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Which adiposity index is best? Comparison of five indicators and their ability to identify type 2 diabetes risk in a population study

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ABSTRACT

Aims: We compared ability of five adiposity indicators [body mass index (BMI), waist circumference (WC), waist-to-height ratio (WHtR), waist-by-height$^{0.5}$ (WHT.5R), and a body shape index (ABSI)] to identify current diabetes and their prospective associations with diabetes.

Methods: Baseline data were from 7,979 participants of UK Whitehall II study, of whom 7,488 diabetes-free participants were followed-up (median = 16.0 years) for incident diabetes (n = 940). According to five indices' cut-points, participants were separately classified into low-value groups and high-value groups. We cross-sectionally investigated ability of the indicators to identify existing diabetes by receiver operating characteristic curve analysis, and explored prospective associations between elevated indices and diabetes using Cox regression analysis.

Results: Waist-based indicators were superior to BMI in identifying diabetes. High WHtR (≥0.5) demonstrated the highest multivariable-adjusted HR [2.64 (95 % CI 2.29, 3.03)]. Across all indicators, associations between elevated indicators and diabetes were stronger in younger participants. In combined analyses, "low BMI but high WHtR" had higher risk for diabetes [2.20 (95 % CI 1.65, 2.95)] than "high BMI but low WHtR" [1.34 (95 % CI 1.05, 1.70)].

Conclusion: Waist-based indicators are more strongly associated with diabetes than BMI. WHtR, an easy-to-calculate, waist-based index with a sex- and race-independent cut-point, may be useful for diabetes prevention.

1. Introduction

Obesity, traditionally defined as an excess of body fat leading to impaired health, is a global public health epidemic [1]. For example, in the UK, it is predicted that around 35 % of all adults will be obese by 2025 [2]. Some observational studies suggest that obesity, associated with adipose dysfunction and insulin resistance, increases type 2 diabetes risk [3,4].

Body mass index (BMI), as an index of body weight in kilograms divided by height in square meter (kg/m^2), is the most commonly used method to assess obesity in clinical practice with relevant guidelines (i.e., 25 to 30 kg/m^2 of BMI is overweight; ≥30 kg/m^2 is obese) [5,6], and has been widely accepted as a quick and simple tool to classify patients into different risk categories [7]. However, BMI does not distinguish fat from muscle or between different body fat distributions [8]. Additionally, it has been observed that individuals with a more central (or abdominal) fat distribution are at greater health risk than those with peripheral fat [9], thus, when we look at obesity, we cannot ignore the corresponding risk associated with elevated waist circumference (WC) while considering excessive body weight (i.e., BMI). Furthermore, several studies indicated that some indices related to abdominal obesity, such as WC or waist-to-height ratio (WHtR), may more accurately predict diabetes risk than BMI [10,11]. Compared with WC, WHtR is an index of abdominal obesity corrected for height, and its recognized cut-off value is 0.5 (<0.5 is considered normal), regardless of sex or ethnicity [12]. Besides BMI and WC, a number of novel adiposity indicators for defining obesity continue to emerge. For example, waist-by-height$^{0.5}$ (WHT.5R) is a new indicator developed in 2016 [13], which apportions less importance to height than WHtR. WHT.5R has been shown to better predict cardiometabolic risks, including high lipids and blood glucose, than BMI [13]. A body shape index (ABSI), proposed in 2012, was specifically developed as a transformation of WC, with

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minimal correlation to height, weight, or BMI [14]. Complementary to BMI, the primary goal of this index is to capture the excess risk attributed to elevated WC. It was initially used to predict premature mortality risk [15]. However, these two newly-developed indices do not yet have accepted cut-points like BMI and WHtR, and at present, there are limited studies that simultaneously compare traditional indicators and these new ones with respect to their ability to discriminate current or future diabetes risk.

Furthermore, fat distribution is influenced by factors such as sex and age, which means that these relevant characteristics should be taken into account when applying anthropometry to assess an individual's health risk. Some studies suggested the existence of sex differences and age differences in the strength of association between adiposity indices and diabetes [16–18]. For example, a cross-sectional study of 35,256 Chinese adults aged 20–74 years showed that the association between elevated adiposity indicators and diabetes was stronger in their younger participants [18]. This suggests that stratification by sex and age is necessary when investigating the association between obesity and diabetes. However, the potential differences in sex and age remain inconclusive because there is limited biological evidence that can explain them and results of relevant studies were not entirely consistent [19]. In addition, few studies examined multiple (>3) obesity indicators along with investigating multiple important relevant stratification factors, and it remains unclear which adiposity index is more appropriate for assessing diabetes risk in specific demographic groups (such as women or men). In order to better understand and utilize different adiposity indicators, we conducted corresponding stratification analyses.

In this large, population-based study, we aimed to compare the ability of five adiposity indices (BMI, WC, WHtR, WHT.5R, and ABSI) to cross-sectionally identify individuals with diabetes, and to compare their prospective associations with incident diabetes, in order to assess future diabetes risk among those without diabetes at baseline.

2. Methods

2.1. Data collection

This study used data from the UK Whitehall II (WHII) study. Briefly, the WHII is an ongoing cohort study of 10,308 British civil servants aged 35–55 years, of whom 3,413 (33.1 %) were females, at initial enrollment (Phase 1), recruited from 20 London-based offices during 1985–1988 [20,21]. Phase 1 of WHII included a clinical examination and a self-administered questionnaire to collect data covering demographics, health status and lifestyle factors. Subsequent phases of information collection have alternated between questionnaire alone and questionnaire accompanied by a clinical examination [22,23]. The University College London ethics committee approved the study. Use of human material conformed to the Declaration of Helsinki.

2.2. Subjects

For the purposes of the present study, Phase 3 of the Whitehall II study (1991–1994) was considered the baseline, as it was the first phase to collect anthropometric measurements. Participants without data on height, weight, or waist circumference at Phase 3 were excluded. The remaining participants constituted the cross-sectional study sample (Sample I), which was used to compare the ability of different adiposity indices to identify individuals with diabetes at baseline. For the prospective analyses, participants with diabetes at Phase 3 were further excluded to form Sample II, which was used to examine the prospective association between adiposity indices and incident diabetes during follow-up until Phase 9 (2007–2009), the most recent diabetes screening phase. A flow chart of sample construction is shown in Fig. 1.

2.3. Assessments

The exposures are five adiposity indicators, including body mass index (BMI) [weight (kg)/height^2 (m^2)], waist circumference (WC)

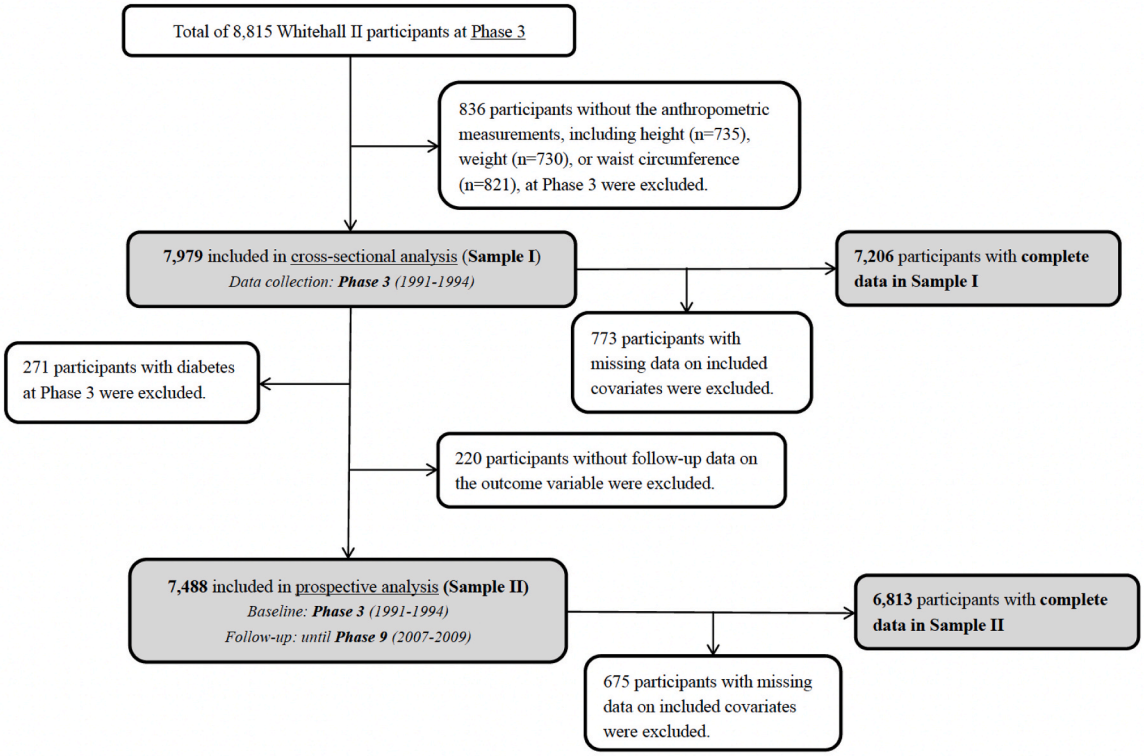


Fig. 1. Flow chart of the included participants.

[cm], waist-to-height ratio (WHtR) [WC (cm)/height (cm)], waist-by-height$^{0.5}$ (WHT.5R) [WC (cm)/Height$^{0.5}$ (cm$^{0.5}$)], and a body shape index (ABSI) [WC (m)/(BMI$^{2/3}$ (kg/m^2)$^{2/3}$ × height$^{1/2}$ (m$^{1/2}$))]. Of these, weight was measured with all items of clothing removed except underwear, using an electronic Soehnle scale with a digital readout. Height was measured with a stadiometer with the individual standing erect with the head in the Frankfort plane, and WC was measured using a fiberglass tape measure at 600 g tension as the smallest circumference at or below the costal margin [24].

To enable quantitative comparison of the exposures, we used standardized values of the exposures in cross-sectional analyses. For the longitudinal part of the analyses, we dichotomized the adiposity indicators according to their cut-points. For indicators with an accepted cut-point, we chose that recognized value, and for indicators without an accepted cut-off value, we selected the 75th population-wide percentile as its cut-point [5,12,25,26]. That is, Sample II were separately divided into low-value group (reference group) [BMI: <25 kg/m^2; WC: <90 cm for men and < 80 cm for women; WHtR: <0.5; WHT.5R < 75th population-wide centile (6.911 cm$^{0.5}$); ABSI < 75th population-wide percentile (0.078 m$^{7/6}$/kg$^{2/3}$), respectively] and high-value group (≥corresponding cut-off value), i.e., different adiposity indicators were used as different criteria to classify individuals into low or high level of obesity-related risk. To ensure comparability of different exposures and minimize the loss of numerical information when converting to categorical variables, in the longitudinal part, we additionally analyzed the associations of the standardized exposure variables (i.e., continuous) with diabetes risk.

Subgroup analyses were based on sex (male or female), age (<50 or ≥ 50 years, median age), ethnicity (white or non-white), and baseline cardiovascular disease (CVD) diagnosis (yes or no) for investigating differences in the strength of association between elevated adiposity indices and diabetes among different stratifications. For further examining health effect of different categories of obesity, such as excessive weight alone, abdominal obesity alone or both, we consider individuals' weight and WC at the same time. According to the different levels of BMI and WHtR combination, the Sample II was subdivided into 4 groups: (1) low BMI (<25 kg/m^2) and low WHtR (< 0.5) (reference group), (2) high BMI (≥25 kg/m^2) and low WHtR (< 0.5), (3) low BMI (< 25 kg/m^2) and high WHtR (≥0.5), (4) high BMI (≥25 kg/m^2) and high WHtR (≥0.5).

Potential confounding factors were age (continuous), sex (female or male), ethnicity (white or non-white), smoking (never smoker, ex-smoker, or current smoker), drinking (current not drinking, current moderate, current heavy), socioeconomic position (high, intermediate or low) [22], baseline CVD diagnosis (yes or no), CVD medication (yes or no), family history of diabetes (yes or no), physical activity (active, moderately active, or inactive) [27], dietary behavior (frequency of fruit and vegetables consumed; <daily or ≥ daily) [22], and menopause status (female subjects only: yes or no).

The outcome, type 2 diabetes, was defined as having a fasting plasma glucose ≥ 7.0 mmol/L, a 2-hour postload glucose ≥ 11.1 mmol/L at clinical examination, a physician diagnosis of type 2 diabetes, use of diabetes medication, or a hospital record of diabetes between 1991 and 2009 [28,29]. For each participant, follow-up time began on the date that anthropometric measurements were taken in Phase 3 and ended on the occurrence of diabetes, death, emigration, or the end date of last survey, whichever occurred first.

2.4. Statistical analyses

Continuous variables were presented as means ± standard deviations (SDs) or median (25th-75th percentiles), and categorical variables as frequencies with proportions. Differences between groups were assessed using two-sided t-tests or Wilcoxon rank-sum tests for continuous variables, and chi-squared tests for categorical variables. Correlations between the five adiposity indicators were estimated using Pearson's correlation coefficient (r).

In cross-sectional analyses, receiver operating characteristic (ROC) curves were constructed for each of the five indices, and the area under the ROC curve (AUC) was calculated to identify the index with the highest AUC as the strongest discriminator for diabetes. For each index, AUCs were calculated using two models: one without covariates and one with the inclusion of age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication. In both models, apart from the adiposity index being evaluated, all other variables included in the model were kept the same to ensure a fair comparison across indices. These covariates were treated as potential confounders, rather than predictive variables, to specifically evaluate the independent discriminatory contribution of each adiposity measure.

In cohort analyses, Cox regression analysis was used to estimate hazard ratios (HRs) and 95 % confidence interval (CI) for time to diabetes in relation to the elevated adiposity indices. For each adiposity index separately, three Cox models were estimated. Model$_1$ did not include any covariate. Adjusted covariates for the Model$_2$ included age, sex, and ethnicity. For the Model$_3$, besides the covariates of the Model$_2$, we additionally included smoking, drinking, socioeconomic position, baseline CVD diagnosis, CVD medication, family history of diabetes, physical activity, and dietary behavior. The differences in cumulative event (diabetes) rates were evaluated based on different combinations of BMI and WHtR, using log-rank test, and its results were presented as Kaplan-Meier (K-M) curves. Additionally, we did two-by-two comparisons between the four groups and used the Benjamini-Hochberg method to adjust the significance level. We also used Cox regression analysis to estimate HRs and 95 % CI for diabetes in relation to different levels of BMI and WHtR combination. Proportional hazards assumption was tested based on Schoenfeld residuals. P values of the global test indicated that the assumption was not entirely met. However, according to the K-M curves for the exposures (**Supplementary Fig. S1**) and the graphs of the scaled Schoenfeld residuals against the transformed time (**Supplementary Fig. S2**), the violation of proportionality was not extreme, which means a single HR for the exposure can still be a reasonable summary of the data [30].

