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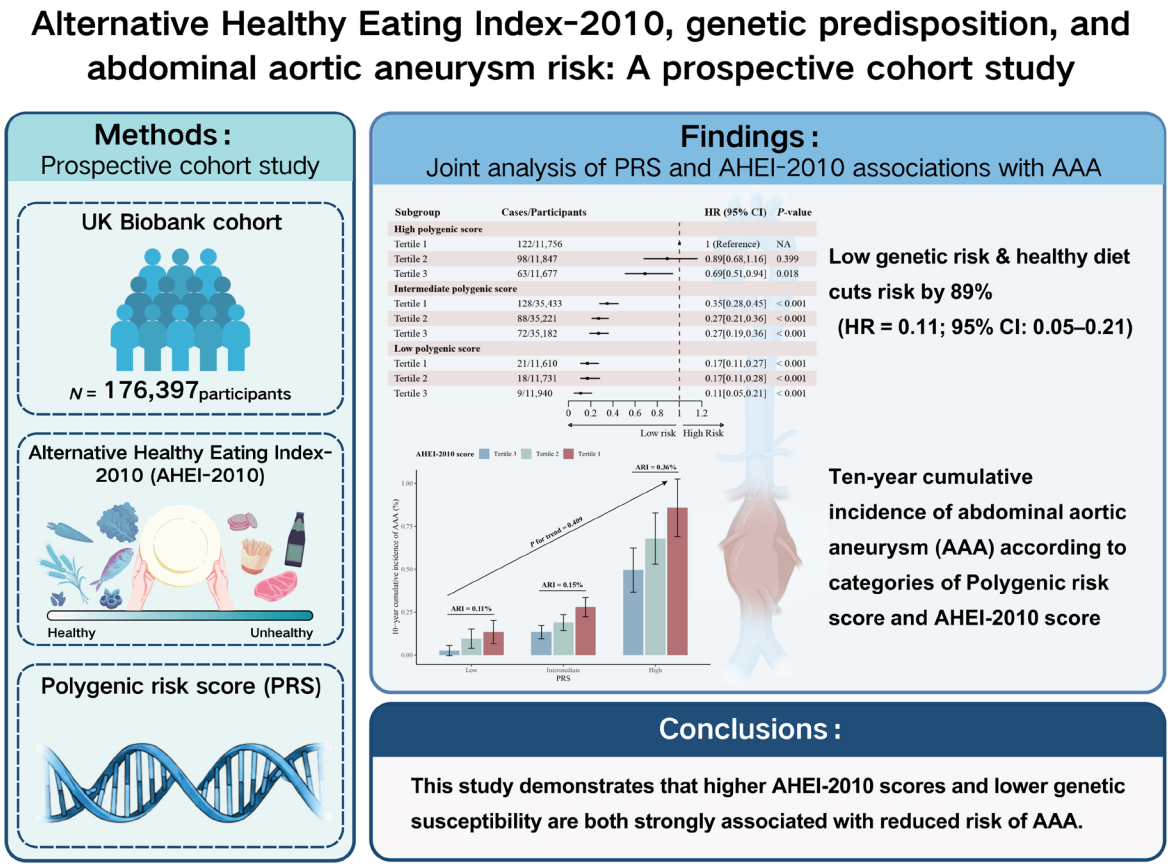
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Alternative Healthy Eating Index-2010, genetic predisposition, and abdominal aortic aneurysm risk

A prospective cohort study

Xinyi Liu, MD[a], Zhiyu Qiao, MD[a], Haiou Hu, MD[a], Yipeng Ge, MD[a], Chengnan Li, MD[a] Junming Zhu, MD[a,*]

Graphical abstract



Abstract

Background: Abdominal aortic aneurysm (AAA) is a progressive vascular disease with high mortality after rupture. Although genetic susceptibility is a major determinant of AAA risk, the role of dietary quality in its development remains unclear. We therefore investigated the association between adherence to the Alternative Healthy Eating Index-2010 (AHEI-2010) and incident AAA, and examined whether this association was modified by genetic susceptibility, as measured by a polygenic risk score (PRS).

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[a] Department of Cardiovascular Surgery, Beijing Aortic Disease Center, Beijing Anzhen Hospital of Capital Medical University, Beijing, China.

* Correspondence: Junming Zhu, Department of Cardiovascular Surgery, Beijing Aortic Disease Center, Beijing Anzhen Hospital of Capital Medical University, No. 2 Anzhen Road, Chaoyang District, Beijing 100029, China (e-mail: anzhenzjm@ccmu.edu.cn).

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Methods: We analyzed 176,397 UK Biobank participants who completed at least one web-based 24-h dietary recall. Dietary quality was assessed using the AHEI-2010, and a PRS for AAA was derived using the PRS–continuous shrinkage method. Incident AAA cases were ascertained through hospital inpatient and death registry records, and Cox proportional hazards models estimated hazard ratios (HRs) with multivariable adjustment. Interactions and joint effects of AHEI-2010 and PRS were further examined.

Results: Over a mean follow-up of 13.3 years, 619 AAA cases occurred. Higher AHEI-2010 scores were associated with a lower risk of AAA (HR for highest vs. lowest tertile = 0.73; 95% confidence interval [CI]: 0.59–0.89; HR per 1-standard deviation increase = 0.88; 95% CI: 0.81–0.96), with a linear dose–response relationship. Fruit and whole-grain intake were independently protective. Participants in the highest PRS quintile had a more than 2-fold increased risk of AAA (HR = 2.36; 95% CI: 2.10–3.36). No significant multiplicative or additive interactions were detected between diet and genetics; however, joint analyses showed that individuals with low PRS and high AHEI-2010 scores had nearly 90% lower risk of AAA compared with those with high PRS and low AHEI-2010 scores (HR = 0.11; 95% CI: 0.05–0.21).

Conclusion: This study demonstrates that higher AHEI-2010 scores and lower genetic susceptibility are both strongly associated with reduced risk of AAA. These findings suggest that integrating dietary strategies and genetic screening into targeted AAA prevention programs may have substantial utility.

