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Association between cardiometabolic index and biological ageing among adults: a population-based study

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Abstract

Background Cardiovascular health (CVH) is closely associated with ageing. This study aimed to investigate the association between cardiometabolic index (CMI), a novel indicator of cardiometabolic status, and biological ageing.

Methods Cross-sectional data were obtained from participants with comprehensive CMI and biological age data in the National Health and Nutrition Examination Survey from 2011 to 2018. Biological age acceleration (BioAgeAccel) is calculated as the differences between biological age and chronological age, and that biological age is derived from a model incorporating eight biomarkers. Weighted multivariable regression, sensitivity analysis, and smoothing curve fitting were performed to explore the independent association between CMI and the acceleration of biological age. Subgroup and interaction analyses were performed to investigate whether this association was consistent across populations.

Results In 4282 subjects ≥ 20 years of age, there was a positive relationship between CMI and biological age. The BioAgeAccel increased 1.16 years for each unit CMI increase [1.16 (1.02, 1.31)], and increased 0.99 years for per SD increase in CMI [0.99 (0.87, 1.11)]. Participants in the highest CMI quartile had a BioAgeAccel that was 2.49 years higher than participants in the lowest CMI quartile [2.49 (2.15, 2.83)]. In stratified studies, the positive correlation between CMI and biological age acceleration was not consistent across strata. This positive correlation was stronger in female, diabetes, and non-hypertension populations.

Conclusions CMI is positively correlated with biological ageing in adults in the United States. Prospective studies with larger sample sizes are required to validate our findings.

Keywords Cardiometabolic index, Biological ageing, NHANES

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Background

Ageing is characterised by a progressive decline in physiological function [1]. A UN report estimated that the world's population is ageing at an accelerating rate; over 65 years old contribute to 9% of the population, and this number is expected to rise to 16% by 2050 [2]. There is significant heterogeneity in the ageing process and health outcomes of older individuals [3]. Determining an individual's biological age (BA) is necessary to better understand and prevent ageing. Much progress has been achieved in developing biological ageing clocks using biomedical data, such as DNA methylation [4], transcriptomics [5], proteomics [6], and organ-level characteristics [7], over the last two decades. Previous studies have established many composite BA predictors [8–10], demonstrating the potential of predicting BA using blood biochemical markers. Among the various recognised approaches for estimating BA, the Klemera and Doubal (KD) method [11] has been reported to outperform traditional methods in predicting mortality outcomes. In the current study, the ageing process was represented by biological age and biological age acceleration (BioAgeAccel).

As the body ages, the metabolic function of the systems decreases. Elevated cardiometabolic risk factors, such as obesity and hypertension, can lead to increased oxidative stress and inflammation, which are known to accelerate biological aging processes [12, 13].The cardiometabolic index (CMI) is a novel metabolic index introduced in 2015 by Ichiro Wakabayashi [12] that combines clinical measures of triglycerides, high-density lipoprotein cholesterol, and waist-height ratio and accurately reflects both blood the lipid levels and degree of obesity. CMI has been related to several metabolic disorders, including diabetes mellitus, atherosclerosis, ischemic stroke, and hypertension, according to some research. Several studies have investigated the clinical significance of CMI in metabolic disorders, such as atherosclerosis [14], ischaemic stroke [15], hypertension [16], and metabolic-associated fatty liver disease [17], and more significant increases in CMI over time were significantly associated with a greater risk for subsequent cardiovascular events [18]. Ageing and metabolism are inextricably related and dysregulated metabolism is a hallmark of ageing hallmarks [19]. However, the association between CMI and biological ageing has yet to be extensively explored.

We hypothesize a positive relationship between the cardiometabolic index and biological aging. In addition, this relationship is assumed to be nonlinear, as it is expected that the effect may vary at different levels of cardiometabolic risk. This hypothesis is grounded in the premise that at lower levels of cardiometabolic risk, the impact on biological aging may be relatively modest. However, as CMI increases, the cumulative effects of chronic inflammation and oxidative stress—key drivers of aging—are likely to intensify, leading to a more pronounced acceleration of aging processes.

This study aimed to investigate the association between CMI and biological ageing, providing a clinical basis for preventing ageing.

