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Life's Essential 8, Life's Simple 7 and the odds of hyperuricaemia: results from the China Multi-Ethnic Cohort Study

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Abstract

Objective: Life's Essential 8 (LE8) is a new comprehensive metric based on Life's Simple 7 (LS7). Few studies have investigated the association between LE8 and the odds of hyperuricaemia (HUA). This study examined the association between LE8, LS7 with odds of HUA.

Methods: We cross-sectionally analysed data from the China Multi-Ethnic Cohort (CMEC) study. LE8 and LS7 were categorized as low, moderate and high. The CMEC provided an ideal and unique opportunity to characterize the association between LE8, LS7 and the odds of HUA.

Results: Of the 89 823 participants, 14 562 (16.2%) had HUA. A high level of LE8 was associated with lower odds of HUA after full adjustment. The adjusted odds ratios (ORs) were 1 (reference), 0.70 (95% CI 0.67, 0.73) and 0.45 (0.42, 0.48) across low, moderate and high LE8 groups, respectively ($P_{trend} < 0.001$). Similar results were observed in LS7 and HUA. The adjusted ORs were 1 (reference), 0.68 (95% CI 0.65, 0.71) and 0.46 (95% CI 0.43, 0.49) across low, moderate and high LS7 groups, respectively ($P_{trend} < 0.001$). There were significant interactions between LE8 and age, gender, ethnicity and drinking habits on HUA. Receiver operating characteristics analysis showed that the area under the curve for LE8 and LS7 were similar (0.638 and 0.635, respectively).

Conclusion: This study indicated a clearly inverse gradient association between the cardiovascular health metrics LE8 and LS7 and the odds of HUA.

Lay Summary

What does this mean for patients?

Hyperuricaemia (HUA) is a condition where a person has a higher uric acid level in the blood than usual. HUA can lead to the development of various diseases, including gout. Recently the American Heart Association released a new approach to assess people's cardiovascular health: Life's Essential 8 (LE8). LE8 measures health behaviours such as diet and physical activity, as well as factors like blood pressure and body mass index. We wanted to determine whether there is an association between the LE8 score and the risk of HUA. After looking at data from nearly 90 000 Chinese adults, we found that a higher LE8 score was associated with a lower risk of HUA. Our results show that people should be encouraged to take on more healthy behaviours, thus generating a higher LE8 score and lowering their risk of HUA.

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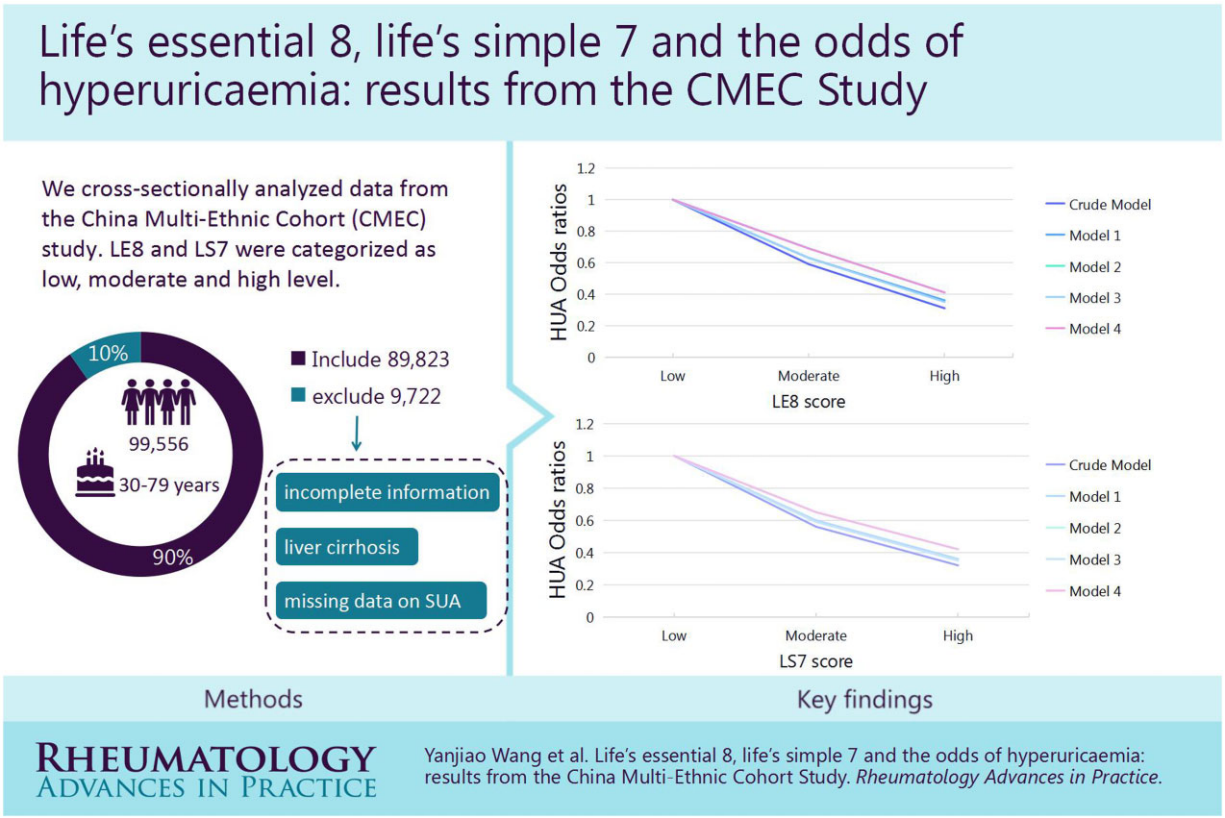
Graphical abstract