Keywords: abdominal aortic aneurysm, alternative healthy eating index-2010, polygenic risk score, UK Biobank

Nonstandard Abbreviations and Acronyms

AAA	abdominal aortic aneurysm
AHEI-2010	Alternative Healthy Eating Index-2010
ARI	absolute risk increase
ASM	appendicular skeletal muscle mass
BMI	body mass index
CI	confidence interval
GWAS	genome-wide association study
HGS	handgrip strength
HR	hazard ratio
ICD-10	International Classification of Diseases, 10th Revision
LD	linkage disequilibrium
PRS–CS	polygenic risk score–continuous shrinkage
PRS	polygenic risk score
SD	standard deviation
SNP	single-nucleotide polymorphism

Highlights

- In 176,397 UK Biobank participants followed for a mean of 13.3 years, higher AHEI-2010 scores were linearly associated with a lower incidence of abdominal aortic aneurysm.
- Among AHEI-2010 components, higher intakes of fruit (excluding juice) and whole grains showed the most consistent inverse associations, and their combined 2-component score demonstrated an even stronger protective association (HR: 0.67) than the overall AHEI-2010 score (HR: 0.73).
- Although no significant gene–diet interaction was detected, joint analyses indicated substantially lower AAA risk among individuals with both low genetic risk and high diet quality, supporting the potential value of integrating dietary strategies with genetic risk stratification for prevention.

1. Introduction

Abdominal aortic aneurysm (AAA) is a progressive vascular condition characterized by a localized dilation of the abdominal aorta exceeding 3 cm or 50% of its normal diameter.[1] AAA often remains clinically silent until rupture, and despite surgical intervention, the associated mortality rate remains high, making it a major cause of cardiovascular death among older adults.[2] Established risk factors include age, male sex, smoking, hypertension, and dyslipidemia; however, these factors alone do not fully account for the occurrence and progression of AAA.[3] Thus, identifying additional modifiable and genetic determinants is essential for improving risk stratification and preventive strategies.

Dietary factors have emerged as important contributors to vascular health.[4] Prior research has examined individual dietary components (such as fruit and vegetable intake,[5] alcohol consumption,[6] coffee,[7] and vitamins[8]) in relation to AAA risk, but findings have been inconsistent and often limited by small sample sizes or cross-sectional designs. More broadly, adherence to healthy dietary patterns has been linked to reduced risks of several cardiovascular diseases. The Alternative ealthy Eating Index-2010 (AHEI-2010) is a widely used dietary quality score that emphasizes high intake of vegetables, fruits, whole grains, nuts, and polyunsaturated fatty acids, alongside limited consumption of red and processed meats, sugar-sweetened beverages, trans fat, and sodium.[9] Higher AHEI-2010 scores have consistently been associated with lower incidence of coronary heart disease, stroke, and total mortality.[10] However, whether dietary quality, assessed comprehensively by AHEI-2010, is associated with AAA risk has not been systematically evaluated in large-scale prospective cohorts.

In parallel, genetic susceptibility is recognized as a major determinant of AAA development.[11] Genome-wide association studies (GWAS) have identified multiple

single-nucleotide polymorphisms (SNPs) associated with AAA, implicating pathways of vascular remodeling, lipid metabolism, and inflammation.[12] Polygenic risk scores (PRS), which aggregate the effects of these variants, provide a quantitative measure of inherited predisposition to AAA. While PRS has shown promise for risk prediction, its integration with lifestyle factors remains limited in AAA research. Emerging evidence in other cardiometabolic diseases suggests that healthy dietary patterns can attenuate the adverse impact of genetic predisposition,[13,14] raising the possibility that diet may modulate genetic risk for AAA. Yet, whether such gene–diet interactions exist for AAA remains unclear.

Taken together, although both dietary and genetic factors have been individually implicated in AAA, existing studies have not provided a clear synthesis of findings nor examined their joint influence. Traditional risk factors such as smoking, hypertension, and dyslipidemia are already well established, but the roles of diet quality and genetic susceptibility—2 potentially modifiable and clinically relevant domains—remain underexplored. To address these gaps, we aimed to investigate the prospective association between adherence to the AHEI-2010 and the incidence of AAA, and to further examine whether dietary quality modifies the effect of genetic susceptibility on AAA risk in the UK Biobank.

2. Methods

2.1. Study design and participants

This research drew on data from the UK Biobank, a large prospective cohort comprising more than 500,000 participants. Individuals aged 37 to 73 years were enrolled between 2006 and 2010 at 22 assessment centers across the UK. At baseline, participants completed a comprehensive touchscreen questionnaire that gathered information on demographics, socioeconomic background, lifestyle behaviors, and medical history. Further details regarding the study design and data collection methods are available in previously published reports.[15] Ethics approval for the UK Biobank was obtained from the North West Multicenter Research Ethics Committee (Ref:11/NW/0382).

In total, 210,965 participants were initially enrolled in this cohort, each of whom completed at least one 24-h online dietary recall at baseline. Participants were excluded if they declined follow-up, had a baseline diagnosis of AAA, were pregnant, or reported implausible energy intakes (< 500 or > 3,500 kcal/day for women and < 800 or > 4,200 kcal/day for men). These cutoffs were based on previous UK Biobank and international dietary validation studies to minimize measurement error from under- or over-reporting of energy intake.[16] Participants of non–White British ancestry, defined according to both self-reported ethnicity and principal component analysis of genetic data provided by the UK Biobank, were also excluded to reduce population stratification bias. In addition, individuals with missing demographic, socioeconomic, or lifestyle data were excluded. After sequential exclusions of 612 prevalent AAA cases, 2,271 participants with implausible energy intake, 24,842 with missing baseline information, and 6,843 with incomplete genetic data, 176,397 participants remained in the final analytic sample (Supplementary Figure S1, Supplemental Digital Content, https://links.lww.com/CARES/A12).

2.2. Dietary assessment and calculation of the AHEI-2010 score

Dietary intake was assessed using the web-based 24-h dietary recall questionnaire, Oxford WebQ. Between April 2009 and June 2012, participants with valid email addresses were invited to complete up to 5 assessments.[16] The Oxford WebQ collected frequency data on the consumption of 206 food items and 32 beverages over the previous 24-h. For participants who completed more than one 24-h recall, the average intake across all available assessments was calculated and used in subsequent analyses.