To address potential bias due to missingness in included covariates (Supplementary **Table S1**), we conducted multivariate imputation (MI) by chained equations, using 30 imputed data sets. All reported P values were 2-sided. All analyses were performed in R (version 4.4.1).

3. Results

3.1. Characteristics of participants

The details of participants' baseline characteristics are shown in Supplementary **Table S2.** A total of 7,979 individuals were available for the cross-sectional analysis (Sample I). In Sample I, the mean age was 50.06 ± 6.03 years and 2,468 (30.9 %) were female. In the cohort part, there were 7,488 persons (Sample II). Further details (baseline characteristics of groups based on different adiposity indicators and participants with complete records) are provided in Supplementary **Table S3** and Supplementary **Table S4**. When the study population was stratified by sex, significant differences were observed in adiposity indices and several demographic and lifestyle characteristics. Men had a larger mean WC and a higher mean WHtR, whereas women had a higher mean BMI and a higher mean age (Supplementary **Table S5**a and **S5**b).

3.2. Correlation analysis

BMI, WC, WHtR, and WHT.5R were strongly correlated with each other and with weight (all r > 0.7). WHtR is the only one of our indicators that is essentially independent of height (r close to 0). ABSI was strongly correlated with WC (r > 0.7) but weakly correlated with BMI (r < 0.3), whereas its correlation with WHtR (r was around 0.6) and WHT.5R (r was around 0.7) was moderately strong (Supplementary Table S6**)**.

Table 1
Area under the receiver operating characteristic curve for five adiposity indicators in relation to diabetes in the Sample I.

Exposures	AUC_1 (95 % CI) Sample I: no covariates	AUC_2 (95 % CI) Sample I: with covariates
BMI	0.588 (0.552, 0.624)	0.722 (0.718, 0.726)
WC	0.604 (0.568, 0.640)	0.724 (0.720, 0.728)
WHtR	0.638 (0.603, 0.674)	0.728 (0.724, 0.732)
WHT.5R	0.624 (0.588, 0.660)	0.726 (0.722, 0.730)
ABSI	0.604 (0.569, 0.640)	0.727 (0.723, 0.731)

AUC_1, among Sample I (sample size = 7,979, number of events = 271), without covariates;
AUC_2, among Sample I (sample size = 7,979, number of events = 271), including covariates for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, CVD medication, and imputation for the missing covariates;
AUC, area under the curve; CVD, cardiovascular disease; CI, confidence interval; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index.

3.3. ROC curve analyses

Of 7,979 participants included in the baseline visit, 271 (3.4 %) had baseline diabetes. Results of ROC curves analyses without inclusion of covariates demonstrated WHtR had a higher AUC (0.638) than other indicators (Table 1). After including covariates, there was a general improvement in the discriminatory ability of our adiposity indicators in identifying diabetes (all AUCs > 0.7). Although the differences in AUCs between the 5 indicators were small, WHtR was the indicator with the largest AUC in imputed data (0.728). For complete data (Supplementary Table S7), ABSI had the highest AUC (0.720). The AUC for BMI was consistently lower than the AUCs for the other 4 measures. When the study population was stratified by sex, waist-based measures consistently showed higher AUCs than BMI in both males and females. WHtR had the highest AUC among males, while WHtR and ABSI shared the highest AUC among females. Overall, AUCs were higher in males than in females across all adiposity indices (Supplementary Table S8).

3.4. Cox regression analyses

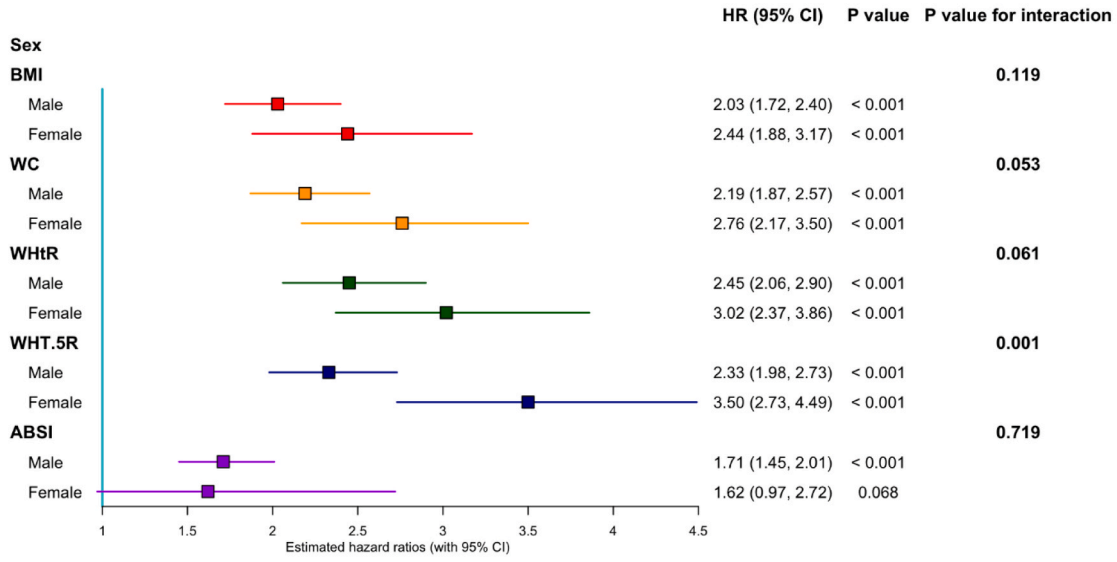
Of 7,488 participants included in the cohort analyses, after a median follow-up of 16.0 years (interquartile range: 14.2–16.5 yrs), 940 individuals developed diabetes, and of those 940, complete covariate data were available for 847. The estimated HRs associated with elevated adiposity indices (high-value group) are shown in Table 2. In all the models, higher levels of adiposity indicators were associated with a greater risk of developing diabetes. In the maximally-adjusted models, WHtR had a relatively higher HR (complete data: 2.67, 95 % CI: 2.31–3.10; imputed data: 2.64, 95 % CI: 2.29–3.03) for incident diabetes than the other indicators. The HRs for the waist-based measures (WC, WHtR, and WHT.5R) were all higher than the HR of BMI. Moreover, when analyses were performed using standardized values of the

Table 2
Estimated hazard ratios for incident diabetes in relation to the five adiposity indices (reference: low-value group).

	N [a] (%)	Diabetes, [a] N (%)	Crude HR_0, [a] (95 % CI)	Adjusted HR_1, [a] (95 % CI)	Adjusted HR_2, [a] (95 % CI)	N [b] (%)	Diabetes, [b] N (%)	Adjusted HR_2 with MI, [b] (95 % CI)
BMI								
Low	3629 (53.3)	277 (7.6)	1	1	1	3949 (52.7)	310 (7.9)	1
High	3184 (46.7)	570 (17.9)	2.54 (2.20, 2.93)	2.41 (2.09, 2.79)	2.25 (1.94, 2.60)	3539 (47.3)	630 (17.8)	2.17 (1.89, 2.49)
WC								
Low	4497 (66.0)	377 (8.4)	1	1	1	4908 (65.5)	415 (8.5)	1
High	2316 (34.0)	470 (20.3)	2.69 (2.35, 3.08)	2.56 (2.23, 2.93)	2.37 (2.06, 2.72)	2580 (34.5)	525(20.3)	2.36 (2.07, 2.70)
WHtR								
Low	4171 (61.2)	310 (7.4)	1	1	1	4557 (60.9)	344 (7.5)	1
High	2642 (38.8)	537 (20.3)	3.04 (2.64, 3.50)	2.88 (2.49, 3.33)	2.67 (2.31, 3.10)	2931 (39.1)	596 (20.3)	2.64 (2.29, 3.03)
WHT.5R								
Low	5167 (75.8)	471 (9.1)	1	1	1	5666 (75.7)	518 (9.1)	1
High	1646 (24.2)	376 (22.8)	2.78 (2.43, 3.19)	2.77 (2.40, 3.18)	2.54 (2.21, 2.93)	1822 (24.3)	422 (23.2)	2.63 (2.29. 3.01)
ABSI								
Low	5140 (75.4)	546 (10.6)	1	1	1	5655 (75.5)	612 (10.8)	1
High	1673 (24.6)	301 (18.0)	1.80 (1.56, 2.07)	1.87 (1.60, 2.20)	1.70 (1.45, 2.00)	1833 (24.5)	328 (17.9)	1.67 (1.43, 1.94)

BMI: low: <25 kg/m^2, high: ≥25 kg/m^2; WC: low: <90 cm for men and < 80 cm for women; high: ≥90 cm for men and ≥ 80 cm for women; WHtR: low: <0.5; high: ≥0.5; WHT.5R: low: <75th population-wide centile (6.911 cm$^{0.5}$), high: ≥75th population-wide centile (6.911 cm$^{0.5}$); ABSI: low: <75th population-wide percentile (0.078 m$^{7/6}$/kg$^{2/3}$), high: ≥75th population-wide percentile (0.078 m$^{7/6}$/kg$^{2/3}$);
Crude HR_0, the Cox regression model did not include any covariate (Model 1);
Adjusted HR_1, the Cox regression model was adjusted for age, sex, and ethnicity (Model 2);
Adjusted HR_2, the Cox regression model was adjusted for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication (Model 3);
Adjusted HR_2 with MI, the imputed Cox regression model was adjusted for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication;
MI, multivariate imputation; CVD, cardiovascular disease; HR, hazard ratio; CI, confidence interval; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index.
[a] Using complete data (n = 6,813) of Sample II, number of events for complete data = 847;
[b] Using Sample II, sample size = 7,488, number of events = 940, imputation for the missing covariates.

a. sex stratification



b. age stratification

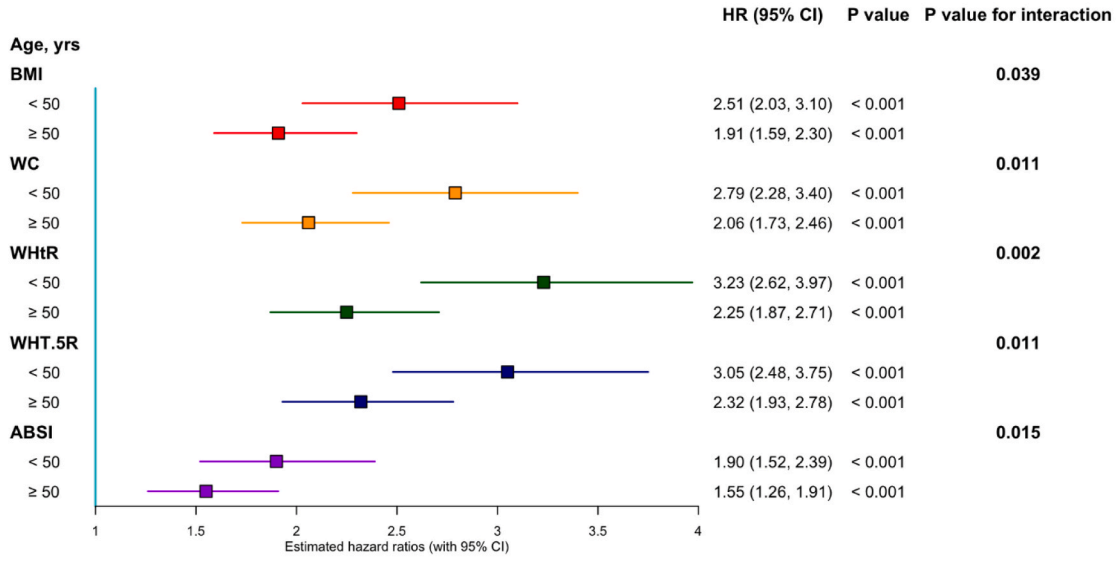
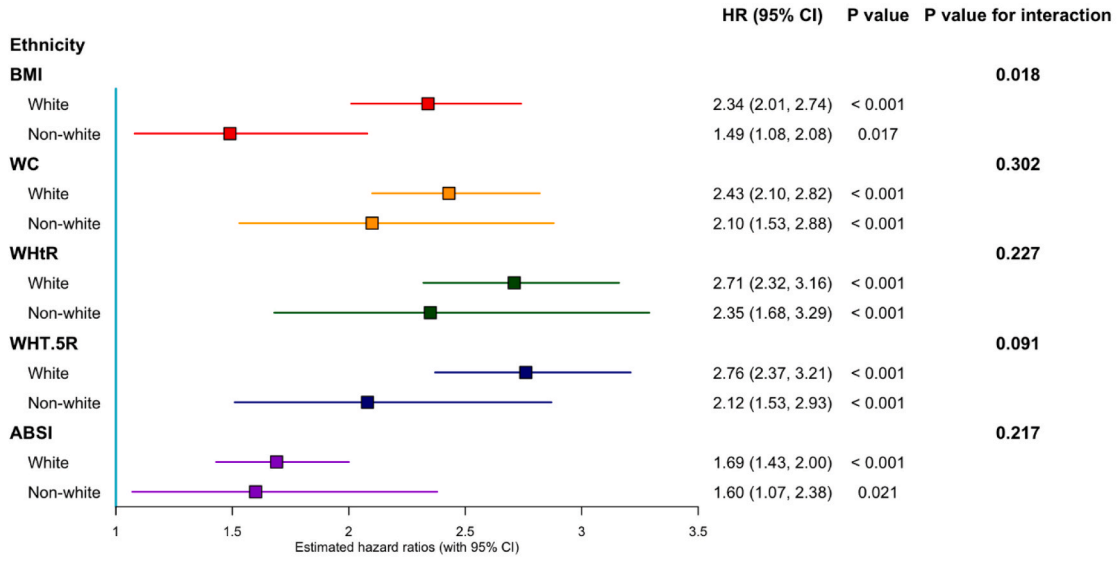


Fig. 2. Subgroup analyses for the associations between the five indices (high-value group) and incident diabetes in Sample II. BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index; HR, hazard ratio; CI, confidence interval; CVD, cardiovascular disease.

exposures (continuous), our results still showed that waist-based measures had larger HRs for diabetes than BMI (Supplementary Table S9).