Methods

Survey description

Data were obtained from the NHANES, a national population-oriented survey by the National Centre for Health Statistics (NCHS) designed to evaluate potential health risk factors and the nutrition status of non-institutionalised citizens across the United States [20]. A complicated, stratified, multistage probability cluster sampling design was devised to select a representative sample of the US population.

All NHANES research protocols were authorized by the NCHS Research Ethics Review Board, and all survey participants or, in the case of participants under 16 years of age, a parent or legal guardian provided written informed consent. The public can access all comprehensive NHANES survey designs and data from www.cdc.gov/nchs/nhanes/.

Study population

Four NHANES cycles from 2011 to 2018 were chosen to investigate the relationship between CMI and biological age acceleration because only these four cycles had comprehensive variables to compute both CMI and biological age.

This study included participants who provided complete CMI and biological age data. A total of 39,156 individuals were initially enrolled, and individuals were excluded if [1] they had missing data on biological age ($n = 27{,}522$) [2], had missing values for CMI ($n = 6{,}510$), or [3] were under the age of 20 years ($n = 842$). Ultimately, 4,282 individuals were included in the analysis (Fig. 1).

Measurement of CMI

CMI was used to measure the diabetes risk and atherosclerosis progression to indicate visceral adipose tissue distribution and function. The CMI score comprises four health factors (triglyceride, high-density lipoprotein, waist circumference, and body height). Height and weight circumferences were measured via physical examination. Triglyceride and high-density lipoprotein levels were assessed in the blood samples. The entire algorithm for calculating the CMI has been published [21].

CMI = (triglycerides (mmol/L)/high-density lipoproteins (mmol/L)) × (waist circumference (cm)/body height (cm)).

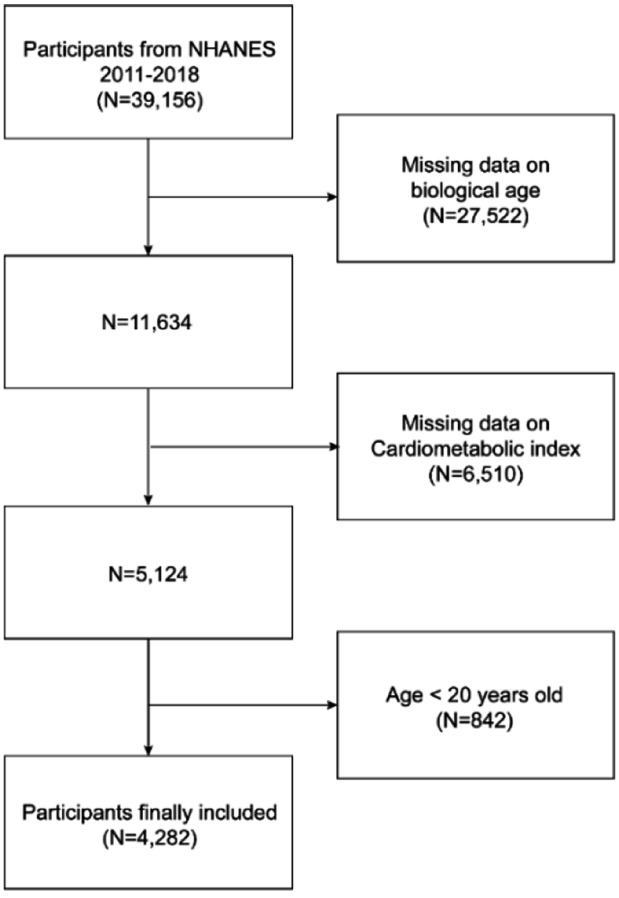

Fig. 1 Flowchart of the sample selection from NHANES 2011–2018

Measurement of biological age

Biological age is a measurement of biological ageing [22]. Klemera proposed an algorithm to determine the biological age on the basis of eight biomarkers (serum albumin, serum creatinine, serum glycated haemoglobin, serum alkaline phosphatase, serum total cholesterol, serum urea nitrogen, C-reactive protein, and systolic blood pressure) [11].

$$BioAge = \frac{\sum_{j=1}^{8}(x_j - q_j)\left(\frac{k_j}{s_j}\right) + \frac{CA}{{s_{BA}}^2}}{\sum_{j=1}^{8}\left(\frac{k_j}{s_j}\right)^2 + \frac{1}{{s_{BA}}^2}}$$

In the above equation, CA represents the chronological age; x represents the value of biomarker j; q, k, and s represent parameters when biomarker j is regressed on CA; and sBA represents a scaling factor equal to the square root of the variance in chronological age explained by the biomarker set.

