

# Nonlinear relationship between muscle quality index and hyperuricemia among non-elderly adults

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## Abstract

**Introduction:** The muscle quality index (MQI) is an increasingly recognized indicator of muscle status. The objective of this research was to analyze the association between MQI and hyperuricemia.

**Material and methods:** We performed a cross-sectional analysis using data from the National Health and Nutrition Examination Survey (NHANES) spanning the years 2011 to 2014. Participants in this study were adults aged 20–59 years. The muscle quality index (MQI) was calculated as the ratio of handgrip strength (HGS) to appendicular skeletal muscle mass (ASM). Hyperuricemia was defined by serum uric acid concentrations exceeding 420  $\mu\text{mol/l}$  (7 mg/dl) in males and 360  $\mu\text{mol/l}$  (6 mg/dl) in females. Assessment of the MQI-hyperuricemia relationship involved logistic regression, analysis of subgroups, and the application of smooth curve fitting.

**Results:** This research included 5,283 participants, of whom 716 had hyperuricemia. The prevalence of hyperuricemia decreased with higher quartiles of MQI ( $p < 0.001$ ). After correcting for confounders, MQI was closely associated with hyperuricemia (OR = 0.59, 95% CI: 0.46–0.77,  $p < 0.001$ ). Smooth curve fitting indicated a nonlinear association between the variables, with a threshold identified at 2.92. Subgroup analyses demonstrated that this association was particularly evident in participants with diabetes.

**Conclusions:** Our findings revealed an inverse correlation between MQI and hyperuricemia, suggesting that preserving adequate muscle quality may be advantageous in reducing the risk of hyperuricemia. Further prospective research is needed.

**Key words:** muscle quality index, hyperuricemia, cross-sectional research, nonlinear relationship, National Health and Nutrition Examination Survey.

## Introduction

Hyperuricemia, a prevalent metabolic disorder, is defined by plasma uric acid concentrations exceeding the normal range [1]. It not only serves as an early warning sign of gout, but also significantly increases the risk of developing hypertension, diabetes, chronic kidney disease, cardiovascular diseases, and mortality [2–5]. Despite the rising global incidence of hyperuricemia and its growing health burden, effective treatment options remain limited [6].

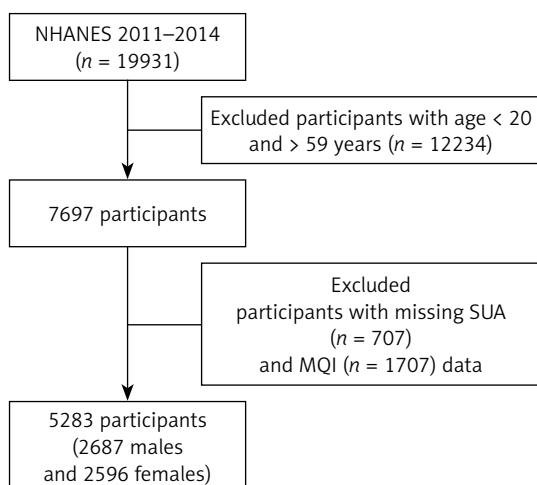
Handgrip strength (HGS), the force produced by the muscle groups of the hand, is a common metric used to assess an individual's overall muscle strength and functional status [7]. Stronger HGS is generally correlated with a lower risk of cardiovascular diseases, reduced mortality rates, and improved quality of life [8]. However, the association between HGS and hyperuricemia is currently controversial. Veronese *et al.* found that elderly patients with hyperuricemia typically exhibit reduced HGS [9]. Some studies have shown a positive correlation between uric acid levels and increased HGS, with higher uric acid levels significantly associated with greater muscle mass, HGS, and bone density [10]. Loss of muscle strength and loss of muscle mass do not parallel each other, and simply considering muscle strength, that is, HGS, may not reflect the true condition of the muscles. Thus, the muscle quality index (MQI) is proposed as a new functional index, which is defined as the ratio of HGS and appendicular skeletal muscle mass (ASM) [11]. MQI provides a more comprehensive assessment of muscle tissue quality by considering both the mass and strength. Current research indicates that MQI is significantly associated with pulmonary function [12], cardiovascular diseases [13], trouble sleeping [14], and periodontal disease [15].

