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FULL RESEARCH PAPER
CVD risk factors

Cardiovascular health and life expectancy free of atherosclerotic cardiovascular disease among patients with type 2 diabetes: a cohort study

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Aims The effect of cardiovascular health (CVH) on the lifetime risk of atherosclerotic cardiovascular disease (ASCVD) in type 2 diabetes (T2DM) patients is unclear. This study examines how CVH is associated with ASCVD-free life expectancy in diabetic patients compared to non-diabetics.

Methods and results This cohort study analysed 246 183 UK Biobank adults, including 10 579 (4.3%) with type 2 diabetes. CVH was measured using the Life's Essential 8 score, which was categorized into low, moderate, and high levels. The main outcome was life expectancy free of ASCVD. For participants with diabetes, the average follow-up was 11.6 years for ASCVD incidence and 5.0 years for mortality after ASCVD. During these times, there were 2211 new ASCVD cases and 591 deaths post-diagnosis. At the age of 40, women with low CVH were expected to live 39.1 (95% confidence interval, CI, 38.6–39.7) years free from ASCVD, which increased to 43.3 years (42.7–43.8) for those with high CVH, while non-diabetic women were predicted to have an ASCVD-free life expectancy of 44.8 years (44.2–45.3). For men at the age of 40, the corresponding numbers were 32.8 years (32.2–33.4), 38.5 years (37.9–39.1), and 39.0 years (38.4–39.6). Of the total life expectancy at age 40, women with low CVH spent 87.5% (95% CI, 87.1–87.9%) of their remaining years without ASCVD, compared to 89.6% (89.2–89.9%) for those with ideal CVH. Women without T2DM spent 89.7% (89.4–90.0%) of their life expectancy free from disease. For men at the same age, the corresponding percentages were 82.9% (82.4–83.4%) for low CVH, 85.6% (85.2–86.0%) for ideal CVH, and 84.4% (84.0–84.8%) for those without T2DM.

Conclusion Higher levels of CVH in individuals with diabetes extend their life expectancy free from ASCVD, bringing it closer to that of individuals without diabetes. We have provided an important addition to the current evidence advocating for CVH-enhancing strategies in diabetic patients.

Lay summary This study evaluated the association between CVH and ASCVD-free life expectancy in patients with T2DM compared to non-diabetic individuals.

- High level of CVH in patients with T2DM reduced the risk of ASCVD incidence and mortality, making their outcomes comparable to non-diabetic participants.
- High CVH of diabetics increased both the length of ASCVD-free life expectancy and the proportion of ASCVD-free years in overall life expectancy compared to non-diabetics.

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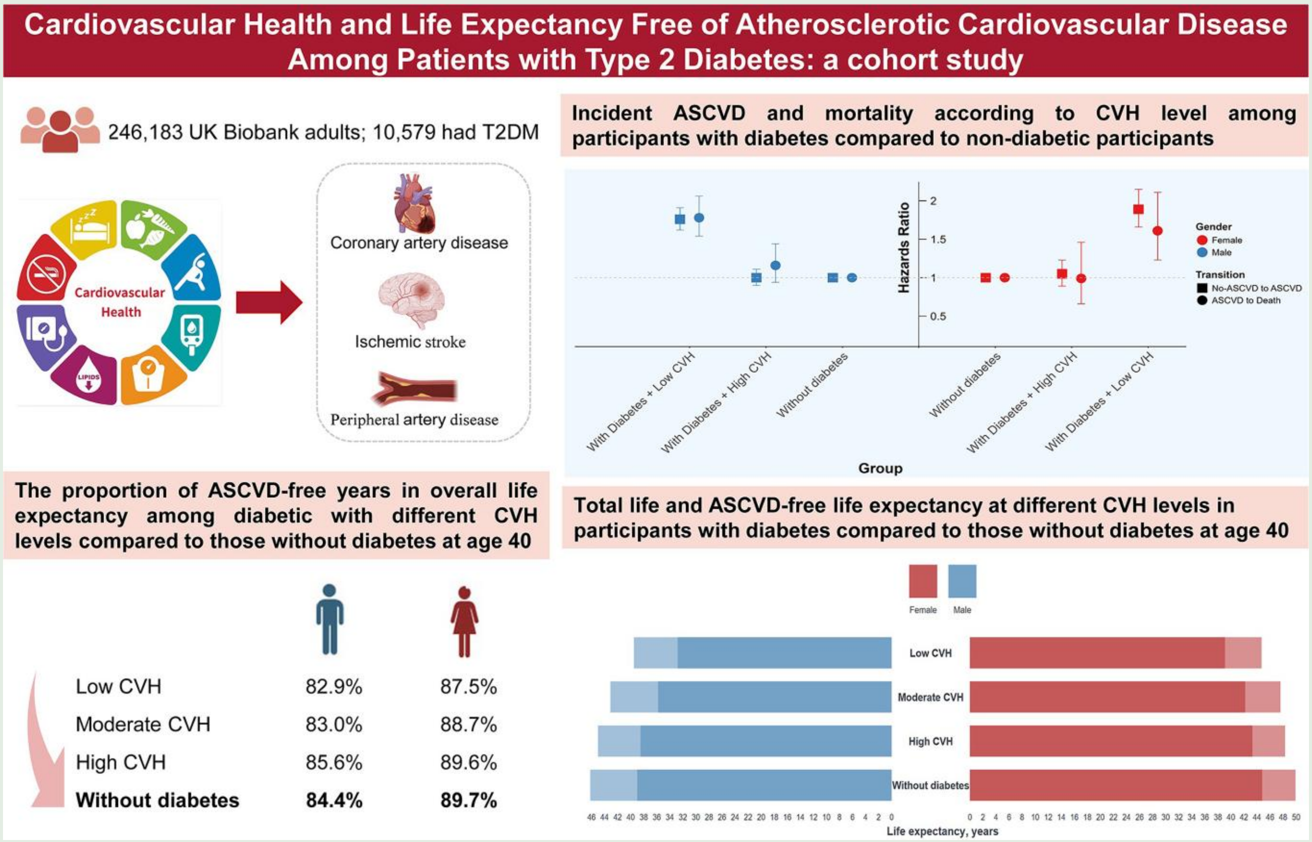
Graphical Abstract



T2DM, type 2 diabetes mellitus; ASCVD, atherosclerotic cardiovascular disease; CVH, cardiovascular health.

