

Joint and Interactive Associations of the Visceral Adiposity Index and Systemic Inflammatory Markers with Serum Uric Acid Levels among Gas Station Workers

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Abstract

Objective: To investigate the independent, joint, and interactive associations of the visceral adiposity index (VAI) and systemic inflammatory markers with serum uric acid (SUA) levels among gas station workers in Guangxi, China, and to explore the nonlinear dose-response relationship between VAI and SUA.

Methods: This cross-sectional study included 902 gas station workers from a petroleum sales enterprise in Guangxi, China. Demographic characteristics, lifestyle factors, occupational information, anthropometric measurements, and laboratory indicators were collected through questionnaire survey, physical examination, and blood testing. VAI was calculated using waist circumference, body mass index, triglycerides, and high-density lipoprotein cholesterol. Four systemic inflammatory markers were evaluated, including the systemic inflammatory response index (SIRI), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII). Multivariable linear regression was used to assess the independent associations of VAI and inflammatory markers with SUA levels. Joint effects were evaluated by cross-classifying VAI and inflammatory markers according to their median values. Multiplicative interactions between VAI and inflammatory markers were examined using interaction terms. Restricted cubic spline analysis was performed to explore the nonlinear association between VAI and SUA.

Results: Among the 902 participants, the median SUA level was 331.00 $\mu\text{mol/L}$ (interquartile range: 278.25–397.75), and the prevalence of hyperuricemia was 25.7%. SUA levels and the prevalence of hyperuricemia increased significantly across VAI tertiles (both $p < 0.001$). After adjustment for age, sex, work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia, participants in the middle and highest VAI tertiles had higher SUA levels than those in the lowest tertile, with β values of 20.72 $\mu\text{mol/L}$ (95% CI: 9.56–31.88) and 57.11 $\mu\text{mol/L}$ (95% CI: 45.44–68.78), respectively (p for trend < 0.001). Restricted cubic spline analysis showed a significant nonlinear dose-response relationship between VAI and SUA (p -overall < 0.001 , p -nonlinear < 0.001), characterized by a rapid increase in SUA at low-to-moderate VAI levels and a plateau at higher VAI levels. None of the four systemic inflammatory markers showed significant independent associations with SUA levels in the fully adjusted model. Joint effect analysis showed that elevated SUA levels were mainly observed in groups with high VAI, whereas isolated elevation of inflammatory markers contributed little to SUA levels. Significant negative multiplicative interactions were observed between VAI and SIRI, NLR, and SII, with interaction p values of 0.031, 0.003, and 0.022, respectively. No significant interaction was observed between VAI and MLR. Simple slope analysis further indicated that the positive association between VAI and SUA was stronger at lower SIRI levels.

Conclusion: Among gas station workers in Guangxi, VAI was positively associated with SUA levels and showed a significant nonlinear dose-response relationship. Systemic inflammatory markers were not independently associated with SUA levels, but SIRI, NLR, and SII modified the association between VAI and SUA. These findings suggest that visceral adiposity may be an important correlate of elevated SUA

in this occupational population, while systemic inflammation may act more as an effect modifier than as an independent predictor.

1 Introduction

Hyperuricemia is a common metabolic abnormality caused by disordered purine metabolism or reduced uric acid excretion. It is an important biochemical basis for the development and progression of gout and is closely associated with multiple chronic noncommunicable diseases, including hypertension, cardiovascular disease, metabolic syndrome, and chronic kidney disease [1, 2]. In recent years, changes in dietary patterns and lifestyles and the increasing prevalence of obesity have been accompanied by a rising prevalence of hyperuricemia, making it an important public health concern [3–5]. Serum uric acid levels are influenced not only by genetic factors, diet, and renal excretion but also by metabolic abnormalities such as obesity, dyslipidemia, insulin resistance, and chronic low-grade inflammation [2, 7–10]. Therefore, identifying factors associated with elevated serum uric acid in occupational populations is important for the early screening and targeted prevention of hyperuricemia.

Visceral fat accumulation is an important metabolic basis of abnormal uric acid metabolism. Compared with subcutaneous fat, visceral fat has greater endocrine and metabolic activity and may contribute to elevated serum uric acid by promoting free fatty acid release, inducing insulin resistance, altering adipokine secretion, and affecting renal uric acid excretion [7–9]. The visceral adiposity index (VAI) is a composite measure based on waist circumference, body mass index, triglycerides, and high-density lipoprotein cholesterol and can indirectly reflect visceral fat accumulation and adipose tissue dysfunction [6]. Compared with a single anthropometric measure, VAI incorporates both body size and lipid-metabolism information and may therefore provide a more comprehensive indication of abnormal fat distribution and related metabolic risk [6, 10]. Previous studies have suggested that VAI is closely associated with hyperuricemia, metabolic syndrome, and cardiorenal metabolic abnormalities; however, whether the association between VAI and serum uric acid is nonlinear in different populations and whether it is modified by inflammatory status remain unclear [10].

Chronic low-grade inflammation is considered an important component of obesity-related metabolic abnormalities. Peripheral blood cells, including neutrophils, lymphocytes, monocytes, and platelets, not only reflect systemic immune-inflammatory status but also participate in oxidative stress, vascular endothelial injury, and metabolic dysfunction [11, 12]. In recent years, increasing attention has been paid to inflammation indices derived from routine blood-cell counts, including the systemic inflammatory response index (SIRI), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII). These markers are convenient and inexpensive and have been used to evaluate inflammatory burden and prognosis in various chronic diseases [13–16]. Uric acid itself has dual biological properties: it has antioxidant activity at certain concentrations, whereas persistent elevation may induce oxidative stress, inflammatory responses, and endothelial dysfunction [11, 12]. Systemic inflammatory status may therefore be involved in the association between visceral adiposity and abnormal uric acid metabolism. Nevertheless, the role of inflammatory markers in the

relationship between VAI and serum uric acid remains uncertain, and consistent evidence is lacking as to whether they act as independent correlates, mediators, or effect modifiers.

Gas station workers constitute an occupational population with distinctive exposure characteristics. Gasoline and its volatile organic compounds contain benzene, toluene, xylene, and other components, and long-term low-dose exposure may induce oxidative stress, inflammatory responses, and metabolic dysfunction [17–19]. Gas station workers also commonly experience shift work, night work, prolonged standing, and irregular work schedules, which may further influence obesity, lipid metabolism, inflammatory status, and uric acid metabolism. Compared with the general community population, metabolic health in occupational groups may be jointly affected by lifestyle factors and occupational exposures and therefore has important public health relevance. However, most existing studies of factors associated with serum uric acid have focused on community populations or clinical patients, while studies of petrochemical occupational groups such as gas station workers remain limited. In addition, most previous studies have considered a single obesity measure or a single inflammatory marker rather than systematically evaluating the independent, joint, and interactive effects of visceral adiposity and multiple systemic inflammatory indices on serum uric acid.

Accordingly, this study used questionnaire, physical-examination, and laboratory data from gas station workers employed by a petroleum sales enterprise in Guangxi to investigate the associations of VAI and four systemic inflammatory indices (SIRI, NLR, MLR, and SII) with serum uric acid. The specific objectives were to: (1) examine the independent association and nonlinear dose-response relationship between VAI and serum uric acid; (2) evaluate the independent associations of the four systemic inflammatory indices with serum uric acid; and (3) investigate the joint effects and multiplicative interactions of VAI and the systemic inflammatory indices. This study aimed to provide epidemiological evidence for the early identification and targeted prevention of abnormal uric acid metabolism in occupational populations.

