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Association Between Life's Essential 8 score and Changes in High-Sensitivity C-Reactive Protein: The Kailuan Prospective Cohort Study

Weizhe Li
Handan Central Hospital
Pei Liang
Handan Mingren Hospital
Lifeng Zhang
Handan Central Hospital
Xin Wang
Handan Central Hospital
Jinjian Zhang
Handan Central Hospital
Haijiao Zhang
Handan Central Hospital
Xiaohui Liu
Handan Central Hospital
Xueliang Ma
Handan Central Hospital
Shasha An
Handan Central Hospital
Yanxiu Wang
Kailuan General Hospital
Kangran Li
heblikr@163.com
Handan Central Hospital
Shouling Wu
Kailuan General Hospital

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Abstract

Life's Essential 8 (LE8) scores cardiovascular health (CVH) using four health behaviors and four health factors. The association between baseline CVH measured by LE8 and longitudinal changes in high-sensitivity C-reactive protein (hs-CRP) remains unclear. We investigated this in 85,911 northern Chinese participants (first exam 2006–2007, complete LE8 data, ≥ 2 hs-CRP measurements). Overall CVH score ranged 0–100 (low/moderate/high groups). Poisson regression assessed incident hs-CRP > 3 mg/L, calculating risk ratios (RRs) and 95% CIs. Over mean 10.32 years, 13,727 incident cases occurred. Baseline CVH level was significantly inversely associated with hs-CRP elevation. After multivariable adjustment, compared with low CVH, the RRs (95% CI) for incident hs-CRP > 3 mg/L were 0.962 (0.956–0.969) for moderate CVH and 0.925 (0.907–0.943) for high CVH. Subgroup analysis showed the health factors score had a more significant impact on longitudinal hs-CRP changes than the health behaviors score. Conclusion: Baseline CVH status based on LE8 is significantly inversely associated with longitudinal hs-CRP changes, with health factors playing a more pronounced role.

Introduction

Inflammation is one of the mechanisms by which the body defends against foreign biological invasion as well as physical and chemical injury.[1] However, persistent low-grade inflammation can lead to damage to the body's own tissues and organs.[2] Studies have confirmed that elevated levels of hs-CRP, a biomarker of inflammation, are associated with an increased risk of cardiovascular diseases.[3–6] Furthermore, among individuals with established cardiovascular diseases, higher hs-CRP levels have been shown to be positively associated with adverse prognoses,[7–9] whereas anti-inflammatory therapy can reduce the risk of adverse outcomes.[9,10] Traditional cardiovascular risk factors, including obesity, hypertension, dyslipidemia, and hyperglycemia, are all linked to unhealthy lifestyle habits.[11,12] Similarly, increased systemic inflammation is also associated with adverse lifestyle patterns.[13] Existing evidence has verified that physical inactivity, prolonged sedentary behavior, cigarette smoking, and dietary patterns are correlated with elevated hs-CRP levels.[14–18]

Although previous studies have investigated factors associated with elevated hs-CRP levels, most have focused on the association of a single factor with hs-CRP[14,15,17,19] and were predominantly cross-sectional in design.[20,21] Since an individual may be concurrently exposed to multiple determinants of hs-CRP, examining the effect of any single factor alone cannot truly and comprehensively reflect the overall profile of factors influencing hs-CRP. In 2022, the American Heart Association proposed LE8, a comprehensive indicator system for evaluating cardiovascular health.[22] To date, no longitudinal studies have explored the association between baseline LE8 score and longitudinal changes in hs-CRP over time. To fill this research gap, the present study was conducted using data from a community-based, real-world prospective cohort in China—the Kailuan Study[23] (Registration number: ChiCTR-TNC-11001489) to investigate the longitudinal association between baseline LE8 score and changes in hs-CRP.

Methods

Study Population

The Kailuan Study is a prospective cohort study initiated by Kailuan General Hospital and 11 affiliated hospitals. During 2006–2007, the first round of physical examinations was conducted among active and retired employees of the Kailuan Group, with a total of 101,510 participants enrolled (81,110 males and 20,400 females, aged 18–98 years). Subsequently, the 2nd, 3rd, 4th, 5th, 6th, and 7th waves of health examinations were performed biennially from 2008 to 2018. As the present study aimed to investigate the longitudinal association between baseline LE8 score and changes in hs-CRP levels, we included participants with complete baseline data and at least two hs-CRP measurements. To minimize potential confounding effects of the coronavirus disease 2019 (COVID-19) pandemic on study outcomes, all follow-up data on hs-CRP in this study were restricted to the 7th health examination conducted before the outbreak of COVID-19. The study was performed according to the guidelines of the Helsinki Declaration and was approved by the Ethics Committee of Kailuan General Hospital (Approval Number: 2006-05) and Handan Central Hospital (Approval Number: 2024-010). All participants agreed to take part in the study and provided informed written consent.

Inclusion and Exclusion Criteria

Inclusion Criteria: (1) Participants from the Kailuan Study cohort; (2) Individuals with intact cognitive function who were able to complete the questionnaires; (3) Participants who underwent health examinations and completed at least two subsequent hs-CRP measurements; (4) Individuals with complete baseline data and LE8 information; (5) Participants who provided written informed consent.