Keywords: Life's Essential 8, Life's Simple 7, uric acid, hyperuricaemia, CMEC.

Key messages

- Epidemiological evidence on the role of LE8 in hyperuricaemia is limited.
- LE8 and LS7 were each independently associated with the odds of hyperuricaemia.
- Our study supports the public health implications of LE8 for the odds of hyperuricaemia assessment.

Introduction

The epidemic of hyperuricaemia (HUA) has attained greater recognition in recent years. HUA has become a major problem in addition to the traditional 'three-high' diseases: hyperlipidaemia, hypertension and hyperglycaemia. Specifically, an increase in uric acid synthesis and a decrease in renal urate excretion can cause HUA, which is defined as serum uric acid (SUA) levels >404.6 μmol/l (6.8 mg/dl) for both men and women [1]. The prevalence of HUA in South Australia and the USA is 16.6% [2] and 14.6% [3], respectively. A study reported that HUA prevalence is 25% in East European hypertensive participants [4]. The prevalence of HUA in China ranges from 13.7 to 18.8% in different regions [5]. Of note, the prevalence of HUA is mostly driven by lifestyle, such as an unhealthy diet [6], sleep deprivation [7] or unfavourable lipid profiles [8].

Recently, the American Heart Association (AHA) released a new approach to assess cardiovascular health, Life's Essential 8 (LE8), which includes four health behaviours (diet, smoking, physical activity and sleep health) and four health factors [BMI, glucose, blood pressure (BP) and non-high-density lipoprotein (HDL) cholesterol]. Life's Essential 7 (LS7) was created in 2010 and was originally defined to include three health behaviours (diet quality, participation in physical activity and smoking exposure) and four health factors (BMI, fasting blood glucose, total cholesterol and BP levels). Each indicator includes three levels (poor, intermediate, ideal) and the criteria for each level are based on 'generally accepted clinical thresholds'. In the updated LE8 in 2022, new indicators about sleep quality and the original indicators have a more detailed calculation method. For example, the calculation of 'blood lipid' uses 'total cholesterol – HDL cholesterol' instead of 'lipid profiles' since non-HDL cholesterol can be measured in a non-fasting state and can be reliably calculated in all people. The score for each of the indicators in the LE8 has been increased compared with the LE7, with a maximum score of 100 (representing the best). The LE8 has a better ability to address intra- and interindividual differences

than Life's Simple 7 (LS7) [9]. The contribution of comprehensive cardiovascular health factors to SUA is debated [10–12]. The discrepancy may be due to study designs, sample sizes or confounding variables.

Whether comprehensive cardiovascular health factors (i.e. the LE8) has effect on HUA has not examined. Therefore this study aimed to investigate the relations between LE8 and the odds of HUA in the China Multi-Ethnic Cohort (CMEC). We also compared the different effects of the LE8 and LS7 on the odds of HUA.