Keywords cardiovascular health • Atherosclerotic cardiovascular disease • Multistate model • Life expectancy • Diabetic patients

Introduction

Diabetes poses a significant global health challenge, impacting ∼1 in 11 adults, with 90% of cases being type 2 diabetes mellitus (T2DM).[1] Research has demonstrated that T2DM significantly elevates the risk of atherosclerotic cardiovascular disease (ASCVD), the primary cause of mortality and morbidity among T2DM patients,[2] and is associated with substantial reductions in life expectancy.[3] Consequently, there is growing interest in identifying cost-effective preventive strategies for ASCVD among T2DM patients in recent years.[4–6]

In 2022, the American Heart Association (AHA) introduced Life's Essential 8 (LE8), an updated cardiovascular health (CVH) metric that integrates lifestyle factors and biological measurements, including blood lipids, blood pressure, and blood glucose levels.[7] Previous studies have demonstrated that T2DM patients with higher CVH scores have lower risks of cardiovascular diseases[8,9] and mortality.[10,11] However, studies that examine the relationship between CVH and overall health quality related to ASCVD—taking into account both incident ASCVD and mortality together—in individuals with diabetes are lacking. Life expectancy free from ASCVD is such a comprehensive metric, and assessing years of life spent without ASCVD will provide easily understood information for policymakers and the public when planning future healthcare needs.

This study utilizes a multistate model to evaluate the association between CVH and life expectancy free from ASCVD among diabetic individuals, in comparison to non-diabetic individuals, within a large UK cohort. Our objective is to address critical knowledge gaps by exploring how improvements in CVH can mitigate the adverse overall health impacts related to diabetes.

Method

Study population

The UK Biobank is a large, population-based cohort study with detailed protocols available publicly.[12] Approximately 500 000 individuals, aged 40–69 years, were recruited from 22 evaluation centres across England, Scotland, and Wales between 2006 and 2010. Upon enrolment, participants completed questionnaires and interviews, underwent physical examinations, and provided blood samples. Mortality and disease diagnoses were tracked through linkage to the national death registers and hospital inpatient records. All participants provided written informed consent. UK Biobank study received ethical approval from the National Information Governance Board for Health and Social Care, the North West Multi-center Research Ethics Committee in England and Wales, and the Community Health Index Advisory Group in Scotland (http://www.ukbiobank.ac.uk/ethics/). The present research has been conducted using the UK Biobank Resource (www.ukbiobank.ac.uk) under application number 86898. We excluded participants who had prevalent ASCVD or cancer, those diagnosed with type 1 diabetes at baseline or follow-up, or those with incomplete baseline data on age, sex, and CVH metrics.

Diabetes diagnosis

Participants with type 2 diabetes at baseline were identified by integrating various data sources, according to previous studies.[13] The presence of diabetes was determined through self-reports, hospital records, prescriptions for glucose-lowering or diabetes-related medications, or a glycated haemoglobin A1c (HbA1c) level of 6.5% or higher.

Cardiovascular health measurements

The CVH score was calculated based on eight baseline factors: diet, physical activity, nicotine exposure, sleep pattern, body mass index, blood lipids, blood glucose, and blood pressure. Dietary assessments were aligned with the AHA guidelines for cardiovascular disease, diabetes, and obesity.[14] Specifically, the diet score encompassed 12 food groups: fruits, vegetables, whole grains, refined grains, fish, dairy products, vegetable oil, processed meats, unprocessed red meats, industrial *trans* fats, sugar-sweetened beverages, and sodium. Data on physical activity, nicotine exposure, sleep habits, and medication use were self-reported. Measurements of height, weight, and blood pressure were taken on-site at the assessment centre, while serum cholesterol and glycated haemoglobin levels were analysed centrally in a laboratory. The calculation and scoring details of each CVH metric adhered to the AHA criteria,[7] with minor adjustments for the UK Biobank data, as detailed in a previous publication (see Supplementary material online, Table *S1*).[15] Each CVH metric was scored on a scale from 0 to 100, and the overall CVH score was the average of the scores across all eight metrics. A higher score represents better CVH, and the overall CVH was divided into low (lowest tertile), moderate (second tertile), and high (highest tertile) levels, based on the distribution among diabetic participants. This classification ensured more participants per category in the diabetic group, improving statistical power.

Covariates

We selected covariates based on those commonly used in previously published, similar studies.[9,16,17] Covariates including age, ethnicity, Townsend Deprivation Index (TDI), family history of diabetes, ASCVD, and cancer, as well as the use of aspirin, antihypertensive, and lipid-lowering drugs were collected via a self-administered touchscreen questionnaire. The TDI is a continuous variable, with higher values indicating lower socioeconomic status. This index incorporates variables such as car ownership, household overcrowding, occupation, and employment status. To handle missing covariate data, we utilized multivariate imputation by chained equations.

Outcome

ASCVD outcome was defined as a composite of coronary artery disease, ischaemic stroke, and peripheral artery disease. The identification of ASCVD incidence and mortality was achieved through linkage with hospital admission records and death registries. Outcomes were classified according to the International Classification of Diseases, Tenth Revision (ICD-10) and Ninth Revision (ICD-9), as well as the Office of Population Censuses and Surveys Classification of Interventions and Procedures, Version 4, following previous studies.[18,19] A detailed definition of outcomes is provided in Supplementary material online, Table *S2*.

Statistical analysis

Person-time for each participant was calculated from the enrolment date until the occurrence of an ASCVD diagnosis, death, withdrawal, or the end of the follow-up period (31 August 2022, for Scotland; 31 October 2022, for England; and 31 May 2022, for Wales), whichever occurred first. Population-based multistate life tables were employed to estimate life expectancy free of ASCVD among individuals with diabetes compared to those without diabetes. In constructing the multistate life table, three health states were defined: (1) absence of ASCVD, (2) onset of ASCVD, and (3) death. Three potential state transitions were analysed: (1) from non-ASCVD to incident ASCVD, (2) from non-ASCVD to death, and (3) from ASCVD diagnosis to death among those who developed ASCVD during the study period. Only the participants' initial transition into a new state was recorded, with no transitions back to a previous state being considered.

Firstly, we assessed the relationship between CVH and transitions using Gompertz multistate models among participants with diabetes compared to those without diabetes. The multistate survival models were adjusted for covariates, including age, ethnicity, the Townsend Deprivation Index, a family history of diabetes, ASCVD, and cancer, and the use of aspirin, antihypertensive, and lipid-lowering drugs. Additionally, for the HR of transition 3, the age at ASCVD diagnosis was further adjusted.