2 Materials and Methods

2.1 Data Source and Study Participants

Data for this cross-sectional study were obtained from a survey conducted in Nanning, Guangxi Zhuang Autonomous Region, from July 1 to July 30, 2025. Cluster sampling was used, with gas stations, oil depots, and other primary work sites affiliated with the Guangxi Nanning Petroleum Branch of China Petroleum & Chemical Corporation Sales Co., Ltd. serving as sampling units. On-duty employees in urban Nanning and in Long'an County, Mashan County, Wuming District, Shanglin County, Binyang County, Litang Town, and Hengzhou City underwent questionnaire assessment, physical examination, and laboratory testing. A total of 1,044 workers were surveyed, and 902 were included in the final analysis after application of the eligibility criteria. Inclusion criteria were: (1) age ≥ 18 years; (2) employment in the current position for ≥ 1 year; (3) completion of the questionnaire, physical examination, and laboratory tests; and (4) complete data for the main study variables. Exclusion criteria were: (1)

pregnancy or lactation; (2) severe cardiac, hepatic, renal, hematologic disease, or malignant tumor; (3) use of urate-lowering medication within the preceding 3 months; and (4) missing data for key variables. The study was approved, and all participants provided written informed consent.

2.2 Exposure and Outcome Variables

The exposure variables were the visceral adiposity index (VAI) and four systemic inflammatory indices: the systemic inflammatory response index (SIRI), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII). All inflammatory indices were derived from routine blood-cell counts obtained during the health examination using an automated hematology analyzer. VAI was calculated separately by sex: for men, $VAI = [waist\ circumference / (39.68 + 1.88 \times BMI)] \times (TG / 1.03) \times (1.31 / HDL-C)$; for women, $VAI = [waist\ circumference / (36.58 + 1.89 \times BMI)] \times (TG / 0.81) \times (1.52 / HDL-C)$. Waist circumference was measured in cm, BMI in kg/m², and triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) in mmol/L. The inflammatory indices were calculated as follows: $SIRI = (neutrophil\ count \times monocyte\ count) / lymphocyte\ count$; $NLR = neutrophil\ count / lymphocyte\ count$; $MLR = monocyte\ count / lymphocyte\ count$; and $SII = platelet\ count \times neutrophil\ count / lymphocyte\ count$. All blood-cell counts were expressed as $\times 10^9/L$.

The primary outcome was serum uric acid (SUA), measured in fasting venous blood using an automated biochemical analyzer and expressed in micromoles per liter. SUA was analyzed as a continuous variable in the primary analysis. Hyperuricemia was the secondary outcome used in sensitivity analysis and was defined as SUA > 420 micromoles/L in men or > 360 micromoles/L in women, or a self-reported previous physician diagnosis of hyperuricemia.

2.3 General Questionnaire

A self-designed structured questionnaire was used to collect general information (Additional file 1). Individual-level variables included age, sex, educational attainment (junior high school or below, senior high school or technical secondary school, and junior college or above), and household registration (urban or rural). Occupational variables included average monthly income (< 2,000, 2,000–3,000, 3,001–4,000, 4,001–5,000, or $\geq 5,001$ CNY), work seniority (years), job category (fuel attendant, station manager, administrative position, technical position, or other), daily working hours (< 8, 8–9, 10–11, or ≥ 12 hours), work intensity (very high, high, moderate, low, or very low), and number of night-shift days per month. Behavioral variables included smoking (currently smoking ≥ 1 cigarette/day for ≥ 6 months), drinking (currently drinking ≥ 3 times/week for ≥ 6 months), and physical activity (total number of physically active days per week calculated using the short form of the International Physical Activity Questionnaire). Health conditions included hypertension (self-reported history or systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg), diabetes mellitus (self-reported history or fasting plasma glucose ≥ 7.0 mmol/L), hyperlipidemia (self-reported history or total cholesterol ≥ 6.2 mmol/L, triglycerides ≥ 2.3 mmol/L, low-density lipoprotein cholesterol ≥ 4.1 mmol/L, or high-density lipoprotein cholesterol < 1.0 mmol/L), and body mass index (continuous, kg/m²).

2.4 Statistical Analysis

The database was established in Microsoft Excel, and all statistical analyses were performed using R version 4.5.2.

Normally distributed continuous variables are presented as means and standard deviations, and skewed continuous variables as medians and interquartile ranges [M (P25, P75)]. Between-group comparisons were performed using t tests or analysis of variance for normally distributed variables and rank-sum tests for skewed variables. Categorical variables are presented as frequencies and percentages and were compared using chi-square tests. To reduce the influence of extreme values on model estimates, continuous variables were winsorized at the 1st and 99th percentiles. Variables with marked skewness, including VAI, SIRI, NLR, MLR, SII, triglycerides, and fasting plasma glucose, were natural-log transformed before modeling and the transformed values were used in regression analyses.

With SUA as a continuous outcome, separate linear regression models were fitted to assess the independent associations of VAI and each inflammatory index with SUA. Model 1 was unadjusted; Model 2 adjusted for age and sex; and Model 3 additionally adjusted for work seniority, job category, smoking, drinking, physical activity, hypertension, and hyperlipidemia. Tests for trend were performed by entering the tertile categories as ordinal variables in the fully adjusted model. VAI and each inflammatory index were further dichotomized at their medians and cross-classified into four groups (low VAI + low inflammation, low VAI + high inflammation, high VAI + low inflammation, and high VAI + high inflammation). The low VAI + low inflammation group served as the reference, and multivariable linear regression was used to evaluate joint effects on SUA. Multiplicative interactions were assessed by adding the product of the continuous VAI value and each continuous inflammatory index to the fully adjusted model; interaction analyses using median-based dichotomous groups were also performed as sensitivity analyses. Restricted cubic splines with four knots at the 5th, 35th, 65th, and 95th percentiles were fitted to evaluate the dose-response relationship between VAI and SUA, and the test for nonlinearity was reported. All statistical tests were two-sided, and $P < 0.05$ was considered statistically significant.

2.5 Sensitivity Analyses

Several sensitivity analyses were conducted to examine the robustness of the main findings. Stepwise-adjusted models were additionally presented for the associations of VAI and the systemic inflammatory indices with SUA and for the joint VAI-inflammatory-index groups. Logistic regression was used to repeat the primary analyses with hyperuricemia as a binary outcome. The VAI main-effect and interaction analyses were repeated after excluding participants with $\text{BMI} \geq 40 \text{ kg/m}^2$. Sex-stratified analyses were conducted for the association between VAI and SUA and for the VAI $\times$ SIRI interaction. Variance inflation factors were used to assess collinearity, with $\text{VIF} < 5$ considered to indicate no substantial multicollinearity. Robust standard errors were used to evaluate the robustness of linear-regression estimates, and false discovery rate correction was applied to multiple interaction tests. All tests were two-sided, and $P < 0.05$ was considered statistically significant.

3 Results

3.1 General Characteristics

A total of 902 gas station workers were included and divided into low (T1, n = 301), middle (T2, n = 301), and high (T3, n = 300) VAI tertiles. Baseline characteristics according to VAI tertile are shown in Table 1.

With increasing VAI, age, the proportion of men, the proportion with urban household registration, smoking prevalence, drinking prevalence, and work seniority generally increased, with statistically significant differences among groups (all $P < 0.05$). Participants in T3 were older than those in T1 [42.04 (7.63) vs 39.09 (7.95) years], and the proportion of men was also higher in T3 than in T1 (60.7% vs 39.5%). Smoking and drinking prevalences in T3 were 29.7% and 26.7%, respectively, compared with 16.3% and 17.9% in T1.

