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Prevalence and Determinants of Hyperuricemia in Middle-Aged, Urban Chinese Men

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Abstract

Background: Hyperuricemia is associated with metabolic syndrome and has emerged as a marker for both type 2 diabetes and cardiovascular disease. We estimated the prevalence and lifestyle risk factors of hyperuricemia in middle-aged, urban Chinese men.

Methods: The study included 3,978 urban Chinese men 40–74 years of age from a population-based cohort study, the Shanghai Men's Health Study, who were free of type 2 diabetes at baseline and had provided fasting blood samples. Uric acid concentrations were measured by the uricase method. Hyperuricemia was defined as >7.0 mg/dL. Anthropometric measurements and information on lifestyle factors and disease history were collected by in-person interviews.

Results: One quarter of the study subjects had hyperuricemia. Participants with metabolic syndrome had a higher prevalence of hyperuricemia. Body mass index (BMI), waist-to-hip ratio (WHR), waist circumference, and weight gain (since age 20) were positively associated with the prevalence of hyperuricemia. Physical activity was inversely related to the prevalence of hyperuricemia. The odds ratios for hyperuricemia for quintiles of nonoccupational physical activity were 1.00, 0.80, 0.73, 0.75, and 0.57 (P trend <0.001). Participants with hyperuricemia were less likely to be current smokers, but were more likely to drink alcohol regularly. Beer consumption was associated with higher risk of hyperuricemia compared with consumption of wine or liquor.

Conclusions: In this representative sample of middle-aged, urban Chinese men, hyperuricemia is highly prevalent. Obesity, weight gain in adulthood, and alcohol intake were associated with a higher prevalence of hyperuricemia, whereas daily physical activity and smoking were inversely related to the prevalence of hyperuricemia.

Introduction

Hyperuricemia has been associated with incident insulin resistance[1] and is highly prevalent among individuals with metabolic syndrome.[2–4] A positive association between plasma uric acid and the incidence of type 2 diabetes has been found in some[5–7] but not all studies.[8] Thus, identification of factors associated with the occurrence of hyperuricemia will help in the prevention of type 2 diabetes and cardiovascular disease.

Data on the associations between uric acid levels, physical activity,[9–13] and smoking[9,10,14–17] are limited and inconsistent. Although the association between alcohol and hyperuricemia are better established,[18–22] only four studies have investigated the effect of individual alcoholic beverages with high uric acid levels.[18,19,23,24]

In 2002, we launched a population-based cohort study of 61,504 men, 40–74 years of age in Shanghai, China, the Shanghai Men's Health Study (SMHS). In this report, we evaluate associations between lifestyle factors and the prevalence of hyperuricemia in a subset of SMHS participants who had provided fasting blood samples and were free of type 2 diabetes at baseline. We also present data on how the prevalence of the metabolic syndrome and its individual components differ by hyperuricemia status.

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Methods

The Shanghai Men's Health Study

The SMHS is a population-based cohort study of 61,504 Chinese men between 40 and 74 years of age, who were free of cancer at enrollment and living in urban Shanghai, China. Recruitment for the SMHS was initiated in April, 2002, and completed in June, 2006. A total of 83,058 eligible male residents of eight communities in urban Shanghai were invited to participate by trained interviewers through in-person contact, and 61,504 men who had no prior history of cancer were enrolled in the study (response rate, 74.0%). Reasons for nonparticipation were refusal (21.1%), out of area during enrollment (3.1%), and other miscellaneous reasons, including poor health or hearing problems (1.8%). The study protocols were approved by the Institutional Review Boards of all participating institutes, and all participants provided written, informed consent. Through in-person interviews, information was collected on demographic characteristics, disease history, and lifestyle factors, including dietary intake and physical activity. Participants were measured for weight and circumferences of the waist and hips according to a standard protocol. Participants were asked to provide biological samples including a blood or cheek cell sample and a spot urine sample. In a subcohort of 3,978 participants who had no history of diabetes at baseline and who had provide a fasting blood sample, we measured levels of disease-related biomarkers. This subcohort forms the basis of the current study.

