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Impact of a comprehensive healthy lifestyle score on a broad spectrum of cardiometabolic and related diseases and its incremental value for risk prediction based on the UK biobank database

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Abstract

Background Cardiometabolic and related diseases (CMDs) represent a major global disease burden. While traditional risk factors are well-established, the role of lifestyle factors in primary prevention has not been sufficiently studied.

Methods This study analyzed data from 148,155 participants in the UK Biobank without baseline CMDs. Cox proportional hazards models were used to assess the impact of healthy lifestyle factors (non-current smoking, non-excessive drinking, regular physical activity, healthy diet, and healthy sleep) on 11 types of CMDs (coronary heart disease, type 2 diabetes, atrial fibrillation, hypertension, stroke, heart failure, Alzheimer's disease, non-alcoholic fatty liver disease, valvular heart disease, cardiomyopathy, and pulmonary hypertension). Participants were categorized into four groups based on weighted lifestyle scores. Improvements in predictive models after incorporating lifestyle factors were evaluated.

Results Most individual healthy lifestyle factors were associated with a lower risk of CMDs, while regular physical activity was not independently associated with composite CMD risk in the fully adjusted model; healthy sleep contributed the largest weight to the composite lifestyle score (β = 0.342). Adhering to a healthier lifestyle demonstrated a significant and dose–response association with a reduced risk of composite CMDs. In the fully adjusted model, compared to the "very unhealthy" group, the "very healthy" group exhibited a significantly lower

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risk of developing CMDs (HR: 0.70, 95% CI: 0.67–0.74). Similar inverse associations were observed across several individual CMDs, with the strongest association observed for pulmonary hypertension (HR: 0.34, 95% CI: 0.24–0.50). Incorporating the lifestyle score into traditional risk models was associated with modest improvement in predictive performance. The findings were generally consistent across subgroups and sensitivity analyses.

Conclusions These findings suggest that adherence to a comprehensive healthy lifestyle, including adequate sleep, is associated with reduced risk of various CMDs and may improve risk stratification. The particularly strong association observed for pulmonary hypertension also supports further investigation of lifestyle patterns across a broader range of cardiometabolic and related conditions.

Trial registration Not applicable.

Keywords Cardiometabolic diseases, Healthy lifestyle, Primary prevention, Risk stratification

Introduction

Cardiometabolic and related diseases (CMDs), encompassing a broad spectrum of cardiovascular and metabolic disorders, represent the leading cause of morbidity, mortality, and significant economic burden worldwide, posing a severe global health challenge [1, 2]. Primary prevention of these diseases is a cornerstone of public health policy. Research indicates that modifiable lifestyle factors, including a healthy diet, regular physical activity, smoking cessation, and non-excessive drinking, may play a potentially significant role in this regard. Therefore, the composite healthy lifestyle score has been developed as a crucial epidemiological tool to quantify the cumulative effects of these behaviors on health outcomes. However, traditional lifestyle indices often have significant limitations. For instance, they typically assign equal weight to each component, failing to reflect the varying degrees of impact individual behaviors have on disease risk [3–5]. Moreover, they frequently overlook the critical factor of sleep, despite growing evidence that sleep is an independent determinant in maintaining metabolic homeostasis and cardiovascular function [6, 7]. Furthermore, current understanding may still be incomplete regarding whether the inverse association of a healthy lifestyle consistently manifest across a broader spectrum of CMDs.

This study aims to utilize data from 148,155 participants in the UK Biobank, a large-scale prospective cohort without baseline CMDs, to explore the relationship between a healthier lifestyle and the risk of this broad disease spectrum, which further compares these findings with existing clinical risk models, providing potential new scientific evidence for personalized risk stratification and the optimization of public health intervention strategies.

Methods

Study population

The UK Biobank is a prospective cohort with over 500,000 participants of adults aged 37 to 73 years old recruited from 22 centers across the UK between 2006 and 2010. Participants provided information on lifestyle and other potentially health-related aspects through extensive baseline questionnaires, interviews, and physical measurements. All participants provided written informed consent for this study. The UK Biobank study has approval from the North West Multi-Centre Research Ethics Committee. Additional information regarding the initiative is available in various published works. [8]

Among the 502,367 participants at baseline in UK Biobank, we excluded participants with CMDs at recruitment (n = 169,745); missing data on target lifestyle factors (n = 181,013); or missing data on any of the covariates (n = 3,454). The final sample size was 148,155. The flow chart is shown in Fig. 1.

Healthy lifestyle

This study defined a healthy lifestyle using five modifiable components: smoking, alcohol consumption, physical activity, diet, and sleep. Smoking status was simply categorized as current or non-current [9]. For alcohol, we converted weekly consumption of various beverages into standard units based on UK National Health Service guidelines [10] and Office for National Statistics survey data [11]. Adhering to the healthy guideline of "non-excessive drinking" (< 14 units per week) was the desired behavior. A healthy diet was assessed with a score based on American Heart Association guidelines [12], which assigned one point for more than 2 of the following three items, otherwise 0 points: (1) Total fruit and vegetable intake: ≥ 4.5 pieces or servings per day (1 serving being 3 tablespoons); (2) Total fish intake ≥ 2 times per week; (3) Red meat intake ≤ 5 times per week and processed meat intake ≤ 2 times per week. For physical activity, one point was assigned for "regular physical activity", which was defined as meeting the benchmarks set by the 2019 UK Physical Activity Guidelines (≥ 150 min of moderate or ≥ 75 min of vigorous activity per week) [13]. A point for "high-quality sleep" was given if a participant achieved a score of 4–5 on a composite sleep index that included duration, chronotype, insomnia, snoring, and daytime dozing [14]. Finally, these five factors were

