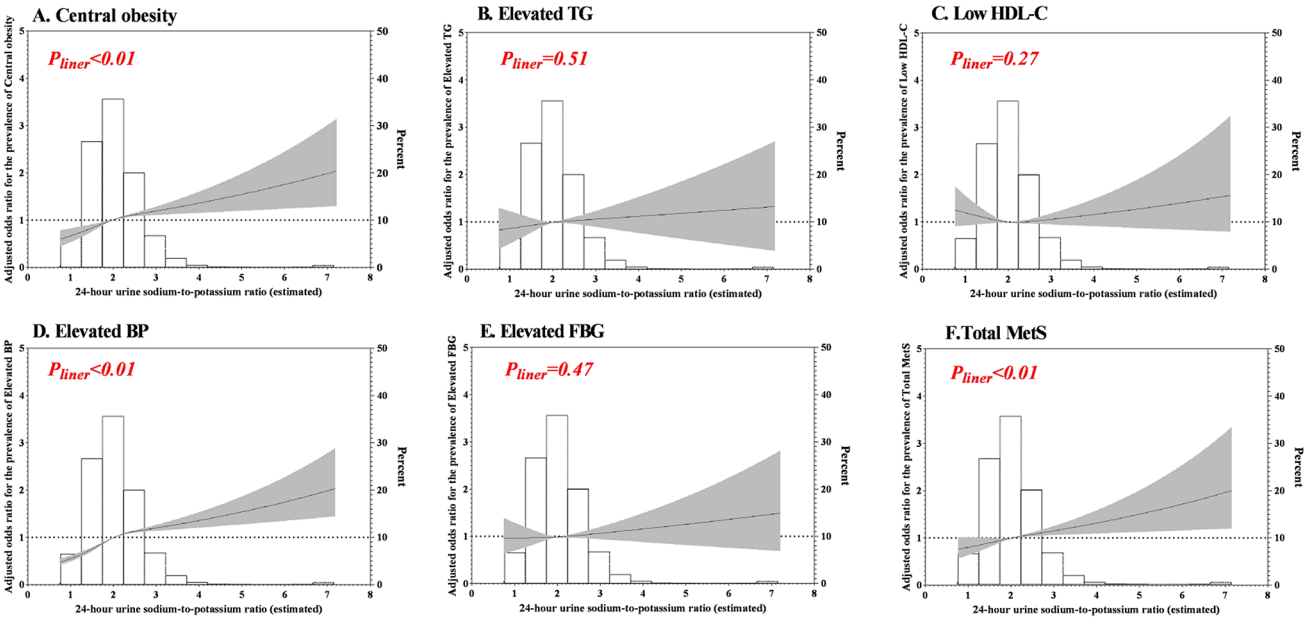

Fig. 4. Adjusted associations of 24-h urine sodium-to-potassium ratio (estimated) with total MetS and its components using RCS in females (**A**) Central obesity; (**B**) Elevated TG; (**C**) Low HDL-C; (**D**) Elevated BP; (**E**) Elevated FBG; (**F**) Total MetS.

transport mechanisms could influence the response to dietary sodium and potassium intake, and contribute to variations in blood pressure and obesity risk[49]. Therefore, personalized dietary advice based on individual genetic characteristics may be needed to optimize sodium and potassium intake and reduce the risk of chronic diseases.

Further analysis showed that higher 24hUNa/KE were more closely linked to increased MetS risk in women than in men, with no significant link found for men. In men, no significant association was seen between 24hUNa/KE and central obesity, but in women, higher 24hUNa/KE levels were significantly associated with increased risk of central obesity. These results emphasize significant gender disparities in the correlation between 24hUNa/KE and MetS. A study conducted on Korean women indicated that imbalances in sodium and potassium might lead to abdominal fat accumulation, caused by changes in lipid metabolism[46]. Another reason may be female-unique physiological attributes, including decreased cardiovascular protection stemming from menopause-induced lower estrogen levels, which could enhance susceptibility to heart conditions[47–50]. In men, 24hUNa/KE correlated positively with elevated blood pressure while showing a significant negative correlation with FBG levels. High levels of sodium consumption result in fluid retention, which in turn raises blood volume and blood pressure. Conversely, potassium possesses antihypertensive properties by facilitating the excretion of sodium. An imbalance between sodium and potassium can also disrupt insulin signaling, thereby contributing to insulin resistance. This is because potassium plays a crucial role in sustaining a healthy intracellular environment and in improving insulin sensitivity[51,52].