Exclusion Criteria: (1) Participants with any missing items in baseline data or LE8 components despite completing two or more hs-CRP measurements; (2) Individuals who declined to participate in the present study.Data Collection and Variable Definitions

Investigators and physical examiners involved in this study were all fixed staff members who had received standardized training, and all questionnaires and examinations were performed in strict accordance with the unified protocols established for the study. Face-to-face interviews were conducted by specially trained investigators to complete questionnaires item by item, including demographic characteristics, occupational status, lifestyle behaviors (sleep, smoking, alcohol consumption, physical activity, and diet), educational level, average monthly household income, medical history, and family history of diseases. physical examinations were performed to obtain data on blood pressure, height, weight, and waist circumference. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Smoking was defined as smoking at least one cigarette per day on average for at least one consecutive year in the past year. Alcohol consumption was defined as drinking at least 100 mL of liquor (alcohol content ≥ 50%) per day on average for at least one consecutive year in the past year. physical activity was defined as engaging in aerobic exercise such as walking, jogging, or ball games for at least 90 minutes per week. Data collection for epidemiological surveys and

biochemical measurements was performed with reference to previously published literature from our research group.[24]

LE8 Score:[22] The LE8 score was assessed according to the algorithm developed by the American Heart Association (AHA). In consideration of the actual settings of the Kailuan Study, definitions and scoring criteria for each component were adapted to establish the Kailuan Study version of the LE8 score (Supplementary Table 2). This version comprises four health behaviors (diet, physical activity, nicotine exposure, and sleep duration) and four health factors (BMI, blood lipids, blood glucose, and blood pressure). Studies employing this adapted version have been recognized in the field and widely applied in relevant research.[25–28] Each cardiovascular health behavior or factor was scored on a scale of 0–100. The overall and domain-specific CVH scores were calculated as the unweighted average of individual component scores, with a total score ranging from 0 to 100.

The dietary score was derived from the AHA-endorsed Dietary Approaches to Stop Hypertension (DASH-style) diet, which emphasizes intake of vegetables, fruits, nuts, legumes, whole grains, and restricted consumption of sodium, sugar-sweetened beverages, red meat, and processed meat. Considering epidemiological characteristics of the Chinese population and available data in the Kailuan Study, the dietary scoring algorithm was adjusted to include intake of salt, high-fat foods, and tea.[25–27] BMI cut-points were modified according to Chinese population criteria:[30] BMI < 23.0 kg/m^2: 100 points, BMI 23.0–24.9 kg/m^2: 75 points, BMI 25.0–29.9 kg/m^2: 50 points, BMI 30.0–34.9 kg/m^2: 25 points, BMI ≥ 35.0 kg/m^2: 0 points. Blood glucose score was determined based on fasting blood glucose (FBG) levels. Blood pressure score was evaluated according to systolic and diastolic blood pressure. For blood lipid scoring, non-high-density lipoprotein cholesterol (non-HDL-C), calculated as total cholesterol minus high-density lipoprotein cholesterol, was used as the indicator.

Participants were categorized into three groups based on total LE8 score:[26] Low CVH group: LE8 score ≤ 49 (n = 14,297), Middle CVH group: 50 ≤ LE8 score ≤ 79 (n = 70,069), High CVH group: LE8 score ≥ 80 (n = 1,545).

Laboratory-Related Tests

All participants were required to fast for at least 8 hours, and 5 mL of antecubital venous blood was collected between 7:00 and 9:00 a.m. Measurements of total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and fasting blood glucose (FBG) were completed within 4 hours. Blood glucose levels were measured using the hexokinase method, with reagents provided by Zhongsheng Beikong Biotechnology (Zhongsheng Beikong, Beijing, china), The CV for this method was less than 2%. All the above assays were measured by auto analyzer (Hitachi 747, Hitachi, Tokyo, Japan).

Measurement of High-Sensitivity C-Reactive Protein Levels

Serum hs-CRP levels were measured using the immunoturbidimetric method with a lower detection limit of 0.1 mg/L. From 2006 to 2009, the Ministry of Health's proficiency testing (Laboratory Proficiency Testing, PT) results for hs-CRP analysis in our laboratory were consistently 100%. Additionally, for each experimental sample, two measurements were conducted daily with a minimum interval of 2 hours, over a span of 20 days, to evaluate measurement precision. The results showed that the intra-assay coefficient of variation (CV) was 6.53%, the inter-assay CV was 4.78%, the intra-day CV was 6.61%, and the total CV was 9.37%. Consistent with previous studies,[20] hs-CRP ≤ 3.0 mg/L was classified as low risk, and hs-CRP > 3.0 mg/L was classified as high risk.

Follow-up

The follow-up started from the time when participants completed their first hs-CRP measurement during the initial health examination in 2006–2007. Follow-up assessments were conducted every two years, with the development of hs-CRP > 3 mg/L defined as the endpoint event. The follow-up was concluded upon completion of the 7th health examination in 2018–2019.

Statistical Analysis

Normally distributed continuous data are described using the mean and standard deviation (SD), compared with the one- way ANOVA. Skewed continuous data are summarized by the median and interquartile range, compared with the Wilcoxon rank-sum test. Categorical variables are described as frequencies and percentages, compared with the $\chi 2$ test. To address the missing values in the covariates, we employed a multiple imputation technique using chained equations. Generalized linear regression was applied to analyze the association between LE8 score and increases in hs-CRP levels. Poisson regression was used to investigate the longitudinal association between different LE8 score groups and incident elevated hs-CRP > 3 mg/L. Two multivariable adjusted models were constructed: Model 1, controlled for age and sex; Model 2, controlled for educational level, family income, drinking status and Follow-up time. Restricted cubic spline (RCS) regression with 3 knots placed at the 10th, 50th, and 90th percentiles of the LE8 score distribution was performed to flexibly model the association between continuous LE8 score and the relative risk of incident hs-CRP > 3 mg/L. The median LE8 score was used as the reference value. Likelihood ratio tests were used to evaluate the overall association and nonlinear trends.