Regarding occupational factors, the distributions of daily working hours, night-shift days, work intensity, and job category differed significantly across VAI tertiles (all $P < 0.05$). Compared with T1, T3 included a smaller proportion of fuel attendants and relatively larger proportions of station managers, administrative staff, and technical staff. Total physical activity did not differ significantly across VAI tertiles ($P = 0.055$).

For metabolic and health-related indicators, the prevalences of hypertension, diabetes mellitus, hyperuricemia, and hyperlipidemia increased markedly with increasing VAI (all $P \leq 0.001$). Hyperuricemia prevalence was 44.0% in T3 compared with 12.3% in T1. BMI and SUA also increased across VAI tertiles; in T3, BMI was 26.01 (3.43) kg/m² and median SUA was 383.50 (321.00, 451.50) micromoles/L, both markedly higher than in T1.

Among the inflammatory indices, SIRI and SII differed significantly across VAI tertiles ($P < 0.001$ and $P = 0.015$, respectively), with relatively higher levels in the high-VAI group. NLR and MLR did not differ significantly among the three groups ($P = 0.594$ and $P = 0.164$, respectively). Educational attainment and monthly income also did not differ significantly across VAI tertiles (both $P > 0.05$).

Table 1
Baseline characteristics according to tertiles of the visceral adiposity index

Variables	Total (n = 902)	T1, low (n = 301)	T2, middle (n = 301)	T3, high (n = 300)	P value
Age, years	40.68 (7.84)	39.09 (7.95)	40.93 (7.69)	42.04 (7.63)	< 0.001
Male sex, n (%)	434 (48.1)	119 (39.5)	133 (44.2)	182 (60.7)	< 0.001
Education, n (%)					0.393
Junior high school or below	74 (8.2)	25 (8.3)	27 (9.0)	22 (7.3)	
Senior high/technical secondary school	425 (47.1)	138 (45.8)	145 (48.2)	142 (47.3)	
Junior/vocational college	293 (32.5)	93 (30.9)	92 (30.6)	108 (36.0)	
Bachelor's degree or above	110 (12.2)	45 (15.0)	37 (12.3)	28 (9.3)	
Monthly income, CNY, n (%)					0.100
<2,000	46 (5.1)	13 (4.3)	18 (6.0)	15 (5.0)	
2,000–2,999	429 (47.6)	156 (51.8)	150 (49.8)	123 (41.0)	
3,000–3,999	278 (30.8)	86 (28.6)	89 (29.6)	103 (34.3)	
4,000–4,999	106 (11.8)	36 (12.0)	33 (11.0)	37 (12.3)	
≥5,000	43 (4.8)	10 (3.3)	11 (3.7)	22 (7.3)	
Urban household registration, n (%)	479 (53.1)	140 (46.5)	161 (53.5)	178 (59.3)	0.007
Smoking, n (%)	194 (21.5)	49 (16.3)	56 (18.6)	89 (29.7)	< 0.001
Drinking, n (%)	181 (20.1)	54 (17.9)	47 (15.6)	80 (26.7)	0.002
Physical activity, days/week	6.50 (3.00, 7.50)	6.50 (3.00, 8.00)	5.50 (2.50, 7.50)	6.50 (3.00, 7.50)	0.055
Work seniority, years	14.63 (9.71)	12.67 (9.47)	14.97 (9.88)	16.26 (9.47)	< 0.001
Daily working hours, n (%)					0.008
<8 hours	51 (5.7)	6 (2.0)	22 (7.3)	23 (7.7)	

Variables	Total (n = 902)	T1, low (n = 301)	T2, middle (n = 301)	T3, high (n = 300)	P value
8–9 hours	216 (23.9)	63 (20.9)	83 (27.6)	70 (23.3)	
10–11 hours	269 (29.8)	94 (31.2)	83 (27.6)	92 (30.7)	
>=12 hours	366 (40.6)	138 (45.8)	113 (37.5)	115 (38.3)	
Night shifts, days/month	10.00 (0.00, 15.00)	10.00 (4.00, 15.00)	10.00 (0.00, 15.00)	10.00 (0.00, 15.00)	0.012
Work intensity, n (%)					0.032
Very high	352 (39.0)	134 (44.5)	104 (34.6)	114 (38.0)	
High	392 (43.5)	121 (40.2)	149 (49.5)	122 (40.7)	
Moderate	152 (16.9)	45 (15.0)	44 (14.6)	63 (21.0)	
Low	4 (0.4)	1 (0.3)	2 (0.7)	1 (0.3)	
Very low	2 (0.2)	0 (0.0)	2 (0.7)	0 (0.0)	
Job category, n (%)					0.009
Fuel attendant	572 (63.4)	211 (70.1)	197 (65.4)	164 (54.7)	
Station manager	175 (19.4)	50 (16.6)	51 (16.9)	74 (24.7)	
Administrative	54 (6.0)	13 (4.3)	18 (6.0)	23 (7.7)	
Technical	63 (7.0)	14 (4.7)	21 (7.0)	28 (9.3)	
Other	38 (4.2)	13 (4.3)	14 (4.7)	11 (3.7)	
Hypertension, n (%)	128 (14.2)	20 (6.6)	35 (11.6)	73 (24.3)	< 0.001
Diabetes mellitus, n (%)	41 (4.5)	4 (1.3)	14 (4.7)	23 (7.7)	0.001
Hyperuricemia, n (%)	232 (25.7)	37 (12.3)	63 (20.9)	132 (44.0)	< 0.001
Hyperlipidemia, n (%)	72 (8.0)	10 (3.3)	15 (5.0)	47 (15.7)	< 0.001
BMI, kg/m2	24.15 (3.57)	22.34 (3.06)	24.11 (3.24)	26.01 (3.43)	< 0.001
SUA, micromoles/L	331.00 (278.25, 397.75)	300.00 (256.00, 356.00)	325.00 (276.00, 381.00)	383.50 (321.00, 451.50)	< 0.001
VAI	1.35 (0.86, 2.19)	0.73 (0.59, 0.86)	1.35 (1.15, 1.58)	2.72 (2.19, 3.69)	< 0.001

Variables	Total (n = 902)	T1, low (n = 301)	T2, middle (n = 301)	T3, high (n = 300)	P value
SIRI	0.81 (0.58, 1.09)	0.74 (0.56, 0.96)	0.82 (0.58, 1.23)	0.87 (0.64, 1.19)	< 0.001
NLR	1.62 (1.29, 2.06)	1.60 (1.25, 2.02)	1.64 (1.30, 2.10)	1.63 (1.29, 2.05)	0.594
MLR	0.22 (0.18, 0.26)	0.21 (0.18, 0.25)	0.22 (0.18, 0.28)	0.22 (0.17, 0.25)	0.164
SII	459.49 (353.34, 607.17)	427.51 (337.45, 577.95)	469.98 (370.28, 613.63)	485.58 (354.93, 617.20)	0.015

Daily working hours are presented using the four original questionnaire categories. VAI, visceral adiposity index; BMI, body mass index; SUA, serum uric acid; SIRI, systemic inflammatory response index; NLR, neutrophil-to-lymphocyte ratio; MLR, monocyte-to-lymphocyte ratio; SII, systemic immune-inflammation index.

3.2 Independent Association Between VAI and SUA

Table 2 presents the multivariable linear-regression results for the association between VAI tertiles and SUA. In the unadjusted model, compared with the low-VAI group (T1), SUA was significantly higher in T2 and T3, with β coefficients of 24.12 micromoles/L (95% CI, 10.80–37.44) and 78.97 micromoles/L (95% CI, 65.64–92.29), respectively; the trend was statistically significant (P for trend < 0.001).