Blood glucose, lipid, and uric acid measurements

At the time of the in-person interview, a 10-mL blood sample was drawn into an EDTA vacutainer tube. The samples were kept in a portable Styrofoam box with ice packs (0–4°C) and were processed within 6 h. All samples were stored at −70°C immediately after processing. One set of samples were shipped to the United States on dry ice, and these samples are currently stored at Vanderbilt University. Among participants who donated a blood sample at baseline (n = 46,169), 12.5% reported having had their last meal at least 8 h prior to the blood draw. For this study, we included the first 3,978 participants who were free of diabetes at baseline and who had provided a fasting blood sample. Blood glucose level and lipid profiles were measured by the Vanderbilt Clinical Nutrition Center using an ACE clinical chemistry system. The levels of uric acid were measured by using the ACE® Uric acid reagent on ACE® Clinical Chemistry System (Alfa Wassermann, Inc., West Caldwell, NJ) following the manufacturer's protocol. Uric acid in plasma was oxidized by uricase to allantoin and hydrogen peroxide. The hydrogen peroxide then oxidatively coupled dichlorohydroxybenzene sulfonic acid (DHBS) and 4-aminoantipyrine (AAP) in a reaction catalyzed by peroxidase, producing a red-colored quinoneimine complex which absorbs strongly at 505 nm.

Physical activity

Detailed information about nonoccupational physical activity was obtained using a validated physical activity questionnaire (PAQ).[25] The questionnaire evaluated physical activity during the 5 years preceding the interview, including participation in leisure-time activities such as regular exercise and sports; daily living activities such as walking, stair climbing, cycling, and household activities; and the daily commuting journey to/from work. Summary energy expenditure values (metabolic equivalent task [MET]-h/day) for these activities were estimated using a compendium of physical activity values.[26] We calculated total nonoccupational physical activity (total METs) by combining the three subtypes of physical activity—leisure time, daily living, and commuting.

Smoking status

Never smokers were defined as participants who had never smoked at least one cigarette per day for more than 6 months, whereas ex-smokers were defined as participants who had smoked at least one cigarette per day for more than 6 months but were not currently smoking. Current smokers were asked how many cigarettes they smoked per day. Participants were then classified according to their current smoking status into five categories: never smokers (n = 727; 18.28%), ex-smokers (n = 243; 6.11%), smoke less than 10 cigarettes per day (n = 857; 21.54%), smoke between 10 and 20 cigarettes per day (n = 1,714; 43.09%), and smoke more than 20 cigarettes per day (n = 437; 10.99%).

Alcohol intake

We asked each participant whether he had ever drunk alcoholic beverages at least once a week for 6 months or more. If the answer was yes, he was asked to provide the usual amount of consumption of rice wine, grape wine, beer, or liquor separately. Participants that had given up drinking were coded as ex-drinkers (n = 149) and were not included in the analysis. One drink was defined as 360 g of beer (12.6 g of ethanol), 103 g of grape wine (12.3 g of ethanol), 30 g of liquor (12.9 g of ethanol),[27] or 103 g of rice wine (12.3 g of ethanol). Participants were then classified into five categories according to their alcohol intake: nondrinkers (n = 2,332; 58.6%), occasional drinkers (less than 0.5 drinks per day; n = 65; 1.63%), light drinkers (0.5–0.99 drinks per day; n = 178; 4.64%), moderate drinkers (1.0–2.99 drinks per day; n = 923; 23.20%), and heavy drinkers (more than 3 drinks per day; n = 509; 12.8%). Because occasional drinkers were a small group, we combined them with light drinkers. We also analyzed individual types of beverages according to intake (none, <1 per day, 1–3 drinks per day, and >3 drinks per day). We combined grape wine and rice wine, because grape wine consumption is not common in this population.

