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Life's essential 8 and risk of subclinical atherosclerosis progression: a prospective cohort study

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ABSTRACT

BACKGROUND Previous studies have demonstrated the benefits of ideal cardiovascular health (CVH) in reducing cardiovascular risk. However, its role in subclinical atherosclerosis (SA) progression remains unclear. We aim to examine the association of CVH, estimated by the American Heart Association's new Life's Essential 8 (LE8), with the progression of SA.

METHODS This prospective cohort study was conducted among 972 asymptomatic Chinese participants and followed up for 5.7 years. The LE8 score (range, 0–100) consisted of blood pressure, lipids, glucose, body mass index, smoking status, diet health, physical activity and sleep health was evaluated in 1998 and 2008–2009. Progression of SA was determined by carotid plaque and coronary artery calcification (CAC) in 2008–2009 and 2013–2014. Log-binomial regression model was used to estimate the association of LE8 score with SA progression.

RESULTS Each 10 points increment in LE8 score was associated with 15.2% (RR: 0.848, 95% CI: 0.797–0.902), 17.7% (RR: 0.823, 95% CI: 0.766–0.884) and 12.0% (RR: 0.880, 95% CI: 0.845–0.916) lower risks of carotid plaque, CAC and overall SA progression, respectively. Compared with participants with non-ideal CVH at both visits, the participants with ideal CVH at both visits had 39.1% (RR: 0.609, 95% CI: 0.494–0.752), 41.0% (RR: 0.590, 95% CI: 0.456–0.764) and 29.7% (RR: 0.703, 95% CI: 0.598–0.825) lower risks of carotid plaque, CAC and overall SA progression, respectively.

CONCLUSIONS Higher LE8 scores were associated with lower risks of SA progression. Besides, long-term maintenance of optimal CVH was more beneficial to prevent SA progression.

Atherosclerotic cardiovascular disease (ASCVD) remains the major contributor to premature mortality and disability, responsible for 327.7 million disability-adjusted life-years globally in 2019.[1,2] In China, the burden of ASCVD kept growing sharply in the past decades due to lifestyle change, population ageing, environmental pollution and so on.[3,4] The initiation and development of ASCVD involve chronic, multistep and complex pathophysiological processes, among which atherosclerosis is a crucial link, and it is affected by many factors including genetic and traditional risk factors.[5,6]

Atherosclerosis has decades-long preclinical phase and may lead to severe health consequences once clinical events occur, thus early detection of atherosclerosis using non-invasive methods may help to identify individuals at risk for atherosclerotic cardi-

ovascular events and subsequently provide them with targeted preventive measures.[7] As well-established surrogates of subclinical atherosclerosis (SA), carotid plaque and coronary artery calcification (CAC) are of increasing clinical significance.[8-10] Population-based studies have also provided compelling evidence for associations of carotid plaque and CAC with the risk of ASCVD.[11-14]

In 2010, the American Heart Association (AHA) issued a report to state the definition and quantification of cardiovascular health (CVH) metrics (Life's Simple 7, LS7) with the goal of CVH promotion,[15] and numerous studies have proved the benefits of keeping ideal CVH in diverse populations subsequently.[16,17] In 2022, the AHA updated it to the Life's Essential 8 (LE8) score by adding sleep indicator and using different scoring method.[18] To date, several studies have evaluated the association between CVH and progression of atherosclerosis,[19-22] while none of them assessed the protective impacts of CVH on progression of atherosclerosis across various vessel beds. In addition, the benefits of long-term maintenance of ideal CVH have not been fully illustrated. To fill this gap, we performed the current study to examine the association of CVH, evaluated by the AHA's new LE8 metrics, with SA progression in a sub-cohort of middle-aged Chinese adults.

METHODS

Study Population

All participants were residents of Beijing and derived from the Prediction for Atherosclerotic Cardiovascular Disease Risk in China (China-PAR) project. Detailed study design and method of the China-PAR project have been published elsewhere.[23] In this study, participants who attended the interview of cardiovascular risk factors in 1998 comprised the baseline visit population, and the follow-up visits and SA examinations were performed in 2008–2009 and 2013–2014.