Furthermore, we calculated the BioAgeAccel [20] by estimating the differences in the biological and chronological age [23]. A negative BioAgeAccel value indicated a lower biological age.

Covariates

Covariates that may affect the relationship between CMI and biological age were also included in our study, including gender (male, female), age (years), race (non-Hispanic White, non-Hispanic Black, Mexican American, or other races), education level (less than high school, high school, or above high school), family poverty-to-income ratio (PIR), alcohol consumption per day, hypertension, and diabetes mellitus (DM), where hypertension and DM were defined according to self-reported information supplied by the individuals. All detailed measurement protocols for these variables are available at www.cdc.gov/nchs/nhanes/.

Statistical analysis

The sample analysis utilised appropriate weights considering the complexity of the NHANES sampling approach. For continuous data, weighted means (standard errors) were utilized, whereas sample sizes (weighted percentages) were employed for categorical variables. The demographics of the participants were assessed according to the CMI quartile using variance analysis. Weighted multivariate linear regression analysis was applied to explore the linear correlations of CMI and BioAgeAccel. Prior to conducting the weighted multivariate linear regression analysis, we rigorously assessed the key assumptions underlying linear regression models. These included normality, constant variance (homoscedasticity), and the absence of influential outliers. To improve the normality of the CMI distribution, a natural log transformation was applied.

Trend analysis was used to explore the linear relationship between CMI and BioAgeAccel after converting CMI from a continuous to categorical variable (quartile). Subgroup analysis was performed to examine the relationship between CMI and BioAgeAccel in individuals of different ages, sexes, education levels, and diabetes status, and interaction tests were conducted to determine whether the associations were constant across subgroups. The interaction tests were adjusted for the following covariates: age, gender, ethnicity, family PIR, education level, hypertension, diabetes, smoking status, alcohol consumption. The nonlinear relationship between CMI and BioAgeAccel was explored using restricted cubic spline. All analyses were conducted using R (version 4.2) and EmpowerStat (version 5.0) software. Statistical significance was determined as a two-sided $P < 0.05$.

Results

Baseline characteristics

A total of 4,282 participants were enrolled, of whom 48.65% were male, with an average chronological age of 50.01 ± 17.32 years and biological age of 51.28 ± 17.36 years. Among the four CMI quartiles, statistically

significant differences were found in age, sex, race, education levels, PIR, smoking status, alcohol consumption, diabetes, hypertension, family PIR, BMI, waist circumference, triglycerides, HDL-C, biological age, and BioAgeAccel scores (all $P<0.05$). Individuals with elevated CMI levels were male, smokers, and had higher BMI, waist circumference, triglycerides, diabetes, hypertension, biological age, BioAgeAccel, and decreased family PIR and HDL-C levels (all $P<0$ 0.05). The baseline characteristics of the participants based on the CMI quartiles are shown in Table 1. Baseline information for the included and excluded populations is presented in Table S1.

Association between CMI and bioageaccel

Table 2 shows the association between the CMI and BioAgeAccel. The results demonstrated a significant positive association between the CMI and BioAgeAccel in both the crude model [1.25 (1.10, 1.40)] and model 2 [1.43 (1.29, 1.58)]. Moreover, this association remained significant in model 3 [1.16 (1.02, 1.31)]. After adjusting for all related covariates, the BioAgeAccel increased 1.16 years for each unit lnCMI increase. Furthermore, the BioAgeAccel increased 0.99 years for per standard deviation (SD) increase in lnCMI [0.99 (0.87, 1.11)].