Body size and weight history

Anthropometric measurements of weight, height, and waist and hip circumferences were taken twice, according to a standard protocol. If the difference between the first two measurements was larger than 1 cm for circumferences or 1 kg for weight, a third measurement was taken. The average of the two closest measurements was applied in the present study. From these measurements, the following variables were created: Body mass index (BMI), weight in kilograms divided by the square of height in meters, and waist-to-hip ratio (WHR), waist circumference divided by hip circumference. Standardized weight change was calculated as the

difference between measured weight at baseline and self-reported weight at age 20 divided by the interval between study recruitment and age 20 (kg/year). Data for weight at age 20 were available for 3,615 participants. We present data on weight gain in 5-year intervals.

Other confounding factors

Usual dietary intake was assessed through an in-person interview using a validated food frequency questionnaire.[28] The Chinese Food Composition Tables[29] were used to estimate intake of nutrients and total energy intake (kcals/day). Sociodemographic factors included in the analyses as potential confounders were age, level of education (none, elementary school, middle/high school, college), income in yuan/year (<500, 500–999, 1,000–1,999, >1,999), occupation (professional, clerical, manual), use of hypertension medication (yes/no), hypertension (blood pressure ≥85/130 mmHg and/or taking hypertension medication), and cardiovascular disease (CVD) at baseline (yes/no).

Hyperuricemia definition and metabolic syndrome criteria for this study

Hyperuricemia was defined as >7.0 mg/dL.[30–32] Participants were classified as having metabolic syndrome according to the Adult Treatment Panel III (ATP III) modified criteria, which use waist circumference cut points that are ethnicity-specific for Chinese men (≥90 cm instead of 102 cm) and a fasting glucose cut point of ≥5.6 mmol/L.[33] Other criteria for metabolic syndrome were serum triglyceride levels >1.70 mmol/L, high-density lipoprotein cholesterol (HDL-C) <1.04 mmol/L, and blood pressure ≥85/130 mmHg or taking medication for hypertension.

Table 1. Characteristics of the Study Population by Hyperuricemia Prevalence

	All participants	*No hyperuricemia*	*Hyperuricemia*	*P value*
Age (median)	48.5	48.0	49.0	0.19
Kcal/day	1928	1937.8	1898.4	0.03
BMI (mean)	23.3	22.9	24.5	<0.001
WHR (mean)	0.89	0.89	0.91	<0.001
Current smoker (%)	75.6	76.9	71.7	<0.01
Alcohol (%)	41.4	39.10	48.1	<0.001
Exercise (%)	21.8	21.7	21.99	0.85
Education (%)				
None	3.2	3.4	2.52	<0.01
Elementary	38.0	38.27	37.66	
Up to high School	40.7	41.45	38.17	
College	18.1	16.86	21.65	
Income level (%)				
<500	19.3	19.08	20.02	0.32
500–999	41.8	42.7	39.30	
1000–1999	29.4	29.0	30.48	
>1999	9.5	9.2	10.16	
Occupation (%)				0.03
Professional	20.3	19.57	22.33	
Clerical	23.1	22.56	24.55	
Manual workers	57.7	57.87	53.12	
Hypertension medication	13.5	10.26	23.09	<0.001
Pre-existing CVD	4.1	3.89	4.91	0.15
Metabolic syndrome prevalence	29.3	21.4	53.1	<0.01
Metabolic syndrome components				
High waist circumference	25.7	21.4	38.8	<0.001
Glucose intolerance	38.9	31.8	60.1	<0.001
High triglycerides	40.4	31.2	68.5	<0.001
Low HDL-C	22.3	21.4	24.9	<0.001
Hypertension	48.6	44.50	60.9	<0.001
Metabolic syndrome number of factors				
None	18.1	22.1	6.0	<0.01
1 factor	27.8	32.0	15.2	
2 factors	24.8	25.5	25.7	
3 factors	16.9	13.5	27.3	
4 factors	10.0	6.5	20.3	
5 factors	2.4	1.3	5.5	

Abbreviations: BMI, body mass index; WHR, waist-to-hip ratio; CVD, cardiovascular disease; HDL-C, high-density lipoprotein cholesterol.