After adjustment for age and sex, the associations were attenuated but remained statistically significant. After further adjustment for work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia, VAI remained positively associated with SUA. Compared with T1, SUA was 20.72 micromoles/L higher in T2 (95% CI, 9.56–31.88) and 57.11 micromoles/L higher in T3 (95% CI, 45.44–68.78), with a clear dose-response trend (P for trend < 0.001). These findings indicate that higher VAI was independently associated with elevated SUA.

Table 2
Multivariable linear-regression association between VAI tertiles and SUA

VAI tertile	Model 1 β (95% CI)	Model 2 β (95% CI)	Model 3 β (95% CI)
T1 (low)	Reference	Reference	Reference
T2 (middle)	24.12 (10.80, 37.44)	21.59 (10.41, 32.78)	20.72 (9.56, 31.88)
T3 (high)	78.97 (65.64, 92.29)	62.33 (50.90, 73.77)	57.11 (45.44, 68.78)
P for trend	< 0.001	< 0.001	< 0.001

β represents the difference in SUA (micromoles/L) compared with T1. Model 1 was unadjusted. Model 2 adjusted for age and sex. Model 3 additionally adjusted for work seniority, smoking, drinking, physical

activity, job category, hypertension, and hyperlipidemia. P for trend was calculated by entering VAI tertiles as an ordinal variable. CI, confidence interval.

3.3 Independent Associations of Systemic Inflammatory Indices With SUA

Table 3 presents the multivariable linear-regression results for the associations between tertiles of the four systemic inflammatory indices and SUA. In the fully adjusted model, none of SIRI, NLR, MLR, or SII was independently associated with SUA at a statistically significant level.

Using the lowest tertile of each inflammatory index as the reference, SUA was 9.68 micromoles/L higher in the highest SIRI tertile, but the difference was not statistically significant (95% CI, -2.16 to 21.52; P = 0.109; P for trend = 0.107). SUA was also 9.48 micromoles/L higher in the highest SII tertile, which did not reach statistical significance (95% CI, -2.33 to 21.30; P = 0.116; P for trend = 0.115). No significant associations were observed for the NLR or MLR tertiles.

These results suggest that, after adjustment for demographic, behavioral, and occupational factors, individual systemic inflammatory indices were not clearly and independently associated with elevated SUA.

Table 3
Multivariable associations of systemic inflammatory-index tertiles with SUA

Inflammatory index	Tertile	β (95% CI), micromoles/L	P value	P for trend
SIRI	T1	Reference		
	T2	8.01 (-3.65, 19.66)	0.178	
	T3	9.68 (-2.16, 21.52)	0.109	0.107
NLR	T1	Reference		
	T2	-3.30 (-15.13, 8.53)	0.584	
	T3	-3.09 (-14.86, 8.68)	0.606	0.609
MLR	T1	Reference		
	T2	6.27 (-5.39, 17.94)	0.292	
	T3	-4.08 (-15.81, 7.66)	0.495	0.493
SII	T1	Reference		
	T2	4.66 (-7.09, 16.41)	0.436	
	T3	9.48 (-2.33, 21.30)	0.116	0.115

β represents the difference in SUA (micromoles/L) compared with T1. Models adjusted for age, sex, work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia. SIRI, systemic inflammatory response index; NLR, neutrophil-to-lymphocyte ratio; MLR, monocyte-to-lymphocyte ratio; SII, systemic immune-inflammation index; CI, confidence interval.

3.4 Joint Effects of VAI and Systemic Inflammatory Indices on SUA

Table 4 presents the associations between the joint VAI-inflammatory-index groups and SUA. Overall, SUA was significantly elevated whenever VAI was high, irrespective of which inflammatory index was considered, whereas isolated elevation of an inflammatory index was associated with only a small or nonsignificant increase in SUA.

For the joint VAI-SIRI groups, compared with the low VAI + low SIRI group, SUA was 13.70 micromoles/L higher in the low VAI + high SIRI group (95% CI, 0.62–26.77; $P = 0.040$). SUA was also significantly higher in the high VAI + low SIRI and high VAI + high SIRI groups, with β coefficients of 49.88 micromoles/L (95% CI, 36.57–63.20; $P < 0.001$) and 44.72 micromoles/L (95% CI, 32.08–57.35; $P < 0.001$), respectively.

The joint NLR, MLR, and SII analyses showed similar patterns. The low VAI + high inflammatory-index groups did not differ significantly from the reference groups, whereas SUA was significantly elevated in both the high VAI + low inflammatory-index and high VAI + high inflammatory-index groups. Beta coefficients for the high-VAI joint groups ranged from 40.25 to 51.77 micromoles/L and were all statistically significant.

These findings indicate that the association of elevated VAI with higher SUA was relatively stable, whereas an isolated increase in an inflammatory index contributed relatively little to SUA.

Table 4
Joint VAI-inflammatory-index groups in relation to SUA

Inflammatory index	Joint group	β (95% CI), micromoles/L	P value
SIRI	Low VAI + low SIRI	Reference	-
	Low VAI + high SIRI	13.70 (0.62, 26.77)	0.040
	High VAI + low SIRI	49.88 (36.57, 63.20)	< 0.001
	High VAI + high SIRI	44.72 (32.08, 57.35)	< 0.001
NLR	Low VAI + low NLR	Reference	-
	Low VAI + high NLR	10.03 (-2.97, 23.04)	0.130
	High VAI + low NLR	51.77 (38.53, 65.01)	< 0.001
	High VAI + high NLR	40.45 (27.53, 53.38)	< 0.001
MLR	Low VAI + low MLR	Reference	-
	Low VAI + high MLR	1.89 (-11.08, 14.86)	0.775
	High VAI + low MLR	40.25 (27.07, 53.44)	< 0.001
	High VAI + high MLR	43.83 (30.46, 57.20)	< 0.001
SII	Low VAI + low SII	Reference	-
	Low VAI + high SII	7.76 (-5.39, 20.91)	0.247
	High VAI + low SII	47.98 (34.66, 61.30)	< 0.001
	High VAI + high SII	41.61 (29.02, 54.20)	< 0.001

VAI and each inflammatory index were dichotomized at their medians. β represents the difference in SUA (micromoles/L) compared with the low VAI + low inflammatory-index group. Models adjusted for age, sex, work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia.

3.5 Multiplicative Interactions of VAI With Systemic Inflammatory Indices

Table 5 presents the multiplicative interactions of VAI with the four systemic inflammatory indices in relation to SUA. In the fully adjusted model, significant multiplicative interactions were observed between VAI and SIRI, NLR, and SII, and all interaction coefficients were negative.

In the continuous-variable interaction analyses, the VAI $\times$ SIRI interaction was statistically significant (β =-5.32, SE = 2.46, P = 0.031); the dichotomous interaction had the same direction and was also significant (β =-18.87, SE = 9.43, P = 0.046). The interaction between VAI and NLR was the most

pronounced. Both the continuous and dichotomous interaction terms were statistically significant, with β coefficients of -5.81 (SE = 1.95, P = 0.003) and - 21.35 (SE = 9.29, P = 0.022), respectively.

The continuous VAI $\times$ SII interaction was also statistically significant (β =-0.01, SE = 0.004, P = 0.022), whereas the dichotomous interaction was not significant (β =-14.13, SE = 9.37, P = 0.132). No significant multiplicative interaction was observed between VAI and MLR.

Overall, SIRI, NLR, and SII may modify the association between VAI and SUA. The negative interaction coefficients suggest that the positive association between VAI and SUA may be stronger at lower inflammatory levels, whereas the marginal effect of VAI on SUA may be attenuated at higher inflammatory levels. To visualize the interaction between VAI and SIRI, a simple-slope plot was generated from the multivariable linear-regression model containing the continuous VAI $\times$ SIRI interaction. The 25th and 75th percentiles of SIRI represented low and high SIRI levels, respectively. Predicted SUA increased with VAI at both SIRI levels; however, the slope was steeper at low SIRI and flatter at high SIRI, suggesting a stronger positive association between VAI and SUA at lower SIRI. The VAI-SIRI interaction was statistically significant (P for interaction = 0.031), consistent with the negative multiplicative interaction shown in Table 5 (Fig. 1).