Given the uninvestigated link between MQI and hyperuricemia in non-elderly adults, we conducted a cross-sectional analysis of National Health and Nutrition Examination Survey (NHANES) data to examine this association.

## Material and methods

### Study population

Administered by the National Center for Health Statistics (NCHS), a branch of the Centers for Disease Control and Prevention (CDC), NHANES



**Figure 1.** Flowchart depicting the selection of participants

represents one of the most extensive population-based studies in the United States [16]. NHANES adopted a multi-stage, stratified probability sampling methodology to ensure the selection of a nationally representative cohort. Enrolled individuals participated in comprehensive health evaluations, including physical examinations, structured interviews on dietary and health behaviors, and standardized laboratory analyses. The present investigation analyzed datasets derived from the 2011–2014 NHANES survey cycles. Exclusion criteria comprised subjects under 20 or over 59 years of age, and those lacking serum uric acid (SUA) and MQI data (Figure 1). Ultimately, 5,283 participants (2,687 males and 2,596 females) were incorporated into the final analysis of this study.

### Exposure and outcome definitions

The ratio of HGS to ASM is defined as the MQI [17]. A dynamometer is used to measure HGS. Grip strength quantification involved summation of peak values obtained from both hands, with results standardized in kilograms. Measurement of appendicular lean mass (ALM) was conducted with dual-energy X-ray absorptiometry (DXA), focusing on muscular components of upper and lower limbs (kg). DXA technology provided comprehensive body composition analysis, including bone density and soft tissue distribution across major anatomical regions (extremities, trunk, and cephalic segment). The aggregate lean tissue mass in limbs was designated as ASM. SUA values  $> 420 \mu\text{mol/l}$  (7 mg/dl) in men and  $> 360 \mu\text{mol/l}$  (6 mg/dl) in women are diagnostic thresholds for hyperuricemia [18, 19].

### Covariate definitions

To minimize potential confounding, we included the following variables as covariates in our analysis: age, sex, race, marital status, household's annual income, educational background, smoking, presence of diabetes, hypertension, and cardiovascular disease, body mass index (BMI), glycated hemoglobin ( $\text{HbA}_{1c}$ ), triglyceride levels (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), serum creatinine (SCr), and estimated glomerular filtration rate (eGFR) [20]. The eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula [21]. BMI was classified as normal weight ( $< 25 \text{ kg/m}^2$ ), overweight ( $25\text{--}29.9 \text{ kg/m}^2$ ), or obese ( $\geq 30 \text{ kg/m}^2$ ). Information on diabetes and hypertension was obtained via self-report. Cardiovascular disease was ascertained from participant self-reports of myocardial infarction, stroke, heart failure, coronary heart disease, or angina.

## Statistical analysis

Analytical procedures accounted for the complex survey design by incorporating CDC-specified sampling weights across two survey cycles. Descriptive statistics for quantitative measures were reported as weighted mean  $\pm$  standard error, while categorical measures were reported as weighted percentage (standard error). Between-group comparisons were conducted using appropriate weighted statistical tests (weighted Student's *t*-test and  $\chi^2$  test). Logistic and linear regression analyses were conducted to evaluate the relationships between MQI and both hyperuricemia and SUA levels. Model 1 was unadjusted and served as the reference. Model 2 adjusted for demographic variables such as age, sex, and race. Model 3 further incorporated socioeconomic and lifestyle factors, including marital status, annual household income, education level, and smokers. Model 4, extending Model 3, further adjusted for clinical and biochemical parameters: hypertension, diabetes, cardiovascular disease, BMI, TG, LDL-c, HDL-c, SCr, eGFR and HbA<sub>1c</sub>. Smoothed curve fitting and threshold effect analyses were applied to evaluate the association between MQI and hyperuricemia risk among non-elderly adults, and to determine potential inflection points in this relationship. Subgroup and interaction analyses were also conducted. All statistical procedures in this study were executed with R 4.1.1 and EmpowerStats software (<http://www.empowerstats.com>), with significance levels defined a priori at  $p < 0.05$ .