Secondly, we determined the overall sex- and age-specific transition rates for each transition by employing survival analysis with a Gompertz distribution, utilizing age as the time scale. Thirdly, we calculated the baseline prevalence of CVH levels in diabetic patients by sex and in 10-year age groups, as well as the prevalence of participants free from disease. Finally, we combined the overall transition rates, HRs by CVH among diabetic patients compared to non-diabetic individuals, and the prevalence of each CVH level in diabetic patients, along with the prevalence of non-diabetic participants, to calculate weighted transition rates. These rates were then utilized to construct multistate life tables for participants with and without diabetes, extending to age 100. The proportion of life expectancy free from ASCVD was estimated by dividing the life expectancy lived without ASCVD by the total life expectancy at a given age. The 95% confidence intervals (CIs) of the life expectancy estimates were calculated using Monte Carlo simulation (parametric bootstrapping) with 2000 runs.

To ensure the robustness of our findings, we conducted several sensitivity analyses: (1) using waist circumference instead of BMI to construct CVH score, acknowledging research that indicates higher BMI correlates with increased survival in type 2 diabetes patients (a score of 100 points is assigned for a female waist circumference below 80 cm and a male waist circumference below 94 cm; 50 points for waist circumferences between 80 and 87.9 cm for females and between 94 and 101.9 cm for males; no points for waist measurements exceeding 88 cm for females and 102 cm for males); (2) treating CVH as a continuous score by dividing the overall CVH score by every 10 points after the low CVH level, and conducting the analysis within diabetic patients; (3) excluding events occurring in the first 2 years; (4) excluding individuals diagnosed with incident diabetes during follow-up; (5) excluding individuals diagnosed with diabetes before the age of 30 to further minimize the potential influence of type 1 diabetes; (6) Adjusting for additional potential confounders, including education, income, and depression; (7) repeating the analyses without imputing missing covariate values.

Statistical analyses were performed using R, version 4.1.3. All statistical tests were two-sided, with significance defined as $P < 0.05$.

Table 1 Baseline characteristics of participants with and without diabetes

Characteristics[a]	Women				Men			
	With diabetes			Without diabetes	With diabetes			Without diabetes
	Low CVH (*n* = 1305)	Moderate CVH (*n* = 1308)	High CVH (*n* = 1439)	(*n* = 124 645)	Low CVH (*n* = 2278)	Moderate CVH (*n* = 2268)	High CVH (*n* = 1981)	(*n* = 110 959)
Age (years)	57.4 ± 7.33	58.9 ± 7.35	57.9 ± 8.14	55.5 ± 7.99	57.7 ± 7.34	59.5 ± 7.08	59.8 ± 7.13	55.6 ± 8.18
White ethnicity	1154 (88.4)	1121 (85.7)	1200 (83.4)	119 038 (95.5)	2040 (89.6)	2063 (91.0)	1694 (85.5)	106 064 (95.6)
Townsend Deprivation Index	−0.3 ± 3.35	−0.7 ± 3.33	−1.1 ± 3.14	−1.5 ± 2.91	−0.4 ± 3.37	−0.9 ± 3.26	−1.2 ± 3.13	−1.5 ± 3.00
CVH score								
Total CVH	47.3 ± 6.67	60.5 ± 2.84	72.5 ± 5.22	68.0 ± 10.56	47.1 ± 6.63	60.3 ± 2.83	71.4 ± 4.70	64.8 ± 10.21
Diet	29.0 ± 15.14	34.8 ± 15.27	39.7 ± 15.38	33.6 ± 15.13	25.0 ± 16.27	30.7 ± 16.04	38.1 ± 16.34	28.3 ± 16.84
Physical activity	41.53 ± 41.73	74.3 ± 35.11	91.1 ± 21.81	76.9 ± 35.48	43.8 ± 42.43	76.2 ± 35.00	91.0 ± 21.36	78.1 ± 35.22
Nicotine exposure	64.8 ± 35.84	80.5 ± 26.81	88.2 ± 19.47	77.2 ± 30.52	56.2 ± 36.69	71.7 ± 29.53	84.4 ± 21.25	73.9 ± 32.96
Sleep pattern	77.73 ± 26.73	86.1 ± 21.78	92.7 ± 15.01	89.7 ± 18.71	80.2 ± 24.56	87.9 ± 19.37	93.5 ± 14.03	89.7 ± 18.14
Body mass index	26.0 ± 24.98	39.3 ± 28.14	66.5 ± 24.06	73.3 ± 28.59	33.9 ± 24.96	46.0 ± 26.53	66.2 ± 24.84	68.4 ± 25.81
Blood lipid	40.7 ± 29.48	54.1 ± 27.52	66.6 ± 24.06	48.1 ± 29.68	47.1 ± 28.39	60.9 ± 25.34	72.5 ± 20.10	44.8 ± 27.86
Blood glucose	77.5 ± 30.55	88.4 ± 20.06	94.9 ± 12.59	100.00	73.5 ± 31.35	87.7 ± 20.67	94.0 ± 14.26	100.00
Blood pressure	21.1 ± 20.47	26.9 ± 23.55	40.0 ± 25.31	44.2 ± 26.85	17.5 ± 19.29	21.6 ± 20.85	31.9 ± 24.25	35.5 ± 25.45
Family history								
Diabetes	652 (50.0)	685 (52.4)	716 (49.8)	27 105 (21.7)	988 (43.4)	1003 (44.2)	927 (46.8)	21 892 (19.7)
ASCVD	859 (65.8)	883 (67.5)	932 (64.8)	72 907 (58.5)	1362 (59.8)	1346 (59.3)	1194 (60.3)	59 954 (54.0)
Cancer	492 (37.7)	501 (38.3)	474 (32.9)	44 026 (35.3)	834 (36.6)	857 (37.8)	676 (34.1)	39 316 (35.4)
Medication								
Antihypertensive	784 (60.1)	801 (61.2)	657 (45.7)	18 123 (14.5)	1451 (63.7)	1495 (65.9)	1149 (58.0)	19 121 (17.2)
Glucose-lowering	626 (48.0)	682 (52.1)	692 (48.1)	0.00	1188 (52.2)	1293 (57.0)	1107 (55.9)	0.00
Lipid-lowering	746 (57.2)	830 (63.5)	812 (56.4)	9670 (7.8)	1421 (62.4)	1566 (69.0)	1409 (71.1)	14 377 (13.0)
Aspirin	414 (31.7)	436 (33.3)	438 (30.4)	8109 (6.5)	855 (37.5)	989 (43.6)	845 (42.7)	11 422 (10.3)
Diabetes duration (years)	5.2 ± 6.08	5.4 ± 6.69	5.5 ± 6.92	0.00	6.4 ± 8.12	6.3 ± 8.01	6.5 ± 8.82	0.00
Presence of prediabetes[b]	0.00	0.00	0.00	16 067 (12.9)	0.00	0.00	0.00	13 853 (12.5)
Age of ASCVD diagnosis (years)	67.9 ± 7.72	69.6 ± 7.34	68.6 ± 7.87	68.0 ± 7.75	66.7 ± 7.73	68.5 ± 7.03	69.1 ± 6.82	66.8 ± 7.88

CVH, cardiovascular health; ASCVD, atherosclerotic cardiovascular disease.
[a]Data were mean (standard deviation) or count (percentage) for continuous and categorical variables.
[b]Prediabetes was defined as a glycated haemoglobin (HbA1c) level of 5.7–6.4% in participants without a prior diagnosis of diabetes.