Table 5
Multiplicative interactions of VAI with four systemic inflammatory indices in relation to SUA

Inflammatory index	Continuous VAI $\times$ index β (SE)	P value	Dichotomized VAI $\times$ index β (SE)	P value
SIRI	-5.32 (2.46)	0.031	-18.87 (9.43)	0.046
NLR	-5.81 (1.95)	0.003	-21.35 (9.29)	0.022
MLR	-22.13 (19.01)	0.244	1.69 (9.27)	0.857
SII	-0.01 (0.004)	0.022	-14.13 (9.37)	0.132

Models adjusted for age, sex, work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia. The continuous interaction term was the product of $\ln(\text{VAI})$ and $\ln(\text{inflammatory index})$. β is the interaction coefficient; SE, standard error. The dichotomous interaction was the product of the high/low VAI group and the high/low inflammatory-index group.

3.6 Dose-Response Relationship

Restricted cubic spline analysis was used to evaluate the dose-response relationship between VAI and SUA, with knots placed at the 5th, 35th, 65th, and 95th percentiles of VAI and adjustment for age, sex, work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia. VAI showed a significant overall association with SUA and a significant nonlinear relationship (P overall < 0.001; P nonlinear < 0.001). SUA generally increased as VAI increased. The increase was rapid at lower VAI levels and gradually attenuated at higher levels, producing a nonlinear pattern that was initially steep and subsequently flatter (Fig. 2).

3.7 Network Toxicology

Based on screening of the Comparative Toxicogenomics Database and GeneCards, 74 BTEX-related targets and 576 targets related to abnormal uric acid metabolism were identified. Their intersection yielded 16 potential common targets: CYP2E1, CCL2, ALB, TNF, MUC1, PTGS2, TP53, CXCL8, NFKB1, CYP2D6, UGT1A6, UGT1A7, IGF2BP2, RELA, CASP3, and APP. These targets were imported into the STRING database, with the species restricted to *Homo sapiens*, to construct a protein-protein interaction network. The network contained 16 nodes and 67 interaction edges, with an average node degree of 8.38. ALB, TNF, PTGS2, TP53, CASP3, CCL2, CXCL8, NFKB1, RELA, and APP had relatively high degree values, suggesting that they may be key candidate targets through which volatile organic compounds encountered at gas stations influence abnormal uric acid metabolism. Gene Ontology enrichment analysis showed that the common targets were primarily enriched in biological processes including response to tumor necrosis factor, cellular response to chemical stimulus, response to xenobiotic stimulus, response to cytokine, regulation of small-molecule metabolic processes, and regulation of cell death. Kyoto Encyclopedia of Genes and Genomes enrichment analysis indicated involvement of the IL-17 signaling pathway, TNF signaling pathway, AGE-RAGE signaling pathway in diabetic complications, and non-alcoholic fatty liver disease pathway (Fig. 3). These findings suggest that volatile organic compounds commonly encountered at gas stations may contribute to abnormal uric acid metabolism through xenobiotic metabolism, inflammatory cytokine signaling, oxidative-stress-related responses, regulation of small-molecule metabolism, and cellular injury.

3.8 Sensitivity Analyses

Multiple sensitivity analyses were performed to examine the robustness of the main findings, and the results are presented in Supplementary Tables S1-S8. Stepwise-adjusted models showed that the positive association between VAI and SUA remained stable in the unadjusted, age- and sex-adjusted, and fully adjusted models. Independent associations between the systemic inflammatory indices and SUA were generally unstable and were not statistically significant after full adjustment. Results for the joint VAI-inflammatory-index groups were also consistent: SUA was markedly elevated in the high-VAI groups, whereas isolated high inflammatory-index groups showed smaller or nonsignificant increases. When hyperuricemia was analyzed as a binary outcome, higher VAI remained positively associated with hyperuricemia. The positive association between VAI and SUA remained stable after exclusion of participants with BMI ≥ 40 kg/m². In sex-stratified analyses, a positive association between VAI and SUA was observed in both men and women, whereas the VAI $\times$ SIRI interaction was mainly observed in women. Collinearity diagnostics showed VIF values below 5 in all models, indicating no substantial multicollinearity. Models based on robust standard errors produced results broadly consistent with the primary analyses. After false discovery rate correction, some interactions between VAI and the inflammatory indices remained statistically significant. Overall, the positive association between VAI and SUA was robust, whereas the interactions of VAI with inflammatory indices were relatively sensitive to stratification and model specification and should be interpreted cautiously as exploratory effect-modification signals.

4 Discussion

This study systematically evaluated the relationships of the visceral adiposity index (VAI) and four systemic inflammatory indices with SUA among gas station workers employed by a petroleum sales enterprise in Guangxi. The principal findings were as follows. First, VAI was significantly and positively associated with SUA, and the association remained stable after adjustment for demographic, behavioral, and occupational factors. Compared with the lowest VAI tertile, the highest tertile had an approximately 57-micromoles/L higher SUA level, suggesting that visceral fat accumulation may be an important correlate of elevated SUA in this occupational population. Second, restricted cubic spline analysis indicated that the relationship between VAI and SUA was not simply linear but followed a clear nonlinear dose-response pattern: SUA increased rapidly with VAI in the low-to-moderate range, whereas the increase gradually flattened at higher VAI levels. Third, none of the four systemic inflammatory indices—SIRI, NLR, MLR, and SII—was independently associated with SUA at a statistically significant level, suggesting that these indices themselves may not be the main independent correlates of elevated SUA in this population. Fourth, interaction analyses showed negative multiplicative interactions of VAI with SIRI, NLR, and SII, and simple-slope analysis further showed a stronger positive association between VAI and SUA at lower SIRI levels. Together, these findings suggest that visceral fat accumulation may be a central correlate of abnormal uric acid metabolism in gas station workers, whereas systemic inflammatory indices may act more as effect modifiers than as independent main-effect factors.

Hyperuricemia is a common metabolic abnormality closely associated with the risks of gout, cardiovascular disease, metabolic syndrome, and chronic kidney disease and has become an important public health problem [1–5]. Obesity, particularly visceral fat accumulation, is considered an important basis of abnormal uric acid metabolism. Compared with conventional obesity measures, VAI integrates waist circumference, body mass index, triglycerides, and high-density lipoprotein cholesterol and can, to some extent, reflect both visceral fat accumulation and adipose tissue dysfunction [6]. Previous research has shown that visceral adipose tissue is not merely an energy-storage organ but also a metabolically active endocrine organ. Excess accumulation may elevate uric acid through insulin resistance, lipid-metabolism disorders, oxidative stress, and altered renal urate excretion [7–10]. The stable positive association between VAI and SUA observed in this study is broadly consistent with these findings.

This study further identified a nonlinear dose-response relationship between VAI and SUA. The restricted cubic spline curve showed that SUA increased rapidly with VAI at lower VAI levels and gradually plateaued once VAI became high. This pattern suggests diminishing marginal effects of visceral adiposity on SUA. One possible explanation is that during the early or moderate stages of visceral fat accumulation, insulin resistance, increased free fatty acid release, and lipid-metabolism abnormalities may exert particularly sensitive effects on uric acid production and excretion. Once visceral fat accumulation is substantial, the relevant metabolic pathways may approach saturation, and further increases in VAI may make a smaller marginal contribution to SUA. Because this was a cross-sectional study, the spline results indicate only a nonlinear association and cannot establish a specific threshold

effect or causality. Longitudinal studies are needed to verify the stability and clinical relevance of this nonlinear relationship.