Sensitivity analysis: To verify the robustness of the main findings, sensitivity analyses were conducted after excluding participants with hs-CRP > 10 mg/L (acute-phase inflammation) at the last follow-up, those with follow-up duration < 2 years, cancer patients, and those taking antihypertensive, lipid-lowering, or glucose-lowering medications.

All analyses were conducted using SAS (version 9.4) and R (version 4.4.2). A two-sided $P < 0.05$ was considered statistically significant.

Results

Baseline Characteristics of the Study Population

A total of 85 911 participants with complete baseline LE8 data and at least two hs-CRP measurements were included in the final analysis. The mean follow-up duration was 10.32 ± 3.27 years. Among them, 67 871 were male (79.00%) and 18040 were female (21.00%), with a mean age of 50.58 ± 11.94 years. Within the LE8 component scores, sleep duration score was the highest (87.36 ± 22.02), followed by fasting blood glucose score (85.57 ± 24.00), while dietary intake score was the lowest (38.73 ± 15.24). Regarding demographic characteristics, participants with higher LE8 scores were younger, had a higher proportion of females, higher educational attainment, and higher per capita monthly household income compared with those with lower LE8 scores. For chronic disease conditions, participants in higher LE8 score groups exhibited lower prevalence of cancer and lower proportions of antihypertensive, hypoglycemic, and lipid-lowering medication use, all with statistically significant differences ($P < 0.001$). Among all participants, the median hs-CRP level was 0.78 (0.30, 2.00) mg/L, and participants with higher LE8 scores had significantly lower hs-CRP levels than those with lower scores. From the low to high CVH groups, the median increments in hs-CRP concentration were 0.31 mg/L, 0.28 mg/L, and 0.30 mg/L, respectively, with significant between-group differences ($P < 0.001$). Furthermore, from baseline to the end of follow-up, the detection rates of hs-CRP > 3 mg/L increased from 18.58% to 25.75% in the low CVH group, 17.25% to 20.41% in the middle CVH group, and 11.39% to 14.50% in the high CVH group, with significant intergroup differences ($P < 0.001$). Data are presented in Table 1.

Table 1
Baseline characteristics according to baseline Life's Essential 8 cardiovascular health status

Characteristics	Overall	Low CVH (0–49 points)	Moderate CVH (50–79 points)	High CVH (80–100 points)	*P-value*
Participants, n (%)	85 911	14 297 (16.6)	70 069 (81.6)	1 545 (1.8)	
Age, years	50.58 ± 11.94	51.55 ± 10.32	50.50 ± 12.14	45.01 ± 14.79	<0.001
Men, n (%)	67 871(79.00)	13 330(93.24)	53 787(76.76)	754(48.8)	<0.001
Current drinker, n (%)	32 636(37.99)	8 939(62.52)	23 123(33.00)	574(37.15)	<0.001
High school or above, n (%)	17 689(20.59)	2 479(17.34)	14 319(20.44)	891(57.67)	<0.001
Income >1000 RMB, n (%)	5 697(6.63)	993(6.95)	4 438(6.33)	266(17.22)	<0.001
Tumor [n (%)]	290(0.34)	50(0.35)	231(0.33)	9(0.58)	0.2291
Use of antihypertensive medication, n (%)	9 463(11.01)	3 455(24.17)	5 970(8.52)	38(2.46)	<0.001
Use of antidiabetic medication, n (%)	2 013(2.34)	852(5.96)	1 154(1.65)	7(0.45)	<0.001
Use of lipid-lowering medication, n (%)	828(0.96)	303(2.12)	517(0.74)	8(0.52)	<0.001
Life's Essential 8 scores (out of 100 points)					
Mean total CVH score	59.29 ± 9.96	43.70 ± 5.11	61.95 ± 6.87	82.91 ± 3.23	<0.001
LE8 Behaviors score					
Mean diet score	38.73 ± 15.24	29.86 ± 14.27	39.91 ± 14.22	67.51 ± 15.53	<0.001

Abbreviations: CVH, cardiovascular health; Normally distributed continuous data are presented as (mean ± SD); IQR: interquartile range (P25-P75); LE8: Life's Essential 8; Age: age of participants at entry into the cohort; Non-HDL-C: non-high-density lipoprotein cholesterol; hs-CRP_1: high-sensitivity C-reactive protein at first examination; hs-CRP_2: high-sensitivity C-reactive protein at last examination; CRP_d: increase in hs-CRP concentration (follow-up minus baseline); Follow-up: Time from entry into the cohort to completion of the last hs-CRP measurement.