Table 2 Incident ASCVD and mortality according to CVH level among participants with diabetes compared with non-diabetic participants

Transition	CVH level	Women		Men	
		No. of cases/person-years	HR (95% CI)	No. of cases/person-years	HR (95% CI)
Incident ASCVD	CVH level in diabetic patients				
	Low	256/15 553.9	1.89 (1.66–2.15)	652/24 932.4	1.76 (1.62–1.91)
	Moderate	190/15 859.8	1.21 (1.04–1.40)	570/225 571.2	1.33 (1.21–1.45)
	High	164/17 742.6	1.05 (0.89–1.23)	379/23 127.3	1.00 (0.90–1.11)
	Without diabetes	8173/1 595 596.0	1 [reference]	14 501/1 366 986.0	1 [reference]
Mortality among those without ASCVD	CVH level in diabetic patients				
	Low	90/13 540.9	1.65 (1.33–2.05)	224/20 448.7	1.84 (1.60–2.12)
	Moderate	92/14 386.8	1.48 (1.20–1.84)	204/21 489.3	1.39 (1.20–1.61)
	High	87/16 499.2	1.38 (1.11–1.71)	149/20 410.2	1.17 (0.99–1.38)
	Without diabetes	4506/1 531 401.0	1 [reference]	5617/1 257 828.0	1 [reference]
Mortality among those with ASCVD	CVH level in diabetic patients				
	Low	62/1226.1	1.61 (1.23–2.11)	217/3239.8	1.78 (1.54–2.06)
	Moderate	34/969.3	1.05 (0.74–1.49)	156/2896.8	1.25 (1.05–1.47)
	High	27/848.9	0.99 (0.66–1.46)	95/1914.9	1.16 (0.94–1.44)
	Without diabetes	1244/40 941.6	1 [reference]	2814/74 081.8	1 [reference]

ASCVD, atherosclerotic cardiovascular disease; CVH, cardiovascular health; HR, hazard ratio; CI, confidence interval.
Multistate survival analysis using the Gompertz distribution was utilized to compute hazard ratios (HRs) and 95% confidence intervals (CIs), with the absence of diabetes serving as the reference. The analysis was adjusted for age, ethnicity, the Townsend Deprivation Index, a family history of diabetes, ASCVD, and cancer, and the use of aspirin, antihypertensive, and lipid-lowering drugs. Additionally, for the HR of transition 3, the age at ASCVD diagnosis was further adjusted.

Results

Cohort characteristics

The study included 246 183 participants after excluding 256 172 individuals who had type 1 diabetes, prevalent ASCVD or cancer, or had missing data on key variables (CVH factors, age, and sex) (see Supplementary material online, Figure *S1*). Among the 246 183 participants, 10 579 (4.3%) had T2DM at baseline. For participants with diabetes, the mean follow-up durations were as follows: 11.6 years (SD = 3.4) for the incidence of ASCVD, 5.0 years (SD = 4.0) for mortality post-ASCVD, and 12.7 years (SD = 2.2) for mortality in the absence of ASCVD. During these periods, there were 2211 new ASCVD cases, 591 deaths following an ASCVD diagnosis, and 846 deaths without prior ASCVD diagnosis. In contrast, participants without diabetes had a mean follow-up of 12.6 years (SD = 2.5) for ASCVD incidence, 5.1 years (SD = 4.0) for mortality post-ASCVD, and 13.1 years (SD = 1.6) for mortality in the absence of ASCVD. Over the follow-up period, there were 22 674 new cases of ASCVD, 4058 deaths following ASCVD diagnosis, and 10 123 deaths without a preceding ASCVD diagnosis.

The baseline characteristics of the study are presented in *Table 1*. Of the participants, 52.3% were female, with a mean age 55.6 years (SD = 8.07). Diabetic individuals had lower overall CVH scores compared to non-diabetic individuals, with scores of 60.5 vs. 68.0 for women and 59.1 vs. 64.8 for men. Among the 235 604 participants without diabetes, the distribution across CVH levels was as follows: 14.7% (n = 34 547) had low CVH, 29.1% (n = 68 546) had moderate CVH, and 56.5% (n = 132 511) had high CVH. Conversely, within the diabetic population, 33.9% (n = 3583) had low CVH, 33.8% (n = 3576) had moderate CVH, and 32.3% (n = 3420) had high CVH. Supplementary material online, Table *S3* outlines the characteristics of participants with complete data on age, sex, and CVH components and for those with missing data. Compared to those included in the analysis, excluded participants were more likely to be female, less likely to be White, and had a lower Townsend Deprivation Index.

Incident atherosclerotic cardiovascular disease and mortality

Using non-diabetic individuals as the reference group, it was found that higher CVH levels in diabetic patients were associated with a lower ASCVD and mortality risk. Among diabetic women, those with low CVH had a 1.89-fold increased ASCVD hazard compared to non-diabetic women, while those with high CVH had no significant difference in ASCVD hazard. Similarly, diabetic men with low CVH had a 1.76-fold higher ASCVD hazard compared to non-diabetic men; whereas those with high CVH had no significant difference. Additionally, no significant difference in post-ASCVD mortality was observed between diabetics with high CVH and non-diabetics, as shown in *Table 2*. The findings remained consistent when using waist circumference instead of BMI to calculate CVH, and after further adjustment for education, household income, and depression. Similarly, the results were unaffected by excluding outcomes within the first 2 years of follow-up, cases of incident diabetes, or patients with an early diabetes diagnosis (before age 30). Furthermore, the associations were unchanged when using the non-imputed data (see Supplementary material online, Table *S4* and *S5*). Analysis of participants with prediabetes revealed a pattern consistent with that observed in diabetes: individuals with prediabetes who achieved a high CVH score showed no increased risk of cardiovascular disease events and mortality compared to participants with normal glucose regulation (see Supplementary material online, Table *S6*).