This relationship remained statistically significant after CMI was grouped into quartiles ($P<0.01$). Participants in the highest CMI quartile had a BioAgeAccel that was 2.49 years higher than participants in the lowest CMI

Table 1 Baseline characteristics of participants by cardiometabolic index quartiles among U.S. Adults

		Cardiometabolic index				*P*-value
Characteristics	**Overall (*N* = 4282)**	**Q1 (*N* = 1071)**	**Q2 (*N* = 1070)**	**Q3 (*N* = 1070)**	**Q4 (*N* = 1071)**	
Age, year	50.01 ± 17.32	44.75 ± 17.63	49.21 ± 17.17	50.41 ± 16.76	49.94 ± 15.07	< 0.001
Gender, n (%)						< 0.001
Male	2083 (48.65)	420 (39.23)	507 (47.34)	536 (50.06)	656 (61.25)	
Female	2199 (51.35)	651 (60.77)	563 (52.66)	534 (49.94)	415 (38.75)	
Race/ethnicity, n (%)						< 0.001
Non-Hispanic White	1454 (33.96)	688 (64.22)	689 (64.41)	639 (59.76)	732 (68.35)	
Non-Hispanic Black	917 (21.42)	163 (15.18)	135 (12.60)	105 (9.77)	47 (4.43)	
Mexican American	664 (15.51)	58 (5.46)	91 (8.49)	106 (9.92)	125 (11.65)	
Other races	1247 (29.12)	162 (15.14)	155 (14.50)	220 (20.55)	167 (15.57)	
Education level, n (%)						< 0.001
< High school	910 (21.25)	104 (9.58)	124 (11.58)	149 (13.93)	171 (15.97)	
High school	983 (22.96)	224 (20.92)	256 (23.90)	297 (27.75)	287 (26.84)	
> High school	2389 (55.79)	744 (69.50)	690 (64.51)	624 (58.33)	612 (57.18)	
Alcohol drinks/day	2.38 ± 1.82	2.22 ± 1.52	2.43 ± 1.94	2.35 ± 1.81	2.52 ± 2.08	< 0.001
Smoking status, n (%)	1873 (43.48)	418 (39.02)	443 (41.44)	506 (47.32)	581 (54.24)	< 0.001
Yes	2405 (56.22)	653 (60.98)	627 (58.56)	564 (52.68)	490 (45.76)	
No	1598 (37.32)	214 (19.95)	329 (30.77)	417 (38.95)	463 (43.24)	
Hypertension, n (%)	2684 (62.68)	857 (80.05)	741 (69.23)	653 (61.05)	608 (56.76)	
Yes						
No						
Diabetes, n (%)						< 0.001
Yes	806 (18.82)	56 (5.19)	92 (8.58)	179 (16.72)	265 (24.74)	
No	3476 (81.32)	1015 (94.81)	978 (91.42)	891 (83.28)	805 (75.26)	
Family PIR	2.93 ± 1.53	3.13 ± 1.56	3.03 ± 1.61	2.82 ± 1.58	2.85 ± 1.55	< 0.001
BMI	29.58 ± 7.02	24.69 ± 4.77	29.17 ± 6.21	31.23 ± 6.82	33.36 ± 6.96	< 0.001
Height, cm	166.53 ± 9.96	167.68 ± 9.36	168.40 ± 10.05	167.83 ± 9.98	169.59 ± 9.95	< 0.001
Waist circumference, cm	100.56 ± 16.76	87.48 ± 11.96	99.86 ± 14.59	105.40 ± 15.42	111.63 ± 15.54	< 0.001
Triglyceride, mg/dL	113.38 ± 89.62	51.45 ± 15.89	80.54 ± 20.49	114.31 ± 26.29	207.80 ± 107.52	< 0.001
HDL-C, mg/dL	54.42 ± 16.67	71.12 ± 18.93	57.5 ± 12.19	49.87 ± 9.99	40.61 ± 8.33	< 0.001
CMI	1.58 ± 2.02	0.39 ± 0.12	0.83 ± 0.14	1.44 ± 0.25	3.54 ± 2.58	< 0.001
lnCMI	0.07 ± 0.85	-1.00 ± 0.37	-0.21 ± 0.17	0.35 ± 0.17	1.15 ± 0.42	< 0.001
lnCMI/SD	0.09 ± 1.00	1.18 ± 0.43	-0.24 ± 0.20	0.41 ± 0.20	1.35 ± 0.50	< 0.001
Biological age, year	51.28 ± 17.36	43.90 ± 17.01	49.09 ± 16.45	50.93 ± 16.16	51.82 ± 14.67	< 0.001
BioAgeAccel, year	0.72 ± 4.90	-0.86 ± 3.98	-0.12 ± 3.90	0.52 ± 4.35	1.88 ± 4.65	< 0.001

Continuous variables are expressed as mean ± SD. Categorical variables are expressed as frequency n (%). *Q* quartile, *PIR* the ratio of income to poverty, *BMI* body mass index, *HDL-C* high-density lipoprotein cholesterol, *BioAgeAccel* biological age acceleration