Fig. 2 illustrates the results of subgroup analyses to identify potential modifiers of the associations between the five indices and incident diabetes in Sample II. We found strong statistical evidence for effect modification only for age 50 (P value for interaction < 0.05), whereby the association between each adiposity indicator and diabetes was stronger in people aged under 50 at baseline, compared with those aged 50 and older. Additionally, in both age groups, elevated WHtR and WHT.5R had the highest HRs for diabetes, suggesting the strong association of the two indices with diabetes. Regarding the other potential modifiers we considered, generally, among females, the white, and persons with baseline CVD, elevated adiposity indicators except ABSI appear to confer a higher risk increase of developing diabetes than males, the non-white, and individuals without baseline CVD, but the observed differences are small in most cases, and the statistical evidence

c. ethnicity stratification



d. baseline CVD diagnosis stratification

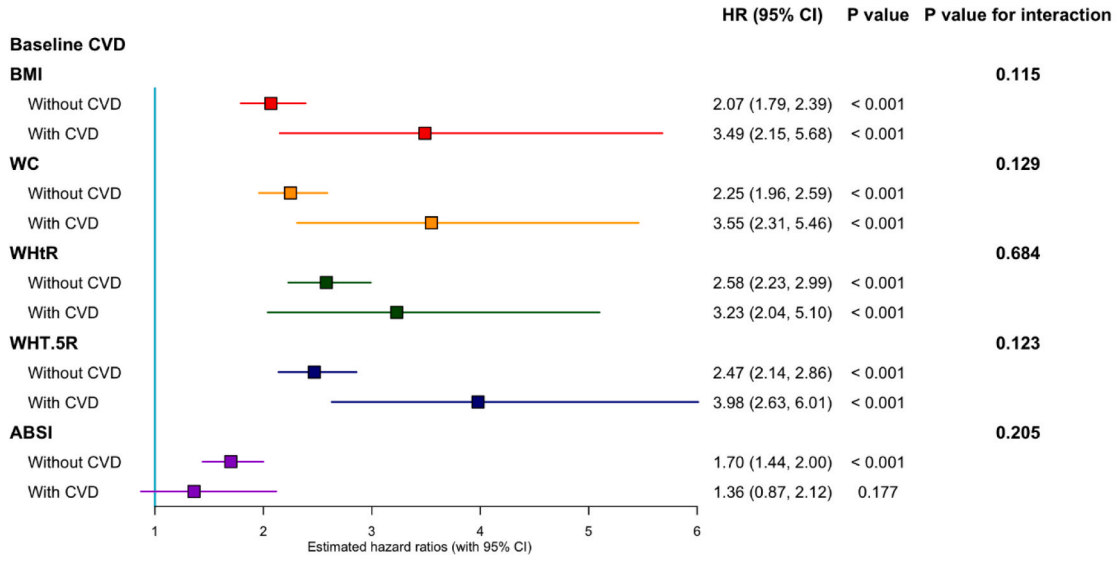


Fig. 2. (*continued*).

for most interactions is weak. More information is shown in Supplementary Table S10 and Supplementary Table S11.

3.5. Combining BMI and WHtR as metrics for diabetes risk

Supplementary Fig. S3a and S3b show Kaplan-Meier plots for time to diabetes event, stratified by different levels of BMI and WHtR combination. Comparisons between the groups showed strong evidence of differences in incidence of diabetes between the groups (all $P < 0.001$) except for "low BMI and high WHtR" and "high BMI and high WHtR" (Supplementary Table S12), which was not statistically significant according to the Benjamini-Hochberg procedure ($P > 0.05$). Hence, it was inferred that the cumulative diabetes rate in the "low BMI and high WHtR" group was significantly higher than that in the "high BMI and low WHtR" group. Meanwhile, in Cox regression analysis (Table 3), the "low BMI and high WHtR" group also showed a higher diabetes risk than the "high BMI and low WHtR" group in both the complete (HR_2: 2.34 vs 1.48) and imputed data (HR_2: 2.20 vs 1.34).

4. Discussion

This study explored the ability of different adiposity indicators to cross-sectionally identify existing diabetes, as well as their prospective association with incident diabetes. The results showed that waist-based

Table 3
Crude and adjusted hazard ratios for diabetes in relation to combination of BMI and WHtR (reference: low BMI and low WHtR).

Groups	N [a] (%)	Diabetes, [a] N (%)	Crude HR_0, [a] (95 % CI)	Adjusted HR_1, [a] (95 % CI)	Adjusted HR_2, [a] (95 % CI)	N[b] (%)	Diabetes, [b] N (%)	Adjusted HR_2 with MI, [b] (95 % CI)
low BMI and low WHtR	3299 (48.3)	218 (6.6)	1	1	1	3597 (48.0)	248 (6.9)	1
high BMI and low WHtR	872 (13.0)	92 (10.6)	1.67 (1.31, 2.13)	1.57 (1.23, 2.00)	1.48 (1.16, 1.90)	960 (12.8)	96 (10.0)	1.34 (1.05, 1.70)
low BMI and high WHtR	330 (4.7)	59 (17.9)	3.12 (2.34, 4.15)	2.56 (1.90, 3.45)	2.34 (1.73, 3.15)	352 (4.7)	62 (17.6)	2.20 (1.65, 2.95)
high BMI and high WHtR	2312 (34.0)	478 (20.7)	3.50 (2.98, 4.10)	3.30 (2.80, 3.88)	3.03 (2.57, 3.57)	2579 (34.4)	534 (20.7)	2.90 (2.49, 3.39)

Crude HR_0, the Cox regression model did not include any covariate (Model 1);
Adjusted HR_1, the Cox regression model was adjusted for age, sex, and ethnicity (Model 2);
Adjusted HR_2, the Cox regression model was adjusted for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication (Model 3);
Adjusted HR_2 with MI, the imputed Cox regression model was adjusted for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication;
MI, multivariate imputation; CVD, cardiovascular disease; HR, hazard ratio; CI, confidence interval; BMI, body mass index; WHtR, waist-to-height ratio.
[a] Using complete data (n = 6,813) of Sample II, number of events for complete data = 847;
[b] For Sample II, sample size = 7,488, number of events = 940, imputation for the missing covariates.

measures were superior to BMI in identifying current diabetes. Although all elevated adiposity indices were associated with a higher incident diabetes risk, the waist-based measures had stronger associations than BMI. Persons with low BMI but high WHtR had a higher risk of developing diabetes than individuals with high BMI but low WHtR. This study provided information on the capacity of waist-based indicators to identify diabetes risk, providing the evidence of the rationale for routine monitoring of waist-based indicators in general practice, in addition to BMI.

First, in the cross-sectional analyses, we investigated the ability of different adiposity indicators to identify existing diabetes, and found that waist-based measures performed better than BMI, even though the differences are small, which is consistent with some studies [11,31]. For example, a *meta*-analysis including 39 relevant studies showed WC and WHtR were better at discriminating diabetes than BMI [11]. The explanation may be in part due to abdominal fat distribution is a more important risk factor for diabetes than general obesity [32]. Compared with BMI, waist-based measures can better reflect the accumulation of abdominal fat or ectopic fat. Additionally, although the AUCs given by WC and WHT.5R were almost as high as that of WHtR, the AUC of WHtR was slightly higher with or without controlling for covariates. It may be because WHtR is the only one of the three waist-based measures that is essentially uncorrelated with height according to our correlation analyses (height and WHtR: r was close to 0), which means it may reflect better on individual abdominal fat and reduce the influence of height on the results to some extent. As for ABSI, after including covariates, its AUC increased considerably, being only lower than WHtR in the imputed data and slightly higher than WHtR in the complete data. It suggested that for our study population, ABSI is no less capable of discriminating current diabetes than other waist-based measures, and better than BMI. However, ABSI has disadvantages for clinical applications: it is complex to compute and due to its small absolute value, individual differences in values are usually not distinguishable until 3 or 4 decimal places. Additionally, we found that, in both sexes, waist-based adiposity indices generally had higher AUCs than BMI, which is consistent with our findings in the overall population. While AUCs were higher in men than in women across all adiposity indices, this difference may be partly explained by sex-related differences in fat distribution and metabolic risk [33]. At a given adiposity level, men tend to have a higher proportion of visceral fat than women, which is strongly associated with diabetes risk [34]. Furthermore, type 2 diabetes is often diagnosed at a younger age and lower BMI in men than in women [35], which may suggest that equivalent levels of adiposity indices correspond to a higher immediate diabetes risk in men. These differences might contribute to the higher discriminative performance (AUC) of adiposity indices observed in men in this study.

For prospective association between the elevated adiposity indicators and diabetes, we found that elevated waist-based measures had higher HRs for incident diabetes than BMI, which is consistent with some studies [16,36]. For instance, a *meta*-analysis including 15 eligible studies with 120,012 persons indicated that WHtR and WC had a greater association with diabetes than BMI [36]. As mentioned before, abdominal obesity, which is highly associated with visceral fat [37], is a more important risk factor compared with general obesity, thus, measures of abdominal obesity, such as WC, WHtR, and WHT.5R, were more strongly associated with diabetes risk than BMI. Meanwhile, waist-based measures reflect not only visceral fat but also subcutaneous abdominal fat, which is strongly associated with insulin resistance and diabetes [38]. Additionally, our study showed high-value group by WHtR cut-point had the highest HR for incident diabetes, with a 42 % higher risk of developing diabetes than high-value group by BMI cut-off, indicating that “0.5 of WHtR” appears to be a better public health tool for classifying individuals into obesity-related diabetes risk categories than the often-used BMI cut-off of 25 kg/m^2. Although ABSI was developed as a transformation of WC, and increased ABSI was strongly associated with incident diabetes in our study, it produced a relatively weaker

association than the three waist-based measures of this study, which is broadly in agreement with some studies [16,39]. The nature of the variables used in the ABSI calculation and the relationships between the variables may be a potential explanation for the results. ABSI was proposed to associate body shape with mortality, statistically independently of BMI [14]. However, our outcome is diabetes, not death. Additionally, a study showed that ABSI was weakly correlated with metabolic syndrome and cardiometabolic risk which are highly associated with diabetes [40]. Furthermore, although our findings showed that increased WC seems to be more strongly associated with the diabetes than increased BMI, it does not imply that the effect of BMI gain on diabetes can be disregarded. Hence, the weak correlation of ABSI with BMI may also contribute to the current results.

In stratification analyses of prospective associations, although we have not found many statistically significant interactions between elevated adiposity indices and sex, age, ethnicity, or baseline CVD diagnosis for diabetes, overall, our measures (except ABSI) had relatively higher absolute HRs in women and relatively younger participants. These findings may be related to the following points. (1) The observation that elevated adiposity indicators were more strongly associated with diabetes risk in women is consistent with some studies [16,41]. We speculate that this may be because women tend to have a higher body fat percentage than men and fat distribution varies by sex [33]. An increase in WC appears to have a greater effect on diabetes risk than an equivalent gain in weight. When women are overweight or obese, they preferentially deposit fat subcutaneously [42], and their excess subcutaneous fat may contribute more to increased WC than in men [41], potentially elevating their diabetes risk. Additionally, the majority of the women in our study were either postmenopausal (50.9 % in our data) or currently perimenopausal. During this period, abdominal fat accumulation, especially visceral fat, becomes more pronounced in females [43]. Consequently, the association between visceral adipose tissue and diabetes risk in women is likely to become stronger, indicating their higher future diabetes risk compared with males. However, sex difference is still controversial. For example, a relevant study suggested the association between increased adiposity indices and diabetes risk was generally stronger in men [17]. Currently, there is no clear biological mechanism to explain the potential sex difference. (2) In our study, we found a weaker association between obesity and diabetes in older people (50 + years) compared with younger people, suggesting that measuring adiposity indicators may be particularly useful in the relatively younger individuals in our data. A similar finding was reported in a related study [18]. However, the reason for this finding remains unclear. We hypothesized that it may be because age is a strong influencing factor for diabetes, which may lead to a high risk of developing the disease. Consequently, the association between adiposity indicators and diabetes may be correspondingly reduced in the older age group. Further research with a broader age range is warranted. (3) Notably, our study had relatively low precision in estimating the HRs for non-white individuals and those with baseline CVD due to the small sample size (<10 %) in these subgroups.

Although central-obesity indices are thought to be more strongly associated with diabetes than BMI, we cannot completely ignore the influence of weight gain, and among our three waist-based measures, WHtR, a height-adjusted measure of WC with an accepted single cut-point, is more stable as it is less influenced by race and sex. Therefore, we further studied the effects of different levels of BMI and WHtR combinations on diabetes development, and found that "low BMI but high WHtR" group (4.7 % of sample II; "inaccurately" classified as low obesity-related risk by BMI criteria alone) had a higher risk of developing diabetes than the "high BMI but low WHtR" group (classified as high obesity-related risk by BMI criteria alone), which suggests excessive WC may have a greater effect on diabetes development than abnormal BMI. This result also indicates that when assessing diabetes risk based on obesity (or overweight status), we should not rely solely on BMI being within the normal range, as individuals with normal BMI but elevated WHtR are also at high diabetes risk. A cross-sectional study of 46,979 participants reported similar results, providing support to our findings [44]. Therefore, more people may benefit from including "maintaining normal waist-based indicators in addition to BMI" in their health management.

Even though our baseline (1991–1994) and follow-up (2007–2009) data are historical, it is well documented that the prevalence of both obesity and type 2 diabetes has continued to rise substantially from the early 1990s to the present [2,45]. These long-term trends suggest that a middle-aged individual today may face a higher absolute risk of developing diabetes than a counterpart during our study period. However, the relative associations between adiposity indices and diabetes risk are thought to be broadly consistent over time, which may be explained by stable underlying biological mechanisms [46]. Therefore, compared with BMI, the observed greater discriminative ability and stronger association of waist-based measures (such as WHtR) in relation to diabetes risk are likely to remain applicable in present-day clinical and public health settings.

There are some limitations in this study. First, our study used only a single measurement of the adiposity indicators, assuming it represents adulthood; however, these indices may change during the follow-up period. Hence, our future research will further explore this topic using repeated measurements of adiposity indicators. Second, when doing stratification analyses, the sample size for some subgroups, such as non-whites and individuals with baseline CVD, was small. This implies low statistical power to detect differences between subgroups and reduced precision in the estimates. Moreover, the predominance of White participants (over 90 %) may limit the generalizability of the findings to other populations, particularly those with markedly different ethnic compositions. Third, participants with a single fasting glucose measurement $\geq$ 7.0 mmol/L or 2-hour postload glucose $\geq$ 11.1 mmol/L, without confirmation by a second test, were classified as having diabetes, which may have led to some degree of misclassification. In addition, hemoglobin A1c (HbA1c) was not included in the diagnostic criteria, as it was not collected until Phase 7 in the WHII. Forth, data on fat content and distribution were not available for Phase 3 of WHII. Thus, we were unable to directly investigate the association between the adiposity indices and body fat or visceral fat. Fifth, since there are no recognized cut-offs for WHT.R and ABSI, we chose the 75th percentile as their cut-points [47]; however, the values (i.e., 75th percentile) may vary when the study population changes. However, we also analyzed the association between standardized values of the five indices and diabetes (avoiding some drawbacks of a dichotomous approach), reaffirming that waist-based measures are more strongly associated with diabetes risk than BMI.