The hazard ratios for ASCVD, post-ASCVD mortality, and mortality without ASCVD were lower in patients with moderate or high CVH compared to those with low CVH, regardless of T2DM status. In diabetic women, high CVH was associated with a 45% reduction in ASCVD hazard and a 37% reduction in post-ASCVD mortality compared to those with low CVH. Similarly, non-diabetic women with high CVH had a 50% lower ASCVD hazard and a 38% reduction in mortality following ASCVD. Diabetic men showed a 42% reduction in ASCVD hazard and a 36% reduction in post-ASCVD mortality, while non-diabetic men had a 47% and 36% reduction, respectively. There was no significant interaction between CVH level and diabetes status in terms of ASCVD incidence or mortality (see *Table 3*).

Life expectancy free from atherosclerotic cardiovascular disease

In patients with diabetes, an increase in CVH levels was associated with an extension in both total life expectancy and life expectancy free from ASCVD, compared with non-diabetic individuals. At the age of 40, diabetic women with low CVH exhibited a total life expectancy of 44.7 years (95% CI, 43.9–45.6), with 39.1 years (38.6–39.7) free from ASCVD. Those with high CVH expected a life expectancy of 48.2 years (47.4–49.1), with 43.3 years (42.7–43.8) ASCVD-free. In contrast, non-diabetic women had a total life expectancy of 50.0 years (49.2–50.8), with an estimated 44.8 years (44.2–45.3) predicted to be ASCVD-free. Similar patterns were observed in men at age 40. Diabetic men with low CVH are expected to have a life expectancy of 39.5 years (38.6–40.5), with an ASCVD-free expectancy of 32.8 years (32.2–33.4). This figure significantly increased for those with high CVH to 45.1 years (44.2–46.0), with 38.5 years (37.9–39.1) ASCVD-free. Non-diabetic men had a total life expectancy of 46.3 years (45.4–47.2), with 39.0 years (38.4–39.6) ASCVD-free (*Figure 1*).

Consistent with these findings on absolute life expectancy, at age 40, the percentage of remaining life without ASCVD increased from 87.5% to 89.6% in diabetic women as CVH improved from low to high, reaching the nearly same level as non-diabetic women (89.7%). Similarly, for men, this proportion rose from 82.9% (low CVH) to 85.6% (high CVH), approaching and even exceeding the 84.4% observed in non-diabetic men (*Table 4*). These findings remained unchanged when analyses were performed using unimputed data (see Supplementary material online, Figure S1, Supplementary material online, Table S6). Accordingly, the proportion of life expectancy spent with ASCVD decreased as CVH improved among individuals with diabetes, compared to those without diabetes (see Supplementary material online, Table S7).

Discussion

In this large, population-based longitudinal cohort analysis, it demonstrated that diabetic patients experienced progressively lower risks of incident ASCVD and overall mortality as CVH levels improved using non-diabetic individuals as the reference group. Diabetic patients with high CVH exhibited no significant excess risk of incident ASCVD and subsequent mortality compared to non-diabetics. Additionally, increasing CVH levels among diabetics was correlated with a reduction in the disparity in total and ASCVD-free life expectancy compared to non-diabetics. Furthermore, improved CVH levels in diabetic participants were also associated with a higher proportion of life expectancy free from ASCVD, compared to non-diabetic individuals.

This research indicated that diabetic patients with high levels of CVH did not exhibit a significantly higher risk for the onset of ASCVD and subsequent mortality compared to non-diabetic individuals. Similarly, participants with prediabetes showed no increased risk when

Table 3 Incident ASCVD and mortality by CVH level among participants with and without diabetes

Gender	Transition	Presence of diabetes	Low CVH		Moderate CVH		High CVH		*P* for interaction
			No. of cases/ person-years	HR (95% CI)	No. of cases/ person-years	HR (95% CI)	No. of cases/ person-years	HR (95% CI)	
Women	Incident ASCVD	With diabetes	256/15 553.9	1 [reference]	190/15 859.8	0.65 (0.54–0.78)	164/17 742.6	0.55 (0.45–0.68)	0.066
		Without diabetes	1727/188 433.3	1 [reference]	2782/408 332.4	0.75 (0.70–0.79)	3664/998 830.6	0.50 (0.47–0.53)	
	Mortality among those without ASCVD	With diabetes	90/13 540.9	1 [reference]	92/14 386.8	0.88 (0.66–1.18)	87/16 499.2	0.81 (0.60–0.96)	0.053
		Without diabetes	882/175 343.5	1 [reference]	1312/386 617.0	0.67 (0.62–0.73)	2312/969 440.3	0.58 (0.53–0.63)	
	Mortality among those with ASCVD	With diabetes	62/1226.1	1 [reference]	34/969.3	0.64 (0.42–0.98)	27/848.9	0.63 (0.39–0.99)	0.996
		Without diabetes	360/8886.5	1 [reference]	404/14 099.5	0.66 (0.57–0.76)	480/17 955.6	0.62 (0.54–0.71)	
Men	Incident ASCVD	With diabetes	652/24 932.4	1 [reference]	570/25 571.2	0.78 (0.70–0.88)	379/23 127.3	0.58 (0.51–0.66)	0.780
		Without diabetes	3639/230 435.9	1 [reference]	5343/442 782.5	0.74 (0.71–0.77)	5519/693 767.4	0.53 (0.51–0.55)	
	Mortality among those without ASCVD	With diabetes	224/20 448.7	1 [reference]	204/21 489.3	0.78 (0.64–0.94)	149/20 410.2	0.64 (0.52–0.79)	0.947
		Without diabetes	1294/203 941.5	1 [reference]	2027/402 952.9	0.77 (0.72–0.82)	2296/650 933.7	0.60 (0.56–0.65)	
	Mortality among those with ASCVD	With diabetes	217/3239.8	1 [reference]	156/2896.8	0.69 (0.56–0.85)	95/1914.9	0.64 (0.50–0.82)	0.378
		Without diabetes	854/18 941.9	1 [reference]	1064/27 542.2	0.80 (0.73–0.88)	896/27 597.6	0.64 (0.58–0.71)	

ASCVD, atherosclerotic cardiovascular disease; CVH, cardiovascular health; HR, hazard ratio; CI, confidence interval.
Multistate survival analysis using the Gompertz distribution was utilized to compute HRs (and 95% CIs), with low CVH serving as the reference. The analysis was adjusted for age, ethnicity, the Townsend Deprivation Index, a family history of diabetes, ASCVD, and cancer, the duration of diabetes (for diabetic patients), the use of diabetes medications (for diabetic patients), and the use of aspirin, antihypertensive, and lipid-lowering drugs. Additionally, for the HR of transition 3, the age at ASCVD diagnosis was further adjusted.