No significant independent associations were found between SIRI, NLR, MLR, or SII and SUA. The relationship between uric acid and inflammation is complex. On the one hand, uric acid has antioxidant properties; on the other, persistently elevated uric acid may participate in inflammatory processes through oxidative stress, endothelial dysfunction, and activation of innate immunity [11, 12]. SIRI, NLR, MLR, and SII are all inflammatory indices derived from peripheral blood-cell counts and have been used to assess inflammatory burden or prognosis in multiple chronic diseases [13–16]. However, they are not inflammation-specific biomarkers and may be affected by infection, stress, sleep, medication, timing of blood collection, and short-term physiological fluctuations. Therefore, in a relatively young and generally healthy occupational population, blood-cell-derived inflammatory indices may be insufficient to independently explain differences in SUA.

Notably, although none of the four inflammatory indices had a significant independent effect, VAI showed multiplicative interactions with SIRI, NLR, and SII, all in a negative direction. Simple-slope analysis showed that the positive association between VAI and predicted SUA was steeper at low SIRI and flatter at high SIRI. This suggests that inflammatory indices may not be independent correlates of SUA but may alter the strength of the VAI-SUA association under different inflammatory backgrounds, thereby acting as effect modifiers. NLR displayed a pattern of a nonsignificant independent effect but a significant interaction, also suggesting that it may more strongly reflect modulation of a metabolic association by immune-inflammatory status than a direct relationship with elevated SUA. Because interaction analyses are sensitive to model specification, variable scale, and covariate adjustment, these results should be regarded as exploratory and require confirmation in other occupational populations or prospective cohorts.

From a mechanistic perspective, the association between VAI and elevated SUA may occur primarily through nonspecific metabolic pathways. Visceral fat accumulation promotes free fatty acid release and induces insulin resistance and hyperinsulinemia. Insulin resistance may increase SUA by promoting renal tubular urate reabsorption, reducing uric acid excretion, and enhancing purine metabolism [7–9]. Visceral-fat-related dyslipidemia and oxidative stress may also affect glomerular and tubular function and thereby indirectly alter uric acid excretion [8, 9]. In the present study, inflammatory indices were not independently associated with SUA but showed modifying effects in interaction analyses, suggesting that inflammation may not be the principal intermediate pathway by which VAI influences SUA but may instead reflect differences in the immune-inflammatory background across metabolic states. A hypothetical interpretation of the negative interaction is that, when inflammation is low, the visceral adiposity and lipid-metabolism abnormalities represented by VAI exert a more prominent influence on SUA; when inflammation is high, the body may already be in a more complex metabolic-immune state, and the marginal effect of VAI on SUA may be attenuated. Because this study was cross-sectional, this explanation remains hypothetical and requires longitudinal and mechanistic investigation.

The gas station workers in this study have distinctive occupational-health characteristics. Gasoline and its volatile organic compounds contain benzene, toluene, xylene, and other constituents, and long-term low-dose exposure may be associated with oxidative stress, inflammatory responses, and altered hepatic and renal function [17–19]. Gas station workers also commonly experience shift work, night work, irregular work schedules, and distinctive physical-activity patterns, all of which may be related to obesity, dyslipidemia, and abnormal uric acid metabolism. Attention to visceral fat accumulation and uric acid abnormalities in this occupational population therefore has public health relevance. This study further used network toxicology to explore potential molecular mechanisms linking volatile organic compounds commonly encountered at gas stations to abnormal uric acid metabolism. The results indicated a shared target network between BTEX-related targets and targets associated with uric acid metabolic abnormalities; ALB, TNF, PTGS2, TP53, CASP3, CCL2, CXCL8, NFKB1, RELA, and APP had relatively high network connectivity. Enrichment analyses suggested that these targets mainly involved responses to xenobiotic chemical stimuli, TNF/IL-17 inflammatory signaling, regulation of small-molecule metabolism, and regulation of cell death. These findings have some biological consistency with the epidemiological associations among VAI, systemic inflammatory indices, and SUA observed in this study. Visceral fat accumulation may influence SUA through dyslipidemia, insulin resistance, and altered renal urate excretion, whereas occupational exposure to volatile organic compounds may further disturb metabolic homeostasis through cytochrome-P450-mediated xenobiotic metabolism, activation of inflammatory factors, and oxidative stress. It should be emphasized that the network-toxicology analysis was based on public-database predictions and that individual-level BTEX exposure doses or biomarkers were unavailable; accordingly, these results provide only potential mechanistic clues and cannot be interpreted as causal evidence.

From an occupational-health-management perspective, the findings suggest that, in addition to routine SUA testing, occupational health examinations should give attention to waist circumference, BMI, triglycerides, HDL-C, and other routinely measured indicators related to visceral adiposity. Workers with high VAI may benefit from early health management incorporating lifestyle intervention, weight management, and metabolic-risk assessment. Workers with long employment histories at gas stations, frequent night shifts, or clustering of metabolic abnormalities may also require strengthened occupational-exposure protection, health education, and continuous follow-up to reduce the risks of abnormal uric acid metabolism and related chronic diseases.

This study has several strengths. First, it focused on gas station workers, addressing the limited evidence from occupational populations compared with general community samples and clinical patients. Second, it included VAI and four inflammatory indices-SIRI, NLR, MLR, and SII-and evaluated their relationships with SUA from multiple perspectives, including independent effects, joint effects, interactions, and nonlinear dose-response associations. Third, questionnaire, physical-examination, and laboratory data allowed adjustment for multiple potential confounders, including age, sex, work seniority, smoking, drinking, physical activity, job category, and relevant disease histories, thereby improving the robustness of the findings. Fourth, network-toxicology exploration was added to the epidemiological analyses and provided complementary evidence for potential mechanistic links among volatile organic

compounds at gas stations, inflammatory responses, metabolic dysfunction, and uric acid abnormalities.

This study also has several limitations. First, the cross-sectional design cannot establish the temporal sequence among VAI, inflammatory indices, and SUA and therefore cannot support causal inference. Elevated SUA itself may also affect inflammatory or metabolic status, creating the possibility of reverse causation or bidirectional associations. Second, VAI, SUA, and blood-cell-derived inflammatory indices were each based on a single measurement and may have been influenced by short-term diet, fluid intake, exercise, sleep, infection, stress, and timing of blood collection, resulting in measurement error. Third, despite adjustment for multiple covariates, residual confounding may remain. High-purine foods, sugar-sweetened beverage intake, amount of alcohol consumption, renal-function indices, menopausal status, diuretic use, and use of other medications affecting uric acid metabolism were not fully controlled. Fourth, SIRI, NLR, MLR, and SII are convenient and economical blood-cell-derived indices but lack inflammatory specificity; they cannot replace classical inflammatory biomarkers such as C-reactive protein, interleukin-6, and tumor necrosis factor-alpha and cannot reflect local inflammation in adipose tissue. Fifth, VAI is an indirect surrogate for visceral adiposity rather than a direct measure of visceral fat area by computed tomography, magnetic resonance imaging, or dual-energy X-ray absorptiometry and may therefore be subject to misclassification. Sixth, multiple inflammatory indices and interaction terms were tested, creating the possibility of chance-positive findings due to multiple comparisons; the interaction results should therefore be considered exploratory. Seventh, participants were gas station workers from one petroleum sales enterprise in Guangxi, and a healthy-worker effect may be present; generalization to other regions, industries, or the general population should be cautious. Finally, occupational exposure was not quantitatively assessed using personal benzene-series concentrations, urinary metabolites, cumulative exposure doses, or similar measures. Consequently, the role of occupational exposure in the relationships among visceral adiposity, inflammation, and uric acid metabolism could not be evaluated. The network-toxicology analysis was also largely based on public-database predictions and lacked experimental validation and individual exposure data; the mechanistic interpretations should therefore remain exploratory.