5. Conclusion

This study adds evidence that waist-based measures may be more strongly associated with diabetes risk than BMI. As an easy-to-calculate, WC-derived index with a sex- and race-independent cut-point, WHtR monitoring in general practice, alongside BMI, may play a role in diabetes prevention.

CRediT authorship contribution statement

Cunrong Huang: Writing – original draft, Visualization, Validation, Software, Methodology, Formal analysis, Conceptualization. **Andre Lopes:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Formal analysis. **Annie Britton:** Writing – review & editing, Supervision, Resources, Project administration, Data curation, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.diabres.2025.112268.

References

[1] González-Muniesa P, Mártinez-González MA, Hu FB, Després JP, Matsuzawa Y, Loos RJF, et al. Obesity. Nat Rev Dis Primers 2017;3:17034. https://doi.org/10.1038/nrdp.2017.34.
[2] Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. Lancet. 2017;390:2627-42. DOI: 10.1016/S0140-6736(17)32129-3.
[3] Wang Y, Rimm EB, Stampfer MJ, Willett WC, Hu FB. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. Am J Clin Nutr 2005;81:555–63. https://doi.org/10.1093/ajcn/81.3.555.
[4] Kivimäki M, Kuosma E, Ferrie JE, Luukkonen R, Nyberg ST, Alfredsson L, et al. Overweight, obesity, and risk of cardiometabolic multimorbidity: pooled analysis of individual-level data for 120 813 adults from 16 cohort studies from the USA and Europe. Lancet Public Health 2017;2:e277–85. https://doi.org/10.1016/S2468-2667(17)30074-9.
[5] Jensen MD, Ryan DH, Apovian CM, Ard JD, Comuzzie AG, Donato KA, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults: a report of the American college of cardiology/American heart association task force on practice guidelines and the obesity society. Circulation 2014;129:S102–38. https://doi.org/10.1161/01.cir.0000437739.71477.ee.
[6] Tsigos C, Hainer V, Basdevant A, Finer N, Fried M, Mathus-Vliegen E, et al. Management of obesity in adults: European clinical practice guidelines. Obes Facts 2008;1:106–16. https://doi.org/10.1159/000126822.
[7] Piché ME, Tchernof A, Després JP. Obesity phenotypes, diabetes, and cardiovascular diseases. Circ Res 2020;126:1477–500. https://doi.org/10.1161/circresaha.120.316101.
[8] Soto González A, Bellido D, Buño MM, Pértega S, De Luis D, Martínez-Olmos M, et al. Predictors of the metabolic syndrome and correlation with computed axial tomography. Nutrition 2007;23:36–45. https://doi.org/10.1016/j.nut.2006.08.019.
[9] Kissebah AH, Vydelingum N, Murray R, Evans DJ, Hartz AJ, Kalkhoff RK, et al. Relation of body fat distribution to metabolic complications of obesity. J Clin Endocrinol Metab 1982;54:254–60. https://doi.org/10.1210/jcem-54-2-254.
[10] Seo DC, Choe S, Torabi MR. Is waist circumference ≥102/88cm better than body mass index ≥30 to predict hypertension and diabetes development regardless of sex, age group, and race/ethnicity? Meta-analysis Prev Med 2017;97:100–8. https://doi.org/10.1016/j.ypmed.2017.01.012.
[11] Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. Obes Rev 2012;13:275–86. https://doi.org/10.1111/j.1467-789X.2011.00952.x.
[12] Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0·5 could be a suitable global boundary value. Nutr Res Rev 2010;23:247–69. https://doi.org/10.1017/s0954422410000144.
[13] Nevill AM, Duncan MJ, Lahart IM, Sandercock GR. Scaling waist girth for differences in body size reveals a new improved index associated with cardiometabolic risk. Scand J Med Sci Sports 2017;27:1470–6. https://doi.org/10.1111/sms.12780.
[14] Krakauer NY, Krakauer JC. A new body shape index predicts mortality hazard independently of body mass index. PLoS One 2012;7:e39504. https://doi.org/10.1371/journal.pone.0128972.
[15] Krakauer NY, Krakauer JC. Untangling waist circumference and hip circumference from body mass index with a body shape index, hip index, and anthropometric risk indicator. Metab Syndr Relat Disord 2018;16:160–5. https://doi.org/10.1089/met.2017.0166.
[16] Boonpor J, Parra-Soto S, Talebi A, Zhou Z, Carrasco-Marin F, Petermann-Rocha F, et al. Associations and predictive performance of 11 anthropometric measures with incident type 2 diabetes: A prospective cohort study from the UK Biobank. Obesity (Silver Spring) 2023;31:2648–57. https://doi.org/10.1002/oby.23849.
[17] Ye M, Robson PJ, Eurich DT, Vena JE, Xu JY, Johnson JA. Anthropometric changes and risk of diabetes: are there sex differences? A longitudinal study of Alberta's tomorrow project. BMJ Open 2019;9:e023829. https://doi.org/10.1136/bmjopen-2018-023829.
[18] Zhang Y, Gu Y, Wang N, Zhao Q, Ng N, Wang R, et al. Association between anthropometric indicators of obesity and cardiovascular risk factors among adults in Shanghai. China BMC Public Health 2019;19:1035. https://doi.org/10.1186/s12889-019-7366-0.
[19] Mongraw-Chaffin ML, Peters SAE, Huxley RR, Woodward M. The sex-specific association between BMI and coronary heart disease: a systematic review and meta-analysis of 95 cohorts with 1·2 million participants. Lancet Diabetes Endocrinol 2015;3:437–49. https://doi.org/10.1016/S2213-8587(15)00086-8.
[20] Marmot M, Brunner E. Cohort Profile: the Whitehall II study. Int J Epidemiol 2005;34:251–6. https://doi.org/10.1093/ije/dyh372.
[21] Marmot MG, Smith GD, Stansfeld S, Patel C, North F, Head J, et al. Health inequalities among British civil servants: the Whitehall II study. Lancet 1991;337:1387–93. https://doi.org/10.1016/0140-6736(91)93068-k.
[22] Ding C, O'Neill D, Britton A. Trajectories of alcohol consumption in relation to all-cause mortality in patients with cardiovascular disease: a 35-year prospective cohort study. Addiction 2022;117:1920–30. https://doi.org/10.1111/add.15850.
[23] Britton A, Milne B, Butler T, Sanchez-Galvez A, Shipley M, Rudd A, et al. Validating self-reported strokes in a longitudinal UK cohort study (Whitehall II): Extracting information from hospital medical records versus the Hospital Episode Statistics database. BMC Med Res Methodol 2012;12:83. https://doi.org/10.1186/1471-2288-12-83.
[24] Machado-Fragua MD, Fayosse A, Yerramalla MS, van Sloten TT, Tabak AG, Kivimaki M, et al. Association of metabolic syndrome with incident dementia: role of number and age at measurement of components in a 28-year follow-up of the whitehall ii cohort study. Diabetes Care 2022;45:2127–35. https://doi.org/10.2337/dc22-0206.
[25] Alberti KG, Zimmet P, Shaw J. The metabolic syndrome–a new worldwide definition. Lancet 2005;366:1059–62. https://doi.org/10.1016/s0140-6736(05)67402-8.
[26] Ross R, Neeland IJ, Yamashita S, Shai I, Seidell J, Magni P, et al. Waist circumference as a vital sign in clinical practice: a Consensus Statement from the IAS and ICCR Working Group on Visceral Obesity. Nat Rev Endocrinol 2020;16:177–89. https://doi.org/10.1038/s41574-019-0310-7.
[27] Singh-Manoux A, Hillsdon M, Brunner E, Marmot M. Effects of physical activity on cognitive functioning in middle age: evidence from the Whitehall II prospective cohort study. Am J Public Health 2005;95:2252–8. https://doi.org/10.2105/AJPH.2004.055574.
[28] Barbiellini Amidei C, Fayosse A, Dumurgier J, Machado-Fragua MD, Tabak AG, van Sloten T, et al. Association between age at diabetes onset and subsequent risk of dementia. JAMA 2021;325:1640–9. https://doi.org/10.1001/jama.2021.4001.
[29] Tabák AG, Jokela M, Akbaraly TN, Brunner EJ, Kivimäki M, Witte DR. Trajectories of glycaemia, insulin sensitivity, and insulin secretion before diagnosis of type 2 diabetes: an analysis from the Whitehall II study. Lancet 2009;373:2215–21. https://doi.org/10.1016/S0140-6736(09)60619-X.
[30] Stensrud MJ, Hernán MA. Why test for proportional hazards? JAMA 2020;323:1401–2. https://doi.org/10.1001/jama.2020.1267.
[31] Gracey M, Burke V, Martin DD, Johnston RJ, Jones T, Davis EA. Assessment of risks of "lifestyle" diseases including cardiovascular disease and type 2 diabetes by anthropometry in remote Australian Aborigines. Asia Pac J Clin Nutr 2007;16:688–97.
[32] Taylor AE, Ebrahim S, Ben-Shlomo Y, Martin RM, Whincup PH, Yarnell JW, et al. Comparison of the associations of body mass index and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-cause mortality: a study using data from 4 UK cohorts. Am J Clin Nutr 2010;91:547–56. https://doi.org/10.3945/ajcn.2009.28757.
[33] Blaak E. Gender differences in fat metabolism. Curr Opin Clin Nutr Metab Care 2001;4:499–502. https://doi.org/10.1097/00075197-200111000-00006.
[34] Ohlson LO, Larsson B, Svärdsudd K, Welin L, Eriksson H, Wilhelmsen L, et al. The influence of body fat distribution on the incidence of diabetes mellitus. 13.5 years of follow-up of the participants in the study of men born in 1913. Diabetes 1985;34:1055–8. https://doi.org/10.2337/diab.34.10.1055.
[35] Kautzky-Willer A, Harreiter J, Pacini G. Sex and gender differences in risk, pathophysiology and complications of type 2 diabetes mellitus. Endocr Rev 2016;37:278–316. https://doi.org/10.1210/er.2015-1137.
[36] Kodama S, Horikawa C, Fujihara K, Heianza Y, Hirasawa R, Yachi Y, et al. Comparisons of the strength of associations with future type 2 diabetes risk among anthropometric obesity indicators, including waist-to-height ratio: a meta-analysis. Am J Epidemiol 2012;176:959–69. https://doi.org/10.1093/aje/kws172.
[37] Rattarasarn C, Leelawattana R, Soonthornpun S, Setasuban W, Thamprasit A, Lim A, et al. Regional abdominal fat distribution in lean and obese Thai type 2 diabetic women: relationships with insulin sensitivity and cardiovascular risk factors. Metabolism 2003;52:1444–7. https://doi.org/10.1016/s0026-0495(03)00257-9.

[38] Goodpaster BH, Thaete FL, Simoneau JA, Kelley DE. Subcutaneous abdominal fat and thigh muscle composition predict insulin sensitivity independently of visceral fat. Diabetes 1997;46:1579–85. https://doi.org/10.2337/diacare.46.10.1579.
[39] Hardy DS, Stallings DT, Garvin JT, Xu H, Racette SB. Best anthropometric discriminators of incident type 2 diabetes among white and black adults: A longitudinal ARIC study. PLoS One 2017;12:e0168282. https://doi.org/10.1371/journal.pone.0168282.
[40] Amirabdollahian F, Haghighatdoost F. Anthropometric indicators of adiposity related to body weight and body shape as cardiometabolic risk predictors in british young adults: superiority of waist-to-height ratio. J Obes 2018;2018:8370304. https://doi.org/10.1155/2018/8370304.
[41] Langenberg C, Sharp SJ, Schulze MB, Rolandsson O, Overvad K, Forouhi NG, et al. Long-term risk of incident type 2 diabetes and measures of overall and regional obesity: the EPIC-InterAct case-cohort study. PLoS Med 2012;9:e1001230. https://doi.org/10.1371/journal.pmed.1001230.
[42] Gavin KM, Bessesen DH. Sex differences in adipose tissue function. Endocrinol Metab Clin North Am 2020;49:215–28. https://doi.org/10.1016/j.ecl.2020.02.008.
[43] Garaulet M, Pérez-Llamas F, Baraza JC, Garcia-Prieto MD, Fardy PS, Tébar FJ, et al. Body fat distribution in pre-and post-menopausal women: metabolic and anthropometric variables. J Nutr Health Aging 2002;6:123–6.
[44] Liu XC, Liu YS, Guan HX, Feng YQ, Kuang J. Comparison of six anthropometric measures in discriminating diabetes: A cross-sectional study from the national health and nutrition examination survey. J Diabetes 2022;14:465–75. https://doi.org/10.1111/1753-0407.
[45] NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in diabetes prevalence and treatment from 1990 to 2022: a pooled analysis of 1108 population-representative studies with 141 million participants. Lancet. 2024;404:2077-93. DOI: 10.1016/S0140-6736(24)02317-1.
[46] Klein S, Gastaldelli A, Yki-Järvinen H, Scherer PE. Why does obesity cause diabetes? Cell Metab 2022;34(1):11–20. https://doi.org/10.1016/j.cmet.2021.12.012.
[47] Pedersen MM, Ekstrøm CT, Sørensen TIA. Emergence of the obesity epidemic preceding the presumed obesogenic transformation of the society. Sci Adv 2023;9:eadg6237. https://doi.org/10.1126/sciadv.adg6237.