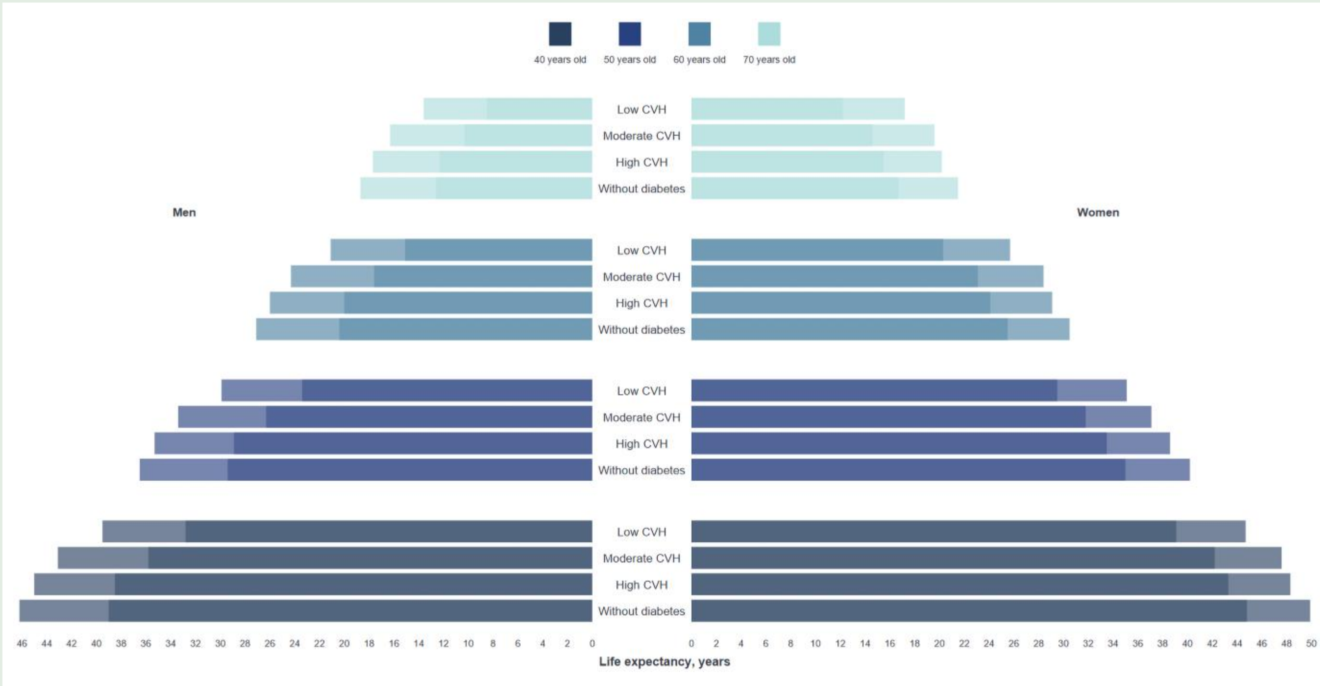


Figure 1 Total life expectancy and life expectancy with and without ASCVD across CVH levels among participants with diabetes compared with the participants without diabetes. ASCVD, atherosclerotic cardiovascular disease; CVH, cardiovascular health. Life expectancy was calculated using weighted transition rates, adjusted for age, ethnicity, the Townsend Deprivation Index, a family history of diabetes, ASCVD, and cancer, and the use of aspirin, antihypertensive, and lipid-lowering drugs, as well as age at ASCVD diagnosis. Each bar represents the life span of individuals, with the darker colour depicting life expectancy free from ASCVD.

Table 4 Life expectancy free from ASCVD by CVH level

Age	CVH level	Women		Men	
		LE without ASCVD, year (95% CI)	Proportion[a], per cent (95% CI)	LE without ASCVD, year (95% CI)	Proportion[a], per cent (95% CI)
40	Low CVH	39.1 (38.6–39.7)	87.5 (87.1–87.9)	32.8 (32.2–33.4)	82.9 (82.4–83.4)
	Moderate CVH	42.2 (41.7–42.7)	88.7 (88.4–89.1)	35.8 (35.2–36.4)	83.0 (82.6–83.5)
	High CVH	43.3 (42.7–43.8)	89.6 (89.2–89.9)	38.5 (37.9–39.1)	85.6 (85.2–86.0)
	Without diabetes	44.8 (44.2–45.3)	89.7 (89.4–90.0)	39.0 (38.4–39.6)	84.4 (84.0–84.8)
50	Low CVH	29.5 (29.0–30.1)	84.1 (83.6–84.6)	23.4 (22.8–24.0)	78.1 (77.5–78.7)
	Moderate CVH	32.5 (32.0–33.1)	85.9 (85.5–86.3)	26.3 (25.7–268)	78.6 (78.0–79.1)
	High CVH	33.5 (33.0–34.1)	86.9 (86.6–87.3)	28.9 (28.3–29.5)	81.9 (81.5–82.4)
	Without diabetes	35.0 (34.5–35.6)	87.2 (86.8–87.6)	29.4 (28.8–30.0)	80.6 (80.0–81.1)
60	Low CVH	20.3(19.8–20.9)	79.0 (78.4–79.6)	15.1 (14.6–15.7)	71.5 (70.7–72.3)
	Moderate CVH	23.1 (22.6–23.7)	81.6 (81.0–82.1)	17.6 (17.0–18.2)	72.3 (71.6–73.0)
	High CVH	24.1 (23.6–24.7)	83.0 (82.5–83.5)	20.0 (19.4–20.5)	76.9 (76.2–77.5)
	Without diabetes	25.5 (25.0–26.0)	83.5 (83.1–84.0)	20.4 (19.8–21.0)	75.2 (74.6–75.9)
70	Low CVH	12.2 (11.7–12.8)	71.1 (70.3–72.0)	8.5 (8.0–9.0)	62.1 (61.0–63.5)
	Moderate CVH	14.6 (14.1–15.2)	74.8 (74.1–75.5)	10.3 (9.8–10.8)	63.4 (62.5–64.4)
	High CVH	15.5 (15.0–16.0)	76.9 (76.2–77.6)	12.3 (11.8–12.8)	69.6 (68.8–70.4)
	Without diabetes	16.7 (16.2–17.2)	77.9 (77.3–78.6)	12.6 (12.1–13.2)	67.6 (66.7–68.4)