## Results

### Baseline characteristics of the study population

The cohort comprised 5,283 participants (mean age = 9.38 years; 51.27% male), stratified by hyperuricemia status into two comparative groups (Table I). Compared to the non-hyperuricemia group, the proportion of men, hypertension, diabetes, overweight/obesity, TC, TG, LDL-c, SCr, HGS, ASM, and SUA were significantly higher in the hyperuricemia group ( $p < 0.05$ ). Conversely, the hyperuricemia group had significantly lower HDL-c, eGFR, and MQI ( $p < 0.05$ ) (Table I). Significant differences in racial composition were also observed between the two groups ( $p < 0.05$ ).

### Clinical features of hyperuricemia participants according to MQI

Study participants were categorized into four equal groups by MQI quartile distribution (Table II). Comparative analysis demonstrated that individuals in the top MQI quartile were characterized by lower mean age and a higher proportion of male

participants ( $p < 0.01$ ). This group also showed higher levels of education and exhibited significantly lower rates of hypertension, diabetes, cardiovascular diseases, and obesity ( $p < 0.05$ ). Moreover, their HbA<sub>1c</sub>, TG, ASM, and SUA levels were lower, while their HDL-c, SCr, and HGS levels were higher ( $p < 0.05$ ). It is noteworthy that as the levels of MQI progressively increase, the prevalence of hyperuricemia gradually decreases (17.33% vs. 14.11% vs. 12.47% vs. 8.88%,  $p < 0.001$ ).

### Association between MQI and hyperuricemia

Table III shows a negative correlation between MQI and hyperuricemia with or without correction for confounding factors ( $p < 0.001$ ). Multivariable-adjusted logistic analysis revealed a significant inverse association between MQI and hyperuricemia risk, with each 1-unit increment corresponding to a 41% lower probability (OR = 0.59, 95% CI: 0.46–0.77,  $p < 0.001$ ). Quartile-based logistic analysis of MQI demonstrated an inverse dose-response relationship with hyperuricemia prevalence, with progressively lower rates observed in higher quartiles after full covariate adjustment ( $p$  for trend = 0.001). The linear regression model (Table IV), employing SUA concentrations as the outcome measure and MQI as the predictor, demonstrated a significant inverse relationship between these variables ( $\beta = -0.13$ , 95% CI -0.22–0.05,  $p = 0.003$ ). Moreover, results from the smooth curve fitting analysis and threshold effect analysis confirmed that MQI is inversely associated with the risk of hyperuricemia. This relationship is nonlinear, and the threshold point is 2.92 (Table V, Figure 2).

### Subgroup analysis

Subgroup analyses were systematically used to examine potential effect modification of the MQI-hyperuricemia relationship, with results visually presented in Figure 3. Factors including age, sex, hypertension, cardiovascular disease, and BMI did not significantly affect these relationships ( $p > 0.05$ ). However, this association was notably stronger in participants with diabetes (OR = 0.29, 95% CI: 0.14–0.64) compared to those without diabetes (OR = 0.64, 95% CI: 0.49–0.84) ( $p$  for interaction = 0.048).

## Discussion

The current study addresses a gap in the existing literature by examining the association between MQI and hyperuricemia risk. We identified a significant, nonlinear inverse association between MQI and the likelihood of hyperuricemia among non-elderly adults. Moreover, subgroup