Supplementary Table S1 Missingness in the included covariates for the participants

	Sample I (7979) **Missing, N (%)**	**Sample II (7488)** **Missing, N (%)**
Age	0 (0)	0 (0)
Sex	0 (0)	0 (0)
Ethnicity	28 (0.4)	20 (0.3)
Smoking	630 (7.9)	543 (7.3)
Drinking	174 (2.2)	131 (1.7)
Socioeconomic position	173 (2.2)	129 (1.7)
Physical activity	167 (2.1)	124 (1.7)
Dietary behavior	172 (2.2)	129 (1.7)
CVD diagnosis	0 (0)	0 (0)
CVD medication	0 (0)	0 (0)
Family history of diabetes	122 (1.5)	116 (1.5)
Menopause status (female)	60/2468 (2.4)	42/2295 (1.8)

CVD, cardiovascular disease

Supplementary Table S2 Characteristics of the participants in Phase 3 (baseline time)

Characteristic	Sample I, N (%) N = 7979 (100)	Sample II, N (%) N = 7488 (100)
Baseline Diabetes, N (%)	271/7979 (3.4)	0/7488 (0)
Age, mean ± SD, yrs	50.06 ± 6.03	49.95 ± 6.01
< 50	4231/7979 (53.0)	4035/7488 (53.9)
≥ 50	3748/7979 (47.0)	3453/7488 (46.1)
Sex, N (%)		
Male	5511/7979 (69.1)	5193/7488 (69.4%)
Female	2468/7979 (30.9)	2295/7488 (30.6)
Ethnicity, N (%)		
White	7188/7951 (90.4)	6827/7468 (91.4)
Non-white	763/7951 (9.6)	641/7468 (8.6)
Height, mean ± SD, cm	171.86 ± 9.45	172.01 ± 9.4
Weight, mean ± SD, kg	74.88 ± 12.74	74.83 ± 12.67
BMI, mean ± SD, kg/m^2	25.32 ± 3.74	25.26 ± 3.68
WC, mean ± SD, cm	83.82 ± 11.49	83.65 ± 11.41
WHtR, mean ± SD	0.49 ± 0.06	0.49 ± 0.06
WHT.5R, mean ± SD, cm$^{0.5}$	6.39 ± 0.83	6.38 ± 0.82
ABSI, mean ± SD, m$^{7/6}$/kg$^{2/3}$	0.07 ± 0.01	0.07 ± 0.01
SBP, mean ± SD, mm Hg	120.70 ± 13.63	120.47 ± 13.53
DBP, mean ± SD, mm Hg	79.75 ± 9.41	79.66 ± 9.38
TC, mean ± SD, mmol/L	6.49 ± 1.15	6.48 ± 1.16
TG, mean ± SD, mmol/L	1.49 ± 1.13	1.46 ± 1.10
LDL-C, mean ± SD, mmol/L	4.39 ± 1.03	4.39 ± 1.04
HDL-C, mean ± SD, mmol/L	1.43 ± 0.41	1.44 ± 0.41
Fasting glucose, mean ± SD, mmol/L	5.24 ± 0.69	5.20 ± 0.47
CRP, [a] median (IQR), mg/L	0.89 (0.45, 1.90)	0.87 (0.44, 1.83)
IL-6, [a] median (IQR), pg/mL	1.41 (1.03, 2.07)	1.39 (1.02, 2.03)
Baseline CVD diagnosis, N (%)		
Yes	738/7979 (9.2)	682/7488 (9.1)
No	7241/7979 (90.8)	6806/7488 (90.9)
Family history of CVD (angina, MI, stroke), N (%)	3671/7039 (52.2)	3450/6603 (52.2)
Family history of diabetes, N (%)	906/7857 (11.5)	799/7372 (10.8)
Family history of hypertension, N (%)	3077/6855 (44.9)	2891/6427 (45.0)
Smoking Status, N (%)		
Never smoker	3452/7349 (47.0)	3286/6945 (47.3)
Ex-smoker	2839/7349 (38.6)	2680/6945 (38.6)
Current smoker	1058/7349 (14.4)	979/6945 (14.1)
Drinking, N (%)		
Current not drinking	1513/7805 (19.4)	1378/7357 (18.7)
Current moderate (1-14 unit/w)	4399/7805 (56.4)	4178/7357 (56.8)
Current heavy (>14 unit/w)	1893/7805 (24.3)	1801/7357 (24.5)
Fruit/vegetable consumption, N (%)		
< Daily	3040/7807 (38.9)	2840/7359 (38.6)
≥ Daily	4767/7807 (61.1)	4519/7359 (61.4)
Physical activity		
Inactive	1610/7812 (20.6)	1467/7364 (19.9)
Moderate	2780/7812 (35.6)	2649/7364 (36.0)
Active	3422/7812 (43.8)	3248/7364 (44.1)
Education level, N (%)		
Low	300/5989 (5.0)	284/5619 (5.1)
Middle	3120/5989 (52.1)	2909/5619 (51.8)
High	2569/5989 (42.9)	2426/5619 (43.2)
Socioeconomic position, N (%)		
Low	1297/7806 (16.6)	1143/7359 (15.5)
Intermediate	2973/7806 (38.1)	2872/7359 (39.0)
High	3536/7806 (45.3)	3344/7359 (45.4)

[a] Not follow normal distribution, shown with median (25th–75th percentiles);

SD, standard deviation; IQR, interquartile range; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index; CVD, cardiovascular disease; MI, myocardial infarction; TC, total

cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6; Drinking: current moderate (1-14 unit/w), current heavy (>14 unit/w); Socioeconomic position: defined using either current or last recorded employment grade; Physical activity: active (>2.5 hrs/week of moderate physical activity or >1 hr/week of vigorous physical activity), inactive (<1 hr/week of moderate physical activity and <1 hr/week of vigorous physical activity), or moderately active (if not active or inactive).

Supplementary Table S3a Characteristics of the Sample II in Phase 3 *according to BMI criterion*

Characteristic	BMI < 25 kg/m^2, N (%) N = 3949 (52.7)	BMI ≥ 25 kg/m^2, N (%) N = 3539 (47.3)	P value [a]
Age, mean ± SD, yrs	49.48 ± 6.00	50.47 ± 5.98	< 0.001
Sex (Female), N (%)	1196/3949 (30.3)	1099/3539 (31.1)	0.487
Ethnicity (White), N (%)	3643/3936 (92.6)	3184/3532 (90.1)	< 0.001
Height, mean ± SD, cm	172.53 ± 9.30	171.44 ± 9.49	< 0.001
Weight, mean ± SD, kg	67.67 ± 9.09	82.81 ± 11.24	< 0.001
SBP, mean ± SD, mm Hg	117.84 ± 13.33	123.40 ± 13.15	< 0.001
DBP, mean ± SD, mm Hg	77.33 ± 9.01	82.25 ± 9.10	< 0.001
TC, mean ± SD, mmol/L	6.32 ± 1.11	6.66 ± 1.18	< 0.001
TG, mean ± SD, mmol/L	1.22 ± 0.91	1.73 ± 1.22	< 0.001
LDL-C, mean ± SD, mmol/L	4.24 ± 1.01	4.55 ± 1.04	< 0.001
HDL-C, mean ± SD, mmol/L	1.53 ± 0.42	1.34 ± 0.37	< 0.001
Fasting glucose, mean ± SD, mmol/L	5.14 ± 0.46	5.26 ± 0.47	< 0.001
CRP, [b] median (IQR), mg/L	0.63 (0.32, 1.25)	1.22 (0.66, 2.53)	< 0.001
IL-6, [b] median (IQR), pg/mL	1.25 (0.93, 1.78)	1.58 (1.15, 2.35)	< 0.001
Baseline CVD diagnosis, N (%)	304/3949 (7.7)	378/3539 (10.7)	< 0.001
Family history of CVD (angina, MI, stroke), N (%)	1782/3480 (51.2)	1668/3123 (53.4)	0.078
Family history of diabetes, N (%)	359/3902 (9.2)	440/3470 (12.7)	< 0.001
Family history of hypertension, N (%)	1493/3387 (44.1)	1398/3040 (46.0)	0.131
Smoking Status, N (%)			< 0.001
Never smoker	1835/3687 (49.8)	1451/3258 (44.5)	
Ex-smoker	1333/3687 (36.2)	1347/3258 (41.3)	
Current smoker	519/3687 (14.1)	460/3258 (14.1)	
Drinking, N (%)			< 0.001
Current not drinking	718/3902 (18.4)	660/3455 (19.1)	
Current moderate (1-14 unit/w)	2292/3902 (58.7)	1886/3455 (54.6)	
Current heavy (>14 unit/w)	892/3902 (22.9)	909/3455 (26.3)	
Fruit/vegetable consumption, N (%)			< 0.001
< Daily	3040/3900 (36.4)	1420/3459 (41.1)	
≥ Daily	4767/3900 (63.6)	2039/3459 (58.9)	
Physical activity			< 0.001
Inactive	1610/3903 (17.9)	1467/3461 (22.2)	
Moderate	2780/3903 (36.6)	2649/3461 (35.3)	
Active	3422/3903 (45.5)	3248/3461 (42.6)	
Education level, N (%)			< 0.001
Low	133/2932 (4.5)	151/2687 (5.1)	
Middle	1409/2932 (48.1)	1500/2687 (51.8)	
High	1390/2932 (47.4)	1036/2687 (43.2)	
Socioeconomic position, N (%)			< 0.001
Low	510/3902 (13.1)	633/3457 (18.3)	
Intermediate	1646/3902 (42.2)	1226/3457 (35.5)	
High	1746/3902 (44.7)	1598/3457 (46.2)	

[a] T-test or Wilcoxon rank-sum test for continuous variables and chi-square test for categorical variables;

[b] Not follow normal distribution, shown with median (25th-75th percentiles);

BMI, body mass index; SD, standard deviation; IQR, interquartile range; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6

Supplementary Table S3b Characteristics of the Sample II in Phase 3 *according to WC criterion*

Characteristic	WC < cut-point, N (%) N = 4908 (65.5)	WC ≥ cut-point, N (%) N = 2580 (34.5)	P value [a]
Age, mean ± SD, yrs	49.47 ± 5.99	50.85 ± 5.94	< 0.001
Sex (Female), N (%)	1603/4908 (32.7)	692/2580 (26.8)	< 0.001
Ethnicity (White), N (%)	4504/4892 (92.1)	2323/2576 (90.2)	0.006
Height, mean ± SD, cm	171.38 ± 9.29	173.22 ± 9.51	< 0.001
Weight, mean ± SD, kg	69.04 ± 9.25	85.84 ± 10.86	< 0.001
SBP, mean ± SD, mm Hg	118.15 ± 13.15	124.89 ± 13.14	< 0.001
DBP, mean ± SD, mm Hg	77.66 ± 8.96	83.46 ± 8.98	< 0.001
TC, mean ± SD, mmol/L	6.36 ± 1.13	6.71 ± 1.17	< 0.001
TG, mean ± SD, mmol/L	1.23 ± 0.87	1.90 ± 1.33	< 0.001
LDL-C, mean ± SD, mmol/L	4.28 ± 1.02	4.58 ± 1.03	< 0.001
HDL-C, mean ± SD, mmol/L	1.52 ± 0.42	1.29 ± 0.36	< 0.001
Fasting glucose, mean ± SD, mmol/L	5.15 ± 0.46	5.28 ± 0.46	< 0.001
CRP, [b] median (IQR), mg/L	0.67 (0.35, 1.32)	1.40 (0.78, 2.87)	< 0.001
IL-6, [b] median (IQR), pg/mL	1.26 (0.94, 1.81)	1.69 (1.22, 2.52)	< 0.001
Baseline CVD diagnosis, N (%)	370/4908 (7.5)	312/2580 (12.1)	< 0.001
Family history of CVD (angina, MI, stroke), N (%)	2213/4334 (51.2)	1237/2269 (53.4)	0.008
Family history of diabetes, N (%)	458/4847 (9.4)	341/2525 (13.5)	< 0.001
Family history of hypertension, N (%)	1879/4246 (44.3)	1012/2181 (46.4)	0.107
Smoking Status, N (%)			< 0.001
Never smoker	2276/4569 (49.8)	1010/2376 (42.5)	
Ex-smoker	1657/4569 (36.3)	1023/2376 (43.1)	
Current smoker	636/4569 (13.9)	343/2376 (14.4)	
Drinking, N (%)			< 0.001
Current not drinking	913/4847 (18.8)	465/2510 (18.5)	
Current moderate (1-14 unit/w)	2865/4847 (59.1)	1313/2510 (52.3)	
Current heavy (>14 unit/w)	1069/4847 (22.1)	732/2510 (29.2)	
Fruit/vegetable consumption, N (%)			< 0.001
< Daily	1771/4845 (36.6)	1069/2514 (42.5)	
≥ Daily	3074/4845 (63.4)	1445/2514 (57.5)	
Physical activity			< 0.001
Inactive	889/4848 (18.3)	578/2516 (23.0)	
Moderate	1725/4848 (35.6)	924/2516 (36.7)	
Active	2234/4848 (46.1)	1014/2516 (40.3)	
Education level, N (%)			< 0.001
Low	178/3684 (4.8)	106/1935 (5.5)	
Middle	1829/3684 (49.6)	1080/1935 (55.8)	
High	1677/3684 (45.5)	749/1935 (38.7)	
Socioeconomic position, N (%)			0.005
Low	708/4847 (14.6)	435/2512 (17.3)	
Intermediate	1934/4847 (39.9)	938/2512 (37.3)	
High	2205/4847 (45.5)	1139/2512 (45.3)	

[a] T-test or Wilcoxon rank-sum test for continuous variables and chi-square test for categorical variables;

[b] Not follow normal distribution, shown with median (25th-75th percentiles);

WC, waist circumference; WC cut-point: 90 cm for men and 80 cm for women; SD, standard deviation; IQR, interquartile range; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6.