ASCVD, atherosclerotic cardiovascular disease; CVH, cardiovascular health; LE, life expectancy; CI, confidence interval.
LEs have been calculated with hazard ratios adjusted for age, ethnicity, the Townsend Deprivation Index, a family history of diabetes, ASCVD, and cancer, and the use of aspirin, antihypertensive, and lipid-lowering drugs, as well as age at ASCVD diagnosis.
[a]Proportion is computed by dividing the number of years lived with ASCVD by the total life expectancy at a specific age.

compared to those with normal glucose regulation. This conclusion is consistent with previous research findings, where CVH was represented by Life's Simple 7. The study demonstrated that patients with diabetes or prediabetes with more than five components of Life's Simple 7 had lower or no significant excess risks for cardiovascular disease compared to participants with normal glucose regulation.[9] However, our results indicated that high CVH levels among diabetic women did not reduce the mortality risk for those without ASCVD to levels comparable to non-diabetic individuals. One potential explanation for this finding could be our method of stratifying CVH into tertiles, with 'High CVH' defined as a CVH score >65.6, which is below the AHA's recommended level threshold of 80 or above. This approach was adopted to ensure greater statistical power in our analysis. Given an adequate sample size, it will be valuable to conduct specialized research to determine whether achieving the AHA-recommended high CVH level, or a more stringent standard, would have a stronger impact on the overall ASCVD progression and mortality.

Furthermore, our stratified analysis demonstrated that the association between higher CVH and reduced risk of ASCVD and its slower progression to mortality was consistent for both participants with and without diabetes. This suggests that CVH level and diabetes status act as independent contributors of ASCVD and mortality. Therefore, the substantial risk reduction associated with higher CVH in participants with diabetes likely represents an additive benefit, rather than a modification of the underlying disease process conferred by diabetes itself. This distinction is clinically crucial. While our findings champion the message of risk mitigation—that improving CVH can bring the risk for individuals with diabetes closer to that of non-diabetic individuals—it should not be interpreted as reducing the need for standard diabetes care and glycaemic management. Moreover, our baseline characteristics data revealed that diabetic patients generally exhibited lower CVH levels than non-diabetic patients. Specifically, a greater proportion of diabetic individuals had low CVH levels, while fewer had high CVH levels. Therefore, there is considerable potential for promoting CVH as a strategy for preventing cardiovascular diseases among diabetic patients.

To the best of our knowledge, this is the first study to evaluate the associations between CVH and lifestyle with life expectancy free from ASCVD in diabetic patients compared to non-diabetic individuals. Previous research has underscored the significance of CVH in extending disease-free life expectancy in the general population. An analysis of UK Biobank data demonstrated that individuals with high levels of CVH had significantly longer life expectancies free from major chronic illnesses, including CVD, diabetes, cancer, and dementia, across various socioeconomic strata.[15] Li *et al.*[20] discovered that maintaining a healthy lifestyle, which includes never smoking, maintaining a body mass index of 18.5–24.9, engaging in moderate to vigorous physical activity, consuming alcohol in moderation, and achieving a higher diet quality score, was linked to an extended lifespan free from diabetes, cardiovascular diseases, and cancer. In the Chicago Health and Aging Project, adopting a healthy lifestyle characterized by five key factors (non-smoking, a healthy diet, cognitive activities, physical activities, and moderate alcohol consumption) was found to correlate with increased life expectancy, alongside a greater proportion of years lived without Alzheimer's dementia.[21] Research utilizing three sub-cohorts from the Rotterdam Study indicated that an overall healthy lifestyle—encompassing a nutritious diet, physical activity, non-smoking, maintaining a healthy weight, and abstaining from or minimally consuming alcohol—positively influenced overall life expectancy, and the number of years lived free from heart failure.[22] Our study extended the role of CVH and lifestyle from the general population to individuals with diabetes, first demonstrating that high CVH levels can compensate for the shortened life expectancy and ASCVD-free life expectancy associated with diabetes. Building on previous studies that showed high CVH reduces the incidence of ASCVD and mortality in diabetic populations, our work further demonstrated that elevated CVH mitigates the negative influence of diabetes on shortened lifespan and years of life spent free of ASCVD. This provides policymakers and individuals with more easily understandable evidence for promoting high CVH levels within the diabetic community. However, in addition to cardiovascular disease, complications for diabetic patients also include chronic kidney disease, retinopathy, neuropathy, and so on. We look forward to future research on the relationship between CVH and other diabetic complications, which will provide a more comprehensive understanding of CVH's role in the health outcomes of diabetes patients.

This study has several limitations: (1) Our analysis was based exclusively on diagnosed diseases recorded within the UK Biobank, potentially overlooking individuals with undocumented conditions. Nevertheless, previous studies have validated the reliability of routinely collected hospital admission data for epidemiological research.[23,24] (2) The CVH metrics were only assessed at baseline; hence, potential variations in CVH over the follow-up period were not accounted for. However, the outcomes of this study can still guide predictions about future health based on an individual's current characteristics. (3) The participants excluded due to missing data were disproportionately male, non-white, and from lower TDI backgrounds. Furthermore, the UK Biobank cohort predominantly consisted of white individuals with higher socioeconomic status, which could introduce selection bias. However, this study investigated the exposure-disease relationship, the generalizability of which doesn't require participants to be representative.[25] (4) Although we adjusted for known biases, the possibility of residual confounding remained, and the observational nature of our study precluded the establishment of causation.

Conclusion

Diabetic patients who maintained high CVH levels were able to offset the risk of transitioning from health to incident ASCVD and subsequent mortality compared to non-diabetic individuals, thereby achieving a life expectancy free from ASCVD that is closer to that of non-diabetic individuals. This study supports the promotion of CVH as a strategy to enhance the quality of lifespan in patients with T2DM.

Supplementary material

Supplementary material is available at *European Journal of Preventive Cardiology*.

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Author contributions

J.W., X.L., and Y.W. were involved in the conception and design of the study. Y.W. analysed and interpreted the results and wrote the first draft of the manuscript. J.W., X.L., D.S., S.N., and Y.H. edited and reviewed the manuscript draft. All authors approved the final version of the manuscript. J.W., X.L., and Y.W. are the guarantors of this work and, as such, had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

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Conflict of interest: None declared.