**Table I.** Baseline characteristics of non-elderly population in NHANES from 2011 to 2014, weighted

Parameter	Overall (N = 5,283)	Non-hyperuricemia (N = 4,567)	Hyperuricemia (N = 716)	P-value
Age [years]	39.38 ±0.38	39.33 ±0.41	39.75 ±0.45	0.399
Male sex, % (SE)	51.27 (0.77)	47.16 (0.89)	78.56 (1.49)	< 0.001
Race, % (SE)				0.017
Mexican American	11.00 (1.42)	10.22 (1.46)	7.83 (1.42)	
Non-Hispanic Black	64.19 (1.30)	10.86 (1.28)	11.92 (1.73)	
Non-Hispanic White	6.45 (2.69)	63.83 (2.74)	66.58 (2.85)	
Other Hispanic	8.45 (0.98)	6.72 (1.03)	4.71 (0.99)	
Other races	11.00 (0.73)	8.37 (0.69)	8.96 (1.37)	
Annual household income (under \$20,000), % (SE)	13.25 (1.27)	13.34 (0.53)	12.71 (0.87)	0.653
Education level (above high school), % (SE)	65.45 (2.06)	65.61 (2.13)	64.37 (2.77)	0.623
Married, % (SE)	52.01 (1.55)	51.55 (1.65)	55.07 (2.39)	0.160
Smokers, % (SE)	41.79 (1.27)	41.39 (1.45)	44.43 (2.47)	0.304
Hypertension, % (SE)	22.82 (0.91)	20.47 (0.90)	38.43 (2.69)	< 0.001
Diabetes, % (SE)	5.22 (0.41)	5.22 (0.44)	7.18 (0.94)	0.040
Cardiovascular disease, % (SE)	3.20 (0.38)	3.13 (0.41)	3.68 (0.95)	0.570
BMI [kg/m <sup>2</sup> ] % (SE)				< 0.001
Normal weight	32.34 (1.10)	35.52 (1.25)	11.32 (1.27)	
Overweight	33.61 (0.91)	33.19 (0.95)	36.45 (2.30)	
Obesity	34.04 (1.00)	31.29 (1.21)	52.23 (2.02)	
HbA <sub>1c</sub> (%)	5.50 ±0.02	5.49 ±0.02	5.56 ±0.04	0.109
TC [mmol/l]	4.97 ±0.02	4.93 ±0.02	5.24 ±0.06	< 0.001
TG [mmol/l]	1.43 ±0.05	1.36 ±0.05	1.87 ±0.09	<0.001
HDL-c [mmol/l]	1.35 ±0.01	1.37 ±0.01	1.18 ±0.02	< 0.001
LDL-c [mmol/l]	2.97 ±0.02	2.93 ±0.03	3.18 ±0.06	< 0.001
SCr [μmol/l]	76.52 ±0.44	74.74 ±0.43	88.28 ±0.98	< 0.001
eGFR [ml/min/1.73 m <sup>2</sup> ]	101.12 ±0.46	102.17 ±0.47	94.19 ±1.00	< 0.001
HGS [kg]	77.23 ±0.40	75.66 ±0.41	87.64 ±0.78	< 0.001
ASM [kg]	23.03 ±0.14	22.39 ±0.14	27.29 ±0.29	< 0.001
SUA [mg/dl]	5.34 ±0.03	4.99 ±0.02	7.65 ±0.04	< 0.001
MQI	3.40 ±0.02	3.43 ±0.02	3.25 ±0.02	< 0.001

Values for categorical variables are given as weighted percentage (standard error); for continuous variables, as weighted mean ± standard error. Weighted Student's *t*-test and  $\chi^2$  test were used. BMI – body mass index, HbA<sub>1c</sub> – glycated hemoglobin, TC – total cholesterol, TG – triglyceride, HDL-c – high-density lipoprotein cholesterol, LDL-c – low-density lipoprotein cholesterol, SCr – serum creatinine, eGFR – estimated glomerular filtration rate, HGS – combined handgrip strength, ASM – arm and appendicular skeletal muscle, MQI – muscle quality index.

analyses revealed that the inverse association between MQI and hyperuricemia is especially evident in individuals with diabetes.