To derive the AHEI-2010 score, we applied the scoring algorithm described in previous studies.[17] Briefly, component scores (ranging from 0 to 10) were assigned to 11 dietary categories. Higher scores were given for greater consumption of beneficial foods (e.g., vegetables, fruits, whole grains, nuts, and polyunsaturated fatty acids), whereas lower consumption of detrimental items (e.g., sugar-sweetened beverages, red/processed meat, trans fat, and sodium) contributed to higher scores. The detailed scoring scheme is provided in Supplementary Table 1, Supplemental Digital Content, https://links.lww.com/CARES/A12.

2.3. Polygenic risk score calculation

The PRS for AAA was constructed using the PRS–CS method, a Bayesian regression framework with continuous shrinkage priors.[18] Summary statistics were obtained from the largest GWAS of AAA conducted in European ancestry populations (GWAS Catalog ID: GCST90475994), comprising 7,642 AAA cases and 172,172 controls. Prior to PRS construction, variants with a minor allele frequency < 0.01 or imputation INFO score < 0.8 were excluded, leaving approximately 6,285,195 autosomal SNPs for analysis. PRS–CS leverages external linkage disequilibrium (LD) information from the 1,000 Genomes Project Phase 3 European reference panel (N = 503) and estimates posterior SNP effect sizes while applying continuous shrinkage priors to account for polygenicity and correlation between variants. The method was implemented in the *auto* mode, allowing the global shrinkage parameter (φ) to be learned directly from the data. Individual PRS values were calculated as the sum of the estimated posterior SNP effect sizes across all included variants for each participant. Based on the distribution of PRS values in the analytic sample, participants were categorized into three genetic risk strata: low (bottom quintile, ≤ 20%), intermediate (2nd–4th quintiles,

20%–80%), and high (top quintile, ≥ 80%). There was no sample overlap between the GWAS discovery cohort and the present UK Biobank participants.

2.4. Covariate assessment

Covariates included demographic factors (age, sex, body mass index [BMI], handgrip strength [HGS], and appendicular skeletal muscle mass [ASM]); socioeconomic indicators (employment status, Townsend deprivation index, and educational attainment); lifestyle behaviors; baseline medical history (diabetes, hypertension, dyslipidemia, sarcopenia, other cardiovascular diseases, chronic respiratory diseases, chronic kidney disease, chronic liver disease, and cancer); as well as prior use of medications and vitamin supplements. Supplementary Table 2, Supplemental Digital Content, https://links.lww.com/CARES/A12, provides the diagnostic codes for all chronic diseases.

HGS was measured in kilograms for both hands, and the average of the 2 measurements was used. Body composition was assessed at baseline using a single-frequency segmental body composition analyzer, and bioelectrical impedance analysis was applied to derive muscle and fat mass. ASM was calculated as the sum of lean soft tissue mass in both upper and lower limbs. Sarcopenia was defined according to the criteria of the Foundation for the National Institutes of Health Sarcopenia Project. Lifestyle factors included sleep pattern (categorized as poor, intermediate, or healthy), physical activity level (high, intermediate, or low), sedentary time (low, intermediate, or high), and smoking status. Detailed definitions and cutoff values are presented in Supplementary Table 3, Supplemental Digital Content, https://links.lww.com/CARES/A12.

2.5. Outcomes

The primary outcome was incident AAA (ICD-10 codes I71.3 and I71.4), identified from death registries, primary care records, hospital admissions, or self-reported diagnoses. Onset was defined as the earliest recorded diagnosis. The study endpoint was set as October 15, 2024. Follow-up was calculated from the first 24-h dietary recall (2009–2012) to AAA diagnosis, death, loss to follow-up, or study end, whichever occurred first.

2.6. Statistical analysis

Baseline characteristics were summarized as means (standard deviation [SD]) for continuous variables and counts (percentages) for categorical variables. Group comparisons were performed using one-way analysis of variance for continuous variables and chi-square tests for categorical variables. Kaplan–Meier curves were generated to illustrate the cumulative incidence of AAA across AHEI-2010 categories. Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs), with proportional hazards assumptions evaluated using Schoenfeld residuals. Three progressively adjusted models were constructed: Model 1 accounted for age, sex, education level, employment status, Townsend deprivation index, BMI, HGS and ASM; Model 2 further adjusted for baseline comorbidities, including hypertension, diabetes, dyslipidemia, sarcopenia, cardiovascular disease, cancer, chronic respiratory disease, chronic kidney disease, chronic liver disease, as well as prior use of medications and vitamin supplements; and Model 3 additionally incorporated lifestyle factors, including smoking status, physical activity, sleep pattern, and sedentary behavior. Restricted cubic spline regression was applied to assess potential nonlinear associations, and stratified analyses were conducted by age, sex, BMI category (18.5–24.9 vs. outside this range), history of hypertension, and smoking status.

Several sensitivity analyses were performed to test the robustness of the findings. To reduce the possibility of reverse causation, cases diagnosed within the first 2 years of follow-up were excluded. Participants with major baseline comorbidities, including diabetes, hypertension, dyslipidemia, cardiovascular disease, cancer, chronic respiratory disease, and chronic kidney or liver disease, were also excluded to evaluate the impact of preexisting conditions. A Fine–Gray subdistribution hazards model was applied to account for competing risks from non-AAA mortality or loss to follow-up. Multiple imputation by chained equations was used to handle missing data in demographic, socioeconomic, and lifestyle covariates. Ten imputed datasets were generated with the mice package (version 3.16.0) in *R*, including the exposure, outcome, and all covariates in the imputation model; estimates were pooled using Rubin's rules. In addition, the association between AHEI-2010 and AAA was reassessed in participants who completed at least two 24-h dietary recalls to examine the stability of dietary assessment.

To assess the potential impact of genetic susceptibility, we first applied Cox proportional hazards models to compare the risk of AAA across categories of the PRS. In subsequent analyses evaluating the PRS, all models were additionally adjusted for the top 10 genetic principal components. We further evaluated the interaction and joint effects of the AHEI-2010 score and its components with PRS on AAA incidence. Multiplicative interaction was evaluated by including a cross-product term of AHEI-2010 and PRS in the Cox models, with HRs and 95% CI reported. Additive interaction was assessed by calculating the relative excess risk due to interaction and its corresponding 95% CI. For the joint effect analysis, individuals with both high genetic risk and low AHEI-2010 scores were used as the reference group. Finally, in the joint effect analysis, participants were stratified by PRS categories, and the cumulative incidence of AAA was calculated across tertiles of the AHEI-2010 score within each PRS stratum. The absolute risk increase (ARI) of 10-year AAA incidence was estimated by comparing the

low and high AHEI-2010 score groups, and weighted least squares models were applied to test for trends in ARI across PRS categories.