In conclusion, among gas station workers employed by a petroleum sales enterprise in Guangxi, VAI was significantly and positively associated with SUA and showed a nonlinear dose-response relationship. None of the four systemic inflammatory indices was independently associated with SUA, whereas SIRI, NLR, and SII modified the association between VAI and SUA. Network-toxicology analysis further suggested that volatile organic compounds commonly encountered at gas stations may contribute to abnormal uric acid metabolism through responses to xenobiotic chemical stimuli, inflammatory signaling, regulation of small-molecule metabolism, and regulation of cell death. These findings suggest that visceral fat accumulation may be an important correlate of elevated SUA in this occupational population, whereas inflammatory indices may act more as effect modifiers. Future studies should use prospective designs and incorporate insulin resistance, renal function, oxidative stress, classical inflammatory biomarkers, and quantitative occupational-exposure measures to further clarify the

temporal relationships and potential pathways linking visceral adiposity, inflammatory status, occupational exposure, and abnormal uric acid metabolism.

Declarations

5.1 Ethics approval and consent to participate

Ethics approval was obtained from the Ethics Committee of Youjiang Medical University for Nationalities, Baise, Guangxi, China. The ethics approval number will be provided upon request. All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments. All participants provided written informed consent before taking part in the study.

5.2 Consent for publication

Not applicable.

5.3 Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

5.4 Competing interests

The authors declare that they have no competing interests.

5.5 Funding

The authors received no specific funding for this work.

5.6 Authors' contributions

JW: Conceptualization, Methodology, Writing—Original Draft; ZC: Conceptualization, Methodology; XH: Writing—Review & Editing, Supervision; YL: Investigation, Data Curation, Conceptualization, Supervision, Project administration; JC: Investigation, Data Curation; YP: Conceptualization, Supervision, Project administration, Funding Acquisition. All authors read and approved the final manuscript.

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5.8 Authors' information

Not applicable.

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Figures

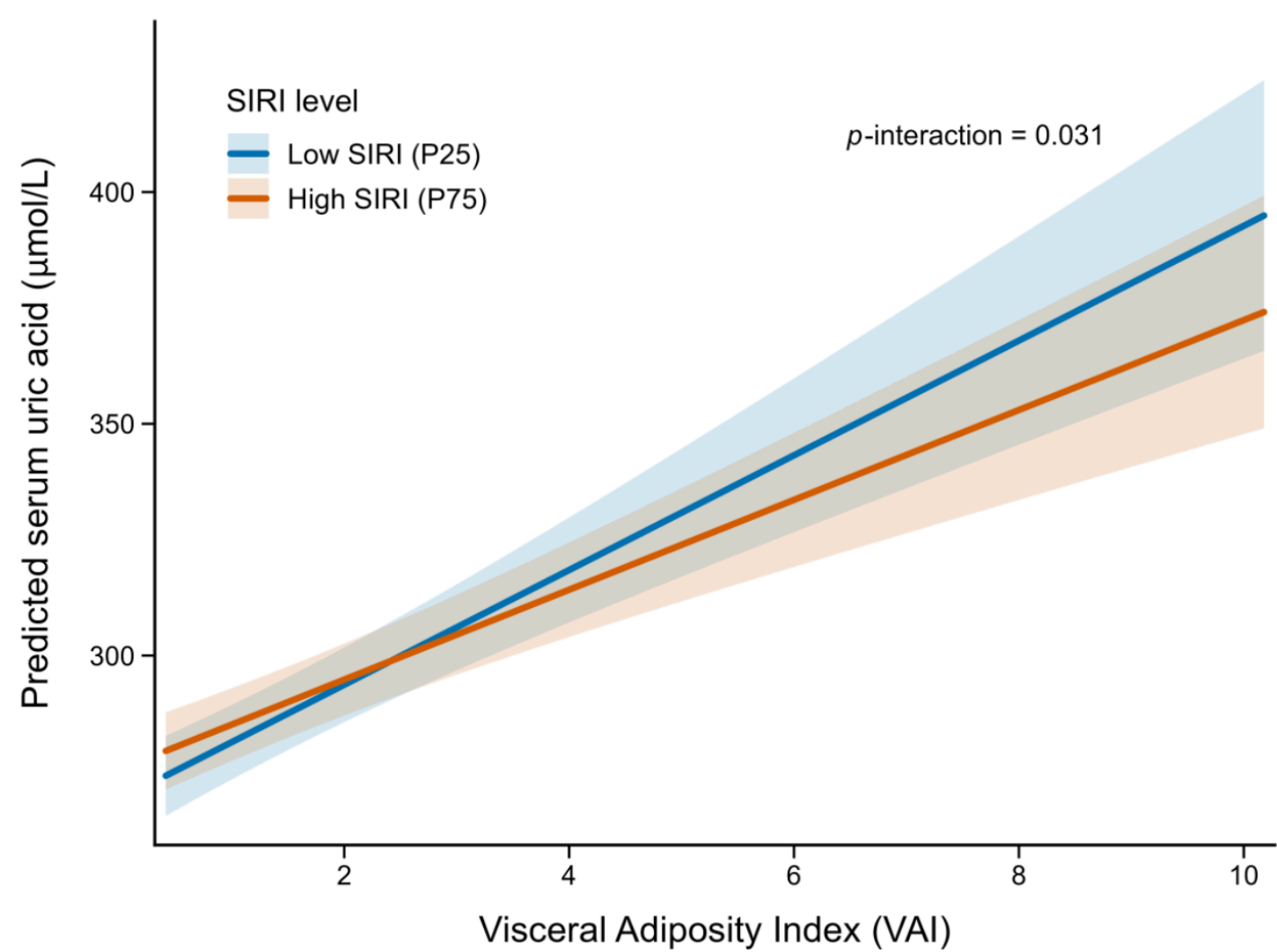


Figure 1

Simple-slope analysis of the interaction between VAI and SIRS in relation to SUA

The figure was generated from a multivariable linear-regression model containing the continuous VAI x SIRS interaction. Predicted SUA was calculated at the 25th percentile (low SIRS) and 75th percentile (high SIRS) of SIRS. Solid lines indicate fitted relationships and shaded areas indicate 95% confidence intervals.

The model adjusted for age, sex, work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia. VAI, visceral adiposity index; SIRI, systemic inflammatory response index.