Table 2 The association between cardiometabolic index and biological age acceleration

Biological age acceleration	Model 1 [β (95% CI)]	Model 2 [β (95% CI)]	Model 3 [β (95% CI)]
Cardiometabolic index			
Continuous			
lnCMI	1.25 (1.10, 1.40)	1.43 (1.29, 1.58)	1.16 (1.02, 1.31)
lnCMI/SD	1.06 (0.94, 1.19)	1.22 (1.10, 1.34)	0.99 (0.87, 1.11)
Category			
Quartile 1	Ref	Ref	Ref
Quartile 2	0.73 (0.38, 1.09)	1.00 (0.66, 1.34)	0.86 (0.53, 1.19)
Quartile 3	1.38 (1.02, 1.74)	1.74 (1.40, 2.09)	1.35 (1.01, 1.69)
Quartile 4	2.74 (2.39,3.09)	3.14 (2.79, 3.48)	2.49 (2.15, 2.83)
P for trend	< 0.0001	< 0.0001	< 0.0001

Model 1: crude model

Model 2: adjusted for age, sex, and ethnicity

Model 3: further adjusted for family PIR, education level, hypertension, diabetes, smoking status and alcohol consumption

quartile [2.49 (2.15, 2.83)]. Furthermore, the restricted cubic spline verified the nonlinear positive relationship between CMI and BioAgeAccel ($P < 0.05$)(Fig. 2).

Subgroup analysis

The subgroup analysis revealed that the correlations between CMI and higher BioAgeAccel scores were not consistent. The association between CMI and biological age acceleration was significantly stronger in women compared to men. The association was more pronounced in individuals with diabetes compared to those without diabetes.

Table 3 Subgroup analysis of the association between CMI and biological age acceleration

Subgroup	BIOAGEACCEL [β (95%CI)]	*P* for interaction
Sex		0.023
Male	1.01 (0.82, 1.21)	
Female	1.33 (1.13, 1.53)	
Age		0.067
< 60 years	1.14 (0.97, 1.30)	
≥ 60 years	0.84 (0.55, 1.12)	
Race		0.068
Non-Hispanic White	1.08 (0.90, 1.26)	
Non-Hispanic Black	1.72 (1.28, 2.17)	
Mexican American	1.22 (0.76, 1.68)	
Other races	1.14 (0.80, 1.48)	
Education		0.560
< High school	1.35 (0.97, 1.73)	
High school	1.18 (0.88, 1.47)	
> High school	1.12 (0.95, 1.30)	
Smoking		0.614
Yes	1.13 (0.93, 1.33)	
	1.20 (1.00, 1.39)	
No	1.20 (1.00, 1.39)	
Hypertension		< 0.001
Yes	0.80 (0.54, 1.05)	
No	1.33 (1.16, 1.50)	
Diabetes		< 0.001
Yes	2.15 (1.73, 2.57)	
No	1.06 (0.91, 1.21)	

Conversely, the association was attenuated in individuals with hypertension compared to those without hypertension.

The interaction test revealed a significant difference in the relationship between CMI and BioAgeAccel across sex, hypertension status, and diabetes status ($P < 0.05$). There was no significant dependence of this positive