All statistical analyses were conducted using *R* software (version 4.4.1; *R* Foundation for Statistical Computing). Two-sided tests were applied, and *P* values < 0.05 were considered statistically significant.

3. Results

3.1. Baseline characteristics of participants

Baseline characteristics of the participants are presented in Table 1. A total of 176,397 individuals were included in the analysis, with a mean age of 56.2 years (SD = 7.9), and 45.6% were men. Compared with participants in the lowest tertile of the AHEI-2010 score, those in the highest tertile were more likely to be women, have higher educational attainment, adhere to healthier lifestyle behaviors, and present with fewer comorbidities and lower use of medications.

3.2. Association between AHEI-2010 score and risk of AAA

During a mean follow-up of 13.3 years (calculated as the average time from baseline to AAA diagnosis, death, or censoring) a total of 619 incident AAA cases were documented. As shown in Supplementary Figure 2, Supplemental Digital Content, https://links.lww.com/CARES/A12, the AHEI-2010 scores were approximately normally distributed in the overall population, with a mean value of about 57. As shown in the Kaplan–Meier curves (Fig. 1A), higher AHEI-2010 scores were significantly associated with a lower cumulative incidence of AAA. In multivariable Cox regression analyses (Table 2), participants

Table 1

Baseline characteristics of study participants according to AHEI-2010 score categories.

Characteristics	AHEI-2010 score			*P* value
	Tertile 1	Tertile 2	Tertile 3	
N	58,799	58,799	58,799	
AHEI scores (range)	(8.4–50.7)	(50.8–63.2)	(63.3–110.0)	
Demographics				
Age (years)	55.3 ± 8.1	56.5 ± 7.8	57.0 ± 7.5	< 0.001
Male (%)	32,768 (55.7%)	26,409 (44.9%)	21,176 (36.0%)	< 0.001
Townsend deprivation index	−1.6 ± 2.8	−1.8 ± 2.7	−1.7 ± 2.7	< 0.001
University or college degree (%)	22,144 (37.7%)	26,244 (44.6%)	29,829 (50.7%)	< 0.001
Employed, student, or retired (%)	54,113 (92.0%)	54,424 (92.6%)	54,592 (92.8%)	< 0.001
BMI	27.6 ± 4.6	26.9 ± 4.4	26.0 ± 4.2	< 0.001
Grip strength (kg)	32.9 ± 11.0	31.2 ± 10.7	29.9 ± 10.3	< 0.001
ASM (kg)	25.0 ± 5.8	23.7 ± 5.5	22.5 ± 5.1	< 0.001
Lifestyle				
Never smoking (%)	31,322 (53.3%)	33,503 (57.0%)	34,560 (58.8%)	< 0.001
Physical activity (%)				
Low	18,869 (32.1%)	15,909 (27.1%)	13,281 (22.6%)	< 0.001
Moderate	19,819 (33.7%)	20,360 (34.6%)	20,364 (34.6%)	
High	20,111 (34.2%)	22,530 (38.3%)	25,154 (42.8%)	
Sleep patterns (%)				
Poor	23,438 (39.9%)	21,377 (36.4%)	18,630 (31.7%)	< 0.001
Moderate	33,348 (56.7%)	34,915 (59.4%)	36,823 (62.6%)	
Good	2,013 (3.42%)	2,507 (4.26%)	3,346 (5.69%)	
Sedentary time (%)				
High	14,619 (24.9%)	12,774 (21.7%)	11,002 (18.7%)	< 0.001
Moderate	20,304 (34.5%)	19,640 (33.4%)	18,738 (31.9%)	
Low	23,876 (40.6%)	26,385 (44.9%)	29,059 (49.4%)	
Medical history				
Hypertension (%)	17,550 (29.8%)	16,335 (27.8%)	14,943 (25.4%)	< 0.001
Diabetes (%)	3,891 (6.62%)	3,417 (5.81%)	2,999 (5.10%)	< 0.001
Dyslipidemia (%)	24,113 (41.0%)	21,162 (36.0%)	17,686 (30.1%)	< 0.001
Cancer (%)	7,418 (12.6%)	7,728 (13.1%)	7,897 (13.4%)	< 0.001
Sarcopenia (%)	33 (0.06%)	24 (0.04%)	11 (0.02%)	< 0.001
Chronic respiratory diseases (%)	7,970 (13.6%)	7,712 (13.1%)	7,267 (12.4%)	< 0.001
Chronic liver disease (%)	197 (0.34%)	175 (0.30%)	127 (0.22%)	< 0.001
Chronic kidney disease (%)	218 (0.37%)	189 (0.32%)	153 (0.26%)	< 0.001
Cardiovascular disease (%)	5,987 (10.2%)	5,718 (9.72%)	5,197 (8.84%)	< 0.001
Number of medications (SD)	2.2 ± 2.4	2.1 ± 2.3	2.0 ± 2.3	< 0.001
Vitamin use (%)	9,472 (16.1%)	15,104 (25.7%)	19,942 (33.9%)	< 0.001

P values were determined using the ANOVA test for continuous variables and the chi-square test for categorical variables.

AHEI-2010 = Alternative Healthy Eating Index-2010; ANOVA = analysis of variance; ASM = appendicular skeletal muscle mass; BMI = body mass index.