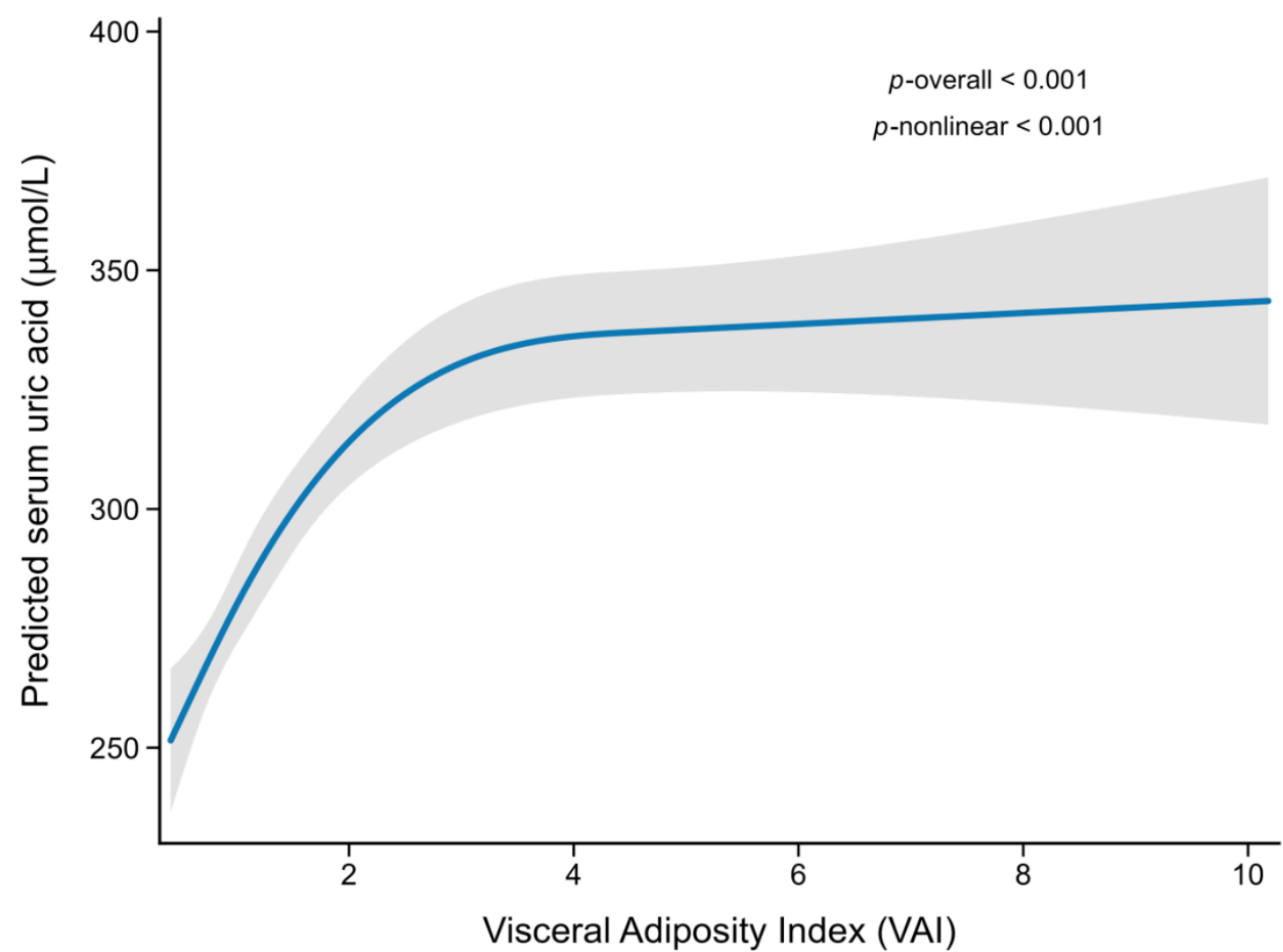
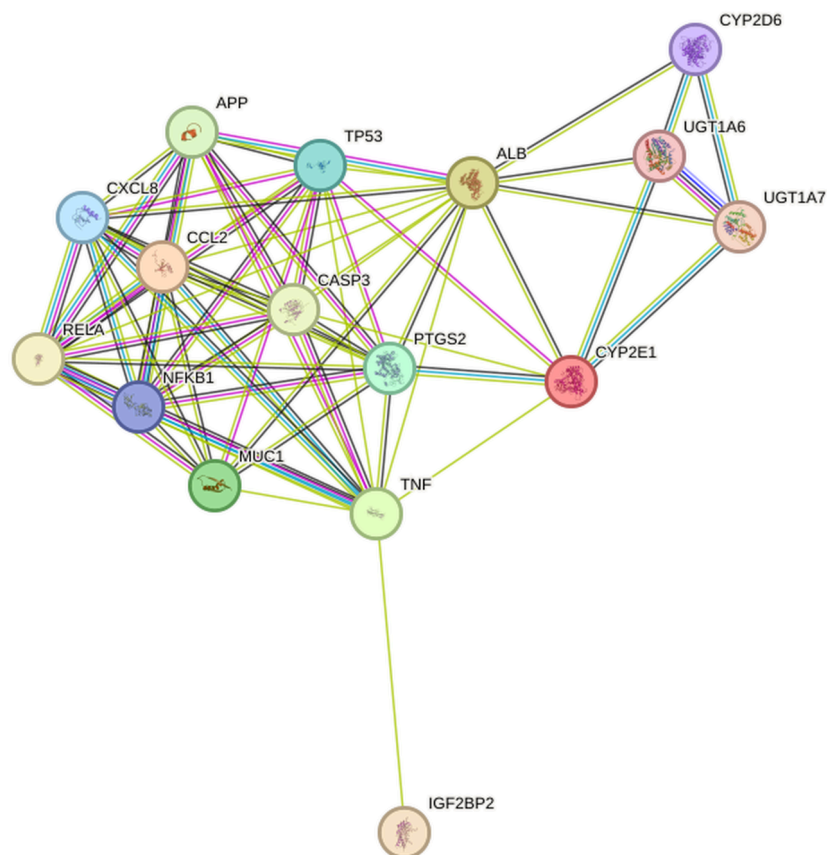


Figure 2

Restricted cubic spline analysis of the association between VAI and SUA

The solid line indicates the fitted association between VAI and SUA, and the shaded area indicates the 95% confidence interval. The model adjusted for age, sex, work seniority, smoking, drinking, physical activity, job category, hypertension, and hyperlipidemia. Knots were placed at the 5th, 35th, 65th, and 95th percentiles of VAI. P overall indicates the significance of the overall association and P nonlinear indicates the significance of nonlinearity.

A



B

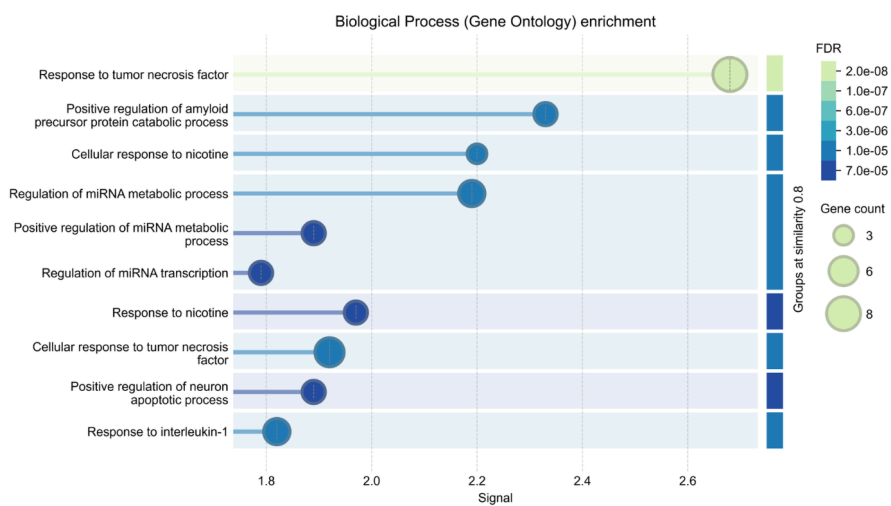


Figure 3

Network-toxicology analysis of potential mechanisms linking volatile organic compounds at gas stations to abnormal uric acid metabolism

A, protein-protein interaction network of the 16 common targets constructed using STRING; nodes represent proteins and edges represent protein interactions. B, Gene Ontology biological-process

enrichment; bubble size represents gene count and color represents the false discovery rate.

Supplementary Files

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