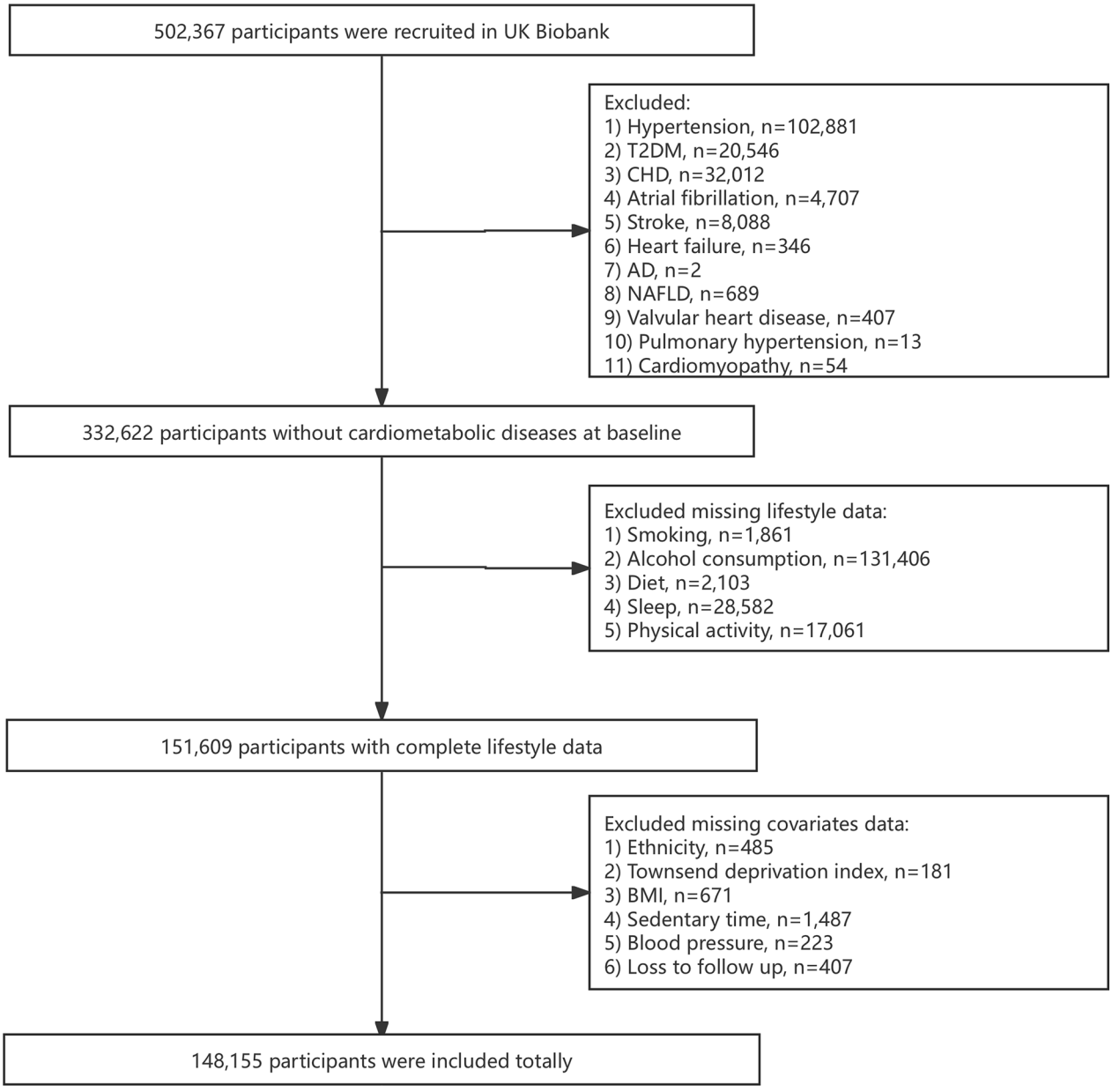


Fig. 1 Flowchart. Abbreviations: AD: Alzheimer's disease; BMI: Body mass index; CHD: Coronary heart disease; NAFLD: Non-alcoholic fatty liver disease; T2DM: Type 2 diabetes mellitus

combined to generate a weighted healthy lifestyle score. Additional details of this calculation are provided in the Supplementary Method 2.

Outcome assessment

The outcome measured in this study was the occurrence of 11 types of CMDs in patients without CMDs at baseline. The UK Biobank collected participants' data from self-reported diseases diagnosed by doctors and hospital inpatient records. Diagnoses were coded according to the International Classification of Disease versions 10 and the Office of Population Censuses and Surveys Classification of Interventions and Procedures version 4. CMDs in this study was defined as the occurrence of ≥ 1 of the following: coronary heart disease (CHD) [15], type 2 diabetes mellitus (T2DM) [16], atrial fibrillation (AF) [17], hypertension [15], stroke [15], heart failure (HF) [18], Alzheimer's disease (AD) [19], Non-alcoholic fatty liver disease (NAFLD) [20], valvular heart disease (VHD) [21], cardiomyopathy [22], and pulmonary hypertension [23]. The date of CMDs was the earliest date of occurrence for any of these cardiometabolic diseases. Detailed information is provided in Supplementary Table 2.

Participants recorded as lost to follow-up before the construction of the final analytic cohort were excluded during the sample selection process, as shown in Fig. 1. For participants included in the final analytic cohort, follow-up was calculated from the date of baseline assessment at the UK Biobank assessment center. Participants were followed until the first diagnosis of any study-defined CMDs, death, or the administrative end of available follow-up on March 7, 2023, whichever occurred first. For participants who developed more than one CMD during follow-up, the earliest diagnosis date was used as the event date for the composite outcome. Participants who did not develop any CMDs were censored at the date of death or March 7, 2023.

Other covariates

The current study included variates of age, sex, ethnicity, body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), Townsend deprivation index (TDI), sedentary time, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), and hemoglobin A1c (HbA1c). Detailed information is provided in Supplementary Method 1, and Supplementary Table 1.