Prior research has paid considerable attention to the association between muscle status and uric acid levels. A population-based study in Korea highlighted a correlation between higher HGS and elevated SUA levels in elderly individuals [22]. In kidney transplant patients, Floriano *et al.* also found that SUA levels were positively related to muscle mass and strength [23]. Xie *et al.* further demonstrated that skeletal muscle mass was

positively associated with hyperuricemia in obese children and adolescents [24]. In our analysis of non-elderly adults, higher HGS and ASM were positively correlated with the prevalence of hyperuricemia. However, the MQI, represented by the HGS/ASM ratio, exhibited a negative correlation with hyperuricemia. This inconsistency may be attributed to the fact that muscle strength diminishes at a faster rate than muscle mass as individuals age [25]. Compared to traditional muscle status indicators, MQI not only considers muscle mass but also evaluates muscle strength, providing a more

**Table II.** Baseline characteristics of non-elderly population according to the quartiles of MQI, weighted

Parameter	Quartile 1	Quartile 2	Quartile 3	Quartile 4	P-value
Age [years]	41.15±0.64	40±0.56	38.9±0.54	37.66±0.44	0.001
Male sex, % (SE)	41.60 (2.36)	50.71 (1.52)	53.29 (1.81)	58.56 (1.67)	< 0.001
Race, % (SE)					< 0.001
Mexican American	9.83 (1.73)	9.9 (1.55)	9.04 (1.15)	10.93 (1.90)	
Non-Hispanic Black	18.87 (2.21)	10.87 (1.49)	8.21 (1.14)	6.9 (0.95)	
Non-Hispanic White	58.96 (3.22)	64.62 (3.25)	67.67 (2.62)	64.82 (3.23)	
Other Hispanic	6.04 (1.17)	7.73 (1.01)	6.13 (1.35)	5.88 (1.02)	
Other races	6.3 (1.07)	6.87 (0.74)	8.96 (0.75)	11.47 (1.22)	
Annual household income (under \$20,000), % (SE)	15.4 (1.66)	12.87 (1.43)	12.55 (1.33)	12.43 (1.95)	0.236
Education level (above high school), % (SE)	62.65 (2.71)	68.54 (2.05)	69.16 (2.20)	60.89 (2.71)	< 0.001
Married, % (SE)	48.15 (2.06)	53.51 (2.49)	54.12 (2.07)	51.78 (2.13)	0.098
Smokers, % (SE)	40.11 (2.20)	41.06 (2.17)	41.16 (1.87)	44.76 (2.18)	0.355
Hypertension, % (SE)	31.7 (1.75)	24.73 (1.50)	19.29 (1.34)	16.51 (1.42)	< 0.001
Diabetes, % (SE)	11.8 (0.96)	5.95 (0.91)	3.16 (0.57)	1.66 (0.33)	< 0.001
Cardiovascular disease, % (SE)	5.31 (0.79)	2.8 (0.60)	2.87 (0.73)	2.03 (0.38)	< 0.001
BMI [kg/m <sup>2</sup> ] % (SE)					< 0.001
Normal weight	9.69 (1.16)	21.98 (1.76)	37.89 (1.57)	57.78 (1.78)	
Overweight	22.14 (1.50)	36.60 (1.32)	40.62 (1.56)	33.64 (1.98)	
Obesity	68.18 (1.48)	41.41 (1.65)	21.49 (1.54)	8.57 (1.14)	
HbA <sub>1c</sub> (%)	5.83 ±0.04	5.52 ±0.02	5.37 ±0.02	5.33 ±0.02	< 0.001
TC [mmol/l]	4.95 ±0.04	4.99 ±0.04	4.97 ±0.04	4.96 ±0.04	0.869
TG [mmol/l]	1.56 ±0.11	1.44 ±0.09	1.44 ±0.09	1.47 ±0.07	0.017
HDL-c [mmol/l]	1.26 ±0.01	1.33 ±0.01	1.36 ±0.02	1.42 ±0.02	< 0.001
LDL-c [mmol/l]	3.01 ±0.04	2.96 ±0.05	3.00 ±0.04	2.90 ±0.04	0.169
SCr [μmol/l]	76.56 ±1.37	75.42 ±0.47	76.88 ±0.60	77.20 ±0.62	0.029
eGFR [ml/min/1.73 m <sup>2</sup> ]	100.49 ±0.68	101.21 ±0.64	100.72 ±0.78	102.05 ±0.69	0.410
HGS [kg]	65.93 ±0.79	75.55 ±0.69	80.00 ±0.85	86.33 ±0.79	< 0.001
ASM [kg]	25.57 ±0.33	23.78 ±0.21	22.26 ±0.23	20.76 ±0.20	< 0.001
SUA [mg/dl]	5.51 ±0.06	5.42 ±0.04	5.29 ±0.05	5.16 ±0.05	< 0.001
Hyperuricemia, % (SE)	17.33 (1.75)	14.11 (1.50)	12.47 (1.34)	8.88 (1.42)	< 0.001