Characteristics	Overall	Low CVH (0–49 points)	Moderate CVH (50–79 points)	High CVH (80–100 points)	*P-value*
Mean physical activity score	53.33 ± 24.50	43.39 ± 29.79	54.92 ± 22.54	73.33 ± 26.15	<0.001
Mean tobacco/nicotine exposure score	63.30 ± 46.00	23.30 ± 38.97	70.81 ± 43.25	93.09 ± 22.91	<0.001
Mean sleep health score	87.36 ± 22.02	74.10 ± 28.21	89.89 ± 19.61	95.22 ± 12.86	<0.001
LE8 Factors score					
Mean body mass index score	67.65 ± 24.52	54.80 ± 22.19	69.80 ± 24.11	89.06 ± 17.99	<0.001
Mean blood lipids (non–HDL-C) score	73.10 ± 28.65	53.80 ± 28.80	76.60 ± 27.09	92.59 ± 17.08	<0.001
Mean blood glucose score	85.57 ± 24.00	70.55 ± 29.57	88.35 ± 21.65	98.09 ± 9.10	<0.001
Mean blood pressure score	46.38 ± 34.10	27.45 ± 27.43	49.39 ± 33.80	85.21 ± 25.42	<0.001
hs-CRP_1, median (IQR)	0.78(0.30,2.00)	1.00(0.40, 2.27)	0.73(0.29, 1.95)	0.50(0.20, 1.40)	<0.001
hs-CRP_2, median (IQR)	1.10(0.50, 2.60)	1.40 (0.60, 3.10)	1.10 (0.45, 2.50)	0.90 (0.43, 2.00)	<0.001
CRP_d	0.29 (-0.70, 1.40)	0.31 (-0.70,1.68)	0.28 (-0.70,1.37)	0.30 (-0.40,1.10)	<0.001
hs-CRP_1>3mg/L [n (%)]	14 922 (17.37)	2 657 (18.58)	12 089 (17.25)	176 (11.39)	<0.001
hs-CRP_2>3mg/L [n (%)]	18 205 (21.19)	3 682 (25.75)	14 299 (20.41)	224 (14.50)	<0.001
Follow-up	10.32 ± 3.27	9.94 ± 3.37	10.39 ± 3.24	10.77 ± 3.14	<0.001

Abbreviations: CVH, cardiovascular health; Normally distributed continuous data are presented as (mean ± SD); IQR: interquartile range (P25-P75); LE8: Life's Essential 8; Age: age of participants at entry into the cohort; Non-HDL-C: non-high-density lipoprotein cholesterol; hs-CRP_1: high-sensitivity C-reactive protein at first examination; hs-CRP_2: high-sensitivity C-reactive protein at last examination; CRP_d: increase in hs-CRP concentration (follow-up minus baseline); Follow-up: Time from entry into the cohort to completion of the last hs-CRP measurement.

Association between baseline LE8 score and increase in hs-CRP

Generalized linear models were applied to analyze the effect of LE8 score (per 10-point increment) on CRP_d.

After adjustment for age and sex in Model 1, LE8 score was inversely associated with CRP_d (β =−0.103, 95% CI: −0.146 to − 0.005, P < 0.001). After further adjustment for baseline hs-CRP, educational level, income, and drinking status in Model 2, this inverse association was strengthened (β = −0.203, 95% CI: −0.244 to − 0.162, P < 0.001). Stratified analyses showed that both the LE8 health behavior score and health factor score were inversely associated with CRP_d. Among individual health behavior components, sleep duration score and nicotine exposure score were significantly and inversely correlated with CRP_d, whereas dietary score and physical activity score showed no statistically significant differences (P > 0.05). Notably, in Model 1, the health factor score was positively associated with CRP_d (β = 0.046, P = 0.0003). After multivariable adjustment in Model 2, however, the health factor Score also became inversely associated with CRP_d (β = −0.065, P < 0.001). Data are presented in Table 2.

Table 2
Association between per 10-point increase in LE8 score and increase in hs-CRP

LE8 score	Model 1		Model 2	
	***β* (95% *CI*)**	***P*-value**	***β* (95% *CI*)**	***P*-value**
LE8 score	-0.103(-0.146, -0.005)	<0.001	-0.203(-0.244, -0.162)	<0.001
LE8 Behaviors score	-0.177(-0.213, -0.142)	<0.001	-0.143(-0.177, -0.109)	<0.001
Mean diet score	0.001(-0.025, 0.028)	0.9042	-0.024(-0.050, 0.001)	0.0609
Mean physical activity score	0.015(-0.001, 0.032)	0.0775	-0.002(-0.018, 0.013)	0.7376
Mean tobacco/nicotine exposure score	-0.075(-0.094, -0.057)	<0.001	-0.036(-0.054, -0.018)	<0.001
Mean sleep health score	-0.065(-0.075, -0.055)	<0.001	-0.054(-0.065, -0.043)	<0.001
LE8 Factors score	0.046(0.021, 0.072)	0.0003	-0.065(-0.089, -0.041)	<0.001
LE8: Life's Essential 8.				
Model 1 adjusted for age and sex.				
Model 2 further adjusted for baseline hs-CRP, education, income and drinking status on the basis of Model 1.				

Poisson Regression Analysis of Different Baseline LE8 Scores and Incident hs-CRP

> 3 mg/L

In this study, hs-CRP was used as the dependent variable (hs-CRP≤3mg/L coded as 1, hs-CRP > 3 mg/L coded as 2), and baseline CVH groups were used as independent variables. After excluding participants with baseline hs-CRP > 3 mg/L, Poisson regression was used to analyze the effect of different baseline LE8 scores on incident hs-CRP > 3 mg/L. After adjustment for age and sex in Model 1, using the low CVH group as the reference, the RR (95% CI) for incident hs-CRP>3mg/L were 0.962 (0.956–0.968) in the middle CVH group and 0.924 (0.906–0.942) in the high CVH group. After further adjustment for educational level, family income, drinking status and Follow-up time in Model 2, the corresponding RR (95%CI) were 0.962 (0.956–0.969) and 0.925 (0.907–0.943), respectively. When LE8 score was analyzed as a continuous variable (per 10-point increment), both models showed a significant inverse association

with the risk of incident hs-CRP > 3 mg/L, with RR (95%CI) of 0.978 (0.976–0.981) and 0.978 (0.976–0.981), respectively. Stratified subgroup analyses indicated differential effects of health behavior and health factor scores on hs-CRP levels, with a more pronounced effect observed for the health factor score (Table 3).