Methods

Study design and participants

Details of the CMEC study have been described previously [13]. Briefly, the survey was conducted in 2018–2019 with 99 556 adults ages 30–79 years enrolled from Chongqing, Yunnan, Sichuan, Guizhou and Xizang in southwest China. The population in these five places includes Tibetans, Yi, Miao, Bai, Buoyi and Dong ethnic groups, in addition to the Han population that we included in our cohort. After identifying the region, we used multistage stratified cluster sampling to obtain samples from the community population. In the first stage, we selected one or two minority settlements for each ethnic group as our study sites, with special consideration given to settlements from plains, basins, rural areas and areas with high levels of air pollution in order to reflect the differences in geographic conditions. In the second phase, the local Centers for Disease Control and Prevention (CDC) selected one to eight communities per settlement (depending on the size of the community) and, most importantly, ethnic structure, taking into account that participants who met our inclusion criteria were invited to participate in our study. During the survey, well-trained health interviewers administered electronic questionnaires to obtain information on demographics, lifestyles (nicotine exposure, drinking habits) and diet. Fasting blood samples were collected. Rigorous quality control measures were implemented in the CMEC, including audio recordings of all interviews to detect data errors, electronic questionnaires to avoid skipping answers, interviews conducted by well-trained interviewers in the local dialect and a central server for managing data. The participants in the CMEC were from a diverse geographic area and had a diversity of diet habits, economic status and ethnic groups. Participants with liver cirrhosis (4429), incomplete information on LE8 or LS7 components ($n = 4405$) or missing data on SUA ($n = 909$) were excluded, leaving 89 823 participants for the primary analysis. All the participants provided written informed consent. Ethical approval was received from the Sichuan University Medical Ethical Review Board (K2020022, K2016038).

Assessment of SUA

Fasting blood samples were obtained and biochemical analyses of SUA were performed by the standard enzymatic method. We classified participants as having HUA if the SUA was >6.8 mg/dl.

Assessments of LE8 and LS7

The LE8 consisted of four healthy behaviours and four health factors [14] (Supplementary Table S1, available at *Rheumatology Advances in Practice* online): diet, physical activity, tobacco exposure, sleep duration, BMI, non-HDL cholesterol, glucose and BP. The score for diet was calculated using dietary components such as dairy products, fruits, vegetables, whole grains, legumes, processed meat and sodium. A modified Dietary Approaches to Stop Hypertension (DASH) index was used to score the participants' adherence to the DASH diet. Compared with the reference DASH index, we dropped the sweetened beverages component and nuts component, and additionally replaced the low-fat dairy component with total dairy products, because of limited information and very low regular consumption of them in our population. Moderate–vigorous physical activity (based on minutes of moderate or greater intensity activity per week; the longer the time, the higher the score; there are seven levels in total and the score increases by 20 points for every 30-min increase in time and 100 points for >150 min), tobacco use (current smokers, never smokers and former smokers were classified by years of abstinence, and the impact of 'living with a smoker' was considered) and sleep duration (average hours of sleep per night; either too much or too little sleep will affect the final score; 7–9 h of sleep time is 100 points) were collected by electronic questionnaires. BMI was calculated as weight divided by the square of the height (100 points: <25 kg/m^2; 70 points: 25.0–29.9; 30 points: 30.0–34.9; 15 points: 5.0–39.9; 0 points: ≥40.0). BP was measured three times (100 points: <120/<80 mmHg; 75 points: 120–129/<80; 50 points: 130–139 or 80–89; 25 points: 140–159 or 90–99; 0 point: ≥160 or ≥100; subtracting 20 points if the person is currently receiving relevant treatment). Non-HDL cholesterol was defined as total cholesterol – HDL cholesterol (100 points: <130; 60 points: 130–159; 40 points: 160–189; 20 points: 190–219; 0 points: ≥220). Blood samples were used to assess fasting blood glucose (FBG) and glycosylated haemoglobin A1c (HbA1c) (100 points: no history of diabetes and FBG <100 mg/dl or HbA1c <5.7%; 60 points: no diabetes and FBG 100–125 mg/dl or HbA1c 5.7–6.4%); 40 points: diabetes with HbA1c <7.0%; 30 points: diabetes with HbA1c 7.0–7.9%; 20 points: diabetes with HbA1c 8.0–8.9%; 10 points: diabetes with HbA1c 9.0–9.9%; 0 points: diabetes with HbA1c ≥10.0%). The LE8 scores were classified into low (<495), moderate (≥495–<590) and high (≥590). The components of the LS7 included diet, smoking exposure, physical activity, total cholesterol, BMI, blood glucose level and BP. Each individual component of the LS7 was divided into three levels: poor, intermediate and high (Supplementary Table S2, available at *Rheumatology Advances in Practice* online). LS7 scores ranged from 0–14: low (0–8), moderate (9–10) and high (11–14).