Data availability

This research has been conducted using the UK Biobank Resource under application number 86898. UK Biobank data are available through registration on the UK Biobank (www.ukbiobank.ac.uk).

References

1. Zheng Y, Ley SH, Hu FB. Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. *Nat Rev Endocrinol* 2018;**14**:88–98.
2. Wong ND, Sattar N. Cardiovascular risk in diabetes mellitus: epidemiology, assessment and prevention. *Nat Rev Cardiol* 2023;**20**:685–695.
3. Sattar N, Rawshani A, Franzén S, Rawshani A, Svensson AM, Rosengren A, *et al.* Age at diagnosis of type 2 diabetes Mellitus and associations with cardiovascular and mortality risks. *Circulation* 2019;**139**:2228–2237.
4. American Diabetes Association Professional Practice Collaborators. 4. Comprehensive medical evaluation and assessment of comorbidities: standards of care in diabetes-2025. *Diabetes Care* 2025;**48**:S59–S85.
5. Ried-Larsen M, Rasmussen MG, Blond K, Overvad TF, Overvad K, Steindorf K, *et al.* Association of cycling with all-cause and cardiovascular disease mortality among persons with diabetes: the European prospective investigation into cancer and nutrition (EPIC) study. *JAMA Intern Med* 2021;**181**:1196–1205.
6. Kristensen FP, Sanchez-Lastra MA, Dalene KE, del Pozo Cruz B, Ried-Larsen M, Thomsen RW, *et al.* Leisure-time physical activity and risk of microvascular complications in individuals with type 2 diabetes: a UK Biobank study. *Diabetes Care* 2023;**46**: 1816–1824.
7. Lloyd-Jones DM, Allen NB, Anderson CA, Black T, Brewer LC, Foraker RE, *et al.* Life's essential 8: updating and enhancing the American Heart Association's construct of cardiovascular health: a presidential advisory from the American Heart Association. *Circulation* 2022;**146**:e18–e43.
8. He P, Li H, Ye Z, Liu M, Zhou C, Wu Q, *et al.* Association of a healthy lifestyle, life's essential 8 scores with incident macrovascular and microvascular disease among individuals with type 2 diabetes. *J Am Heart Assoc* 2023;**12**:e029441.
9. Wang T, Lu J, Su Q, Chen Y, Bi Y, Mu Y, *et al.* Ideal cardiovascular health metrics and Major cardiovascular events in patients with prediabetes and diabetes. *JAMA Cardiol* 2019;**4**:874–883.
10. Sun J, Li Y, Zhao M, Yu X, Zhang C, Magnussen CG, *et al.* Association of the American Heart Association's new "life's essential 8" with all-cause and cardiovascular disease-specific mortality: prospective cohort study. *BMC Med* 2023;**21**:116.
11. Li W, Xing A, Xu W, Guo L, Gao X, Zhou S, *et al.* Life's essential 8 in relation to cardiovascular disease and mortality in individuals with diabetes. *JACC Asia* 2024;**4**:456–464.
12. Sudlow C, Gallacher J, Allen N, Beral V, Burton P, Danesh J, *et al.* UK Biobank: an open access resource for identifying the causes of a wide range of complex diseases of middle and old age. *PLoS Med* 2015;**12**:e1001779.
13. Elliott J, Bodinier B, Bond TA, Chadeau-Hyam M, Evangelou E, Moons KG, *et al.* Predictive accuracy of a polygenic risk score-enhanced prediction model vs a clinical risk score for coronary artery disease. *JAMA* 2020;**323**:636–645.
14. Mozaffarian D. Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: a comprehensive review. *Circulation* 2016;**133**:187–225.
15. Wang X, Ma H, Li X, Heianza Y, Manson JE, Franco OH, *et al.* Association of cardiovascular health with life expectancy free of cardiovascular disease, diabetes, cancer, and dementia in UK adults. *JAMA Intern Med* 2023;**183**:340–349.
16. Han H, Cao Y, Feng C, Zheng Y, Dhana K, Zhu S, *et al.* Association of a healthy lifestyle with all-cause and cause-specific mortality among individuals with type 2 diabetes: a prospective study in UK Biobank. *Diabetes Care* 2022;**45**:319–329.
17. Liu G, Li Y, Hu Y, Zong G, Li S, Rimm EB, *et al.* Influence of lifestyle on incident cardiovascular disease and mortality in patients with diabetes Mellitus. *J Am Coll Cardiol* 2018; **71**:2867–2876.
18. Patel AP, Wang M, Kartoun U, Ng K, Khera AV. Quantifying and understanding the higher risk of atherosclerotic cardiovascular disease among South Asian individuals: results from the UK Biobank prospective cohort study. *Circulation* 2021;**144**:410–422.
19. Ai S, Zhang J, Zhao G, Wang N, Li G, So HC, *et al.* Causal associations of short and long sleep durations with 12 cardiovascular diseases: linear and nonlinear Mendelian randomization analyses in UK Biobank. *Eur Heart J* 2021;**42**:3349–3357.
20. Li Y, Schoufour J, Wang DD, Dhana K, Pan A, Liu X, *et al.* Healthy lifestyle and life expectancy free of cancer, cardiovascular disease, and type 2 diabetes: prospective cohort study. *BMJ* 2020;**368**:l6669.
21. Dhana K, Franco OH, Ritz EM, Ford CN, Desai P, Krueger KR, *et al.* Healthy lifestyle and life expectancy with and without Alzheimer's dementia: population based cohort study. *BMJ* 2022;**377**:e068390.
22. Limpens MA, Asllanaj E, Dommershuijsen LJ, Boersma E, Ikram MA, Kavousi M, *et al.* Healthy lifestyle in older adults and life expectancy with and without heart failure. *Eur J Epidemiol* 2022;**37**:205–214.
23. Eastwood SV, Mathur R, Atkinson M, Brophy S, Sudlow C, Flaig R, *et al.* Algorithms for the capture and adjudication of prevalent and incident diabetes in UK biobank. *PLoS One* 2016;**11**:e0162388.
24. Rannikmäe K, Ngoh K, Bush K, Al-Shahi Salman R, Doubal F, Flaig R, *et al.* Accuracy of identifying incident stroke cases from linked health care data in UK Biobank. *Neurology* 2020;**95**:e697–e707.
25. Fry A, Littlejohns TJ, Sudlow C, Doherty N, Adamska L, Sprosen T, *et al.* Comparison of sociodemographic and health-related characteristics of UK Biobank participants with those of the general population. *Am J Epidemiol* 2017;**186**:1026–1034.