Furthermore, when conducting subgroup analyses, researchers uncovered a positive correlation between the risk of MetS and 24hUNa/KE in individuals who are under the age of 60, indicating that there is a corresponding 4% rise in the risk of developing MetS per every standard deviation increase in the 24hUNa/KE ratio. This finding underscores the importance of monitoring sodium and potassium levels in the urine as potential indicators of MetS risk in younger populations. However, no significant association was observed between the 24hUNa/KE and MetS risk in individuals over the age of 60. This discrepancy could be attributed to a variety of factors, including but not limited to the possibility that the sample size of older participants was smaller, which might have affected the statistical power of the analysis. It is also plausible that age-related physiological changes or differences in dietary habits could influence the relationship between urinary electrolyte excretion and MetS risk in older individuals[53–59]. Elevated levels of urinary sodium excretion may be indicative of excessive dietary salt intake, while reduced potassium excretion in the urine often reflects an insufficient intake of fruits and vegetables[20,60]. The interaction between the irreversibility of the physiological ageing process and the accelerated exposure to risk factors in modern society is the essential paradox of age as a risk factor for chronic disease. Baseline systolic blood pressure is elevated in the elderly population due to vascular sclerosis, reflecting the reduced compensatory reserve of their vasculature[61]. When the sodium-to-potassium ratio is increased, the reduced elasticity of the sclerotic vessels is unable to buffer the haemodynamic changes, leading to more pronounced systolic pressure fluctuations[62].

Our study has several limitations. First, the study exclusively targets individuals undergoing medical check-ups, potentially restricting the applicability of its findings to the broader population with varied dietary patterns. Second, On-site measurements were utilized to estimate urinary sodium and potassium levels (random urine samples), rather than relying on 24-h collections, which might affect precision because of daily fluctuations

in diet among the Chinese population. Spot urine only captures sodium and potassium concentrations at the time of collection compared to 24-h urine, whereas human metabolism is circadian and dietary. In addition, a short-term high-sodium/high-potassium diet significantly alters spot urine electrolyte concentrations, while factors such as water intake and sweating during exercise can dilute or concentrate urine. In populations with variable dietary patterns (e.g. cross-regional, multi-ethnic studies), the errors of the spot urine method are magnified. Third, we did not collect data on medications for hypercholesterolemia, which could have led to an underestimation of its prevalence and its correlation with urinary excretions, despite the fact that treated individuals generally constitute less than 5% of the population in China[63]. Fourth, most of the participants were city residents near Changsha, potentially differing in food habits from those in rural areas. The homogeneity of the sample introduces uncertainty when noticed relationships were the applied into diverse socioeconomic and cultural settings. Lastly, the cross-sectional nature of our study does not allow for the establishment of causal relationships between exposure and outcomes.

Conclusion

Despite these limitations, a noteworthy and positive association has been pinpointed between sodium and potassium ratio and the presence of MetS, along with its various components. This connection is especially pronounced when it comes to central obesity and elevate blood pressure. Interestingly, this correlation appears to be considerably more robust in women. The 24hUNa/KE ratio was significantly associated with the risk of MetS. This provides a promising avenue for intervention strategies aimed at mitigating the effects of MetS and associated health complications.

Data availability

Data described in the manuscript, code book, and analytic code will be made available upon request pending application and approval. Data are available from corresponding author Yuexiang Qin.

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Declarations

Ethics approval and consent to participate

Study protocol and consent forms were approved by the Ethics Committee of the Third Xiangya Hospital (2020-S498), with all procedures followed by the World Medical Association Declaration of Helsinki, and all essential permissions were obtained from the government and health commission. This study utilized a broad consent form, which was signed by each participant undergoing a physical examination prior to their examination. All personal information was anonymized during analysis and reporting to ensure confidentiality and privacy.

Additional information

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