In the main analysis, 2008–2009 visit was used as the baseline because the first atherosclerosis measurement was performed in 2008–2009. Of the 1462 participants who underwent examinations for SA including carotid ultrasound and cardiac computed tomography during 2008–2009, 192 participants with a history of ASCVD at entry or inadequate information on CVH metrics were excluded. A total of 298 participants were further excluded in the analysis regarding to atherosclerosis progression because of loss to follow-up and the final analysis was limited to 972 participants (Figure S1). In the analysis of the association of changes in CVH with SA progression, 25 participants with missing data on CVH in 1998 were further excluded and 947 participants were included. The China-PAR project was approved by the Institutional Review Board at Fuwai Hospital in Beijing. All participants provided written informed consents before baseline examination. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline[24].

Data Collection

A standardized questionnaire was used to obtain demographic characteristics (including age, gender and educational background), medical history (including physician-diagnosed hypertension, diabetes mellitus and hyperlipidemia), smoking status, alcohol intake, physical activity, sleep health, habitual dietary intake, and ASCVD family history of each participant. Information on use of antihypertensive, lipid-lowering and antidiabetic medications within the past two weeks prior to the visit was also collected. Height, weight, and blood pressure (BP) were measured by trained staff. Height and weight were measured twice and the average of two measurements was used to calculate body mass index (BMI) as kilograms divided by height in squared meters. Systolic and diastolic BP were defined as the average of three BP measurements. Blood samples were drawn from the antecubital vein after at least a 10-h fasting to measure glucose and lipids levels.

Determination of Cardiovascular Health

The CVH of each participant was estimated by the AHA's LE8 score. The 8 components of LE8 score, including 4 health behaviors (diet health, physical activity, smoking status and sleep health) and 4 health factors (BMI, blood lipids, blood glucose, and BP) were quantified according to the AHA's definition. Habitual dietary intake, physical activity and sleep health were evaluated by self-reported in-

formation. A modified healthy diet score for Chinese was defined in accordance with the Dietary Guidelines for Chinese Residents, including consumption of fruits/vegetables, fish, soybean products, red meat and tea.[25] Habitual dietary intake included daily intake of 5 typical diet items assessed by a standard food frequency questionnaire. Physical activity was defined as minutes spent on light/moderate/vigorous physical activity per week over the previous 12 months. Smoking status was measured by cigarettes consumption and years quitting smoking and sleep health was measured by average hours of sleep per night. Non-high-density lipoprotein cholesterol (non-HDL) was calculated as total cholesterol minus high-density lipoprotein cholesterol. Each metric ranged from 0 to 100 points, and the overall LE8 score for each participant was calculated as the unweighted average of all 8 component metric scores. The participants were categorized into 3 CVH levels as the AHA recommended: (1) overall LE8 scores of 80 to 100 were considered as high CVH; (2) 50 to 79, moderate CVH; (3) 0 to 49, low CVH. Since the median LE8 score of participants in this study was about 60, we further stratified participants into 3 levels according to their LE8 score from 1998 survey to 2008–2009 survey: (1) participants retained LE8 score < 60 at both visits (low-stable group); (2) participants with LE8 score < 60 at either of the two visits (unstable group); (3) and participants retained LE8 score ≥ 60 at both visits (high-stable group). The definition and scoring for each component metric of LE8 was showed in Supplementary Table S1.

Definition of Subclinical Atherosclerosis

Carotid ultrasound was conducted using an iE33 ultrasonography (Philips Healthcare, Netherland) with 7.5-MHz linear-array transducers by experienced sonographers, according to a standardized scanning protocol. A plaque was defined as any focal protrusion of a carotid intima-media thickness (cIMT) value ≥ 1.5 mm or > 50% thicker than the adjacent cIMT by visual assessment.[9] Carotid plaque score was determined by the total number of plaques detected in each segment of bilateral carotid artery, including common carotid artery, the carotid bifurcation, and the internal carotid artery.[26] CAC was measured with a 64-row LightSpeed VCT scanner (GE Healthcare, Milwaukee, WI, USA). CAC score (CACS) was calculated using the Agatston method for each major epicardial vessel (left main, left anterior descending, left circumflex and right coronary artery), and total CACS was calculated as the sum of CACS of four epicardial vessels.[27]

Progression of carotid plaque was defined as an increase in plaque score between baseline and follow-up measurements ($plaque_{T1} - plaque_{T0} \geq 1$). Progression of CAC was defined as difference of square root transformed CACS ≥ 2.5 ($\sqrt{CACS_{T1}} - \sqrt{CACS_{T0}} \geq 2.5$).[28] Progression of overall SA was defined as the presence of significant progression of either carotid plaque or CAC. Log changes in carotid plaque and CACS were calculated as log-transformed changes between two measurements[29]. For example, participants with negative change of CACS were treated as having no CACS change, and CAC changes of other participants were calculated using the following formula: log CACS change = $\log_e(CACS_{T1} - CACS_{T0} + 1)$.