Traditional models

This study compared the predictive ability between traditional risk prediction models and specified healthy lifestyle models incorporating five lifestyle factors described in our study: (1) The Framingham Risk Score (FRS) for CHD prediction [24]; (2) The Finnish Diabetes Risk Score (FINDRISC) for T2DM [25]; (3) The CHARGE-AF model for AF risk [26]; (4) The QStroke algorithm applied for stroke risk. [27]

Statistical analysis

Continuous variables were expressed as mean ± standard deviation (SD) if they satisfied normal distribution, otherwise as median [interquartile range (IQR)]. Categorical variables were expressed as percentages (%). Group comparisons were performed using χ^2 tests, the Wilcoxon–Mann–Whitney test, or Student's t-test, depending on the data type.

To construct the weighted healthy lifestyle score, we first estimated the β coefficients of the five healthy lifestyle factors in relation to composite cardiometabolic disease using Cox proportional hazards models in the full analytic cohort. The absolute values of these β coefficients were then divided by the sum of the absolute β coefficients to generate the relative weight for each lifestyle factor. Each participant's weighted lifestyle score was calculated by multiplying each binary lifestyle factor by its corresponding weight and then summing the weighted components. Therefore, higher scores indicated a healthier overall lifestyle pattern. To assess the robustness of this data-driven weighting approach, we conducted sensitivity analyses using weights derived from a randomly selected one-third subset of the analytic cohort and using a simple unweighted lifestyle score. To visually demonstrate the contribution of each lifestyle factor to the risk score, we constructed a nomogram based on the results of the Cox model. Subsequently, each β coefficient was standardized by dividing it by the sum of all β coefficients, then multiplied by the binary variable representing individual lifestyle (unhealthy = 0, healthy = 1) and summed to obtain the final score for each participant. Based on this score, participants were divided into four groups: very unhealthy group ([0, 0.25), reference group), unhealthy group ([0.25, 0.50)), healthy group ([0.50, 0.75)), and very healthy group ([0.75, 1.0)).

We employed Cox proportional hazards models to calculate hazard ratios (HRs) and 95% confidence intervals (CIs) for assessing the associations between different lifestyle groups, individual lifestyle factors, and composite CMD as well as 11 specific CMDs. Three progressively adjusted models were constructed in the multivariate analysis: Model 1 adjusted for age, sex, and ethnicity; Model 2 additionally adjusted for TDI, BMI, and sedentary time based on Model 1; Model 3 further adjusted for DBP, SBP, TC, TG, LDL, HDL, and HbA1c based on Model 2. Kaplan–Meier survival curves were plotted to visualize disease-free survival differences across lifestyle groups, with comparisons made using the log-rank test. To evaluate the incremental predictive value of the weighted lifestyle score, we compared traditional risk models with and without the weighted lifestyle score for 10-year incident disease risk. Time-dependent AUC, continuous net reclassification improvement, and integrated discrimination improvement were calculated at the 10-year horizon to assess changes in discrimination and reclassification while accounting for censoring during follow-up. To verify the robustness of the results, multiple subgroup and sensitivity analyses were conducted. Subgroup analyses were stratified by age, sex, ethnicity, BMI, Townsend deprivation index, and sedentary time, with interaction tests performed. Sensitivity analyses included: deriving β coefficients from a random one-third of the dataset and validating them in the remaining two-thirds; analyzing using an unweighted summation score; and excluding participants who experienced outcome events or died within the first two years of follow-up to minimize reverse causality bias. Given the multiple comparisons across disease-specific outcomes, false discovery rate correction was applied using the Benjamini–Hochberg method. For disease-specific outcome analyses, false discovery rate correction was applied across the 33 fully adjusted comparisons, including

three lifestyle-score group comparisons with the very unhealthy group as the reference across 11 individual cardiometabolic and related disease outcomes. Subgroup analyses and prediction-performance comparisons were considered exploratory and interpreted cautiously.

All statistical analyses were two-sided, with $P < 0.05$ considered statistically significant.

Results

Baseline characteristics

This study ultimately included 148,155 UK Biobank participants without CMD at baseline. The median follow-up period was 13.2 years. Based on weighted healthy lifestyle scores (Supplementary Method 3), participants were divided into four groups: Very Unhealthy Group (n = 7,083), Unhealthy Group (n = 28,623), Healthy Group (n = 62,148), and Very Healthy Group (n = 50,301). As shown in Table 1, compared to groups with less healthy lifestyles, those with healthier lifestyles were generally younger, had a higher proportion of females, lower BMI and blood pressure levels, and a lower Townsend deprivation index. Also, participants with higher lifestyle scores had a better lipid profile compared to the very unhealthy group. Baseline characteristics of included and excluded participants were shown in Supplementary Table 3.

Construction of healthy lifestyle score and its association with composite CMD risk

To construct a weighted healthy lifestyle score, this study first used Cox proportional hazards models to assess the associations between five lifestyle factors and the risk of composite CMD, with the regression coefficients (β coefficients) serving as weights. The results of this model indicated that healthy sleep contributed the highest weight (standardized β coefficient = 0.342) (Supplementary Method 3). The nomogram constructed for this weighted model showed that the line segment representing healthy sleep was the longest (Supplementary Fig. 1), consistent with the β coefficient results.

Subsequently, the fully adjusted Cox models (Supplementary Table 4) revealed that non-current smoking (HR: 0.77, 95% CI: 0.74–0.80), non-excessive drinking (HR: 0.91, 95% CI: 0.89–0.93), healthy diet (HR: 0.95, 95% CI: 0.93–0.97), and healthy sleep (HR: 0.89, 95% CI: 0.86–0.90) were all independently associated with a reduced risk of composite CMD. No significant association was observed between physical activity and composite CMD risk (HR: 1.00, 95% CI: 0.97–1.02). The frequency distribution plot demonstrated that the weighted healthy lifestyle score, integrating these factors, exhibited a broad multimodal distribution in the study population (Supplementary Fig. 2).