Table 3
Poisson regression analysis of baseline LE8 score and incident hs-CRP > 3 mg/L

hs-CRP$_2$>3mg/L	**Low CVH (0–49 points)**	**Moderate CVH (50–79 points)**	**High CVH (80–100 points)**	**Per 10-point decrease**	***P*-value**
LE8 score					
Cases, n (%)	2692 (23.12)	10852 (18.72)	183 (13.37)		
Model 1	Reference	0.962 (0.956–0.968)	0.924 (0.906–0.942)	0.978 (0.976–0.981)	<0.001
Model 2	Reference	0.962 (0.956–0.969)	0.925 (0.907–0.943)	0.978 (0.976–0.981)	<0.001
LE8 Behaviors score					
Cases, n (%)	4407 (20.25)	9139 (18.94)	181 (18.45)		
Model 1	Reference	0.981 (0.976–0.987)	0.958 (0.938–0.979)	0.991 (0.989–0.993)	<0.001
Model 2	Reference	0.982 (0.977–0.988)	0.960 (0.940–0.981)	0.992 (0.990–0.994)	<0.001
LE8 Factors score					
Cases, n (%)	2267 (24.02)	8396 (20.07)	3064 (15.54)		
Model 1	Reference	0.974 (0.967–0.981)	0.946 (0.938–0.954)	0.987 (0.986–0.989)	<0.001
Model 2	Reference	0.974 (0.967–0.981)	0.946 (0.938–0.953)	0.987 (0.968–0.989)	<0.001
LE8: Life's Essential 8. RR, relative risk; CI, confidence interval.					
Model 1 adjusted for age and sex.					
Model 2 further adjusted for education, income and drinking status follow-up on the basis of Model 1.					

Sensitivity Analysis

To verify the robustness of the main findings and exclude potential confounding effects on hs-CRP, sensitivity analyses were performed. Exclusion criteria included: hs-CRP > 10 mg/L at the last follow-up (indicating acute-phase inflammation), follow-up time < 2 years, history of cancer at baseline, and use of antihypertensive, hypoglycemic, or lipid-lowering medications at baseline. Results showed even more significant between-group differences in the increase in hs-CRP levels across CVH groups. The median increments in hs-CRP were 0.49 mg/L, 0.41 mg/L, and 0.36 mg/L in the low, middle, and high CVH groups, respectively, with statistically significant differences (P < 0.001). The detection rates of incident hs-CRP>3mg/L were 19.84%, 16.24%, and 11.52% in the three groups, respectively, also with significant differences (P < 0.001), which were consistent with the main analyses (Table 4).

Table 4
Progression of hs-CRP in different LE8 groups (sensitivity analysis)

Characteristics	**Overall**	**Low CVH (0–49 points)**	**Moderate CVH (50–79 points)**	**High CVH (80–100 points)**	***F*/X^2**	***P*-value**
Participants, n (%)	60 229	8 225 (13.7)	50 711 (84.2)	1 293 (2.1)		
hs-CRP_1	0.52 (0.21, 1.10)	0.66 (0.30, 1.27)	0.50 (0.20, 1.09)	0.40 (0.20, 0.94)	312.79	<0.001
hs-CRP_2	1.00 (0.42, 2.18)	1.20 (0.50, 2.50)	0.98 (0.40, 2.10)	0.80 (0.40, 1.81)	211.67	<0.001
CRP_d	0.42 (-0.20, 1.47)	0.49 (-0.20, 1.69)	0.41 (-0.20, 1.43)	0.36(-0.10, 1.20)	28.59	<0.001
hs-CRP_2>3mg/L [n (%)]	10 015 (16.63)	1 632 (19.84)	8 234 (16.24)	149 (11.52)	91.17	<0.001
Follow-up	10.61 ± 2.99	10.38 ± 3.00	10.64 ± 2.99	10.91 ± 2.96	33.77	<0.001

Abbreviations: hs-CRP_1: high-sensitivity C-reactive protein at first examination; hs-CRP_2: high-sensitivity C-reactive protein at last examination; CRP_d: increase in hs-CRP concentration (follow-up minus baseline); Follow-up: Time from entry into the cohort to completion of the last hs-CRP measurement.

Further Poisson regression analyses confirmed the above trend. Compared with the low CVH group, the RR (95% CI) for incident hs-CRP > 3 mg/L were 0.965 (0.958–0.972) in the middle CVH group and 0.929 (0.911–0.948) in the high CVH group. Subgroup analyses stratified by health behavior and health factors revealed that the influence of the health factor score on hs-CRP levels remained more prominent. (Table 5)

Table 5
sensitivity analysis

hs-CRP$_2$>3mg/L	Low CVH (0–49 points)	Moderate CVH (50–79 points)	High CVH (80–100 points)	Per 10-point decrease	*P*-value
LE8 score					
Cases, n (%)	1 632 (19.84)	8 234 (16.24)	149 (11.52)		
Model 1	Reference	0.964 (0.957–0.971)	0.927 (0.909–0.945)	0.980 (0.977–0.982)	<0.001
Model 2	Reference	0.965 (0.958–0.972)	0.929 (0.911–0.948)	0.980 (0.977–0.982)	<0.001
LE8 Behaviors score					
Cases, n (%)	3 039 (17.03)	6 866 (16.48)	110 (15.19)		
Model 1	Reference	0.987 (0.981–0.992)	0.961 (0.939–0.985)	0.993 (0.991–0.995)	<0.001
Model 2	Reference	0.989 (0.983–0.995)	0.964 (0.941–0.988)	0.994 (0.991–0.996)	<0.001
LE8 Factors score					
Cases, n (%)	1 247 (21.06)	6 201 (17.47)	2 567 (13.64)		
Model 1	Reference	0.972 (0.964–0.981)	0.946 (0.937–0.954)	0.987 (0.986–0.989)	<0.001
Model 2	Reference	0.972 (0.964–0.980)	0.945 (0.936–0.954)	0.987 (0.986–0.989)	<0.001

LE8, Life's Essential 8.