Statistical Analysis

Demographic characteristics and cardiovascular risk factors by CVH levels were presented as mean ± SD or median (interquartile range) for continuous variables and number of subjects with percentage for categorical variables. Differences in means or percentage among groups were examined with analysis of variance or chi-square test, respectively.

Since the incidence of outcomes (progression of carotid plaque and CAC) in this study were greater than 10%, odds ratios would overestimate the risks. Thus, log-binominal regression models were used to estimate relative risk (RR) and corresponding 95% confidence interval (CI) for progression of atherosclerosis associated with CVH (estimated by LE8 score). Also, we used multivariable linear regression models to examine the relationship between CVH and changes of carotid plaque and CACS for sensitivity analysis. To fit model assumptions, log changes in carotid plaque and CACS were used as outcome variables. LE8 score was categorized into three levels and the level of low CVH was considered as the reference. Tests for trend of effect sizes across CVH levels were performed by entering the median value of each category as a continuous variable in the models. We examined the possible nonliner dose-response relationship between CVH and

atherosclerosis progression with restricted cubic splines. The main models were adjusted for baseline age, gender, family ASCVD history, alcohol intake and levels of educational attainment. To avoid over-adjustment, cardiovascular risk factors included in CVH metrics (e.g., anti-hypertensive and lipid-lowering therapy) were not adjusted as covariates in the main models. Additional subgroup analyses stratified by age group (< 55 and ≥ 55 years), gender (men and women) and educational level (less than high school and high school or above) were performed to examine the potential effect modification by baseline characteristics and multiplicative interactions were tested.

To assess the effect of CVH change on the risk of atherosclerosis progression, we divided participants into 3 groups and the low-stable group was considered as the reference. Tests for trend of effect sizes across groups were performed by entering the group as numeric variable in the models. Log-binominal regression models were used to estimate the RR and corresponding 95% CI for SA progression associated with CVH change and the same covariates as main model were adjusted. Subgroup analysis was performed and the characteristics used for stratification were described above.

All statistical analyses were performed using R statistical software (Version 4.0.0, R Project for Statistical Computing). *P* values are two-sided and significant at < 0.05.

RESULTS

Baseline Characteristics

Baseline demographic characteristics of 972 participants are summarized in Table 1, stratified by CVH levels. Participants with a mean (SD) age of 53.9 (6.5) years at baseline were followed for an average of 5.7 ± 0.5 years. At baseline, 108 (11.1%) participants had high CVH, 722 (74.3%) had moderate CVH and 142 (14.6%) had low CVH. Participants with higher levels of CVH had a lower prevalence of hypertension, diabetes mellitus and dyslipidemia. What's more, participants with higher levels of CVH at baseline are more likely to have a higher educational level than those with low CVH.

Figure 1 displays the mean score and standard error of each component of LE8 score for participants overall and by sex and age group. Mean score of diet was lowest and sleep was highest for overall participants. Women had extremely higher score for smoking status than men and generally similar scores for BMI, blood lipids and sleep health. Compared with individuals younger than 55 years old, those aged over 55 years old had lower scores for BMI, blood glucose, blood lipids, BP and physical activity, and higher scores for smoking status.

Association of CVH with Progression of Subclinical Atherosclerosis

As shown in Table 2 and Figure 2, higher LE8 scores were significantly associated with lower risks of carotid plaque and CAC progression after adjusting covariates, with RRs of 0.848 (95% CI: 0.797–0.902) and 0.823 (95% CI: 0.766–0.884) for each 10 points increment in LE8 score, respectively. Moreover, compared with participants with low CVH, those with high CVH had lower risks for carotid plaque and CAC progression, with RRs of 0.324 (95% CI: 0.181–0.578) and 0.286 (95% CI: 0.137–0.598) respectively. For overall SA progression, the RR for each 10 points increment in LE8 score was 0.880 (95% CI: 0.845–0.916), and RR for high level of CVH was 0.354 (95% CI: 0.226–0.554) compared with low CVH level. As shown in Supplementary Table S2, similar results were observed when progression of carotid plaque and CACS were treating as continuous variables. As shown in Supplementary Table S3, all LE8 metrics were negatively associated with risk of atherosclerosis progression.