Table 1 Baseline characteristics of participants by healthy lifestyle score

	Total (n = 148,155)	Very unhealthy (n = 7,083)	Unhealthy (n = 28,623)	Healthy (n = 62,148)	Very healthy (n = 50,301)	P_{value}
Age, years, median [IQR]	55.00 [48.00, 61.00]	52.00 [46.00,60.00]	55.00 [48.00, 61.00]	56.00 [49.00, 62.00]	55.00 [48.00, 61.00]	< 0.001
Sex, male, n (%)	70,787 (47.78)	5003 (70.63)	17,494 (61.12)	31,686 (50.98)	16,604 (33.01)	< 0.001
Ethnicity, white, n (%)	144,946 (97.83)	6876 (97.08)	28,015 (97.88)	60,867 (97.94)	49,188 (97.79)	< 0.001
BMI, kg/m^2, median [IQR]	25.80 [23.53, 28.44]	26.77 [24.24, 29.50]	26.53 [24.24, 29.07]	25.98 [23.74, 28.60]	25.07 [22.94, 27.60]	< 0.001
Sedentary time, median [IQR], h	5.00 [3.00, 6.00]	5.00 [4.00, 7.00]	5.00 [4.00, 6.00]	5.00 [4.00, 6.00]	4.00 [3.00, 5.00]	< 0.001
Blood pressure (BP)						
SBP, mmHg, median [IQR]	135.00 [123.00, 147.00]	136.00 [125.00, 148.00]	137.00 [126.00, 149.00]	135.00 [124.00, 148.00]	132.00 [121.00, 145.00]	< 0.001
DBP, mmHg, median [IQR]	81.00 [74.00, 88.00]	82.00 [76.00, 89.00]	82.00 [76.00, 89.00]	81.00 [75.00, 88.00]	79.00 [73.00, 86.00]	< 0.001
Townsend deprivation index, median [IQR]	-2.51 [-3.85, -0.33]	-1.58 [-3.37, 1.45]	-2.24 [-3.69, 0.25]	-2.56 [-3.88, -0.48]	-2.68 [-3.95, -0.69]	< 0.001
TC, mmol/L [IQR]	5.80 [5.13, 6.53]	5.81 [5.14, 6.53]	5.86 [5.19, 6.60]	5.81 [5.15, 6.54]	5.74 [5.08, 6.47]	< 0.001
TG, mmol/L [IQR]	1.37 [0.98, 2.00]	1.67 [1.13, 2.48]	1.50 [1.05, 2.19]	1.38 [0.98, 2.00]	1.27 [0.93, 1.82]	< 0.001
LDL-C, mmol/L [IQR]	3.61 [3.09, 4.17]	3.65 [3.10, 4.21]	3.65 [3.12, 4.23]	3.61 [3.10, 4.18]	3.56 [3.05, 4.13]	< 0.001
HDL-C, mmol/L [IQR]	1.48 [1.24, 1.76]	1.36 [1.16, 1.63]	1.45 [1.22, 1.73]	1.48 [1.25, 1.76]	1.51 [1.26, 1.77]	< 0.001
HbA1c, mmol/mol [IQR]	34.40 [32.10, 36.60]	34.90 [32.50, 37.30]	34.50 [32.20, 36.80]	34.20 [32.00, 36.50]	34.30 [32.10, 36.60]	< 0.001
Healthy lifestyle factors						< 0.001
Non-current smoking, n (%)	133,536 (90.13)	2306 (32.56)	21,458 (74.97)	60,906 (98.00)	48,866 (97.15)	
Non-excessive drinking, n (%)	77,317 (52.19)	0 (0.00)	2201 (7.69)	24,815 (39.93)	50,301 (100.00)	
Regular physical activity, n (%)	110,124 (74.33)	3379 (47.71)	22,590 (78.92)	45,990 (74.00)	38,165 (75.87)	
Healthy diet, n (%)	110,462 (74.56)	2679 (37.82)	21,073 (73.62)	46,250 (74.42)	40,460 (80.44)	
Healthy sleep, n (%)	93,840 (63.34)	0 (0.00)	4964 (17.34)	38,575 (62.07)	50,301 (100.00)	

BMI, body mass index; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; HDL-C, high-density lipoprotein cholesterol; IQR, interquartile range; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides

Table 2 The association between healthy lifestyle and cardiometabolic and related diseases

	Total (Event)	Model 1		Model 2		Model 3	
		HR (95% CI)	P_{value}	HR (95% CI)	P_{value}	HR (95% CI)	P_{value}
Very unhealthy	7,083(2,171)	1.00 [Reference]		1.00 [Reference]		1.00 [Reference]	
Unhealthy	28,623(8,097)	0.83 (0.79–0.87)	< 0.001	0.85 (0.81–0.90)	< 0.001	0.87 (0.83–0.91)	< 0.001
Healthy	62,148 (15,241)	0.71 (0.68–0.74)	< 0.001	0.75 (0.72–0.79)	< 0.001	0.77 (0.73–0.80)	< 0.001
Very Healthy	50,301 (10,222)	0.62 (0.59–0.65)	< 0.001	0.69 (0.66–0.73)	< 0.001	0.70 (0.67–0.74)	< 0.001

Model 1: adjusted for sex, age, ethnicity (white, non-white)

Model 2: additionally adjusted for Townsend deprivation index, body mass index, sedentary time

Model 3: additionally adjusted for SBP, TC, TG, DBP, LDL, HDL and HbA1c

CI, confidence interval; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; HDL, high-density lipoprotein cholesterol; HR, hazard ratio; LDL, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides

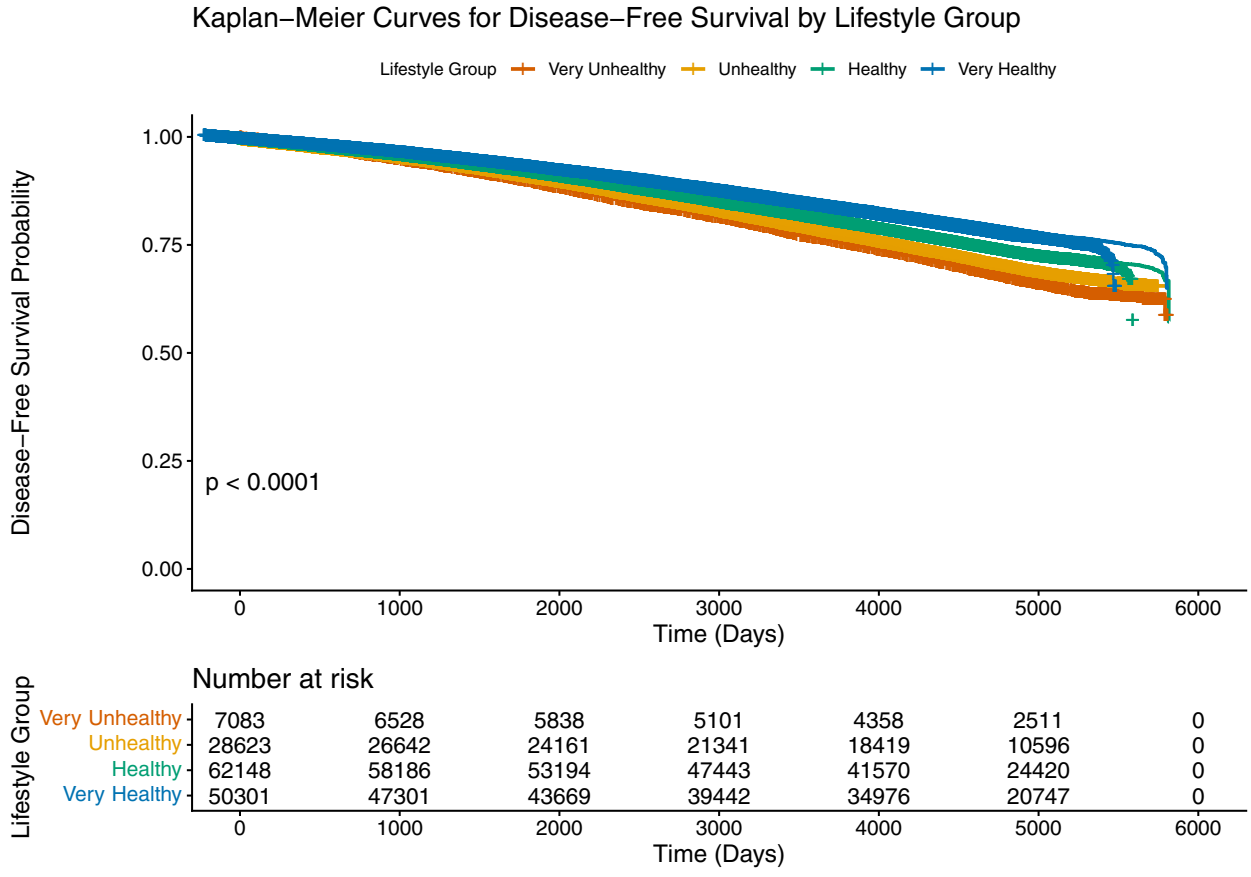


Fig. 2 Kaplan–Meier curves for cardiometabolic and related disease-free survival stratified by lifestyle groups

Healthy lifestyle and cardiometabolic diseases

The fully adjusted Cox proportional hazards model revealed a significant dose–response relationship between the weighted healthy lifestyle score and the risk of composite CMD (Table 2). Using the "very unhealthy" group as the reference, the HRs for the "unhealthy", "healthy", and "very healthy" groups progressively decreased with higher lifestyle scores, with values of 0.87 (95% CI: 0.83–0.91), 0.77 (95% CI: 0.73–0.80), and 0.70 (95% CI: 0.67–0.74), respectively. Kaplan–Meier survival analysis similarly demonstrated that healthier lifestyles were associated with higher disease-free survival rates (P < 0.0001) (Fig. 2). When this analysis was applied to 11 specific CMDs, the inverse association persisted for most individual diseases, including T2DM, CHD, stroke, HF, hypertension, and VHD (Supplementary Table 5). The most pronounced effect was observed for pulmonary hypertension, where the "very healthy" group exhibited a 66% lower risk compared to the "very unhealthy" group (HR: 0.34, 95% CI: 0.24–0.50). After false discovery rate correction for multiple disease-specific comparisons, most of these associations remained statistically significant, whereas the associations for AD, NAFLD, and cardiomyopathy were not statistically significant.

Table 3 Comparison of model performance for 10-year incident disease risk before and after adding the weighted lifestyle score

Exposures	Continuous NRI (95% CI)	P_{value}	IDI (95% CI)	P_{value}	AUC (95% CI)	ΔAUC	P_{value}
CHD							
[a]Basic model	Ref	-	Ref	-	0.7283 (0.7229–0.7338)	-	Ref
Basic model + weighted lifestyle score	0.161(0.123–0.187)	< 0.001	0.0004 (0.0002–0.0006)	< 0.001	0.7295 (0.7240–0.7350)	0.0012	< 0.001
T2DM							
[b]Basic model	Ref	-	Ref	-	0.8001 (0.7934–0.8070)	-	Ref
Basic model + weighted lifestyle score	0.277 (0.239–0.298)	< 0.001	0.0017 (0.0011–0.0023)	< 0.001	0.8013 (0.8001–0.8142)	0.0012	< 0.001
Atrial fibrillation							
[c]Basic model	Ref	-	Ref	-	0.6694 (0.6643–0.6756)	-	Ref
Basic model + weighted lifestyle score	0.198 (0.165–0.221)	< 0.001	0.0012 (0.0010–0.0019)	< 0.001	0.6959 (0.6872–0.7034)	0.0265	< 0.001
Stroke							
[d]Basic model	Ref	-	Ref	-	0.7122 (0.7101- 0.7213)	-	Ref
Basic model + weighted lifestyle score	0.210 (0.175–0.229)	< 0.001	0.0014 (0.0011–0.0018)	< 0.001	0.7211 (0.7145- 0.7298)	0.0089	< 0.001