Covariate measurement

Covariates included age, sex, ethnic group, drinking habits (never, sometimes, usually and always), socio-economic status (SES), medication use, hepatic function [alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyl transferase (GGT)] and creatinine. SES was defined as income, educational attainment, occupational prestige and insurance [15]. The scoring algorithm for SES is shown in Supplementary Table S3, available at *Rheumatology Advances in Practice* online. Income (in Yuan) was divided into the following: <12 000, 12 000–19 999, 20 000–59 999, 60 000–99 999, 100 000–199 999 and ≥200 000. Education was categorized as no formal education, primary school, junior high school, high school, upper

high school and college education or above. Occupation was classified into four groups: unemployed, primary industry, secondary industry and tertiary industry. Higher values indicated higher SES status. SES had three levels: poor (<6), intermediate (≥6–<10) and high SES (≥10).

Statistical analyses

Distribution of the LE8 and LS7 scores among participants is shown in Supplementary Fig. S1, available at *Rheumatology Advances in Practice* online. Linear regression was used to compare continuous characteristics across levels of the LE8 (low, moderate and high) and LS7 (low, moderate and high). Characteristics between HUA and without HUA were compared by *t*-test for continuous variables and the chi-squared test for categorical variables.

Logistic regression was used to assess adjusted odds ratios (ORs) and 95% CIs for the independent relationships of the LE8 and LS7 with HUA. Potential confounders were included based on prior knowledge. We presented five sets of models to demonstrate the impact of key confounding variables. Model 1 was the crude model. Model 2 was adjusted for age, sex, ethnicity, drinking and SES. Model 3 was adjusted for model 2 plus ALT, AST and GGT. Model 4 was adjusted for model 2 plus creatinine. Model 5 was adjusted for model 2 plus ALT, AST, GGT and creatinine. We analysed the relations between each component of the LE8 or LS7 and the odds of HUA, with all components of the LE8 or LS7 simultaneously included for analyses. Restricted cubic spline (RCS) was used to model the non-linear association between the LE8 (continuous), LS7 (continuous) and HUA.

To assess the robustness of our results, we conducted sensitivity analyses. Some studies indicated that the concentration of SUA that was able to discriminate cardiovascular health status was 5.6 mg/dl [16]. Thus a sensitivity analysis for SUA was also conducted. Statistical analyses were performed using R version 4.2.2 (R Foundation for Statistical Computing, Vienna, Austria) and SPSS 26.0 for Windows (IBM, Armonk, NY, USA). Two-sided *P*-values <0.05 were considered statistically significant.

Results

Descriptive characteristics

Of the 89 823 participants (35 602 males, 54 221 females), 14 562 (16.2%) had HUA. The characteristics of the participants with or without HUA are shown in Supplementary Table S4, available at *Rheumatology Advances in Practice* online. Individuals who developed HUA tended to be older, male, have drinking habits, have unfavourable hepatic function, have higher levels of creatinine and to have a high SES. Compared with those who were in the low LE8 group, participants in the high LE8 group tended to be younger, female, of Han ethnicity, drink less and have favourable hepatic or renal function (Table 1).

Table 1. Characteristics of participants according to LE8 and LS7 status.

	LE8 score			LS7 score		
Characteristics	Low	Moderate	High	Low	Moderate	High
Participants, *n*	29 946	29 354	30 523	33 800	30 707	25 316
Age, years, mean	55.6	52.2	46.9	55.4	51.5	46.3
Women, %	61.1	61.7	80.1	43.2	63.6	79.4
Han ethnicity, %	55.9	56.2	63.0	55.9	56.8	63.7
Drinking, %						
Never	49.1	58.9	63.4	52.7	59.1	60.7
Sometimes or usually	28.8	29.7	31.2	27.4	29.7	33.6
Always	22.1	11.4	5.4	19.9	11.2	5.7
SES	6.99	7.07	7.36	6.95	7.08	7.47
Hepatic function, mean						
ALT, U/l	28.2	24.2	20.5	28.0	23.6	20.1
AST, U/l	28.6	26.8	24.7	28.4	26.5	24.6
GGT, U/l	54.7	35.7	25.4	52.6	34.3	24.9
Creatinine, μmol/l	75.0	69.4	64.7	74.2	69.0	64.5
Serum uric acids (μmol/l)	356.1	318.0	285.8	351.4	313.4	285.2

SES was a combination of household income, educational attainment, occupational prestige and insurance.