Statistical analysis

Associations between hyperuricemia, BMI quintiles, WHR quintiles, waist circumference quintiles, standardized weight gain categories, smoking categories, physical activity categories, and alcohol intake categories were investigated by using unconditional logistic regression analysis. The analyses were adjusted for age, environmental factors, use of hypertension medicine, hypertension status, and pre-existing CVD. Tests for trend were performed by entering the categorical variables as continuous parameters in the models. All analyses were performed using SAS (version 9.1), and all tests of statistical significance were based on two-sided probability. All applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during this research.

Results

The prevalence of hyperuricemia in this male population was 25.0%. Participants with hyperuricemia had higher BMI, WHR, waist circumference, and daily energy intake and were more likely to drink alcohol and less likely to smoke than those without the condition (Table 1). Men with hyperuricemia were also more likely to have a college education and a professional job than men without hyperuricemia. The prevalence of hyperuricemia was positively associated with each individual component of metabolic syndrome, the number of metabolic syndrome components present, and the prevalence of metabolic syndrome.

As expected, the association of hyperuricemia with BMI, WHR, waist circumference, and weight gain since age 20 were similar. We also found a graded, positive association between hyperuricemia and each of these parameters (Table 2). The multivariate adjusted odds ratios (ORs) for hyperuricemia across quintiles of WHR were 1.00, 1.29, 2.00, 2.34, and 2.83 (P for trend <0.001), while ORs for waist circumference were 1.00, 1.23, 1.77, 2.47, and 3.16 (P for trend <0.001). After adjustment for BMI, the ORs were attenuated but remained significant. Compared with men with no weight gain, men who gained 2.8 kg or more per 5-year period had an OR of 3.73 [95% confidence interval (CI), 2.72–5.12]. Further adjustment for WHR attenuated the association.

Table 2. Associations of High Uric Acid Level (>7 mg/dL) and BMI, WHR, Waist Circumference Categories, and Weight Gain

	Cases	*OR1*[a]	*(95% CI)*	*OR2*[b]	*(95% CI)*
BMI					
Quintile 1	142	1.00		1.00	
Quintile 2	156	1.27	0.99–1.64	1.12	0.86–1.46
Quintile 3	188	1.64	1.27–2.10	1.35	1.03–1.76
Quintile 4	236	2.56	1.99–3.29	2.01	1.53–2.65
Quintile 5	274	3.23	2.50–4.17	2.35	1.75–3.16
		P trend <0.001		P trend <0.001	
WHR					
Quintile 1	124	1.00		1.00	
Quintile 2	131	1.29	0.98–1.70	1.11	0.84–1.47
Quintile 3	244	2.00	1.56–2.55	1.58	1.22–2.05
Quintile 4	217	2.34	1.82–3.03	1.77	1.34–2.33
Quintile 5	280	2.84	2.21–3.65	1.92	1.44–2.56
		P trend <0.001		P trend <0.001	
Waist circumference					
Quintile 1	144	1.00		1.00	
Quintile 2	138	1.23	0.95–1.60	1.11	0.85–1.46
Quintile 3	183	1.77	1.38–2.28	1.51	1.14–2.01
Quintile 4	238	2.46	1.93–3.15	1.97	1.44–2.68
Quintile 5	293	3.16	2.47–4.06	2.27	1.56–3.30
		P trend <0.001		P trend <0.001	
Standardized weight gain					
≤0 kg/5 year	72	1.00		1.00	
0–0.6 kg/5 year	58	1.38	0.93–2.05	1.26	0.85–1.88
0.6–1.7 kg/5 year	193	1.82	1.33–2.48	1.55	1.13–2.14
1.7–2.8 kg/5 year	222	2.14	1.56–2.93	1.67	1.19–2.34
>2.8 kg/5 year	371	3.73	2.72–5.12	2.71	1.90–3.85
	P trend <0.001			P trend <0.001	

[a]OR1: Adjusted for age, kcal/day, physical activity, alcohol consumption, smoking, education level, income level, occupation, hypertension medication, hypertension, and pre-existing CVD. For standardized weight gain analysis we included weight at 20 years in the model as well.