We further conducted subgroup analyses stratified by age, gender and educational level at baseline (Supplementary Table S4). We observed a more pronounced effect of CVH on progression of plaque in women than in men, with multivariable-adjusted RRs of 0.820 (95% CI: 0.749–0.897) and 0.928 (95% CI: 0.858–1.003) for each 10 points increment in LE8 score, respectively ($P_{interaction}$ = 0.042). In addition, the association between CVH and atherosclerosis progression was stronger in participants with educational levels of high school or above than those with lower educational levels ($P_{interaction}$ = 0.003).

Association of CVH Change with Atherosclerosis Progression

We further assessed the association of long-term

Table 1 Baseline distribution of characteristics and cardiovascular risk by CVH levels.

Baseline characteristics	Overall (*n* = 972)	CVH levels			*P*-value
		Low CVH (*n* = 142)	Moderate CVH (*n* = 722)	High CVH (*n* = 108)	
Male	445 (45.8%)	106 (74.6%)	323 (44.7%)	16 (14.8%)	< 0.001
Age, yrs	53.9 ± 6.5	55.3 ± 6.8	54.0 ± 6.4	51.1 ± 5.8	< 0.001
BMI, kg/m^2	25.9 ± 3.3	27.4 ± 3.1	25.9 ± 3.3	23.7 ± 2.8	< 0.001
SBP, mmHg	133.5 ± 18.3	145.2 ± 19.0	133.3 ± 17.3	119.9 ± 13.3	< 0.001
DBP, mmHg	83.3 ± 10.6	89.6 ± 10.8	83.2 ± 10.1	75.9 ± 9.1	< 0.001
Total cholesterol, mg/dL	200.6 ± 34.4	217.1 ± 36.2	199.4 ± 33.3	186.2 ± 31.3	< 0.001
LDL-C, mg/dL	119.4 ± 29.3	129.9 ± 32.2	119.5 ± 28.2	106.8 ± 28.4	< 0.001
HDL-C, mg/dL	51.8 ± 12.0	47.7 ± 11.1	51.4 ± 11.4	59.6 ± 13.2	< 0.001
Triglycerides, mg/dL	198.3 (42.6)	178.0 (145.1)	122.7 (87.9)	89.7 (48.8)	< 0.001
Fasting glucose, mg/dL	102.6 ± 28.9	116.4 ± 37.5	102.0 ± 27.9	88.7 ± 6.4	< 0.001
Remnant cholesterol, mg/dL	28.1 ± 14.0	36.9 ± 16.4	27.7 ± 13.3	19.8 ± 8.7	< 0.001
Hypertension	308 (31.7%)	79 (55.6%)	221 (30.6%)	8 (7.4%)	< 0.001
Diabetes	93 (9.6%)	24 (16.9%)	69 (9.6%)	0	< 0.001
Hyperlipidemia	114 (11.7%)	30 (21.1%)	78 (10.8%)	6 (5.6%)	< 0.001
Antihypertensive therapy	246 (25.3%)	59 (41.5%)	181 (25.1%)	6 (5.6%)	< 0.001
Lipid-lowering therapy	50 (5.1%)	12 (8.5%)	35 (4.8%)	3 (2.8%)	0.103
Drinking	361 (37.1%)	83 (58.5%)	249 (34.5%)	29 (26.9%)	< 0.001
High school education or above	396 (40.7%)	51 (35.9%)	280 (38.8%)	65 (60.2%)	< 0.001
Current smoking	310 (31.9%)	105 (73.9%)	203 (28.1%)	2 (1.9%)	< 0.001
Family history of ASCVD	521 (53.6%)	79 (55.6%)	379 (52.5%)	63 (58.3%)	0.457

Data are presented as mean ± SD, median (IQR) or *n* (%). ASCVD: arteriosclerotic cardiovascular disease; BMI: body mass index; CVH: cardiovascular health; DBP: diastolic blood pressure; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; SBP: systolic blood pressure.