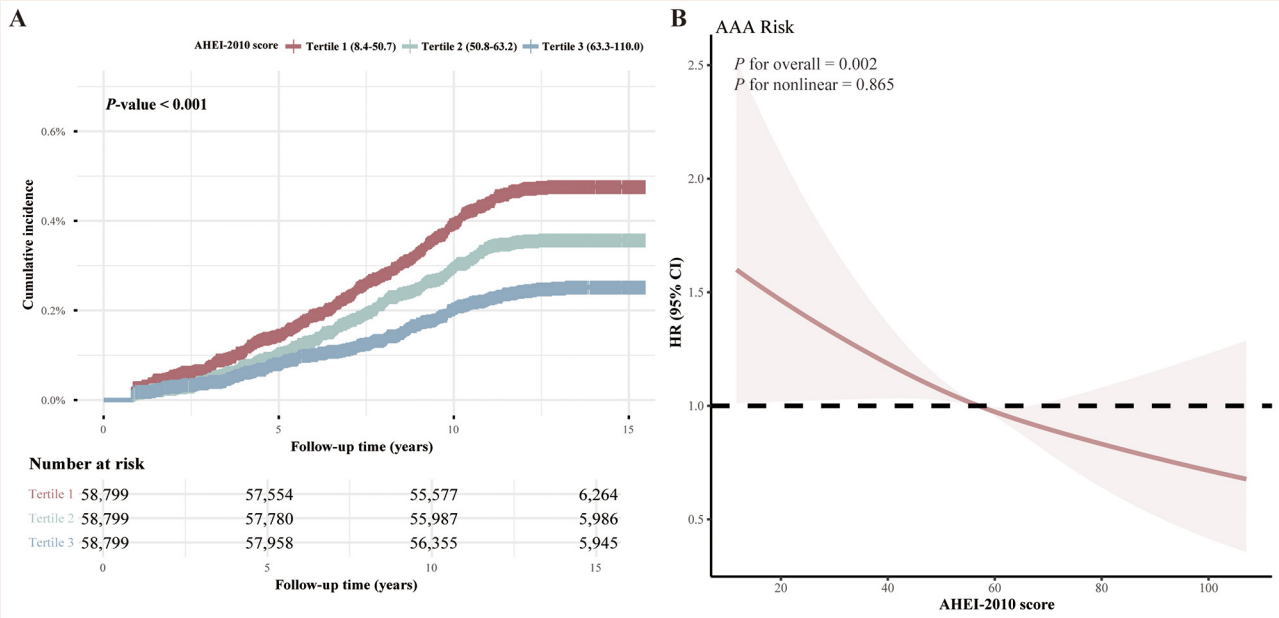


Figure 1. Association between AHEI-2010 score and risk of AAA. (A) Kaplan–Meier curves for cumulative incidence of AAA according to AHEI-2010 score categories, (B) Restricted cubic spline analysis of the association between AHEI-2010 score and AAA risk. Restricted cubic spline analysis adjusted for sex, age, education level, employment status, Townsend deprivation index, body mass index, grip strength, appendicular skeletal muscle mass, prior medication use, vitamin supplementation, medical history (encompassing hypertension, diabetes, dyslipidemia, cardiovascular and cerebrovascular diseases, cancer, sarcopenia, chronic respiratory diseases, chronic kidney disease, and chronic liver disease), smoking history, physical activity, sleep pattern, and sedentary time. AAA = abdominal aortic aneurysm; AHEI-2010 = Alternative Healthy Eating Index-2010.

Table 2

Association between the AHEI-2010 score and AAA incidence

	AHEI-2010 score HR (95% CI)				
Analysis	**Tertile 1**	**Tertile 2**	**Tertile 3**	**Per SD increment**	***P* value**
Cases/Participants	271/58,799	204/58,799	144/58,799	619/176,397	
Model 1	1 (Reference)	0.81 (0.68–0.98)	0.68 (0.55–0.83)	0.86 (0.78–0.93)	< 0.001
Model 2	1 (Reference)	0.82 (0.69–0.99)	0.69 (0.56–0.85)	0.86 (0.79–0.94)	0.001
Model 3	1 (Reference)	0.85 (0.71–1.02)	0.73 (0.59–0.89)	0.88 (0.81–0.96)	0.005

Model 1: Adjusted for sex, age, education level, employment status, Townsend Deprivation Index, body mass index, grip strength, and appendicular skeletal muscle mass. Model 2: Further adjusted for prior medication use, vitamin supplementation, and medical history (including hypertension, diabetes, dyslipidemia, cardiovascular and cerebrovascular diseases, cancer, sarcopenia, chronic respiratory diseases, chronic kidney disease, and chronic liver disease). Model 3: Additionally adjusted for smoking history, physical activity, sleep pattern, and sedentary time.
AHEI-2010 = Alternative Healthy Eating Index-2010; CI = confidence interval; HR = hazard ratio; SD = standard deviation.

in the highest tertile of the AHEI-2010 score had a significantly lower risk of AAA compared with those in the lowest tertile (HR = 0.73; 95% CI: 0.59–0.89) in the fully adjusted model. Moreover, each 1-SD increase in the AHEI-2010 score was associated with a 12% reduction in AAA risk. The proportional hazards assumption was verified using Schoenfeld residuals, with all *P* values > 0.05 (Supplementary Table 4, Supplemental Digital Content, https://links.lww.com/CARES/A12), supporting the validity and robustness of the analyses. The C-index ranged from 0.847 to 0.871, with Model 3 showing the best fit, reflected by the highest C-index and the lowest Bayesian information criterion.

Restricted cubic spline analysis (Fig. 1B) indicated a linear dose–response relationship between AHEI-2010 scores and AAA risk, with knots placed at the 10th (38.8), 50th (56.9), and 90th (75.7) percentiles of the AHEI-2010 distribution. Figure 2 presents the associations of individual AHEI-2010 components with AAA incidence. After adjustment for potential confounders, higher intakes of fruit (excluding juice) and whole grains were significantly associated with reduced AAA risk, whereas no significant associations were observed for the other dietary components. As shown in Supplementary Table 5, Supplemental Digital Content, https://links.lww.com/CARES/A12, we further categorized fruit intake (excluding fruit juice) and whole-grain intake into tertiles to examine their associations with