Model 1 adjusted for age and sex.

Model 2 further adjusted for education, income and drinking status follow-up on the basis of Model 1.

Poisson Regression Analysis of Individual LE8 Component Scores in Relation to Incident hs-CRP > 3 mg/L

Poisson regression was further applied to investigate the associations between individual LE8 component scores (per 10-point increment) and the risk of incident hs-CRP>3mg/L. Incident elevated hs-CRP >3mg/L was set as the dependent variable, and individual LE8 component scores as

independent variables. After adjustment for age, sex, educational level, family income, drinking status and Follow-up time, results were as follows. For health behaviors, higher physical activity score (RR = 0.998; 95% CI: 0.997–0.999) and higher nicotine exposure score (RR = 0.997; 95% CI: 0.997–0.998) were inversely associated with the risk of incident hs-CRP > 3 mg/L. For health factors, higher BMI score (RR = 0.994; 95% CI:0.993–0.995), higher non-HDL-C score (RR = 0.995; 95% CI: 0.994–0.996), and higher blood glucose score (RR = 0.997; 95% CI: 0.996–0.998) also showed significant inverse associations. (Table 6)

Table 6
Poisson regression analysis of the effect of a 10-point increase in each individual LE8 component score on incident hs-CRP > 3 mg/L

LE8 score	**Model 1**		**Model 2**	
	***RR* (95%*CI*)**	***P*-value**	***RR* (95%*CI*)**	***P*-value**
LE8 Behaviors score				
Mean diet score	0.998 (0.997-1.000)	0.1567	0.999 (0.997-1.000)	0.2756
Mean physical activity score	0.998(0.997–0.999)	0.0065	0.998(0.997–0.999)	0.0047
Mean tobacco/nicotine exposure score	0.998(0.997–0.998)	<0.001	0.997(0.997–0.998)	<0.001
Mean sleep health score	1.000 (0.998–1.001)	0.8174	1.000(0.998–1.001)	0.7914
LE8 Factors score				
Mean body mass index score	0.994(0.993–0.995)	<0.001	0.994(0.993–0.995)	<0.001
Mean blood lipids (non–HDL-C) score	0.995(0.994–0.996)	<0.001	0.995(0.994–0.996)	<0.001
Mean blood glucose score	0.997(0.996–0.998)	<0.001	0.997(0.995–0.998)	<0.001
Mean blood pressure score	0.999(0.998-1.000)	0.0811	0.999(0.998-1.000)	0.0888
LE8, Life's Essential 8; RR, relative risk; CI, confidence interval.				
Model 1 adjusted for age and sex.				
Model 2 further adjusted for education, income and drinking status follow-up on the basis of Model 1.				

Subgroup Analysis

Subgroup analyses stratified by covariates demonstrated heterogeneity in the inverse association between LE8 score and the risk of incident hs-CRP > 3 mg/L across different populations. The protective effect was significantly stronger in the female subgroup (RR = 0.966) than in the male subgroup (RR = 0.982). Notably, the association was not statistically significant in participants with diabetes (RR = 0.996, P = 0.992), whereas all other subgroups showed consistent inverse associations. (Table 7)

RCS analysis

Restricted cubic spline (RCS) regression analysis, after adjustment for confounding factors, revealed that the LE8 score was significantly negatively associated with the risk of incident hs-CRP > 3 mg/L (P-overall < 0.0001), with no evidence of a nonlinear trend (P-nonlinear = 0.9528). Figure 1

Disussion

Using data from the Kailuan Study cohort, we demonstrated that favorable health behaviors and health factors were significantly and inversely associated with systemic inflammation as reflected by hs-CRP levels, in a clear dose–response manner. Participants with higher levels of ideal health behaviors and health factors exhibited a substantially lower risk of developing incident hs-CRP>3mg/L over time. Furthermore, the strength of the association was more pronounced for health factors than for health behaviors.

First, the present study identified an inverse association between LE8 score and longitudinal progression of systemic inflammation. Over a mean follow-up of 10.32 years, the median increase in hs-CRP level among the overall study population was 0.29 mg/L. With rising LE8 score, the median increment decreased from 0.31 mg/L in the low-CVH group to 0.30 mg/L in the high-CVH group. Further generalized linear regression analyses revealed that after multivariable adjustment, each 10-point increment in baseline LE8 score was associated with a 0.203 mg/L reduction in the increase in hs-CRP level. Our previous research evaluating cardiovascular health using the Life's Simple 7 (LS7) criteria also supported this inference.[30] Over a mean follow-up of 4.1 years, participants who lost 4–6 ideal cardiovascular health metrics exhibited a significantly higher risk of elevated hs-CRP compared with those maintaining ideal metrics (men: HR = 1.69; 95% CI: 1.38–2.06; women: HR = 1.52; 95% CI: 1.13–2.04). In addition, a study based on the UK Biobank demonstrated that each one-standard-deviation increase in the Composite Inflammatory Lifestyle Score (CILS) was associated with a 22.6% higher hs-CRP concentration relative to the overall population mean after 2 years of follow-up (95% CI: 18.0%–27.2%).[31] Taken together with our current findings, these results consistently indicate that ideal health behaviors and factors are inversely associated with longitudinal changes in systemic inflammation.