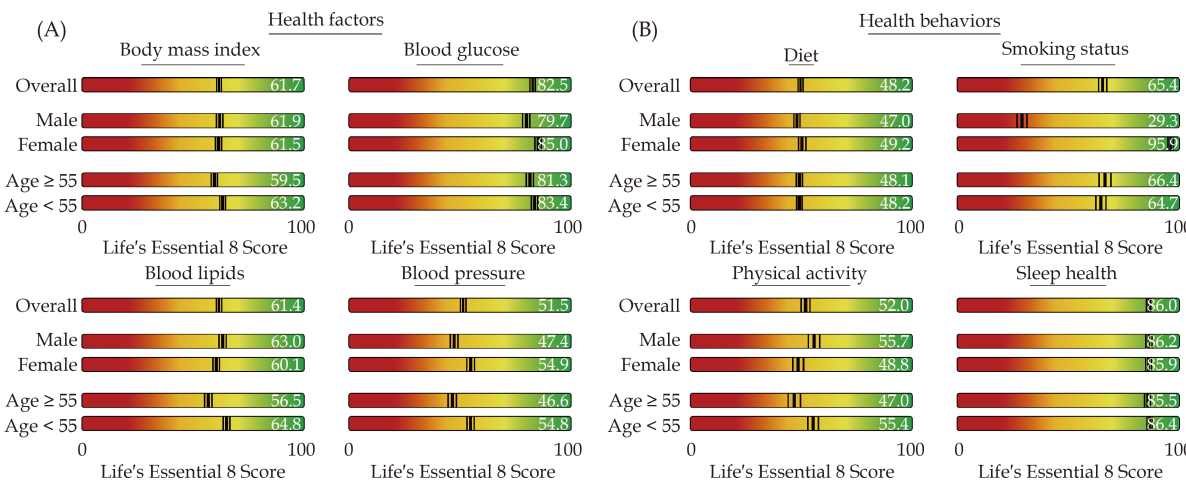

Figure 1 Distribution of LE8 scores at baseline. Mean (thick black bar) and standard error (thin bars) of scores for individual LE8 health factors (A) and behaviors (B), overall and by sex and age group at baseline. LE8: Life's Essential 8.

change of CVH with atherosclerosis progression. As shown in Table 3, 257 (27.5%) participants had low-stable CVH, 268 (28.7%) with unstable CVH, and 410 (43.8%) with high-stable CVH. Compared with those maintained low CVH at both baseline and follow-up visit, individuals with unstable CVH levels tended to have a lower risk for atherosclerosis progression. However, maintaining high CVH at both visits had significant protective effect on atherosclerosis progression, with the RRs of 0.609 (95% CI:

Table 2 Progression of atherosclerosis associated with CVH.

CVH	Progression of carotid plaque		Progression of CAC		Progression of overall SA	
	Events/Total (%)	RR (95% CI)	Events/Total (%)	RR (95% CI)	Events/Total (%)	RR (95% CI)
LE8 score*	324/863 (37.5%)	**0.848 (0.797, 0.902)**	230/745 (30.9%)	**0.823 (0.766, 0.884)**	447/972 (46.0%)	**0.880 (0.845, 0.916)**
CVH levels						
Low CVH	66/128 (51.6%)	1 (ref.)	54/110 (49.1%)	1 (ref.)	92/142 (64.8%)	1 (ref.)
Moderate CVH	247/637 (38.8%)	0.872 (0.728, 1.043)	169/563 (30.0%)	**0.748 (0.601, 0.932)**	338/722 (46.8%)	**0.872 (0.790, 0.963)**
High CVH	11/98 (11.2%)	**0.324 (0.181, 0.578)**	7/72 (9.7%)	**0.286 (0.137, 0.598)**	17/108 (15.7%)	**0.354 (0.226, 0.554)**
P for trend**	-	**< 0.001**	-	**< 0.001**	-	**< 0.001**

Models were adjusted for gender, age, alcohol intake, ASCVD family history and educational level. Statistically significant results are presented in bold. *Association between CVH and progression of atherosclerosis indicators were estimated for each 10 points increase in LE8 score; **Tests for trend were performed by entering the median value of each category as a continuous variable in the models. CAC: coronary artery calcification; CVH: cardiovascular health; LE8: Life's Essential 8; RR: relative risk; SA: subclinical atherosclerosis.