[a] Basic model for CHD is the Framingham Risk Score

[b] Basic model for T2DM is the Finnish Diabetes Risk Score (FINDRISC)

[c] Basic model for atrial fibrillation is the CHARGE-AF model

[d] Basic model for stroke is the QStroke algorithm

AUC, area under the curve; CHD, coronary heart disease; CI, confidence interval; IDI, integrated discrimination improvement index; NRI, continuous net reclassification index; Ref., reference; T2DM, type 2 diabetes mellitus

The strongest inverse association was observed for pulmonary hypertension, where the "very healthy" group had a lower risk than the "very unhealthy" group (FDR-adjusted P < 0.001).

Lifestyle score improves risk prediction models

To evaluate the incremental value of the weighted healthy lifestyle score constructed in this study for existing clinical risk prediction models, we assessed four major CMD by calculating the AUC, continuous NRI, and IDI (Table 3). Adding the weighted lifestyle score to traditional models resulted in statistically significant improvements in AUC, continuous NRI, and IDI for 10-year incident disease risk. Taking CHD as an example, when the weighted lifestyle score was added to the Framingham Risk Score, the AUC increased from 0.7283 (95% CI: 0.7229–0.7338) to 0.7295 (95% CI: 0.7240–0.7350), corresponding to a small absolute increase in AUC of 0.0012 (P < 0.001). The continuous NRI for this model was 0.161 (95% CI: 0.123–0.187; P < 0.001), and the IDI was 0.0004 (95% CI: 0.0002–0.0006; P < 0.001). Similar statistically significant improvements were observed for T2DM, AF, and stroke after incorporating the weighted lifestyle score. However, the absolute increases in AUC were generally modest.

Sensitivity and subgroups analyses

In all three sensitivity analyses, the association between a healthy lifestyle and a reduced risk of composite CMD remained robust (Supplementary Table 6): using β coefficients derived from the training set (a random one-third of the population) to test the validation set (the remaining two-thirds of the population); analyzing with a simple unweighted lifestyle score count; and excluding participants who experienced events within the first two years of follow-up to eliminate potential reverse causation. Supplementary Fig. 3 displays the frequency distribution of the weighted healthy lifestyle score in the validation set (the remaining two-thirds of the population). Supplementary Fig. 4 shows the frequency distribution of the simple count-based total healthy lifestyle score for the entire population.

Subgroup analyses showed that the inverse association between a healthier lifestyle and composite CMD risk was generally consistent across most subgroups (Supplementary Table 7). Stratified analyses by sex, ethnicity, BMI, and sedentary time showed no statistically significant interactions (P for interaction = 0.121, 0.738, 0.281, and 0.291, respectively). However, significant interactions were observed for age (P for interaction < 0.001) and Townsend deprivation index (P for interaction = 0.026), suggesting that the magnitude of the association may differ by age and socioeconomic status.

Discussion

This study, based on a large prospective cohort, systematically evaluated the association between a comprehensive healthy lifestyle score and the risk of 11 CMDs. The main findings can be summarized into four key points: (1) A weighted score composed of five lifestyle factors showed a significant dose–response relationship with a reduced composite risk of multiple CMDs; (2) Healthy sleep was the lifestyle factor with the largest contribution weight among all assessed lifestyle factors; (3) the strongest inverse association was observed for pulmonary hypertension, although this finding should be interpreted

cautiously given the broad ICD-based outcome definition; and (4) adding the weighted lifestyle score to existing clinical risk prediction models provided modest incremental improvement in predictive performance.

It should be noted that the outcomes included in this study represent a broad and clinically heterogeneous spectrum of cardiometabolic and related diseases. Some outcomes, including CHD, T2DM, hypertension, stroke, HF, and AF, are well-established cardiometabolic conditions. Other outcomes, such as AD, NAFLD, VHD, cardiomyopathy, and pulmonary hypertension, may be more appropriately considered cardiometabolic-related conditions because they may share vascular, metabolic, inflammatory, or lifestyle-associated risk pathways. Therefore, the composite outcome in this study should be interpreted as a broad cardiometabolic and related disease spectrum rather than a single homogeneous disease entity. This broader framework allowed us to explore whether healthier lifestyle patterns were associated with a wider range of clinically relevant outcomes, while the disease-specific analyses helped characterize potential heterogeneity across outcomes.

Our main finding—that a healthier combined lifestyle score was associated with a lower risk of CMDs—is consistent with previous research and evidence from meta-analyses [9, 28, 29]. For example, previous studies have shown that lifestyle modifications can be associated with lower incidence of major adverse cardiovascular events, CHD, and T2DM [30–34]. However, this study extends in several aspects. We evaluated up to 11 different CMDs, including some less-studied but clinically equally important conditions, such as VHD and pulmonary hypertension. This systematic assessment suggests that the associations of healthier lifestyle patterns may not be limited to the most common CMDs but may extend across a broader pathophysiological spectrum. Moreover, the inverse association of a healthy lifestyle is not only observed in European and American countries but also demonstrates a similar impact in China [35, 36]. The meta-analysis also confirmed that these inverse associations are generally consistent and significant across individuals from different continents, ethnic groups, and socioeconomic backgrounds. [29, 37]

Many previous studies have adopted a simple summation scoring method when constructing scores, assigning equal weight to each lifestyle factor. For instance, research by Hoevenaar-Blom et al. [38] constructed lifestyle scores by summing scores for factors such as diet, physical activity, smoking, alcohol consumption, and BMI, which demonstrated a significant negative correlation between higher scores (indicating a healthier lifestyle) and the risk of cardiovascular disease. Although this method is effective, its underlying assumption is that each behavior contributes equally to risk reduction. Similar to the studies by Xie et al. [15] and Han et al. [39], this research employs a weighted scoring system based on the β coefficients of factors in the Cox model. This data-driven approach more accurately reflects the differential impacts of various behaviors, revealing that in our cohort, non-current smoking and healthy sleep exert particularly strong influences. This methodological advantage avoids the potential misclassification that may arise from simple summation scoring—for instance, a physically active smoker could receive the same score as a sedentary individual who never smokes.