We not only confirmed an inverse association between ideal health behaviors and factors and systemic inflammation, but also identified them as protective factors against incident hs-CRP>3mg/L. Results from Poisson regression showed that, after multivariable adjustment, participants in the middle and high CVH groups had 3.8% and 7.5% lower risks of incident hs-CRP>3mg/L, respectively, compared with those in the low CVH group. Restricted cubic spline analysis further revealed a linear dose–response

relationship between LE8 score and the risk of incident hs-CRP>3mg/L, with risk decreasing continuously as the score increased. To our knowledge, no similar studies have been published to date. A comparable investigation from the CARDIA cohort assessed cardiovascular health using the LS7 score among 4,405 young adults followed for up to 25 years. Compared with the group maintaining the highest cardiovascular health score trajectory, the lowest trajectory group exhibited a significantly elevated risk of CRP>3mg/L, with corresponding RR (95% CI) values of 2.58 (1.89–3.51) among Black participants and 7.20 (5.09–10.18) among White participants.[32] Although sleep was not included in that study, a longitudinal analysis of older adults from the English Longitudinal Study of Ageing (ELSA) reported that nighttime insomnia symptoms indirectly influenced hs-CRP levels by reducing positive affect over 4 years of follow-up (indirect effect $\beta = 0.006$, $p < 0.05$).[33] Other single-factor longitudinal studies have also demonstrated positive associations of baseline body mass index, hypertension,[19] and tobacco exposure[14] with CRP progression, whereas Mediterranean dietary patterns[15,16] and increased physical activity[17,34] have been shown to reduce hs-CRP levels. The present study extends these findings by indicating that maintaining ideal cardiovascular health status serves as a protective factor against elevations in hs-CRP.

Furthermore, we observed differential magnitudes of effect between health behaviors and health factors on the risk of incident hs-CRP>3mg/L, with health factors showing a stronger impact than behavioral factors. Compared with the low CVH group, the middle and high CVH groups exhibited 2.6% and 5.4% reductions in the risk of incident hs-CRP>3mg/L, respectively, whereas corresponding reductions in the health behavior score subgroups were 1.8% and 4.0%. Similar observations have been reported in previous studies.[20,21] For instance, the SCAPIS study showed that the association of health factors with hs-CRP>3mg/L was stronger than that of health behaviors: relative to the high CVH group, the OR (95% CI) for health factors in the low CVH group was 3.0 (2.7–3.3), while that for behavioral factors was only 1.8 (1.6–2.0).[20] Although such disparities between behavioral and factor-related effects have been noted, the underlying mechanisms remain unclear. We propose two potential explanations: □ Behavioral measures were derived from self-reported data, which may be subject to recall bias, whereas health factors were based on objectively measured indicators with standardized quality control, yielding more reliable results. □ The influence of health behaviors on inflammatory levels may be mediated indirectly through improvements in glucolipid metabolism and blood pressure. Consequently, favorable behaviors may not fully attenuate inflammation in the presence of persistent metabolic abnormalities, whereas favorable metabolic profiles may directly suppress inflammatory pathways.[35,36]

As a nonspecific inflammatory marker, hs-CRP levels are susceptible to confounding by multiple factors. To minimize study bias, we performed sensitivity analyses by excluding individuals with hs-CRP>10mg/L at the last follow-up (indicating acute inflammation), follow-up duration < 2 years, confirmed cancer at baseline, or use of antihypertensive, antidiabetic, or lipid-lowering medications. Results from sensitivity analyses revealed an even stronger inverse association between LE8 score and median increase in hs-CRP. In addition, although baseline characteristics showed that risk factors such as age and male proportion declined with increasing CVH category, the association between LE8 score and the risk of

incident hs-CRP>3mg/L remained statistically significant after multivariable adjustment, consistent with our main findings. We further conducted subgroup analyses across diverse populations. Except for the diabetes subgroup, in which the association was not statistically significant, results from all other subgroups were consistent with the main analysis. Notably, the protective effect of higher LE8 score against incident hs-CRP>3mg/L was significantly stronger in women. This sex disparity may be attributed to the anti-inflammatory and vascular protective effects of estrogen. Estrogen not only helps maintain vascular elasticity and integrity but also improves endothelial function, increases nitric oxide production, enhances vasodilation, and strengthens anti-inflammatory capacity.[37]

Analysis of individual component scores revealed that baseline BMI, blood glucose, and non-HDL-C scores exerted significant effects on incident hs-CRP>3mg/L. Among them, BMI score showed the strongest association: each 10-point increase in BMI score was associated with an approximately 0.6% lower risk of incident hs-CRP>3mg/L. A previous longitudinal study of 6,814 participants similarly reported that BMI was more strongly associated with hs-CRP than other metabolic indicators.[38] Findings from the Hamburg City Health Study (HCHS) cohort further supported this pattern.[39] In middle-aged European adults, hs-CRP concentrations rose stepwise with an increasing number of adverse lifestyle factors, and overweight exhibited the strongest association with hs-CRP (β = 0.68, 95% CI: 0.63–0.73). Mechanistically, obesity promotes a pro-inflammatory phenotypic shift in immune cells, triggers local inflammatory responses in adipose tissue, and impairs the function of anti-inflammatory cells. Activated immune cells subsequently secrete pro-inflammatory cytokines including TNF-α, IL-2, and IL-6, which aggravate systemic inflammation through both local and systemic pathways.[40] Therefore, weight management may be regarded as a central modifiable target for slowing the progression of low-grade chronic inflammation.