Association between levels of the LE8, LS7 and HUA

Table 2 shows the relationship between the levels of the LE8 and odds of HUA. After adjustment for age, sex and ethnicity, we found the odds of HUA decreased monotonously with increasing levels of the LE8. After adjustment for age, sex, ethnicity, drinking and SES, similar results were observed. The adjusted ORs were 1 (reference), 0.64 (95% CI 0.61, 0.67) and 0.38 (95% CI 0.36, 0.40) across low, moderate and high LE8 status, respectively ($P_{trend} < 0.001$). These results were almost unchanged after further adjustment for hepatic function, creatinine or both. The numerical results of the relationships between the LE8 score and HUA are presented in Supplementary Table S5, available at *Rheumatology Advances in Practice* online. We examined the non-linear relationship between the LE8 score and HUA using RCS models. Significant non-linear relationships were observed for the LE8 (Fig. 1) and LS7 (Fig. 2) and HUA. After being fully adjusted, we found the prevalence of HUA was negatively correlated with the LE8 and LS7. In addition, we added the reference line when the OR is 1, which means that when the score of the LE8 or LS7 exceeds this cut point value, the OR of HUA will be <1. When we analysed the individual factor of the LE8 and included all the metrics in the model (Supplementary Table S6, available at *Rheumatology Advances in Practice* online), we found that higher scores for physical activity, BMI, lipids and BP were protective factors for HUA, while nicotine exposure was a risk factor for HUA. However, individuals who had a high level of sleep health had no significant excess risk [1.01 (95% CI 0.96, 1.07)] after full adjustment. Similar results were observed in the LS7 analyses (Table 2, Supplementary Table S7, available at *Rheumatology Advances in Practice* online).

Supplementary Fig. S2, available at *Rheumatology Advances in Practice* online, shows the results of the area under the curve (AUC) analyses. According to the receiver operating characteristics curve, it can be deduced that the LE8 and LS7 had good discriminative performance for differentiating HUA (AUCs 0.638 and 0.635, respectively).

Stratified analyses

In the subgroup analyses (Table 3), the association with a high level of LE8 appeared greater in females [OR 0.32 (95% CI 0.29, 0.36), $P_{interaction} < 0.001$], in ethnic minority groups [OR 0.43 (95% CI 0.39–0.47), $P_{interaction} < 0.001$] and in those never drinking [OR 0.45 (95% CI 0.41, 0.49),

Table 2. ORs and 95% CIs for levels of LE8 and LS7 with the odds of HUA

Score	Low	Moderate	High	P_{trend}
LE8				
Cases, *n*	8158	4326	2078	
Adjusted for age, sex and ethnicity	1 (reference)	0.64 (0.61, 0.66)	0.37 (0.36, 0.40)	<0.001
Multivariable adjusted[a]	1 (reference)	0.64 (0.61, 0.67)	0.38 (0.36, 0.40)	<0.001
Multivariable adjusted[a] + hepatic function[b]	1 (reference)	0.70 (0.67, 0.73)	0.45 (0.42, 0.47)	<0.001
Multivariable adjusted[a] + creatinine	1 (reference)	0.64 (0.61, 0.67)	0.38 (0.36, 0.40)	<0.001
Multivariable adjusted[a] + all[c]	1 (reference)	0.70 (0.67, 0.73)	0.45 (0.42, 0.48)	<0.001
LS7				
Cases, *n*	8651	4189	1722	
Adjusted for age, sex and ethnicity	1 (reference)	0.61 (0.59, 0.64)	0.38 (0.36, 0.40)	<0.001
Multivariable adjusted[a]	1 (reference)	0.62 (0.59, 0.64)	0.38 (0.36, 0.41)	<0.001
Multivariable adjusted[a] + hepatic function[b]	1 (reference)	0.68 (0.65, 0.71)	0.45 (0.42, 0.48)	<0.001
Multivariable adjusted[a] + creatinine	1 (reference)	0.62 (0.59, 0.65)	0.39 (0.37, 0.41)	<0.001
Multivariable adjusted[a] + all[c]	1 (reference)	0.68 (0.65, 0.71)	0.46 (0.43, 0.49)	<0.001

[a] Adjusted for age, sex, ethnicity, drinking and SES.
[b] Hepatic function included ALT, AST and GGT.
[c] Adjusted for age, sex, ethnicity, drinking, SES, diuretics, ALT, AST, GGT and creatinine.