[b]OR2: As above plus WHR for BMI and standardised weight gain analysis and BMI for WHR and waist circumference analysis.

Abbreviations: BMI, body mass index; WHR, waist-to-hip ratio; OR, odds ratio; CI, confidence interval; CVD, cardiovascular disease.

Total non-occupational physical activity, which included leisure time, daily living, and commuting-related physical activity, was inversely related to the prevalence of hyperuricemia (Table 3). The ORs for ≤3, >3–6, >6–9, >9–12, and >12 non-occupational METs were 1.00, 0.80, 0.73, 0.75, and 0.57 ($P < 0.001$), respectively, in fully adjusted analyses (including BMI and WHR), showing a dose–response relationship. Daily living–related physical activity was associated with a lower risk of hyperuricemia, whereas no association between leisure-time physical activity and hyperuricemia was found. We repeated analyses of both leisure-time physical activity and daily living physical activity with mutual adjustment and found similar results (data not shown in tables).

Current smokers were less likely to have hyperuricemia (Table 4). The ORs for never smokers, ex-smokers, and smokers of >0–10, >10–20, >20 cigarettes per day were 1.00, 1.09, 0.77, 0.82, and 0.7, respectively, in analysis adjusted for age, kcal/day, BMI, WHR, physical activity, alcohol intake, income, occupation, education, established CVD, hypertension status, and antihypertensive medication use. Alcohol intake was associated with a higher risk of hyperuricemia (Table 4). All three types of alcoholic beverages were associated with higher prevalence of hyperuricemia. Consumption of beer was associated with the highest prevalence of hyperuricemia as compared with liquor or wine consumption (with grape wine and rice wine combined).

Discussion

The prevalence of hyperuricemia in this population was 25%. Obesity, weight gain since age 20 years, and alcohol consumption were each associated with higher prevalence of hyperuricemia, whereas current smoking and physical activity were associated with lower prevalence of hyperuricemia.

Table 3. Associations of High Uric Acid Level (>7 mg/dL) and Physical Activity

	Cases	*OR1*[a]	*(95% CI)*	*OR2*[b]	*(95% CI)*
Leisure-time physical activity METs					
None	777	1.00		1.00	
≤3	166	0.95	0.77–1.16	0.93	0.75–1.15
>3	53	0.89	0.63–1.25	0.93	0.66–1.32
		P trend 0.42		*P* trend 0.51	
Daily living physical activity METs					
≤3	269	1.00		1.00	
>3–6	346	0.73	0.60–0.89	0.77	0.62–0.93
>6–9	221	0.76	0.62–0.95	0.82	0.66–1.03
>9–12	96	0.62	0.47–0.82	0.68	0.51–0.90
>12	64	0.54	0.39–0.74	0.56	0.40–0.79
		P trend <0.001		*P* trend <0.001	
Total METs					
≤3	161	1.00		1.00	
>3–6	289	0.76	0.60–0.96	0.80	0.63–1.02
>6–9	246	0.66	0.52–0.85	0.73	0.57–0.94
>9–12	171	0.69	0.53–0.90	0.75	0.57–0.99
>12	129	0.50	0.38–0.67	0.57	0.42–0.76
		P trend <0.001		*P* trend <0.001	

[a]OR1: Adjusted for age, kcal/day, alcohol consumption, smoking, education level, income level, occupation, hypertension medication, hypertension, and pre-existing CVD.

[b]OR2: All of the above, plus BMI and WHR.

Abbreviations: OR, odds ratio; CI, confidence interval; METs, metabolic equivalent tasks; BMI, body mass index; WHR, waist-to-hip ratio.

The prevalence of hyperuricemia in our population was similar to the prevalence reported among Han Chinese in Taiwan (26.1%),[34] the age-standardized prevalence reported in adults in urban Qingdao, China (25.3%), and the prevalence reported in Japan (24.4%).[35] However, the prevalence in our population was lower than the prevalence reported in Taiwanese adults (30.6%).[36] Another study conducted in the coastal cities of eastern China reported a prevalence of hyperuricemia in men of 18.32%, which is lower than in our population.