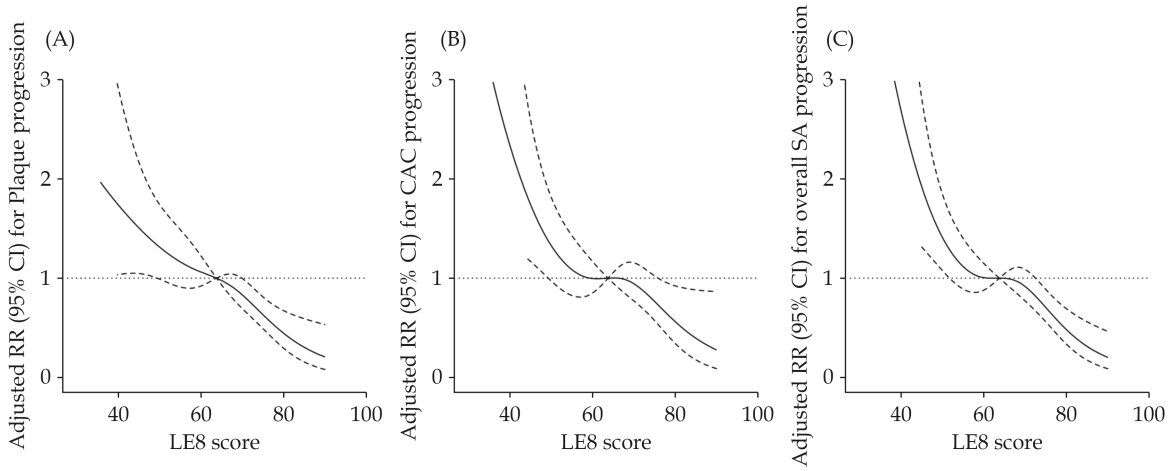

Figure 2 Multivariable-adjusted association between CVH estimated by LE8 score and progression of atherosclerosis. This panel displays relative risks for progression of carotid plaque (A), progression of CAC (B), and progression of overall SA (C) based on CVH (estimated by LE8 score), adjusted for gender, age, ASCVD family history and educational level. Solid lines represent point estimates of the relative risks and dashed lines represent 95% confidence intervals. Models were fitted using restricted cubic splines with 3 evenly spaced knots. ASCVD: atherosclerotic cardiovascular disease; CAC: coronary artery calcification; CVH: cardiovascular health; LE8: Life's Essential 8; SA: subclinical atherosclerosis.

Table 3 Estimated relative risk (95% CI) for the change of CVH on progression of atherosclerosis.

CVH change group	Progression of carotid plaque	Progression of CAC	Progression of overall SA
Low-stable (*n* = 178)	1 (ref.)	1 (ref.)	1 (ref.)
Unstable (*n* = 275)	0.872 (0.732, 1.038)	**0.756 (0.594, 0.963)**	0.895 (0.780, 1.027)
High-stable (*n* = 494)	**0.609 (0.494, 0.752)**	**0.590 (0.456, 0.764)**	**0.703 (0.598, 0.825)**
P for trend*	**0.003**	**< 0.001**	**< 0.001**

Groups: (1) low-stable, with LE8 score < 60 at both baseline and follow-up; (2) unstable, with LE8 score < 60 at either baseline or follow-up; (3) high-stable, with LE8 score ≥ 60 at both baseline and follow-up. *Tests for trend were performed by entering the group as numeric variable in models. Models were adjusted for gender, age, alcohol intake, ASCVD family history and educational level. Statistically significant results are presented in bold. CVH: cardiovascular health; CAC: coronary artery calcification; LE8: Life's Essential 8; SA: subclinical atherosclerosis.

0.494–0.752), 0.590 (95% CI: 0.456–0.764) and 0.703 (95% CI: 0.598–0.825) for carotid plaque, CAC and overall SA progression, respectively.

We performed subgroup analyses stratified by age, gender and educational level at baseline (Supplementary Table S5). There was a multiplicative interaction between age group (≥ 55 years and < 55 years) and CVH change ($P_{interaction}$ = 0.035 for carot-

id plaque progression and $P_{interaction}$ = 0.029 for overall SA progression). No significant interaction effect of gender and educational level with CVH change was found in this study.