**Table III.** Logistic regression analysis results of MQI and hyperuricemia

Hyperuricemia	OR (95% CI), p-value			
	Model 1	Model 2	Model 3	Model 4
Continuous				
MQI	0.60 (0.53, 0.68) < 0.001	0.48 (0.42, 0.55) < 0.001	0.46 (0.40, 0.53) < 0.001	0.59 (0.46, 0.77) < 0.001
Categories				
Quartile 1	Ref.	Ref.	Ref.	Ref.
Quartile 2	0.70 (0.57, 0.86) < 0.001	0.60 (0.48, 0.75) < 0.001	0.58 (0.47, 0.73) < 0.001	0.72 (0.50, 1.03) 0.072
Quartile 3	0.59 (0.48, 0.74) < 0.001	0.46 (0.37, 0.58) < 0.001	0.44 (0.35, 0.56) < 0.001	0.56 (0.38, 0.83) 0.004
Quartile 4	0.44 (0.35, 0.55) < 0.001	0.32 (0.25, 0.40) < 0.001	0.30 (0.23, 0.38) < 0.001	0.51 (0.32, 0.80) 0.003
P for trend	< 0.001	< 0.001	< 0.001	0.001

OR – odds ratio, 95% CI – 95% confidence interval. Model 1: non-adjusted. Model 2: adjusted for age, sex, and race. Model 3 adjusted for age, sex, race, marital status, annual household income, education level, and smokers. Model 4: adjusted for age, sex, race, marital status, annual household income, education level, smokers, hypertension, diabetes, cardiovascular disease, BMI, TG, LDL-c, HDL-c, SCr, eGFR, and HbA<sub>1c</sub>.

**Table IV.** Linear regression analysis results of MQI and SUA levels

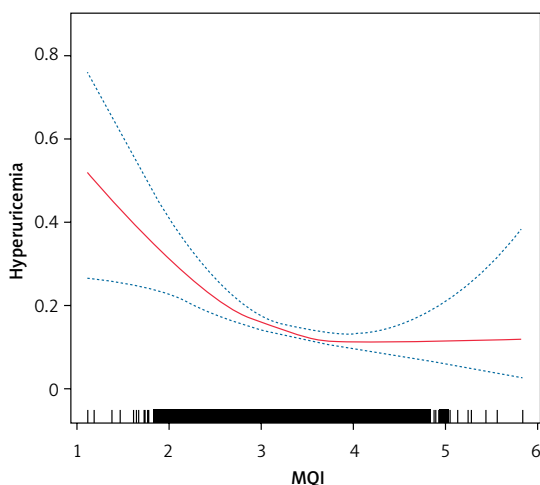
SUA [mg/dl]	$\beta$ (95%CI), <i>p</i> -value			
	Model 1	Model 2	Model 3	Model 4
Continuous				
MQI	-0.25 (-0.30, -0.19) < 0.001	-0.41 (-0.46, -0.36) < 0.001	-0.43 (-0.49, -0.38) < 0.001	-0.13 (-0.22, -0.05) 0.003
Categories				
Quartile 1	Ref.	Ref.	Ref.	Ref.
Quartile 2	-0.14 (-0.24, -0.04) 0.008	-0.26 (-0.35, -0.17) < 0.001	-0.27 (-0.36, -0.18) < 0.001	-0.05 (-0.17, 0.08) 0.455
Quartile 3	-0.27 (-0.37, -0.17) < 0.001	-0.46 (-0.55, -0.37) < 0.001	-0.48 (-0.58, -0.39) < 0.001	-0.17 (-0.31, -0.04) 0.012
Quartile 4	-0.40 (-0.50, -0.29) < 0.001	-0.65 (-0.75, -0.56) < 0.001	-0.68 (-0.78, -0.59) < 0.001	-0.15 (-0.29, -0.00) 0.050
<i>P</i> for trend	< 0.001	< 0.001	< 0.001	0.017