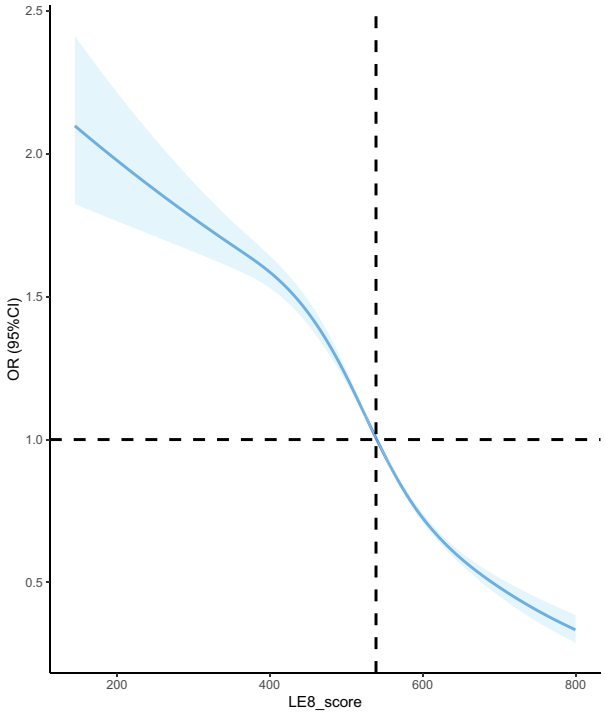


Figure 1. Non-linear relationship of LE8 score with HUA. In the RCS, the model was adjusted for age, sex, ethnicity, drinking, SES, diuretics, ALT, AST, GGT and creatinine. The ORs are shown in the solid blue line and 95% CIs in the shaded area. The threshold for having an OR for HUA <1.0 was >535 for LE8. The black horizontal dashed lines represent the reference line $y = 1$

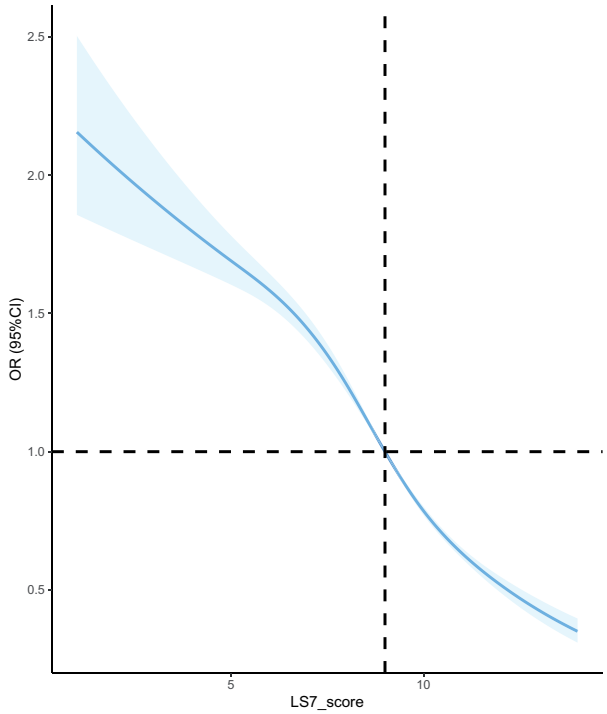


Figure 2. Non-linear relationship of LS7 score with HUA. In the RCS, the model was adjusted for age, sex, ethnicity, drinking, SES, diuretics, ALT, AST, GGT and creatinine. The ORs are shown in the solid blue line and 95% CIs in the shaded area. The threshold for having an OR for HUA <1.0 was >9.0 for LS7. The black horizontal dashed lines represent the reference line $y = 1$

$P_{interaction} < 0.001$]. The association with a high level of the LS7 appeared greater in females [OR 0.32 (95% CI 0.29, 0.36), $P_{interaction} < 0.001$] and in those never drinking [OR 0.45 (95% CI 0.41, 0.50), $P_{interaction} < .001$].

Sensitivity analyses

HUA was also defined as an SUA >7.0 mg/dl for men and 6.0 mg/dl for women. These data are not shown because the results were consistent with the presented results.