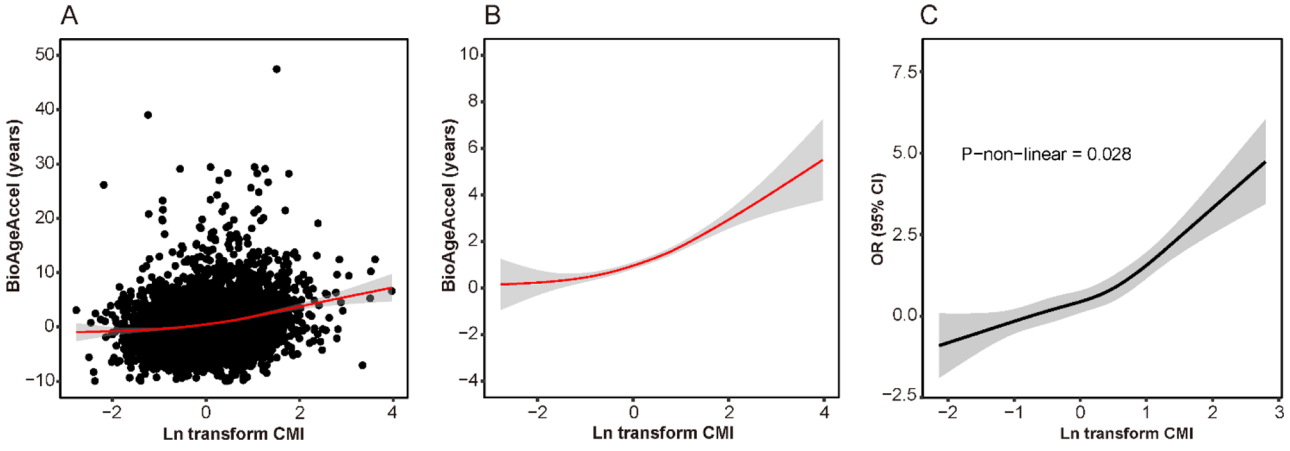

Fig. 2 The association between lnCMI and BioAgeAccel. (**A**) Each black point represents a sample. (**B**) Smooth curve fit between variables. The solid red line represents the smooth curve fit between variables. Grey bands represent the 95% of confidence interval from the fit. (**C**) Analysis of restricted cubic spline regression. ($P = 0.028$)

association on age, race, educational level, or smoking status (all P for interaction > 0.05) (Table 3).

Discussion

In the cross-sectional study of 4,282 adults in the United States, we observed a positive association between CMI and biological age acceleration. Stratified analyses demonstrated that this relationship is not stable across groups.

To the best of our knowledge, this is the first study to explore the relationship between CMI and the acceleration of biological age. CMI, proposed by Japanese scientist Ichiro Wakabayashi, serves as a cardiovascular health indicator and a novel obesity index reflecting visceral adipose tissue dysfunction and distribution. Previous studies have demonstrated the connection between ageing and cardiovascular health [24]. Several studies have demonstrated that changes in individual CVH-related factors significantly influence ageing. Improved cardiovascular health has been associated with lower epigenetic age acceleration [25]. Obesity and cardiovascular metabolic disorders can significantly accelerate biological ageing, as evidenced by various obesity metrics (BMI, body fat percentage, waist circumference, hip circumference, and waist-to-hip ratio) in a cross-sectional investigation of 2,474 Taiwanese adults [26]. The AHA's Life's Simple 7 approach, which promotes a matrix of seven healthy activities, has shown fascinating inverse relationships with ageing biomarkers, such as leukocyte DNA methylation clock and telomere length [27–29]. Adults with high levels of total or individual CVH measures in Life's Essential 8 had a lower risk of all-cause and CVD-specific mortality, according to Sun et al. in another extensive nationally representative sample of US adults [29]. Consistent with previous findings on the impact of cardiovascular health and ageing, our findings indicated a positive relationship between CMI and biological age, suggesting a strong link between cardiovascular health and ageing.

The association between CMI and biological age acceleration was significantly stronger in women compared to men. Women undergo several important hormonal changes throughout their lives, such as the menstrual cycle, pregnancy, breastfeeding, and menopause, and these fluctuations in hormone levels can have an impact on cardiometabolism. For example, the decline in oestrogen levels in postmenopausal women can lead to changes in fat distribution, higher lipid levels and increased insulin resistance, which can accelerate the onset of cardiometabolic problems and biological ageing [30]. On the other hand, women's body fat distribution differs from men's, typically storing more fat in areas such as the hips and thighs, whereas men tend to store fat in the abdomen. Although fat in the hips and thighs is relatively healthier, when a woman's waist-to-hip ratio (WHR) is too high, an increase in abdominal fat can significantly increase cardiometabolic risk, which can accelerate biological age.

This association was more pronounced in diabetics compared to non-diabetics. This may be due to a more pronounced chronic inflammatory response in diabetics, with elevated levels of inflammatory factors such as C-reactive protein (CRP), which damage the endothelial cells of the blood vessels and contribute to the development of atherosclerosis, which accelerates cardiometabolic problems and biological age. In addition, hyperglycaemia and metabolic disorders lead to increased oxidative stress, which damages cellular DNA, proteins and lipids, affecting normal cellular function and lifespan, and thus accelerating biological age [31].