DISCUSSION

Our community-based prospective study indicated that LE8 score was inversely associated with risk of atherosclerosis progression. In addition, long-term maintenance of ideal CVH would further reduce the risk of carotid plaque and CAC progression. Specifically, participants who were female, younger, and educated may benefits more from adhering to ideal CVH.

Consistent with our findings, several previous studies also reported that ideal CVH was considerably associated with decreased prevalence of CAC progression.[19-21] However, the relationship between CVH and progression of cIMT and total plaque area was insignificant, which may be due to the small sample size of only 219 subjects.[22] Nevertheless, these studies were all conducted based on the LS7 algorithm, with metrics evaluated by dichotomous variables. Moreover, other studies conducted in a cohort with nearly half diabetic subjects,[19] included patients with carotid plaque,[22] or included only 6 metrics (lacked diet information)[20] limited the extrapolation of the results. In addition, these studies were mainly conducted in western populations, lacking evidence for Chinese population.

In the current study, there were differences in the association of CVH with atherosclerosis progression among diverse populations. Although the CVH was also inversely associated with the risk of plaque progression in men, the effect was stronger in women and a significant interaction effect was observed. It indicated that women may benefit more from CVH improvement. Additionally, stronger relationship was observed in individuals with higher educational level, who may be more susceptible to CVH. In addition, a significant interaction between CVH change and age group was observed in the analysis of CVH change with atherosclerosis progression. The results of subgroup analyses stressed the importance of improving CVH in female population and maintaining ideal CVH as early as possible from a young age.

With the rapid change of lifestyle globally, the prevalence of CVH components have longitudinal changes over the past decades.[30,31] A series of population-based studies have analyzed the relationship between CVH changes and ASCVD risk, and most of the results suggest that improving CVH levels is related with reduced ASCVD risk.[32-35] One study has analyzed the association between long-term changes in CVH and cross-sectional measures of atherosclerosis, but no study has yet investigated the relationship between changes in CVH and progression of atherosclerosis. In this cohort study, promoting and maintaining an ideal CVH from early life was associated with a reduced risk of cIMT thickening in midlife.[36] In addition, a cohort of Chinese adults suggested that maintaining a stable high CVH level for a long time was associated with lower brachial-ankle pulse wave velocity, a marker of arterial stiffness. [37] Other studies have also suggested the relationship between long-term changes in CVH and other subclinical markers of cardiovascular system, such as left ventricular structural and diastolic function indicators, myocardial stress biomarkers, metabolic syndrome, and C-reactive protein. [38-40] Since most previous studies have only evaluated the effect of CVH at a single visit, evidence to deeply explore the possible impact of CVH changes on atherosclerosis progression was lacked. Our results suggest that having an ideal CVH temporarily may be protective in reducing the risk of atherosclerosis progression, and long-term maintenance of ideal CVH may provide additional cardiovascular benefits. Therefore, strategies are required to encourage people to maintain an ideal CVH level for the sake of primary prevention of ASCVD.

Our study has multiple strengths. This study was conducted in a community-based middle-aged population with repeated imaging examinations over time, making the findings less likely to be affected by selection bias than studies involving patients or elderly population. In addition, we obtained complete and detailed information on each of the CVH metrics, avoiding bias due to missing metrics. Furthermore, considering the repeated CVH measurements and atherosclerosis examination, this study evaluated the association of changes in CVH with atherosclerosis progression, and added to the evidence of cardiovascular benefits from long-term main-

tenance of ideal CVH.

Several limitations of our study should also be noted. First, different definition of diet metric from AHA's definition was used in our study to conform to the dietary habits of Chinese. The scoring was built in accordance with the Dietary Guidelines for Chinese Residents based on the food frequency questionnaire, while AHA recommend that quantiles of DASH-style diet adherence should be used to evaluate diet score. Therefore, diet scores in this study are not comparable to those measured in western populations using the AHA's definition of LE8 score. In addition, due to the limited sample size of this study, cohort studies with large samples are warranted to validate our results.

In summary, higher LE8 score was strongly and independently associated with lower risk of atherosclerosis progression in both carotid and coronary artery. Besides, long-term maintenance of ideal CVH had a stronger protective effect.

DISCLOSURE

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Declaration of Interests

None.

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