Supplementary Table S3c Characteristics of the Sample II in Phase 3 *according to WHtR criterion*

Characteristic	WHtR < 0.5, N (%) N = 4557 (60.9)	WHtR ≥ 0.5, N (%) N = 2931 (39.1)	P value [a]
Age, mean ± SD, yrs	49.26 ± 5.95	51.01 ± 5.94	< 0.001
Sex (Female), N (%)	1646/4557 (36.1)	649/2931 (22.1)	< 0.001
Ethnicity (White), N (%)	4232/4543 (93.2)	2595/2925 (88.7)	0.006
Height, mean ± SD, cm	172.17 ± 9.61	171.77 ± 9.07	0.072
Weight, mean ± SD, kg	69.30 ± 9.88	83.43 ± 11.68	< 0.001
SBP, mean ± SD, mm Hg	117.91 ± 13.09	124.45 ± 13.25	< 0.001
DBP, mean ± SD, mm Hg	77.38 ± 8.89	83.19 ± 9.03	< 0.001
TC, mean ± SD, mmol/L	6.31 ± 1.13	6.73 ± 1.15	< 0.001
TG, mean ± SD, mmol/L	1.19 ± 0.86	1.88 ± 1.28	< 0.001
LDL-C, mean ± SD, mmol/L	4.24 ± 1.02	4.62 ± 1.02	< 0.001
HDL-C, mean ± SD, mmol/L	1.53 ± 0.42	1.28 ± 0.35	< 0.001
Fasting glucose, mean ± SD, mmol/L	5.14 ± 0.46	5.29 ± 0.47	< 0.001
CRP, [b] median (IQR), mg/L	0.64 (0.34, 1.27)	1.36 (0.74, 2.79)	< 0.001
IL-6, [b] median (IQR), pg/mL	1.26 (0.93, 1.80)	1.64 (1.18, 2.43)	< 0.001
Baseline CVD diagnosis, N (%)	332/4557 (7.3)	350/2931 (11.9)	< 0.001
Family history of CVD (angina, MI, stroke), N (%)	2045/4019 (50.9)	1405/2584 (54.4)	0.006
Family history of diabetes, N (%)	419/4495 (9.3)	380/2877 (13.2)	< 0.001
Family history of hypertension, N (%)	1756/3936 (44.6)	1135/2491 (45.6)	0.471
Smoking Status, N (%)			< 0.001
Never smoker	2140/4244 (50.4)	1146/2701 (42.4)	
Ex-smoker	1515/4244 (35.7)	1165/2701 (43.1)	
Current smoker	589/4244 (13.9)	390/2701 (14.4)	
Drinking, N (%)			< 0.001
Current not drinking	840/4503 (18.7)	538/2854 (18.9)	
Current moderate (1-14 unit/w)	2689/4503 (59.7)	1489/2854 (52.2)	
Current heavy (>14 unit/w)	974/4503 (21.6)	827/2854 (29.0)	
Fruit/vegetable consumption, N (%)			< 0.001
< Daily	1599/4501 (35.5)	1241/2858 (43.4)	
≥ Daily	2902/4501 (64.5)	1617/2858 (56.6)	
Physical activity			< 0.001
Inactive	826/4504 (18.3)	641/2860 (22.4)	
Moderate	1620/4504 (36.0)	1029/2860 (36.0)	
Active	2058/4504 (45.7)	1190/2860 (41.6)	
Education level, N (%)			< 0.001
Low	161/3382 (4.8)	123/2237 (5.5)	
Middle	1672/3382 (49.4)	1237/2237 (55.3)	
High	1549/3382 (45.8)	877/2237 (39.2)	
Socioeconomic position, N (%)			0.016
Low	672/4503 (14.9)	471/2856 (16.5)	
Intermediate	1813/4503 (40.3)	1059/2856 (37.3)	
High	2018/4503 (44.8)	1326/2856 (46.4)	

[a] T-test or Wilcoxon rank-sum test for continuous variables and chi-square test for categorical variables;

[b] Not follow normal distribution, shown with median (25th–75th percentiles);

WHtR, waist-to-height ratio; SD, standard deviation; IQR, interquartile range; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6

Supplementary Table S3d Characteristics of the Sample II in Phase 3 *according to WHT.5R criterion*

Characteristic	WHT.5R < 75th population-wide centile, N (%) N = 5666 (75.7)	WHT.5R ≥ 75th population-wide centile N (%) N = 1822 (24.3)	P value [a]
Age, mean ± SD, yrs	49.66 ± 6.01	50.84 ± 5.92	< 0.001
Sex (Female), N (%)	1984/5666 (35.0)	311/1822 (17.1)	< 0.001
Ethnicity (White), N (%)	5178/5648 (91.7)	1649/1820 (90.6)	0.169
Height, mean ± SD, cm	171.52 ± 9.52	173.55 ± 8.87	0.072
Weight, mean ± SD, kg	70.48 ± 9.87	88..36 ± 10.72	< 0.001
SBP, mean ± SD, mm Hg	118.71 ± 13.22	125.95 ± 13.04	< 0.001
DBP, mean ± SD, mm Hg	78.16 ± 9.02	84.30 ± 8.95	< 0.001
TC, mean ± SD, mmol/L	6.40 ± 1.14	6.72 ± 1.16	< 0.001
TG, mean ± SD, mmol/L	1.28 ± 0.89	2.03 ± 1.42	< 0.001
LDL-C, mean ± SD, mmol/L	4.32 ± 1.03	4.60 ± 1.02	< 0.001
HDL-C, mean ± SD, mmol/L	1.50 ± 0.41	1.23 ± 0.33	< 0.001
Fasting glucose, mean ± SD, mmol/L	5.16 ± 0.46	5.31 ± 0.47	< 0.001
CRP, [b] median (IQR), mg/L	0.72 (0.38, 1.42)	1.56 (0.84, 3.12)	< 0.001
IL-6, [b] median (IQR), pg/mL	1.31 (0.96, 1.88)	1.73 (1.24, 2.57)	< 0.001
Baseline CVD diagnosis, N (%)	445/5666 (7.9)	237/1822 (13.0)	< 0.001
Family history of CVD (angina, MI, stroke), N (%)	2575/5003 (51.5)	875/1600 (54.7)	0.027
Family history of diabetes, N (%)	554/5586 (9.9)	245/1786 (13.7)	< 0.001
Family history of hypertension, N (%)	2180/4894 (44.5)	711/1533 (46.4)	0.218
Smoking Status, N (%)			< 0.001
Never smoker	2618/5257 (49.8)	668/1688 (39.6)	
Ex-smoker	1916/5257 (36.4)	764/1688 (45.3)	
Current smoker	723/5257 (13.8)	256/1688 (15.2)	
Drinking, N (%)			< 0.001
Current not drinking	1083/5584 (19.4)	295/1773 (16.6)	
Current moderate (1-14 unit/w)	3262/5584 (58.4)	916/1773 (51.7)	
Current heavy (>14 unit/w)	1239/5584 (22.2)	562/1773 (31.7)	
Fruit/vegetable consumption, N (%)			< 0.001
< Daily	2056/5584 (36.8)	784/1775 (44.2)	
≥ Daily	3528/5584 (63.2)	991/1775 (55.8)	
Physical activity			0.007
Inactive	1084/5587 (19.4)	383/1777 (21.6)	
Moderate	1983/5587 (35.5)	666/1777 (37.5)	
Active	2520/5587 (45.1)	728/1777 (41.0)	
Education level, N (%)			0.060
Low	211/4227 (5.0)	73/1392 (5.2)	
Middle	2153/4227 (50.9)	756/1392 (54.3)	
High	1863/4227 (44.1)	563/1392 (40.4)	
Socioeconomic position, N (%)			0.162
Low	892/5586 (16.0)	251/1773 (14.2)	
Intermediate	2160/5586 (38.7)	712/1773 (40.2)	
High	2534/5586 (45.4)	810/1773 (45.7)	

[a] T-test or Wilcoxon rank-sum test for continuous variables and chi-square test for categorical variables;

[b] Not follow normal distribution, shown with median (25th–75th percentiles);

WHT.5R, waist-by-height$^{0.5}$; WHT.5R: 75th population-wide centile (6.911 cm$^{0.5}$); SD, standard deviation; IQR, interquartile range; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6

Supplementary Table S3e Characteristics of the Sample II in Phase 3 *according to ABSI criterion*

Characteristic	ABSI < 75th population-wide centile, N (%) N = 5655 (75.5)	ABSI ≥ 75th population-wide centile N (%) N = 1833 (24.5)	P value [a]
Age, mean ± SD, yrs	49.54 ± 5.97	51.21 ± 5.97	< 0.001
Sex (Female), N (%)	2239/5655 (39.6)	56/1833 (3.1)	< 0.001
Ethnicity (White), N (%)	5165/5640 (91.6)	1662/1828 (90.9)	0.169
Height, mean ± SD, cm	170.71 ± 9.59	176.02 ± 7.49	< 0.001
Weight, mean ± SD, kg	72.85 ± 12.25	80.92 ± 11.97	< 0.001
SBP, mean ± SD, mm Hg	119.38 ± 13.38	123.82 ± 13.45	< 0.001
DBP, mean ± SD, mm Hg	78.62 ± 9.21	82.86 ± 9.18	< 0.001
TC, mean ± SD, mmol/L	6.43 ± 1.16	6.64 ± 1.13	< 0.001
TG, mean ± SD, mmol/L	1.33 ± 0.93	1.86 ± 1.43	< 0.001
LDL-C, mean ± SD, mmol/L	4.33 ± 1.04	4.56 ± 0.99	< 0.001
HDL-C, mean ± SD, mmol/L	1.49 ± 0.42	1.26 ± 0.33	< 0.001
Fasting glucose, mean ± SD, mmol/L	5.17 ± 0.46	5.28 ± 0.47	< 0.001
CRP, [b] median (IQR), mg/L	0.78 (0.40, 1.62)	1.19 (0.63, 2.41)	< 0.001
IL-6, [b] median (IQR), pg/mL	1.34 (0.98, 1.98)	1.55 (1.17, 2.25)	< 0.001
Baseline CVD diagnosis, N (%)	467/5655 (8.3)	215/1833 (11.7)	< 0.001
Family history of CVD (angina, MI, stroke), N (%)	2569/5005 (51.3)	881/1598 (55.1)	0.009
Family history of diabetes, N (%)	587/5574 (10.5)	212/1798 (11.8)	0.147
Family history of hypertension, N (%)	2201/4904 (44.9)	690/1523 (45.3)	0.794
Smoking Status, N (%)			< 0.001
Never smoker	2574/5229 (49.2)	712/1716 (41.5)	
Ex-smoker	1931/5229 (36.9)	749/1716 (43.6)	
Current smoker	724/5229 (13.8)	255/1716 (14.9)	
Drinking, N (%)			< 0.001
Current not drinking	1111/5556 (20.0)	267/1801 (14.8)	
Current moderate (1-14 unit/w)	3241/5556 (58.3)	937/1801 (52.0)	
Current heavy (>14 unit/w)	1204/5556 (21.7)	597/1801 (33.1)	
Fruit/vegetable consumption, N (%)			< 0.001
< Daily	2017/5555 (36.3)	823/1804 (45.6)	
≥ Daily	3538/5555 (63.7)	981/1804 (54.4)	
Physical activity			0.002
Inactive	1129/5560 (20.3)	338/1804 (18.7)	
Moderate	1938/5560 (34.9)	711/1804 (39.4)	
Active	2493/5560 (44.8)	755/1804 (41.9)	
Education level, N (%)			0.125
Low	218/4251 (5.1)	66/1368 (4.8)	
Middle	2168/4251 (51.0)	741/1368 (54.2)	
High	1865/4251 (43.9)	561/1368 (41.0)	
Socioeconomic position, N (%)			< 0.001
Low	989/5558 (17.8)	154/1801 (8.6)	
Intermediate	2074/5558 (37.3)	798/1801 (44.3)	
High	2495/5558 (44.9)	849/1801 (47.1)	

[a] T-test or Wilcoxon rank-sum test for continuous variables and chi-square test for categorical variables;

[b] Not follow normal distribution, shown with median (25th–75th percentiles);

ABSI, a body shape index; ABSI: 75th population-wide centile (0.078 $m^{7/6}/kg^{2/3}$); SD, standard deviation; IQR, interquartile range; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; BP, blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6

Supplementary Table S4 Characteristics of the *participants with complete data* in Phase 3 (baseline time)

Characteristic	Participants with complete data of Sample I, N (%) N = 7206 (100)	Participants with complete data of Sample II, N (%) N = 6813 (100)
Baseline Diabetes, N (%)	228/7206 (3.2)	0/6813 (0)
Age, mean ± SD, yrs	50.09 ± 6.04	49.97 ± 6.02
< 50	3789/7206 (52.6)	3640/6813 (53.4)
≥ 50	3417/7206 (47.4)	3173/6813 (46.6)
Sex, N (%)		
Male	5013/7206 (69.6)	4753/6813 (69.8)
Female	2193/7206 (30.4)	2060/6813 (30.2)
Ethnicity, N (%)		
White	6564/7206 (91.1)	6266/6813 (92.0)
Non-white	642/7206 (8.9)	547/6813 (8.0)
Height, mean ± SD, cm	171.95 ± 9.35	172.07 ± 9.32
Weight, mean ± SD, kg	74.81 ± 12.67	74.77 ± 12.61
BMI, mean ± SD, kg/m^2	25.27 ± 3.71	25.22 ± 3.67
WC, mean ± SD, cm	83.75 ± 11.48	83.60 ± 11.41
WHtR, mean ± SD	0.49 ± 0.06	0.49 ± 0.06
WHT.5R, mean ± SD, cm$^{0.5}$	6.38 ± 0.83	6.37 ± 0.82
ABSI, mean ± SD, m$^{7/6}$/kg$^{2/3}$	0.07 ± 0.01	0.07 ± 0.01
SBP, mean ± SD, mm Hg	120.66 ± 13.64	120.45 ± 13.54
DBP, mean ± SD, mm Hg	79.75 ± 9.38	79.65 ± 9.35
TC, mean ± SD, mmol/L	6.49 ± 1.15	6.48 ± 1.16
TG, mean ± SD, mmol/L	1.48 ± 1.12	1.46 ± 1.09
LDL-C, mean ± SD, mmol/L	4.39 ± 1.03	4.39 ± 1.04
HDL-C, mean ± SD, mmol/L	1.43 ± 0.41	1.44 ± 0.41
Fasting glucose, mean ± SD, mmol/L	5.24 ± 0.69	5.19 ± 0.47
CRP, [a] median (IQR), mg/L	0.89 (0.45, 1.91)	0.87 (0.44, 1.85)
IL-6, [a] median (IQR), pg/mL	1.41 (1.03, 2.07)	1.39 (1.02, 2.03)
Baseline CVD diagnosis, N (%)		
Yes	688/7206 (9.5)	6178/6813 (90.7)
No	6518/7206 (90.5)	635/6813 (9.3)
Family history of CVD (angina, MI, stroke), N (%)	3327/6394 (52.0)	3151/6043 (52.1)
Family history of diabetes, N (%)	828/7206 (11.5)	742/6813 (10.9)
Family history of hypertension, N (%)	2774/6229 (44.5)	2633/5888 (44.7)
Smoking Status, N (%)		
Never smoker	3389/7206 (47.0)	3226/6813 (47.4)
Ex-smoker	2774/7206 (38.5)	2623/6813 (38.5)
Current smoker	1043/7206 (14.5)	964/6813 (14.1)
Drinking, N (%)		
Current not drinking	1370/7206 (19.0)	1253/6813 (18.4)
Current moderate (1-14 unit/w)	4058/7206 (56.3)	3864/6813 (57.7)
Current heavy (>14 unit/w)	1778/7206 (24.7)	1696/6813 (24.9)
Fruit/vegetable consumption, N (%)		
< Daily	2777/7206 (38.5)	2604/6813 (38.2)
≥ Daily	4429/7206 (61.5)	4209/6813 (61.8)
Physical activity		
Inactive	1451/7206	1326/6813 (19.5)
Moderate	2568/7206	2450/6813 (36.0)
Active	3187/7206	3037/6813 (44.6)
Education level, N (%)		
Low	263/5444 (4.8)	249/5147 (4.8)
Middle	2857/5444 (52.5)	2687/5147 (52.2)
High	2324/5444 (42.7)	2211/5147 (43.0)
Socioeconomic position, N (%)		
Low	1159/7206 (16.1)	1023/6813 (15.0)
Intermediate	2757/7206 (38.2)	2670/6813 (38.2)
High	3290/7206 (45.7)	3120/6813 (45.8)