Discussion

In this large population-based study of 89 823 participants in southwest China, higher levels of the LE8 were associated with lower odds of HUA, independent of age, sex, ethnicity, drinking, SES, hepatic function and creatinine. Compared with those in the low LE8 level, a graded increased protection from HUA was observed from the moderate to high level of the LE8. Similar results were observed in the LS7 and HUA. Our study is a large epidemiological study examining the association between new cardiovascular health metrics

Table 3. ORs for HUA associated with LS7 and LE8 status, stratified by characteristics

Characteristics	LE8 score				LS7 score			
	Low	Moderate	High	$P_{interact}$	Low	Moderate	High	$P_{interact}$
Age group, years				<0.001				<0.001
<50	1.00 (ref)	0.71 (0.66, 0.77)	0.49 (0.45, 0.53)		1.00 (ref)	0.68 (0.64, 0.73)	0.49 (0.45, 0.53)	
≥50	1.00 (ref)	0.71 (0.67, 0.75)	0.48 (0.44, 0.52)		1.00 (ref)	0.70 (0.65, 0.74)	0.51 (0.46, 0.56)	
Sex				<0.001				<0.001
Male	1.00 (ref)	0.75 (0.71, 0.79)	0.57 (0.53, 0.61)		1.00 (ref)	0.73 (0.69, 0.77)	0.59 (0.55, 0.64)	
Female	1.00 (ref)	0.58 (0.53, 0.63)	0.32 (0.29, 0.36)		1.00 (ref)	0.57 (0.52, 0.63)	0.32 (0.29, 0.36)	
Ethnicity				<0.001				0.005
Han	1.00 (ref)	0.75 (0.70, 0.80)	0.47 (0.43, 0.50)		1.00 (ref)	0.71 (0.67, 0.76)	0.46 (0.43, 0.50)	
Ethnic minority groups	1.00 (ref)	0.65 (0.61, 0.70)	0.43 (0.39, 0.47)		1.00 (ref)	0.65 (0.60, 0.69)	0.47 (0.42, 0.42)	
Drinking				<0.001				<0.001
Never	1.00 (ref)	0.70 (0.65, 0.75)	0.45 (0.41, 0.49)		1.00 (ref)	0.66 (0.61, 0.71)	0.45 (0.41, 0.50)	
Sometimes or usually	1.00 (ref)	0.73 (0.67, 0.79)	0.49 (0.44, 0.54)		1.00 (ref)	0.71 (0.65, 0.77)	0.49 (0.44, 0.54)	
Always	1.00 (ref)	0.72 (0.65, 0.79)	0.49 (0.42, 0.57)		1.00 (ref)	0.73 (0.66, 0.80)	0.52 (0.45, 0.61)	
SES				0.07				0.03
Poor status	1.00 (ref)	0.28 (0.05, 1.00)	0.77 (0.24, 2.40)		1.00 (ref)	1.40 (0.41, 4.90)	0.72 (0.18, 2.80)	
Intermediate status	1.00 (ref)	0.64 (0.58, 0.70)	0.46 (0.41, 0.52)		1.00 (ref)	0.68 (0.62, 0.74)	0.46 (0.42, 0.50)	
High status	1.00 (ref)	0.75 (0.68, 0.83)	0.44 (0.39, 0.50)		1.00 (ref)	0.69 (0.62, 0.76)	0.47 (0.42, 0.54)	

Adjusted for age (as appropriate), sex (as appropriate), ethnicity (as appropriate), drinking (as appropriate), SES (as appropriate), diuretics, ALT, AST, GGT and creatinine.

(the LE8) and the odds of HUA. Our study reinforces the relations between the LE8 score, a new enhanced cardiovascular health metric by the AHA, and the odds of HUA.

In this study, the prevalence of HUA was 16.2%, which was higher than that of the healthy Italian population (6.3%) or hypertensive participants (7.3%) [17, 18]. One study reported that the HUA prevalence was 25% in East European hypertensive participants [4]. The prevalence of HUA in China ranges from 13.7 to 18.8% in different regions [5]. The reason for the high HUA prevalence may be attributed to SES and medical conditions. Notably, participants in our study were from less-developed ethnic minority regions. The mean value of SES was 7.14 (range 2–16) and this indicated relatively lower economic achievement in southwest China. Some participants in the current study were from the Yunnan-Guizhou Plateau and Qinghai-Xizang Plateau, where the prevalence of prehypertension and hypertension is very high. Hypertension or antihypertensive treatment might influence the levels of uric acid [19, 20]. Our study is also supported by a report from Yunnan, one of provinces in southwest China, showing the prevalence of HUA is 24.8% in the Bai ethnic group [21].