In our weighted model, healthy sleep had the largest contribution among the five lifestyle factors, suggesting that sleep may play a particularly important role in cardiometabolic health. This finding is clinically relevant because sleep disturbance may affect multiple pathways related to cardiometabolic disease, including blood pressure regulation, glucose metabolism, autonomic balance, inflammation, and neuroendocrine function. From a public health perspective, it also supports the view that sleep should be considered alongside smoking, diet, and physical activity in prevention strategies, rather than being treated as a secondary lifestyle factor. In this regard, our results are closely aligned with the American Heart Association's "Life's Essential 8", which places sleep alongside diet, physical activity, nicotine exposure, body weight, blood lipids, blood glucose, and blood pressure as a core component of cardiovascular health [40]. The prominent contribution of sleep in our analysis provides additional population-based support for this framework and reinforces the importance of integrating sleep health into routine cardiometabolic prevention efforts. Additionally, the meta-analysis of prospective studies revealed a U-shaped relationship between sleep duration and cardiovascular outcomes, indicating that both insufficient and excessive sleep are associated with increased risk [41, 42]. The present findings are consistent with the hypothesis that healthy sleep may represent an important lifestyle component associated with cardiometabolic health. [43] Regarding other lifestyle components, our findings on smoking, alcohol consumption, and diet align with established evidence. The harms of smoking are unequivocal, and our results are consistent with other studies demonstrating that non-current smoking is crucial for cardiovascular health [44]. We found a inverse association of a healthy diet, which is also supported by dietary guidelines advocating for diets rich in fruits, vegetables, whole grains, and limited in processed foods [45–47]. However, in our fully adjusted model, regular physical activity did not maintain an independent statistically significant association with the composite CMDs outcome. This finding should be interpreted cautiously and in the context of the analytic framework used in the present study [48]. The fully adjusted model included metabolic and behavioral

covariates such as BMI, HbA1c, lipid measures, and sedentary time, and such extensive adjustment may have attenuated the observed association. In addition, physical activity was assessed by self-report and categorized according to guideline-based thresholds, which may have reduced sensitivity to differences in activity level. It should also be noted that the composite CMD outcome included 11 heterogeneous diseases, and the strength of association for physical activity may vary across specific endpoints. Therefore, this result should be understood as the fully adjusted association for the composite outcome in our cohort, rather than as evidence against the established importance of physical activity in cardiometabolic prevention. The broader literature and current guidelines continue to support regular physical activity as a key component of disease prevention. [49, 50]

A particularly noteworthy finding of our study was the strong inverse association between a very healthy lifestyle and incident pulmonary hypertension. Participants in the very healthy lifestyle group had a lower risk of ICD-defined pulmonary hypertension than those in the very unhealthy group, representing the strongest association among the individual outcomes examined. However, this finding should be interpreted cautiously because our outcome definition was based on ICD-coded pulmonary hypertension rather than pulmonary arterial hypertension specifically. Pulmonary hypertension is a clinically heterogeneous condition that includes multiple clinical groups with distinct underlying causes and pathophysiological features. [51, 52]

Several potential explanations may partly account for the observed association. Healthy lifestyle patterns may reflect a more favorable overall cardiometabolic profile, lower obesity burden, better vascular health, and reduced cardiopulmonary stress, which may be relevant to pulmonary hypertension across different clinical contexts. In addition, healthy sleep may be particularly relevant, as sleep-related breathing disorders can affect pulmonary haemodynamics and are discussed in relation to pulmonary hypertension in patients with and without chronic respiratory disease [53]. Nevertheless, because of the observational design and the broad ICD-based pulmonary hypertension definition, these explanations should be considered hypothesis-generating rather than causal evidence. Further studies using more specific pulmonary hypertension phenotyping are needed to determine whether healthy lifestyle patterns are differentially associated with specific pulmonary hypertension subtypes, including pulmonary arterial hypertension.

The null or inconsistent associations observed for AD, NAFLD, and cardiomyopathy should also be interpreted cautiously. These results may reflect true differences in the relevance of lifestyle factors across disease pathways, but they may also be influenced by outcome ascertainment. For example, NAFLD and hypertension are commonly diagnosed or monitored in outpatient and primary care settings, whereas our outcome definitions relied on self-reported physician diagnoses and hospital inpatient records. Therefore, milder or asymptomatic cases may have been missed. Similarly, ICD-defined pulmonary hypertension, valvular heart disease, and cardiomyopathy may include heterogeneous clinical entities, which could dilute or obscure subtype-specific associations. These issues may partly explain why associations were stronger and more consistent for several traditional cardiometabolic outcomes than for some related conditions.

Adding the weighted lifestyle score to traditional models resulted in statistically significant improvements in AUC, continuous NRI, and IDI. However, the absolute increases in AUC were generally modest, indicating that the incremental predictive value of the score should be interpreted cautiously. Therefore, these findings should be viewed as evidence of modest improvement in risk stratification rather than substantial enhancement of clinical prediction. The potential value of the weighted lifestyle score may lie in its ability to summarize multiple modifiable behaviors, including sleep, in a simple and interpretable way. Further external validation and clinical utility analyses are needed to determine whether incorporating this score can meaningfully improve risk prediction or decision-making in practice.