[a] Not follow normal distribution, shown with median (25th–75th percentiles);

SD, standard deviation; IQR, interquartile range; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio;

WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6

Supplementary Table S5a Characteristics of the Sample I in Phase 3 according to sex

Characteristic	Male, N (%) N = 5511 (69.1)	Female, N (%) N = 2468 (30.9)	P value [a]
Baseline diabetes	183/5511 (3.3)	88/2468 (3.6)	0.623
Age, mean±SD, yrs	49.75 ± 5.98	50.76 ± 6.08	< 0.001
Ethnicity (White), N (%)	5098/5500 (92.7)	2090/2451 (85.3)	< 0.001
Height, mean±SD, cm	176.36 ± 6.71	161.81 ± 6.49	< 0.001
Weight, mean±SD, kg	78.27 ± 11.21	67.33 ± 12.72	< 0.001
BMI, mean±SD, kg/m^2	25.14 ± 3.18	25.72 ± 4.73	< 0.001
WC, mean±SD, cm	87.47 ± 9.31	75.68 ± 11.73	< 0.001
WHtR, mean±SD	0.50 ± 0.05	0.47 ± 0.07	< 0.001
WHT.5R, mean±SD, cm$^{0.5}$	6.59 ± 0.70	5.95 ± 0.93	< 0.001
ABSI, mean±SD, m$^{7/6}$/kg$^{2/3}$	0.08 ± 0.00	0.07 ± 0.01	< 0.001
SBP, mean±SD, mm Hg	122.00 ± 13.23	117.78 ± 14.05	< 0.001
DBP, mean±SD, mm Hg	81.10 ± 9.19	76.75 ± 9.19	< 0.001
TC, mean±SD, mmol/L	6.47 ± 1.13	6.51 ± 1.21	0.161
TG, mean±SD, mmol/L	1.61 ± 1.23	1.21 ± 0.78	<0.001
LDL-C, mean±SD, mmol/L	4.44 ± 0.99	4.29 ± 1.11	<0.001
HDL-C, mean±SD, mmol/L	1.32 ± 0.35	1.68 ± 0.43	<0.001
Fasting glucose, mean±SD, mmol/L	5.31 ± 0.69	5.09 ± 0.69	< 0.001
CRP, [b] median (IQR), mg/L	0.85 (0.43, 1.73)	1.02 (0.47, 2.36)	<0.001
IL-6, [b] median (IQR), pg/mL	1.35 (1.00, 1.94)	1.57 (1.11, 2.45)	< 0.001
Family history of diabetes, N (%)	571/5434 (10.5)	335/2423 (13.8)	< 0.001
Family history of CVD (angina, MI, stroke), N (%)	2519/4851 (51.9)	1152/2188 (52.7)	0.592
Smoking Status, N (%)			< 0.001
Never smoker	2288/5103 (44.8)	1164/2246 (51.8)	
Ex-smoker	2149/5103 (42.1)	690/2246 (30.7)	
Current smoker	666/5103 (13.1)	392/2246 (17.5)	
Drinking, N (%)			< 0.001
Current not drinking	780/5395 (14.5)	733/2410 (30.4)	
Current moderate	2946/5395 (54.6)	1453/2410 (60.3)	
Current heavy	1669/5395 (30.9)	224/2410 (9.3)	
Fruit/vegetable consumption, N (%)			< 0.001
< Daily	2221/5396 (41.2)	810/2411 (34.0)	
≥ Daily	3175/5396 (58.8)	1592/2411 (66.0)	
Education level, N (%)			< 0.001
Low	201/4177 (4.8)	99/1812 (5.5)	
Middle	2062/4177 (49.4)	1058/1812 (58.4)	
High	1914/4177 (45.8)	655/1812 (36.1)	
Physical activity			<0.001
Inactive	767/5400 (14.2)	843/2412 (35.0)	
Moderate	1975/5400 (36.6)	805/2412 (33.4)	
Active	2658/5400 (49.2)	764/2412 (31.7)	
Socioeconomic position, N (%)			<0.001
Low	356/5395 (6.6)	941/2411 (39.0)	
Intermediate	2590/5395 (48.0)	383/2411 (15.9)	
High	2449/5395 (45.4)	1087/2411 (45.1)	
Menopause, N (%)	NA	1252/2408 (52.0)	NA

[a] T-test or Wilcoxon rank-sum test for continuous variables and chi-square test for categorical variables;

[b] not follow normal distribution, shown with median (25th-75th percentiles);

SD, standard deviation; IQR, interquartile range; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6

Supplementary Table S5b Characteristics of the Sample II in Phase 3 according to sex

Characteristic	Male, N (%) N = 5193 (69.4)	Female, N (%) N = 2295 (30.6)	P value [a]
Age, mean ± SD, yrs	49.66 ± 5.96	50.60 ± 6.07	< 0.001
Ethnicity (White), N (%)	4855/5185 (93.6)	1972/2283 (86.4)	< 0.001
Height, mean ± SD, cm	176.46 ± 6.67	161.95 ± 6.48	< 0.001
Weight, mean ± SD, kg	78.22 ± 11.10	67.16 ± 12.66	< 0.001
BMI, mean ± SD, kg/m^2	25.10 ± 3.13	25.62 ± 4.68	< 0.001
WC, mean ± SD, cm	87.32 ± 9.22	75.34 ± 11.55	< 0.001
WHtR, mean ± SD	0.50 ± 0.05	0.47 ± 0.07	< 0.001
WHT.5R, mean ± SD, cm$^{0.5}$	6.58 ± 0.69	5.92 ± 0.91	< 0.001
ABSI, mean ± SD, m$^{7/6}$/kg$^{2/3}$	0.08 ± 0.00	0.07 ± 0.01	< 0.001
SBP, mean ± SD, mm Hg	121.73 ± 13.14	117.62 ± 13.97	< 0.001
DBP, mean ± SD, mm Hg	80.96 ± 9.16	76.72 ± 9.22	< 0.001
TC, mean ± SD, mmol/L	6.47 ± 1.13	6.50 ± 1.21	0.232
TG, mean ± SD, mmol/L	1.58 ± 1.20	1.19 ± 0.76	< 0.001
LDL-C, mean ± SD, mmol/L	4.44 ± 1.00	4.27 ± 1.11	< 0.001
HDL-C, mean ± SD, mmol/L	1.32 ± 0.35	1.69 ± 0.43	< 0.001
Fasting glucose, mean ± SD, mmol/L	5.26 ± 0.45	5.05 ± 0.47	< 0.001
CRP, [b] median (IQR), mg/L	0.83 (0.43, 1.69)	1.00 (0.46, 2.23)	< 0.001
IL-6, [b] median (IQR), pg/mL	1.34 (0.98, 1.91)	1.55 (1.10, 2.41)	< 0.001
Family history of diabetes, N (%)	501/5120 (9.8)	298/2252 (13.2)	< 0.001
Family history of CVD (angina, MI, stroke), N (%)	2379/4574 (52.0)	1071/2029 (52.8)	0.580
Smoking Status, N (%)			< 0.001
Never smoker	2191/4837 (45.3)	1095/2108 (51.9)	
Ex-smoker	2031/4837 (42.0)	649/2108 (30.8)	
Current smoker	615/4837 (12.7)	364/2108 (17.3)	
Drinking, N (%)			< 0.001
Current not drinking	716/5102 (14.0)	662/2255 (29.4)	
Current moderate (1-14 unit/w)	2801/5102 (54.9)	1377/2255 (61.1)	
Current heavy (>14 unit/w)	1585/5102 (31.1)	216/2255 (9.6)	
Fruit/vegetable consumption, N (%)			< 0.001
< Daily	2084/5103 (40.8)	756/2256 (33.5)	
≥ Daily	3019/5103 (59.2)	1500/2256 (66.5)	
Physical activity			<0.001
Inactive	692/5107 (13.6)	775/2257 (34.3)	
Moderate	1885/5107 (36.9)	764/2257 (33.9)	
Active	2530/5107 (49.5)	718/2257 (31.8)	
Socioeconomic position, N (%)			<0.001
Low	300/5103 (5.9)	843/2256 (37.4)	
Intermediate	2497/5103 (48.9)	375/2256 (16.6)	
High	2306/5103 (45.2)	1038/2256 (46.0)	
Menopause, N (%)	NA	1146/2253 (50.9)	NA

[a] T-test or Wilcoxon rank-sum test for continuous variables and chi-square test for categorical variables;

[b] Not follow normal distribution, shown with median (25th-75th percentiles);

SD, standard deviation; IQR, interquartile range; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index; CVD, cardiovascular disease; MI, myocardial infarction; TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; SBP, systolic blood pressure; DBP, diastolic blood pressure; CRP, C-Reactive Protein; IL-6, Interleukin 6

Supplementary Table S6 Correlation matrix for correlation between different adiposity indicators among Sample I, Sample II, and the participants with complete data from the Sample I and Sample II

a. Sample I (N=7979)

	Weight	Height	BMI	WC	WHtR	WHT.5R	ABSI
Weight	-						
Height	0.55	-					
BMI	0.76	-0.11	-				
WC	0.87	0.39	0.73	-			
WHtR	0.71	-0.01	0.85	0.92	-		
WHT.5R	0.81	0.21	0.81	0.98	0.98	-	
ABSI	0.41	0.49	0.12	0.74	0.59	0.68	-

b. Sample II (N=7488)

	Weight	Height	BMI	WC	WHtR	WHT.5R	ABSI
Weight	-						
Height	0.55	-					
BMI	0.76	-0.11	-				
WC	0.87	0.40	0.73	-			
WHtR	0.71	0.01	0.85	0.92	-		
WHT.5R	0.81	0.22	0.80	0.98	0.98	-	
ABSI	0.42	0.49	0.12	0.74	0.59	0.68	-

c. Participants with *complete data* among the *Sample I* (N=7206)

	Weight	Height	BMI	WC	WHtR	WHT.5R	ABSI
Weight	-						
Height	0.55	-					
BMI	0.77	-0.11	-				
WC	0.87	0.40	0.73	-			
WHtR	0.71	0.00	0.85	0.92	-		
WHT.5R	0.81	0.21	0.80	0.98	0.98	-	
ABSI	0.42	0.49	0.12	0.74	0.59	0.68	-

d. Participants with *complete data* among the *Sample II* (N=6813)

	Weight	Height	BMI	WC	WHtR	WHT.5R	ABSI
Weight	-						
Height	0.55	-					
BMI	0.77	-0.10	-				
WC	0.87	0.41	0.73	-			
WHtR	0.71	0.02	0.84	0.92	-		
WHT.5R	0.81	0.22	0.80	0.98	0.98	-	
ABSI	0.42	0.50	0.12	0.74	0.60	0.69	-

BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index

Supplementary Table S7 Area under the receiver operating characteristic curve for five adiposity indicators in relation to diabetes in the participants with complete data of the Sample I

Exposures	AUC_3 (95% CI) Complete data of Sample I: no covariates	AUC_4 (95% CI) Complete data of Sample I: with covariates
BMI	0.575 (0.536, 0.614)	0.714 (0.677, 0.750)
WC	0.602 (0.563, 0.641)	0.715 (0.678, 0.752)
WHtR	0.629 (0.590, 0.668)	0.718 (0.682, 0.755)
WHT.5R	0.618 (0.579, 0.657)	0.717 (0.680, 0.754)
ABSI	0.614 (0.576, 0.653)	0.720 (0.684, 0.757)

AUC_3, in the participants with complete data among Sample I (sample size=7,206, number of events =228), without covariates;

AUC_4, in the participants with complete data among Sample I (sample size=7,206, number of events =228), including covariates for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication.

AUC, area under the curve; CVD, cardiovascular disease; CI, confidence interval; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index.

Supplementary Table S8 Area under the receiver operating characteristic curve for five adiposity indices in identifying diabetes in Sample I stratified by sex

Exposures	AUC (95% CI) Male: with covariates	AUC (95% CI) Female: with covariates
BMI	0.734 (0.730, 0.738)	0.701 (0.695, 0.707)
WC	0.736 (0.732, 0.740)	0.708 (0.702, 0.714)
WHtR	0.739 (0.735, 0.743)	0.712 (0.706, 0.718)
WHT.5R	0.738 (0.734, 0.742)	0.710 (0.704, 0.716)
ABSI	0.738 (0.734, 0.742)	0.712 (0.706, 0.718)

AUC, among Sample I (sample size=7,979, number of events=271), including covariates for age, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, CVD medication, menopause status (females subjects only), and imputation for the missing covariates;

AUC, area under the curve; CVD, cardiovascular disease; CI, confidence interval; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index

Supplementary Table S9 Estimated hazard ratios (Per 1-SD Increase) for incident diabetes in relation to the five adiposity indices (continuous)

	Adjusted HR_2, [a] (95% CI)	**Adjusted HR_2 with MI, [b] (95% CI)**
Standardized BMI	1.56 (1.48, 1.65)	1.59 (1.51, 1.67)
Standardized WC	1.80 (1.68, 1.93)	1.83 (1.72, 1.96)
Standardized WHtR	1.75 (1.65, 1.86)	1.78 (1.68, 1.89)
Standardized WHT.5R	1.78 (1.67, 1.90)	1.81 (1.70, 1.93)
Standardized ABSI	1.67 (1.51, 1.84)	1.68 (1.52, 1.84)

[a] Using complete data (n=6,813) of Sample II, number of events for complete data=847;

[b] Using Sample II, sample size=7,488, number of events=940, imputation for the missing covariates;

Adjusted HR_2, the Cox regression model was adjusted for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication;

Adjusted HR_2 with MI, the imputed Cox regression model was adjusted for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication;

MI, multivariate imputation; CVD, cardiovascular disease; HR, hazard ratio; CI, confidence interval; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index.