In our study, hyperuricemia prevalence was associated with the presence of metabolic syndrome and its components, which is similar to reports from other studies, including a U.S. representative sample[37] and a Chinese population.[14] Hyperuricemia-induced oxidative stress has been proposed as a cause of metabolic syndrome.[38] Uric acid can act as a pro-oxidant, particularly at increased concentrations, and may be a marker of oxidative stress,[39,40] although it may also have a therapeutic role as an antioxidant.[41,42] It is unclear whether increased concentrations of uric acid in diseases associated with oxidative stress are a protective response or a primary cause.

Our findings on the associations between BMI and WHR and hyperuricemia are in agreement with findings from other cross-sectional studies.[10] In a Japanese population, the incidence of hyperuricemia was associated with BMI [relative risk (RR) = 1.13 for a 2 kg/m^2 increase; 95% CI, 1.02–1.26].[9] Weight loss has been associated with decreases in plasma uric acid.[43] Abdominal obesity is part of the metabolic syndrome,[44] and hyperuricemia has been proposed as a component of the metabolic syndrome. Insulin resistance, frequently found in overweight individuals, may explain part of this association.[45,46]

Some[9,11–13] but not all,[10,47] studies have reported inverse associations between uric acid and physical activity. Exercise may decrease uric acid excretion and accelerate purine degradation.[48] However, although acute exercise lasting between one-half and 3 h appears to elevate serum uric acid in proportion to the intensity of exercise, chronic exercise appears to lower serum uric acid levels.[49] In our study, participating in leisure-time physical activity (exercise and sports) was not associated with hyperuricemia, but we found a dose–response association between physical activity related to daily living and hyperuricemia. Most middle-aged and elderly Chinese men in Shanghai participate in a high level of nonexercise-related physical activity, but have relatively low participation in leisure-time physical activity.[50] The association between physical activity and uric acid could be mediated by the effects of physical activity on insulin sensitivity.[11]

We observed an inverse association between smoking and the prevalence of hyperuricemia. This result is similar to another study, which also found current smoking to be associated with lower incidence of hyperuricemia (RR = 0.65; 95% CI, 0.46–0.92),[9] and is supported by two additional studies.[16,17] However, two other studies found no association between smoking and uric acid.[10,47] It has been suggested that smoking may suppress uric acid levels via the action of the superoxides found in cigarette smoke.[16] Given that the effects

Table 4. Associations of High Uric Acid Level (>7 mg/dL) With Smoking Status and Alcohol Intake

	Cases	*OR1*[a]	*(95% CI)*	*OR2*[b]	*(95% CI)*
Smoking					
Never	203	1.00		1.00	
Ex-smoker	79	1.16	0.84–1.62	1.09	0.78–1.53
Current smoker	714	0.75	0.61–0.92	0.79	0.64–0.98
Smoking status					
Never smoker	203	1.00		1.00	
Ex-smoker	79	1.16	0.84–1.62	1.09	0.78–1.53
>0–10 cigarettes/day	186	0.72	0.56–0.92	0.77	0.60–0.99
>10–20	420	0.77	0.62–0.96	0.82	0.66–1.03
>20	108	0.71	0.52–0.95	0.71	0.53–0.97
Alcohol intake					
Never	516	1.00		1.00	
<1 drink/day	62	1.22	0.89–1.67	1.25	0.91–1.71
1 to <3 drink/day	199	1.36	1.11–1.66	1.35	1.10–1.65
≥3 drink/day	178	2.10	1.68–2.62	2.12	1.68–2.66
Wine[c]					
None	635	1.00		1.00	
<1 drink/day	118	1.08	0.86–1.37	1.10	0.87–1.40
1 to <3 drink/day	142	1.38	1.10–1.73	1.40	1.10–1.77
≥3 drink/day	60	1.89	1.33–2.67	1.85	1.30–2.63
Beer					
None	709	1.00		1.00	
<1 drink/day	163	1.14	0.93–1.40	1.16	0.94–1.43
1 to <3 drink/day	58	1.32	0.95–1.83	1.38	0.99–1.93
≥3 drink/day	25	3.08	1.76–5.36	3.50	1.99–6.17
Liquor					
None	832	1.00		1.00	
<1 drink/day	30	1.33	0.85–2.09	1.25	0.79–1.96
1 to <3 drink/day	45	1.31	0.91–1.89	1.27	0.88–1.84
≥3 drink/day	48	1.55	1.08–2.24	1.43	0.98–2.08