95% CI – 95% confidence interval. Model 1: non-adjusted. Model 2: adjusted for age, sex, and race. Model 3 adjusted for age, sex, race, marital status, annual household income, education level, and smokers. Model 4: adjusted for age, sex, race, marital status, annual household income, education level, smokers, hypertension, diabetes, cardiovascular disease, BMI, TG, LDL-c, HDL-c, SCr, eGFR, and HbA<sub>1c</sub>.

**Table V.** Threshold effect analysis of MQI on hyperuricemia using a two-piecewise linear regression model

Model	OR (95% CI), <i>p</i> -value
Fitting by standard linear model	0.59 (0.46, 0.77), < 0.001
Fitting by two-piecewise linear model	
Breakpoint (K)	2.92
OR1 (< 2.92)	0.28 (0.16, 0.51) < 0.001
OR2 (> 2.92)	0.83 (0.58, 1.18) 0.292
OR2/OR1	2.92 (1.35, 6.31) 0.006
<i>P</i> for logarithmic likelihood ratio	0.007

Adjusted for age, sex, race, marital status, annual household income, education level, smokers, hypertension, diabetes, cardiovascular disease, BMI, TG, LDL-c, HDL-c, SCr, eGFR, and HbA<sub>1c</sub>.

**Figure 2.** Smooth curve fitting results of MQI and hyperuricemia

comprehensive reflection of overall muscle condition [26]. This approach enables earlier prediction of changes in muscle quality and function compared to relying solely on grip strength tests [26]. In this research, we also accounted for the potential confounding effects of body weight. Because both muscle quality and SUA are strongly influenced by body weight and adiposity, we adjusted for BMI in all multivariable models and conducted stratified analyses across different BMI categories. Notably, the inverse association between MQI and hyperuricemia remained robust and statistically significant after these adjustments. These findings indicate that the relationship between muscle quality and hyperuricemia is not solely attributable to the effects of body weight.

Several potential biological mechanisms help to explain these two associations. MQI might influence hyperuricemia risk through its effects on metabolic regulation, including insulin sensitivity and systemic inflammation. Skeletal muscle is the main site of glucose uptake through glucose transporter type 4 (GLUT4), responsible for nearly 80% of total glucose clearance [27]. A decrease in muscle mass leads to altered glucose handling and increased local inflammation, promoting insulin resistance [28]. Insulin resistance is a major factor in the development of hyperuricemia, as it influences uric acid handling in the renal tubules by simultaneously enhancing uric acid reabsorption and excretion [29, 30]. The enhancement of muscle strength may also indirectly reduce uric acid levels by improving insulin resistance. Our study additionally identified a more pronounced link between MQI and hyperuricemia in patients with diabetes, further highlighting the critical role of insulin resistance. Moreover, research has demonstrated that greater