Although the potential mechanisms underlying the relation between cardiovascular health and HUA are not clear, there are several possible explanations. Both the LE8 and LS7 include diet, nicotine exposure, physical activity, BMI, blood glucose, lipids and BP. In our study, we found a high score for diet, physical activity, BMI, lipids and BP had beneficial effects on HUA. With changes in human dietary habits over the past few decades, the prevalence of HUA has been increasing annually [22, 23]. Increased SUA may be due to obesity, leading to increased expression of uric acid and/or decreased breakdown of uric acid [24]. Decreased insulin sensitivity might induce a reduction of urate excretion by stimulating renal tubular sodium–hydrogen exchange [25, 26]. BP stability, increasing bone density and type 2 immunity was related to uric acid metabolism [27–30]. Uric acid is metabolized from endogenous purine degradation and absorption of digested food [31]. In the liver, purines are transformed into hypoxanthine, xanthine and uric acid by xanthine oxidoreductase. Hepatocyte injury disrupts insulin signal transduction and could cause an elevation of purine production by activating the hexose monophosphate shunt [32]. In turn, elevation of SUA could increase the risk of fatty liver disease and induce an unfavourable lipid profile, which is one of the factors of the LE8 and LS7. Our results show that after additional adjustment for hepatic function, the association between cardiovascular health metrics and HUA was attenuated [OR 0.70 (95% CI 0.67, 0.73) *vs* 0.45 (0.42, 0.47) without and with additional adjustment for hepatic function, respectively; $P_{trend} < 0.001$].

In subgroup analyses, the association of cardiovascular health metrics with HUA was stronger in non-drinkers [OR 0.45 (95% CI 0.41, 0.49)]. This may suggest that the protection effect of ideal cardiovascular health metrics on HUA was more evident in non-drinkers. Alcohol is a potential risk factor for increased SUA. A relation between increased alcohol intake and HUA has been shown in many studies [33–37].

In our study, we did not observe that the LE8 had more power to predict HUA than the LS7. The LE8 had more advantages in explaining inter- or intra-individual variances precisely [9], as a newly added lifestyle factor, sleep health, also influences the effect on HUA. The results of the relation between sleep health and HUA are not consistent. A cross-sectional study showed 7–8 h of nocturnal sleep was associated with lower SUA concentrations [5], while longer nocturnal sleep duration was related to decreasing SUA concentrations in obese elderly participants [21]. Jin *et al.* [38] indicated a U-shaped association between sleep duration and cardiovascular health and confirmed 7 h of sleep duration was related to the lowest mortality risk, while some studies observed there was no association between sleep health and HUA [19]. In our study, we did not observe any association between nocturnal sleep duration and the odds of HUA. This was consistent with our previous study that longer daytime napping duration, but not night sleep duration, was independently associated with the risk of HUA in the Yunnan region [7].

The chief strengths of this study include the large sample size, high-quality data and consistent results in the sensitivity

analysis. However, there are some limitations. First, associations between the LE8, LS7 and HUA were cross-sectional, which limits the assessment of temporality. Second, although multiple potential confounding variables were considered, residual confounders that were unmeasured may occur. Third, some variables are self-reported data, such as sleep duration, dietary habits and physical activity, which may incur recall bias. Fourth, urate-lowering therapies and gout status could influence our results, but we lacked these variables and did not consider them in our analysis. Fifth, we do not expect biological effects of cardiovascular health metrics on HUA to differ by race, but our results may not be generalizable to other populations.

In conclusion, this large-scale epidemiological study indicated that a higher level of LE8 was associated with decreased odds of HUA. Similar results were observed in the LS7 analyses. The present findings support the application of the LE8 for assessing HUA risk. Our findings reinforce the importance of improving cardiovascular metrics.

Supplementary material

Supplementary material is available at *Rheumatology Advances in Practice* online.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Authors' contributions

Y.W., J.Y. and Q.M. were responsible for the study concept and design. X.Z., J.Z., Y.W., K.B., L.C., Y.D., T.Y., Y.F. and F.M. were responsible for the acquisition of data. Y.W. and J.Y. were responsible for the interpretation of data. Y.W. drafted the manuscript. J.Y., J.Z. and Y.W. were responsible for critical revision of the manuscript for important intellectual content. Y.W., Q.M. and X.Z. were responsible for statistical analysis. J.Y. and J. Z. were responsible for administrative, technical or material support. J.Y. was responsible for study supervision.

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