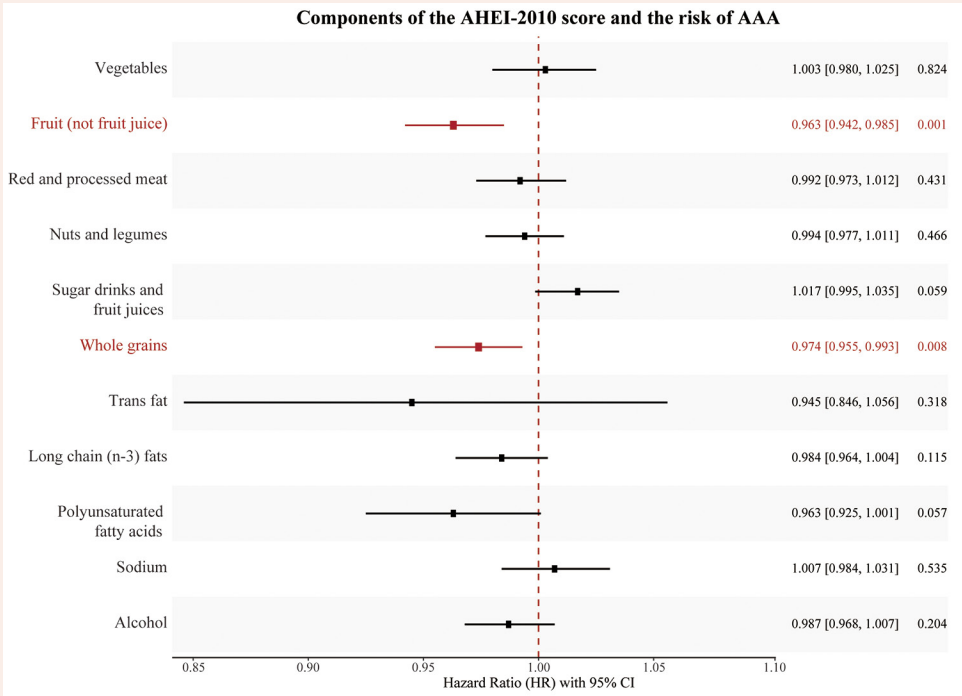


Figure 2. Associations of individual AHEI-2010 components with the risk of AAA. Analyzes were adjusted for sex, age, education level, employment status, Townsend Deprivation Index, body mass index, grip strength, appendicular skeletal muscle mass, prior medication use, vitamin supplementation, medical history (encompassing hypertension, diabetes, dyslipidemia, cardiovascular and cerebrovascular diseases, cancer, sarcopenia, chronic respiratory diseases, chronic kidney disease, and chronic liver disease), smoking history, physical activity, sleep pattern, and sedentary time. AAA = abdominal aortic aneurysm; AHEI-2010 = Alternative Healthy Eating Index-2010.

the incidence of AAA. Compared with participants in the lowest tertile, those in the highest tertile of fruit intake, whole-grain intake, or the combined 2-component score had a significantly lower risk of AAA (fruit: HR = 0.75; 95% CI: 0.63–0.91; whole grains: HR = 0.77; 95% CI: 0.64–0.92; 2-component score: HR = 0.67; 95% CI: 0.53–0.82). As shown in Supplementary Table 6, Supplemental Digital Content, https://links.lww.com/CARES/A12, in the leave-one-out analysis, the inverse association between higher AHEI-2010 scores and lower AAA risk remained stable even after excluding any single component (HR range = 0.85–0.91). These findings suggest that no individual dietary component is solely responsible for the overall association.

As shown in subgroup analyses (Supplementary Table 7, Supplemental Digital Content, https://links.lww.com/CARES/A12), no significant interactions were observed between AHEI-2010 scores and sex, age, BMI, hypertension status, or smoking history. Multiple sensitivity analyses were also conducted to test the robustness of the findings (Supplementary Table 8, Supplemental Digital Content, https://links.lww.com/CARES/A12), including exclusion of cases occurring within the first 2 years of follow-up, application of competing risk models, exclusion of participants with baseline comorbidities, and restriction of analyses to those completing at least two 24-h dietary recalls. The results were consistent with the main analyses, further supporting the robustness and reliability of our findings.

3.3. Genetic risk and the AHEI-2010 Score and its components in relation to AAA risk

As shown in Supplementary Figure 3, Supplemental Digital Content, https://links.lww.com/CARES/A12, the PRS exhibited an approximately normal distribution, with most participants' PRS values centered around a mean of −0.018. As shown in Supplementary Table 9, Supplemental Digital Content, https://links.lww.com/CARES/A12, we constructed a PRS for AAA using the PRS–CS method. Individuals in the highest quintile of genetic risk had a significantly greater risk of AAA compared with those in the lowest quintile (HR = 2.36; 95% CI: 2.10–3.36).

We further examined the potential interaction between genetic risk and the AHEI-2010 score in relation to AAA incidence. As shown in Figure 3A, no significant multiplicative or additive interactions were observed between the PRS and the AHEI-2010 score in relation to AAA risk (P value > 0.05). However, the joint association analysis (Fig. 3B) revealed a markedly elevated risk of AAA. Specifically, compared with participants with both high PRS and low AHEI-2010 scores, those with low PRS and high AHEI-2010 scores had an approximately 89%