In contrast, this association was attenuated in people with hypertension compared to those without hypertension. This may be due to the fact that people with hypertension are usually treated with medications that are effective in controlling blood pressure and reducing the risk of cardiovascular disease. For example, antihypertensive medications such as ACE inhibitors and calcium channel blockers not only lower blood pressure but also improve cardiometabolic health, thereby attenuating the association between CMI and accelerated biological age [32, 33]. In addition, hypertensive patients tend to focus more on lifestyle modifications after diagnosis, such as increasing exercise, improving diet, quitting smoking and limiting alcohol. These lifestyle changes can significantly improve cardiometabolic health and reduce accelerated biological age.

The underlying mechanism through which CMI contributes to the development of biological ageing remains unclear. Individuals with CMI may have an abnormal lipid metabolism, which may account for these findings. Plasma lipid metabolism influences several biochemical pathways and cell types linked to longevity and ageing in healthy adults [34, 35]. Restoring abnormal lipid metabolism is a newly emerging and promising anti-ageing approach [36]. Maintaining a normal BMI promotes healthy life expectancy, extends survival, enhances physical performance in elderly individuals, and alleviates ageing [37, 38].

This study has several advantages. It utilised NHANES data, and a countrywide, population-based sample data set was collected utilizing a standardized approach. All analyses were conducted using appropriate NHANES sampling weights, which enabled the collection of more representative research samples. To make the conclusions more reliable, we adjusted for related confounders. However, the findings of this study should be considered with caution due to some limitations. First, we were unable to establish a causal association between the CMI and biological age due to the cross-sectional design of the

study. Therefore, prospective studies with more extensive sample sizes are required to determine the causality. Furthermore, even after adjusting for relevant confounders, we could not exclude the possibility of other potential covariables, such as drug usage or other comorbidities. Because these details were not collected for NHANES, researchers may have been unable to draw firm conclusions from the data. Finally, the study did not examine biological age on the molecular level and instead relied on clinical biochemical markers. Also, the exclusion of individuals with missing data might limit the generalizability of our findings or potentially introduce selection bias.

Our findings suggest a relationship where cardiometabolic risk may influence biological aging, it is equally plausible that biological aging could contribute to increased cardiometabolic risk. With biological ageing, the elasticity of blood vessel walls decreases and lipid deposition increases, leading to the development of atherosclerosis. This lesion increases the risk of cardiovascular diseases such as coronary heart disease and stroke. The biological ageing process also increases the chronic inflammatory response in the body and increases the level of inflammatory factors, which damages the vascular endothelial cells and promotes the development of atherosclerosis [39]. In addition, aging leads to a decrease in antioxidant capacity and an increase in the generation of reactive oxygen species (ROS), which damages cellular DNA, proteins and lipids, affecting cellular function and increasing the risk of cardiovascular disease. A study analysing 341,159 adults in a multi-stage UK biobank has found that accelerated biological ageing increases the risk of cardiometabolic multimorbidity and mortality [40]. The bidirectional nature of the association between cardiovascular metabolic risk and biological ageing should be emphasised.

Conclusion

Elevated CMI levels are associated with accelerated biological ageing. Prospective studies with larger sample sizes are required to validate our findings.

Abbreviations

NHANES	National Health and Nutrition Examination Survey
CMI	Cardiometabolic index
BMI	Body mass index
PIR	Poverty income ratio
HDL-C	High-density lipoprotein cholesterol
BioAgeAccel	Biological age acceleration

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-025-22053-3.

Supplementary Material 1

Author contributions

NL conceived and designed the study, performed the analyses, wrote the first draft of the manuscript, contributed to the interpretation of the results and critically revised the manuscript. SSP contributed to the interpretation of the results and critically revised the manuscript. KW contributed to the analysis of the results and critically revised the manuscript. QDC、XTC 、LQH and BYW contributed to the interpretation of the results. YL conceived and designed the study, contributed to the interpretation of the results, and critically revised the manuscript.

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Data availability

Datasets analyzed in this study are available at www.cdc.gov/nchs/nhanes/.

Declarations

Ethics approval and consent to participate

All procedures were properly carried out in compliance with relevant guidelines and standards (declaration of Helsinki). The NHANES protocol was approved by the National Center for Health Statistics (NCHS) Ethics Review Board, and each participant provided written informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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