[a]OR1: Adjusted for age, kcal/day, physical activity, smoking, education level, income level, occupation, hypertension medicine, hypertension, and pre-existing CVD. In the case of individual types of alcoholic beverages, each type was adjusted for all other types.

[b]OR2: All of the above, plus BMI and WHR.

[c]Wine: grape wine and rice wine combined.

Abbreviations: OR, odds ratio; CI, confidence interval; CVD, cardiovascular disease; BMI, body mass index; WHR, waist-to-hip ratio.

of smoking on BMI and central obesity may mediate, in part, the effect of smoking on the risk of hyperuricemia, we investigated associations between smoking and hyperuricemia before and after adjustment for BMI and WHR. Adjustment for BMI and or WHR did not change the results.

Alcohol consumption was associated with a higher prevalence of hyperuricemia, similar to the findings of other studies.[18–21] In a Japanese prospective study, the RR for drinking ≥46.0 g/day of ethanol relative to nondrinking was 2.33 (95% CI, 1.55–3.50).[9] Alcohol consumption can elevate serum uric acid levels through increased uric acid synthesis.[51] In our study, consumption of all three individual types of alcoholic drinks (wine, beer, and liquor) was related to the prevalence of hyperuricemia, and beer consumption was associated with a higher prevalence of hyperuricemia as compared with wine and liquor. Beer may increase uric acid levels more than other alcoholic drinks due to its high purine content.[52] Data linking individual alcoholic drinks with hyperuricemia are limited and inconsistent. In one study from Japan, among sake, whisky, beer, and shochu, only beer was associated with elevated serum uric acid levels.[23] In another study conducted in Japan, the type of alcoholic drink was not associated with the risk of hyperuricemia.[19] In a study conducted in the United States (the National Health Examination Survey), both beer and liquor were associated with the risk of hyperuricemia,[18] similar to our results. However, in that study, wine was not associated with uric acid levels.[18] Another study from a Chinese population also reported wine to be associated with higher risk of hyperuricemia, although it is not clear whether the wine category in that study included only rice wine or grape wine, or was a combination of both. Wine in our study was a combination of grape wine and rice wine.

The strengths of this study are the population-based design (representative of the urban male population of Shanghai), the extensive information on confounders, the validated physical activity questionnaire that collected data on leisure-time physical activity and other types of physical activity, and the availability of information on consumption of different alcoholic beverages. Uric acid

level was measured in a centralized lab, which reduced interlab variation. In addition, this population has a high prevalence of smokers, which allowed us to investigate associations between smoking and hyperuricemia prevalence. The main limitation of this study is the cross-sectional design, which prevented us from making any causal inferences based on our results. Another limitation of the study is that we did not have information on other factors that might be related to the prevalence of hyperuricemia, such as kidney disease, skin psoriasis, or family history of gout or hyperuricemia.

In conclusion, our study found that hyperuricemia was prevalent in one quarter of this population of middle-aged Chinese men and was associated with the metabolic syndrome and its factors. Obesity—central obesity, in particular—weight gain, and an adverse lifestyle were associated with a higher prevalence of hyperuricemia in this population. Because hyperuricemia may be an early risk marker for chronic disease development, uric acid may have value as a risk marker for type 2 diabetes and CVD and may be useful in strategies for the prevention of these disorders. Furthermore, measurement of uric acid is inexpensive, easy in terms of preanalytics, and can be performed using routine laboratory methods.

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Author Disclosure Statement

No competing financial interests exist.

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