Cigarette smoking induces systemic oxidative stress, promotes the phenotypic activation of lymphocytes, and triggers inflammatory signaling cascades via NF-κB activation, thereby elevating systemic inflammation.[41] Hyperglycemia and dyslipidemia impair nitric oxide bioavailability and induce endothelial dysfunction, which may further contribute to elevated hs-CRP levels.[42] Conversely, increased dietary fiber intake and adequate sleep improve insulin sensitivity and markedly enhance the expression of endogenous antioxidants. These effects may attenuate telomere attrition, alleviate oxidative stress and mitochondrial dysfunction, and consequently reduce inflammatory status.[43–45] Notably, dietary and sleep scores showed no statistically significant associations in the present study, which may be attributed to differences between the scoring system applied herein and the AHA criteria. Obesity and high-fat diets drive proinflammatory phenotypic switching of immune cells within adipose tissue, generating reactive oxygen species and releasing inflammatory mediators including TNF-α and IL-6, thereby exacerbating systemic inflammation.[42,46] Regular physical activity mitigates inflammatory and oxidative stress signaling in vascular tissue, enhances the bioavailability of antioxidant enzymes and nitric oxide, improves endothelial function and arterial stiffness, and ultimately reduces inflammatory burden.[47] Although hypertension is linked to inflammation, its precise underlying mechanisms remain incompletely elucidated.[48] Collectively, these pro-inflammatory and anti-inflammatory pathways may

partially account for the inverse association between higher LE8 scores and hs-CRP progression observed in the present study.

Strengths and Limitations

This was a large-sample cohort study that confirmed the association between LE8 score and changes in hs-CRP. However, several limitations should be acknowledged. First, participants were grouped according to baseline LE8 score at study entry, and dynamic changes in LE8 during follow-up and their potential impact on hs-CRP were not considered. Nonetheless, our previous research showed that CVH status assessed by LS7 remained relatively stable over a 4-year period. In addition, ideal dietary intake was not precisely evaluated in this study; instead, daily salt intake was used as a surrogate indicator. The relatively imprecise method for estimating salt intake may have influenced the study results. Second, hs-CRP levels can be affected by multiple factors, which may compromise the accuracy of the findings. However, the large sample size and sensitivity analyses restricted to major influencing factors further confirmed the robustness of our results. Third, the study population was predominantly male, which may limit the generalizability of the findings to other populations. Nevertheless, subgroup analyses were performed, and inverse associations between LE8 score and hs-CRP progression were observed in both men and women.

Conclusion

This study demonstrated that in the general population of northern China, the baseline LE8 score was significantly negatively associated with longitudinal changes in hs-CRP levels, with the health factor score showing a more pronounced effect. As a measure of CVH, LE8 may help identify individuals at high risk of elevated inflammation levels at an early stage.

Declarations

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Competing interests

The authors declared no conflicts of interest in this work.

Author Contribution

K.L. and S.W. contributed to the conception or design of the work. W.L., L.Z., X.W., J.Z., H.Z., X.L., X.M., S.A., and Y.W. contributed to the acquisition, analysis, or interpretation of data for the work. W.L. and P.L.

drafted the manuscript. S.W. critically revised the manuscript. All gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

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Data Availability

The datasets generated and/or analysed during the current study are not publicly available due to ethical and privacy restrictions, as they contain participant health information protected under the informed consent agreements. Relevant data supporting the findings are available from the corresponding author upon reasonable request.

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Table

Table 7 is available in the Supplementary Files section.

Figures

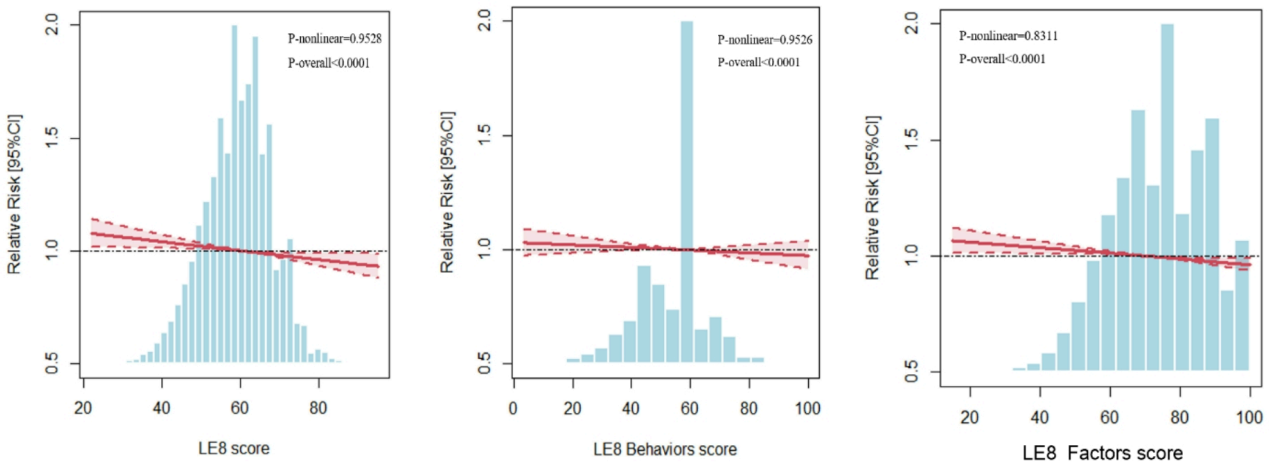


Figure 1

Histogram showing the distribution of LE8 scores within the population.

LE8, Life's Essential 8; RR, relative risk; CI, confidence interval.

Model further adjusted for age and sex, education, income drinking status and follow-up.

Figure legend: The solid red line represents the adjusted relative risk, the red shaded area indicates the 95% confidence interval, the black dashed line denotes the reference line at RR = 1.0, and the light blue histogram shows the distribution of LE8 scores in the study population. All models were adjusted for age, sex, education, income, drinking status and follow-up.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Table7.docx
- SupplementalMaterials.pdf