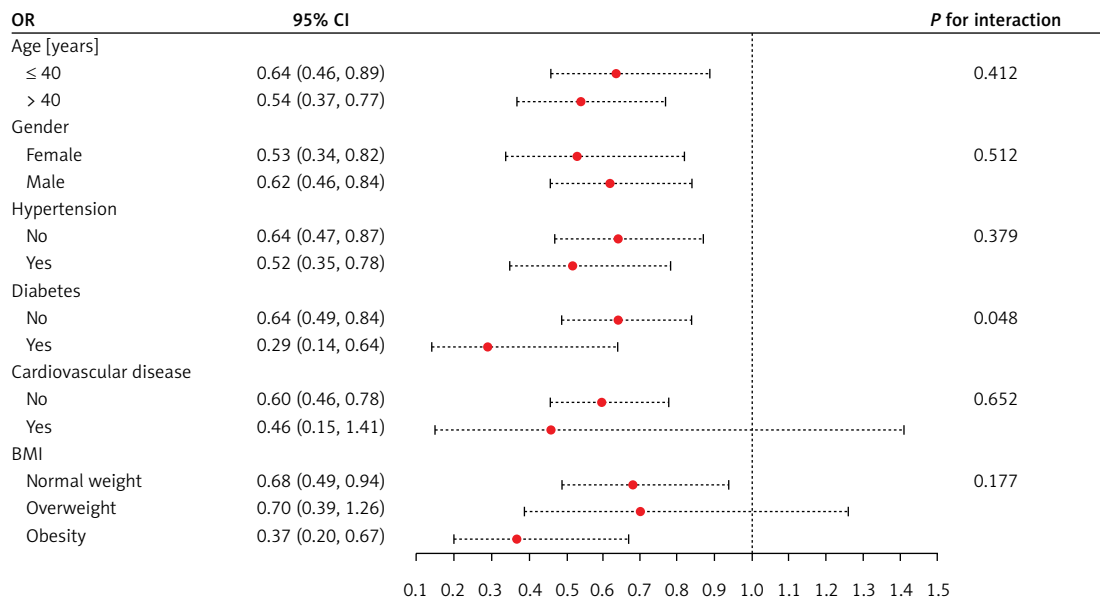


Figure 3. Subgroup analysis

muscle strength is frequently linked to an elevated basal metabolic rate (BMR) [31]. An increase in BMR is generally associated with improvements in metabolic health, such as enhanced insulin sensitivity and reduced adipose tissue [32–34]. These factors help to reduce the production and accumulation of uric acid. Low levels of skeletal muscle strength and mass are significantly correlated with higher levels of circulating inflammatory markers [35, 36]. Chronic inflammation accelerates hyperuricemia by inducing oxidative stress, which damages cell membranes and DNA [37]. This triggers the release and metabolism of intracellular purine nucleotides, leading to excess uric acid production [37]. While it is possible that better muscle quality may reduce the risk of hyperuricemia, the alternative hypothesis suggests that hyperuricemia itself can negatively affect muscle quality through mechanisms involving oxidative stress, as well as lipid and carbohydrate metabolic dysfunction [38–41]. Prior research has also demonstrated that elevated SUA can induce mitochondrial dysfunction, inflammatory responses, and muscle protein degradation, resulting in impaired muscle structure and function [41–43].

Although this study is the first to investigate the MQI-hyperuricemia association, several methodological limitations warrant consideration. First, the study only included participants aged 20–59, which may restrict the applicability of the results to different age populations, such as adolescents and the elderly, potentially overlooking variations in muscle quality and hyperuricemia risk across life stages. Secondly, as discussed above, the cross-sectional nature of this study allows us to identify only an inverse association between MQI and the risk of hyperuricemia, without the ability

to determine causality or the temporal sequence of events. Prospective longitudinal studies with larger and more diverse populations are warranted to further elucidate this relationship. Finally, despite adjusting for numerous confounders, unmeasured factors may still influence the results, warranting further investigation.

In conclusion, our results reveal an inverse association between MQI and hyperuricemia, and maintaining an appropriate MQI level is beneficial for hyperuricemia. However, further prospective research is needed to substantiate this finding.

#### Availability of data and material

The dataset was obtained from the publicly available National Health and Nutrition Examination Survey (NHANES) repository (<https://wwwn.cdc.gov/nchs/nhanes>).

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#### Ethical approval

The study protocol adhered to international ethical guidelines (Declaration of Helsinki) and received formal approval from the National Center for Health Statistics (<https://www.cdc.gov/nchs/nhanes/about/erb.html>) Research Ethics Review Board. Prior to data collection, all subjects vol-

untarily provided documented informed consent following comprehensive explanation of the study procedures.

## Conflict of interest

The authors declare no conflict of interest.

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