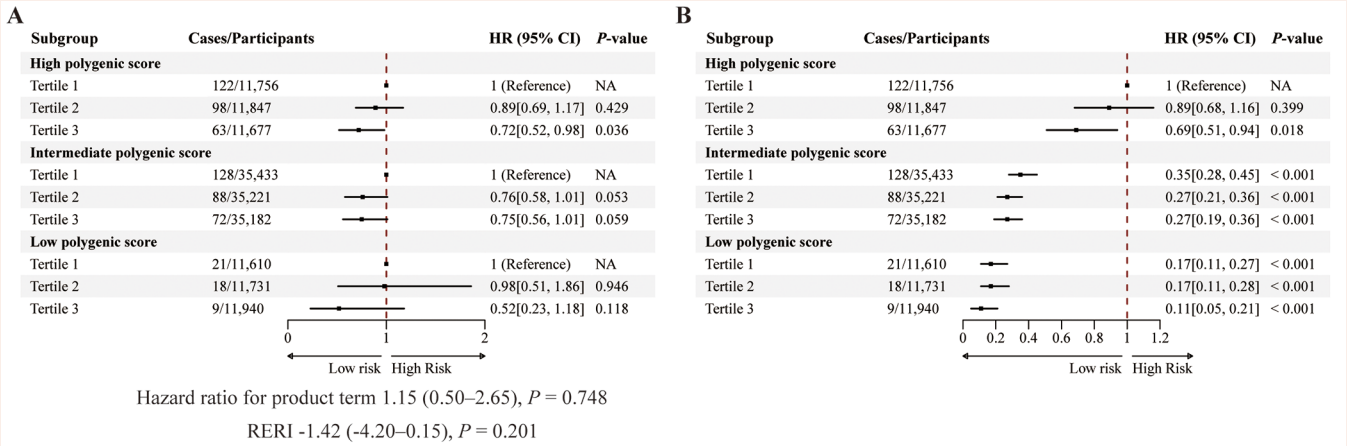


Figure 3. Interaction and joint effects of AHEI-2010 score and polygenic risk score on AAA risk. (A) Interaction of AHEI-2010 score and polygenic risk score on AAA risk. (B) Joint effects of AHEI-2010 score and polygenic risk score on AAA risk. Analyzes were adjusted for sex, age, education level, employment status, Townsend Deprivation Index, body mass index, grip strength, appendicular skeletal muscle mass, prior medication use, vitamin supplementation, medical history (encompassing hypertension, diabetes, dyslipidemia, cardiovascular and cerebrovascular diseases, cancer, sarcopenia, chronic respiratory diseases, chronic kidney disease, and chronic liver disease), smoking history, physical activity, sleep pattern, and sedentary time. AAA = abdominal aortic aneurysm; AHEI-2010 = Alternative Healthy Eating Index-2010.

lower risk of AAA (HR = 0.11; 95% CI: 0.05–0.21). Furthermore, we examined the interaction and joint effects of fruit and whole-grain intake with PRS on the incidence of AAA. As shown in Supplementary Figures S4 and S5, Supplemental Digital Content, https://links.lww.com/CARES/A12, no statistically significant interactions were observed between PRS and the intake of either component. However, compared with participants with high PRS and low intake, those with low PRS and high intake had an approximately 87% lower risk of developing AAA.

As shown in Figure 4, we further examined the joint associations of PRS and the AHEI-2010 score with the 10-year cumulative incidence of AAA. Across all PRS strata, participants with higher AHEI-2010 scores consistently exhibited lower incidence rates compared with those in the lowest tertile. The ARI between the lowest and highest AHEI-2010 tertiles was 0.11% in the low PRS group, 0.15% in the intermediate-PRS group, and 0.36% in the high-PRS group. However, no significant linear trend was observed across PRS categories (*P* for trend = 0.409).

4. Discussion

In this study, we found that higher AHEI-2010 scores were significantly associated with a lower risk of AAA, whereas higher PRS levels were strongly related to an increased risk. Although no statistically significant multiplicative interaction was detected, participants with both low PRS and high AHEI-2010 scores exhibited an approximately 89% reduced risk of AAA. Among the individual dietary components, higher intakes of fruit (excluding juice) and whole grains were also closely associated with lower AAA risk. Taken together, these findings highlight the potential importance of diet quality in the primary prevention of AAA, acting independently of, yet complementing, genetic susceptibility.

Our results extend previous research on the role of dietary patterns in vascular health. While the AHEI-2010 has been consistently linked to reduced risks of coronary heart disease, stroke, diabetes, and all-cause mortality, evidence regarding its role in AAA has been scarce.[9] This study provides novel epidemiological evidence suggesting that adherence to a high-quality diet, as measured by the AHEI-2010, is associated with a substantially lower risk of AAA. This observation complements prior literature emphasizing the adverse effects of unhealthy dietary habits on systemic inflammation, oxidative stress, and vascular remodeling-biological processes that are central to aneurysm formation and rupture.[19] Collectively, our findings suggest that, beyond age and genetic predisposition, overall dietary quality may contribute to AAA risk, underscoring the potential for preventive strategies through nutritional modification. It is worth noting that although multiple dietary assessments were conducted, they may still not accurately reflect individuals' long-term dietary patterns, which is a common limitation in long-term cohort studies. Notably, in stratified analyses, the inverse associations between AHEI-2010 and AAA risk were generally consistent across age, sex, and smoking status, although they appeared more pronounced among men and current smokers. This pattern suggests that individuals with higher baseline vascular risk may particularly benefit from greater adherence to a healthy dietary pattern.

In addition to the overall dietary score, we also evaluated the contribution of individual dietary components. Higher intakes of fruit (excluding juice) and whole grains were independently associated with reduced AAA risk,

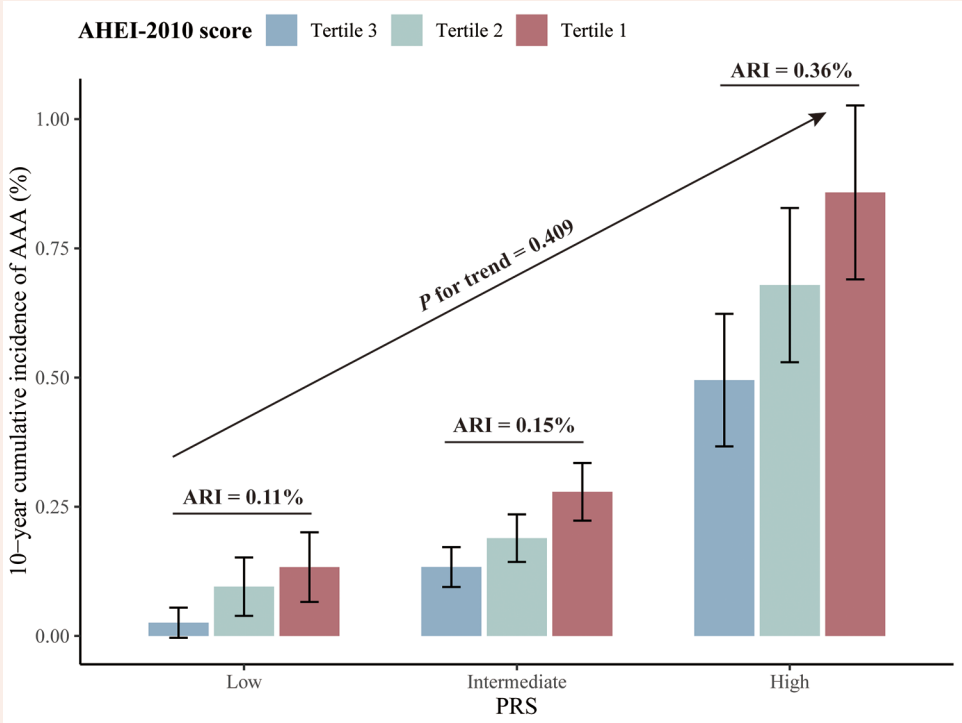


Figure 4. Ten-year cumulative incidence of AAA according to categories of polygenic risk score and AHEI-2010 score. AAA = abdominal aortic aneurysm; AHEI-2010 = Alternative Healthy Eating Index-2010; ARI = absolute risk increase; PRS = polygenic risk score.