This study has several strengths, including its large sample size, prospective design, long-term follow-up, and comprehensive assessment of 11 different CMDs. A series of rigorous subgroup and sensitivity analyses further confirmed the robustness of our findings. Nevertheless, some limitations must also be acknowledged. First, as an observational study, it cannot establish causality. Second, lifestyle factors were assessed through self-reported questionnaires, which may introduce recall bias and social desirability bias. Third, lifestyle was assessed at baseline and may have changed during follow-up. However, in the absence of major health events, core lifestyle habits tend to remain stable in adulthood for most individuals [54]. Fourth, the UK Biobank cohort is not representative of the general UK population; participants were healthier and predominantly of White European descent (97.8% in our sample). While this does not affect the internal validity of the observed associations, caution should be exercised when generalizing the findings to more diverse ethnic groups, as different populations may have distinct risk profiles. Fifth, pulmonary hypertension was identified using ICD codes rather than detailed clinical phenotyping, haemodynamic measurements, or adjudicated subtype classification. Thus, this outcome reflects ICD-defined pulmonary hypertension and should not be interpreted as pulmonary arterial hypertension

specifically. Given the clinical heterogeneity of pulmonary hypertension, potential misclassification and subtype heterogeneity may have influenced the observed association, which should therefore be considered hypothesis-generating. Sixth, outcome misclassification and under-ascertainment may have occurred because disease outcomes were identified using self-reported physician diagnoses and hospital inpatient records. Conditions often diagnosed or managed outside inpatient settings, such as hypertension and non-alcoholic fatty liver disease, may have been under-ascertained. In addition, ICD-based definitions of pulmonary hypertension, VHD, and cardiomyopathy may include clinically heterogeneous entities rather than subtype-specific diagnoses. Seventh, the weighted healthy lifestyle score was derived from Cox regression coefficients estimated in the analytic cohort, and the same cohort was used to evaluate its associations and predictive performance. Although sensitivity analyses using weights derived from a randomly selected subset and using an unweighted lifestyle score yielded broadly consistent results, potential optimism or overfitting due to data-driven weighting cannot be fully excluded. External validation in independent cohorts is needed before this weighted score can be applied in clinical risk prediction. Eighth, although the weighted lifestyle score provided statistically significant incremental predictive value beyond traditional risk models, the absolute increases in the area under the curve were generally small. Therefore, these findings should be interpreted as modest improvement in risk stratification rather than substantial enhancement of clinical prediction. Further external validation and clinical utility analyses are needed before the weighted lifestyle score can be considered for practical risk prediction. Ninth, the composite outcome included a broad and clinically heterogeneous group of cardiometabolic and related diseases. Although this approach allowed us to examine associations across a wider disease spectrum, it may also obscure outcome-specific differences and should not be interpreted as representing a single homogeneous disease process. Tenth, selection bias may have occurred because the main analysis was based on participants with complete data on lifestyle factors and covariates. The final analytic cohort may not fully represent the entire UK Biobank cohort, and the generalizability of the findings should be interpreted with caution. Finally, although false discovery rate correction was applied to disease-specific outcome analyses, subgroup analyses and prediction-performance comparisons were considered exploratory and should be interpreted cautiously.

Conclusion

In conclusion, in this large prospective cohort study, adherence to a comprehensive healthy lifestyle was associated with a lower risk of a broad spectrum of cardiometabolic and related diseases. Among the five lifestyle factors, healthy sleep contributed the greatest weight to the composite lifestyle score, underscoring the importance of incorporating sleep into comprehensive lifestyle assessment. The weighted lifestyle score also provided modest incremental value for 10-year risk prediction beyond traditional risk models. These findings highlight the potential relevance of integrated lifestyle assessment for cardiometabolic and related disease risk stratification and support further investigation of comprehensive lifestyle patterns in disease prevention.

Abbreviations

AD	Alzheimer's disease
AF	Atrial fibrillation
AHA	American heart association
AUC	Area under the curve
BMI	Body mass index
CHARGE-AF	The CHARGE-AF model
CHD	Coronary heart disease
CI	Confidence interval
CMDs	Cardiometabolic diseases
DBP	Diastolic blood pressure
FINDRISC	The Finnish diabetes risk score
FRS	The Framingham risk score
HbA1c	Hemoglobin A1c
HDL	High-density lipoprotein cholesterol
HF	Heart failure
HR	Hazard ratio
IDI	Integrated discrimination improvement index
LDL	Low-density lipoprotein cholesterol
NAFLD	Non-alcoholic fatty liver disease
NRI	Continuous net reclassification index
QStroke	The QStroke algorithm
SBP	Systolic blood pressure
T2DM	Type 2 diabetes mellitus
TC	Total cholesterol
TG	Triglycerides
TDI	Townsend deprivation index
VHD	Valvular heart disease

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13019-026-04485-z.

Additional file 1.

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Author contributions

Xinyue Chen: Writing—original draft, Software, Investigation, Data curation. Sen Han: Writing—original draft, Software, Data curation. Yating Chen: Validation, Investigation. Sheng Yuan: Validation, Investigation. Jining He: Validation, Investigation. Zixiang Ye: Validation, Investigation. Yanjun Song: Supervision, Methodology, Conceptualization. Kefei Dou: Writing—review & editing, Methodology, Conceptualization.

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Data availability
Researchers interested in accessing the data used in this study can apply for access to the UK Biobank by visiting their website (https://www.ukbiobank.ac.uk/enable-your-research/apply-for-access) and submitting an application that includes a research protocol summary and requested data fields. Upon approval by the UK Biobank management team and payment of applicable fees, researchers will be granted access to the dataset.

Declarations

Ethics approval and consent to participate
UK Biobank has been approved by the National Research Ethics Committee of the National Health Service (NHS). All participants signed informed consent.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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