whereas no significant associations were observed for the other components of the AHEI-2010. These results are consistent with prior studies showing that the fiber, antioxidants, and phytochemicals abundant in fruit and whole grains exert vascular protective effects through anti-inflammatory and antioxidant pathways.[20] Specifically, dietary fiber and polyphenols in fruits may reduce systemic inflammation, enhance endothelial nitric oxide bioavailability, and inhibit vascular smooth muscle cell apoptosis, whereas whole grains may help lower circulating lipid levels, improve insulin sensitivity, and alleviate oxidative stress.[19] In contrast, other components (such as vegetables, nuts, or legumes) showed no significant associations, which may reflect several factors. Notably, the combined 2-component score based on fruit and whole-grain intake showed a stronger inverse association with AAA risk than the overall AHEI-2010 score. While comprehensive indices capture overall diet quality, they may be less intuitive and harder to translate into routine practice. In contrast, emphasizing higher fruit and whole-grain consumption provides a simpler, more actionable message that may facilitate clinical counseling and improve adherence at the population level. First, the range of intake for these foods was relatively narrow with low variability in this population, potentially limiting statistical power to detect modest associations. Second, moderate collinearity existed among AHEI components—particularly between vegetables, fruits, and whole grains—which may have attenuated their independent effects when modeled simultaneously. Third, the influence of some components may operate not in isolation but as part of an overall dietary pattern, underscoring the importance of considering overall diet quality rather than individual foods. Although these findings were derived from a single database, they underscore the potential of specific dietary components–particularly fruit and whole grains-as modifiable factors for reducing AAA risk. Accordingly, further well-designed randomized controlled trials are needed to clarify the potential dose-dependent effects of specific dietary factors on the development of AAA. Furthermore, future research incorporating more detailed nutritional assessments and biomarker validation will be essential to clarify the associations between specific nutrients and AAA risk.

Our genetic susceptibility analyses further support the pivotal role of genetic risk in the development of AAA. Consistent with previous GWAS, individuals in the highest quintile of the PRS had more than a 2-fold higher risk of AAA compared with those in the lowest quintile.[21] However, our joint analyses suggest that adherence to a high-quality diet may partly attenuate the adverse impact of genetic susceptibility. Although no significant interactions with PRS were observed for the AHEI-2010 score, fruit intake, or whole grain intake-possibly due to the relatively low incidence of AAA and changes in dietary habits over time-the marked absolute risk differences between categories suggest that dietary factors may still exert meaningful modifying effects. From a public health perspective, the pattern of absolute risk differences is particularly informative. Although relative risks were broadly similar

across PRS strata, the 10-year absolute risk reduction associated with higher AHEI-2010 scores was largest in the high-PRS group (0.36%), compared with 0.11% and 0.15% in the low- and intermediate-PRS groups, respectively. This suggests that individuals at the highest genetic risk may obtain the greatest absolute benefit from improving diet quality, even in the absence of statistically significant multiplicative interaction terms. Similar gene–diet interactions have been reported in coronary artery disease, type 2 diabetes, and hypertension,[13] but this is among the first studies to investigate such relationships in AAA. These findings suggest that adherence to a healthy diet may confer meaningful protection even among genetically susceptible individuals. Nevertheless, further studies in larger cohorts are warranted to validate the potential interplay between diet and genetic predisposition.

This study has several important strengths. First, it was conducted within a large prospective cohort, which strengthens the validity and reliability of the results. Second, to our knowledge, this is the first study to systematically investigate the associations of the AHEI-2010 score and its components with the risk of AAA. Moreover, we examined the potential contributions of both genetic predisposition and dietary patterns to AAA development, offering novel insights that may inform future prevention strategies.

Despite these strengths, some limitations should be considered. Although extensive adjustments were made for potential confounders, residual confounding due to unmeasured factors cannot be completely ruled out. This also includes the possible influence of genetic batch effects in the PRS analyses. In addition, the UK Biobank does not provide detailed data on aneurysm diameter, precluding evaluation of aneurysm size as an outcome. Dietary intake was assessed through self-reported 24-h recalls, which may not accurately capture long-term dietary exposures, particularly as dietary habits can change over time. Fourth, because some data were missing and only participants who completed the dietary assessments were included, our analyses may be subject to potential selection bias. However, this concern is likely limited, as the analytic sample size remained large, and previous evaluations of the Oxford WebQ subsample have shown that it is broadly comparable to the overall UK Biobank cohort in terms of age, sex, and socioeconomic characteristics.[22] Finally, the study was limited to White participants in the UK who completed dietary assessments, which may restrict the generalizability of our findings to other populations.

5. Conclusion

In summary, this large prospective study suggests that adherence to a healthier dietary pattern, as indicated by a higher AHEI-2010 score, was associated with a lower risk of AAA, with fruit and whole grain intake showing the strongest inverse relationships. Although genetic susceptibility remains a strong determinant of AAA risk, participants with both favorable dietary patterns and low genetic risk had the lowest observed risk. These findings indicate that maintaining a healthy diet may help mitigate AAA risk across genetic backgrounds, warranting further investigation in mechanistic and interventional studies.

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Ethical statement

Ethics approval for the UK Biobank study was granted by the North West Multi-centre Research Ethics Committee (Reference: 11/NW/0382). All participants provided written informed consent prior to enrollment. The present secondary analysis was exempted from additional ethical review and conducted in accordance with the principles of the Declaration of Helsinki.

Conflicts of interest

The authors have no conflicts of interest to disclose.

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Data availability statement

This study was conducted under UK Biobank application number 145937. The UK Biobank data are available on application to the UK Biobank.

Author contributions

Conceptualization: Xinyi Liu.
Data curation: Zhiyu Qiao, Haiou Hu.
Formal analysis: Xinyi Liu, Yipeng Ge, Chengnan Li.
Methodology: Xinyi Liu. Software: Zhiyu Qiao.
Supervision: Junming Zhu. All authors reviewed and approved the manuscript.

AI statement

The authors declare that no Gen AI was used in the creation of this manuscript.

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