Supplementary Table S10 Cumulative event (diabetes) rates for different subgroups at the end of follow-up

Subgroup	Participants with complete data of Sample II			Sample II			
	N (%)	Diabetes, N (%)	P value	N (%)	Diabetes, N (%)	Median follow-up time, yrs	P value
Sex			0.489				0.311
Male	4753 (69.8)	582 (12.2)		5193 (69.4)	638 (12.3)	16.1	
Female	2057 (30.2)	265 (12.9)		2295 (30.6)	302(13.2)	16.0	
Age, yrs			<0.001				<0.001
< 50	3640 (53.4)	374 (10.3)		4035 (53.9)	420 (10.4)	16.1	
≥50	3173 (46.6)	473 (14.9)		3453 (46.1)	520 (15.1)	16.0	
Ethnicity			<0.001				<0.001
White	6266 (92.0)	697 (11.1)		6827 (91.4)	767 (11.2)	16.1	
Non-white	547 (8.0)	150 (27.4)		641 (8.6)	172 (26.8)	15.3	
Baseline CVD diagnosis			0.002				0.004
No	6178 (90.7)	743 (12.0)		6806 (90.9)	830 (12.2)	16.1	
Yes	635 (9.3)	104 (16.4)		682 (9.1)	110 (16.1)	16.0	

CVD, cardiovascular disease; P value for the differences in number of event (diabetes) between different subgroups

Supplementary Table S11 Subgroup analyses for incident diabetes in relation to high-value group by different adiposity indicators in cohort part

Stratification	HR, [a] (95% CI)	P value [a]	P value for interaction [a]	HR, [b] (95% CI)	P value [b]	P value for interaction [b]
Sex						
BMI			0.148			0.119
Male	2.11 (1.77, 2.50)	< 0.001		2.03 (1.72, 2.40)	< 0.001	
Female	2.56 (1.94, 3.38)	< 0.001		2.44 (1.88, 3.17)	< 0.001	
WC			0.118			0.053
Male	2.22 (1.87, 2.62)	< 0.001		2.19 (1.87, 2.57)	< 0.001	
Female	2.72 (2.11, 3.51)	< 0.001		2.76 (2.17, 3.50)	< 0.001	
WHtR			0.152			0.061
Male	2.52 (2.10, 3.01)	< 0.001		2.45 (2.06, 2.90)	< 0.001	
Female	3.02 (2.33, 3.91)	< 0.001		3.02 (2.37, 3.86)	< 0.001	
WHT.5R			0.018			0.001
Male	2.32 (1.97, 2.75)	< 0.001		2.33 (1.98, 2.73)	< 0.001	
Female	3.23 (2.48, 4.22)	< 0.001		3.50 (2.73, 4.49)	< 0.001	
ABSI			0.997			0.719
Male	1.75 (1.47, 2.07)	< 0.001		1.71 (1.45, 2.01)	< 0.001	
Female	1.48 (0.84, 2.62)	0.178		1.62 (0.97, 2.72)	0.068	
Age, yrs						
BMI			0.061			0.039
< 50	2.71 (2.17, 3.38)	< 0.001		2.51 (2.03, 3.10)	< 0.001	
≥ 50	1.92 (1.58, 2.33)	< 0.001		1.91 (1.59, 2.30)	< 0.001	
WC			0.005			0.011
< 50	2.90 (2.35, 3.58)	< 0.001		2.79 (2.28, 3.40)	< 0.001	
≥ 50	2.02 (1.68, 2.43)	< 0.001		2.06 (1.73, 2.46)	< 0.001	
WHtR			0.001			0.002
< 50	3.41 (2.74, 4.25)	< 0.001		3.23 (2.62, 3.97)	< 0.001	
≥ 50	2.23 (1.83, 2.71)	< 0.001		2.25 (1.87, 2.71)	< 0.001	
WHT.5R			0.004			0.011
< 50	3.10 (2.49, 3.86)	< 0.001		3.05 (2.48, 3.75)	< 0.001	
≥ 50	2.19 (1.81, 2.65)	< 0.001		2.32 (1.93, 2.78)	< 0.001	
ABSI			0.033			0.015
< 50	1.94 (1.52, 2.47)	< 0.001		1.90 (1.52, 2.39)	< 0.001	
≥ 50	1.57 (1.27, 1.95)	< 0.001		1.55 (1.26, 1.91)	< 0.001	
Ethnicity						
BMI			0.016			0.018
White	2.44 (2.07, 2.87)	< 0.001		2.34 (2.01, 2.74)	< 0.001	
Non-white	1.48 (1.04, 2.11)	0.028		1.49 (1.08, 2.08)	0.017	
WC			0.154			0.302
White	2.47 (2.11, 2.88)	< 0.001		2.43 (2.10, 2.82)	< 0.001	
Non-white	1.97 (1.41, 2.75)	< 0.001		2.10 (1.53, 2.88)	< 0.001	
WHtR			0.268			0.227
White	2.74 (2.33, 3.21)	< 0.001		2.71 (2.32, 3.16)	< 0.001	
Non-white	2.42 (1.68, 3.47)	< 0.001		2.35 (1.68, 3.29)	< 0.001	
WHT.5R			0.020			0.091
White	2.74 (2.34, 3.21)	< 0.001		2.76 (2.37, 3.21)	< 0.001	
Non-white	1.86 (1.32, 2.63)	< 0.001		2.12 (1.53, 2.93)	< 0.001	
ABSI			0.272			0.217
White	1.71 (1.44, 2.04)	< 0.001		1.69 (1.43, 2.00)	< 0.001	
Non-white	1.71 (1.13, 2.59)	0.011		1.60 (1.07, 2.38)	0.021	
Baseline CVD diagnosis						
BMI			0.067			0.115
Without CVD	2.12 (1.82, 2.47)	< 0.001		2.07 (1.79, 2.39)	< 0.001	
With CVD	4.00 (2.40, 6.69)	< 0.001		3.49 (2.15, 5.68)	< 0.001	
WC			0.087			0.129
Without CVD	2.23 (1.92, 2.59)	< 0.001		2.25 (1.96, 2.59)	< 0.001	
With CVD	3.74 (2.40, 5.82)	< 0.001		3.55 (2.31, 5.46)	< 0.001	
WHtR			0.535			0.684
Without CVD	2.60 (2.22, 3.04)	< 0.001		2.58 (2.23, 2.99)	< 0.001	
With CVD	3.38 (2.11, 5.42)	< 0.001		3.23 (2.04, 5.10)	< 0.001	
WHT.5R			0.107			0.123
Without CVD	2.60 (2.22, 3.04)	< 0.001		2.47 (2.14, 2.86)	< 0.001	
With CVD	3.38 (2.11, 5.42)	< 0.001		3.98 (2.63, 6.01)	< 0.001	

ABSI			0.223		0.205
Without CVD	1.74 (1.47, 2.07)	< 0.001		1.70 (1.44, 2.00)	< 0.001
With CVD	1.41 (0.89, 2.22)	0.141		1.36 (0.87, 2.12)	0.177

[a] Using complete data (n=6,813) of Sample II, number of events for complete data=847;

[b] Using Sample II, sample size=7,488, number of events =940, imputation for the missing covariates;

Sex stratification: controlling covariates for age, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, CVD medication, and menopause status (females subjects only).

Age stratification: controlling covariates for sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication.

Ethnicity stratification: controlling covariates for age, sex, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, baseline CVD diagnosis, and CVD medication.

Baseline CVD diagnosis stratification: controlling covariates for age, sex, ethnicity, smoking, drinking, socioeconomic position, physical activity, dietary behavior, family history of diabetes, and CVD medication.

CVD, cardiovascular disease; HR, hazard ratio; CI, confidence interval; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index

Supplementary Table S12 Log-rank test p-values for pairwise comparisons among the different levels of BMI and WHtR combination

a. Among Sample II

	low BMI and low WHtR	high BMI and low WHtR	low BMI and high WHtR	high BMI and high WHtR
low BMI and low WHtR	-			
high BMI and low WHtR	< 0.001	-		
low BMI and high WHtR	<0.001	<0.001	-	
high BMI and high WHtR	< 0.001	<0.001	0.301	-

b. Among the participants with complete data of Sample II

	low BMI and low WHtR	high BMI and low WHtR	low BMI and high WHtR	high BMI and high WHtR
low BMI and low WHtR	-			
high BMI and low WHtR	< 0.001	-		
low BMI and high WHtR	<0.001	<0.001	-	
high BMI and high WHtR	< 0.001	<0.001	0.399	-

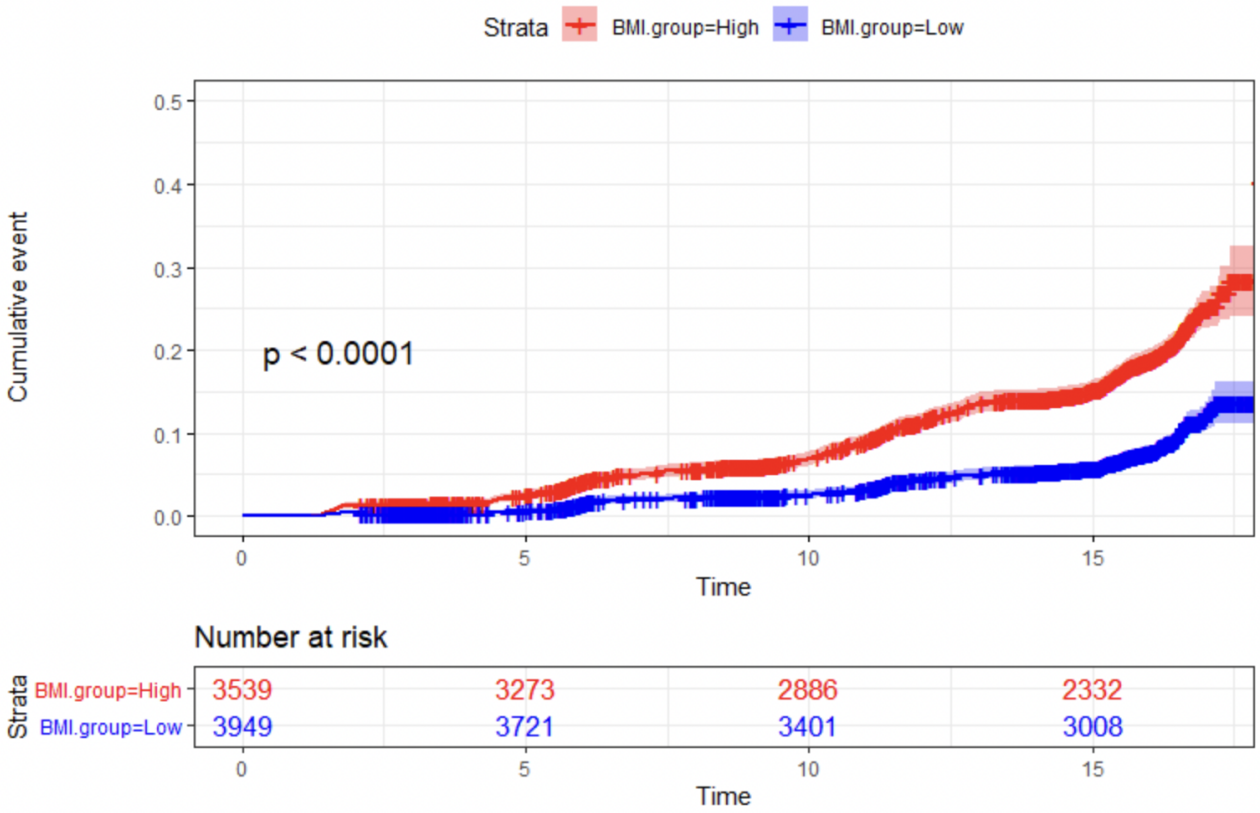
Benjamini-Hochberg method for adjusting significance level; The six p values (i.e., 0.0083, 0.01, 0.0125, 0.0167, 0.025, and 0.05) are judged “significant” by Benjamini-Hochberg procedure.

BMI, body mass index; WHtR, waist-to-height ratio.

Supplementary Fig. S1 Kaplan-Meier curves of the exposed (high-value) and non-exposed (low-value) groups for incident diabetes

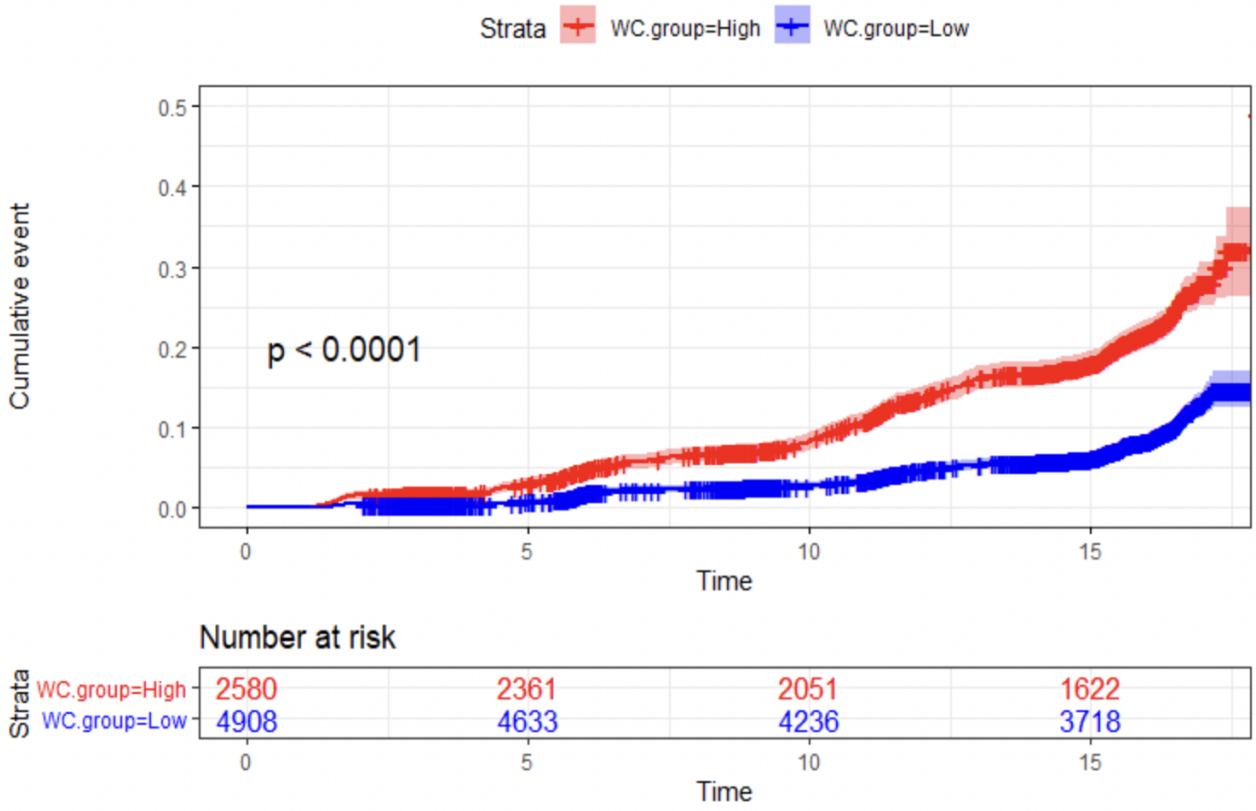
The Kaplan-Meier curves of the exposed and non-exposed groups did not cross during the follow-up time, i.e., one curve was always above the other curve, which means the violation of proportionality was not extreme, and a single HR for the exposures can still be a reasonable summary of the data

a. BMI



Time: time after Phase 3 (year 0) in years;
BMI, body mass index

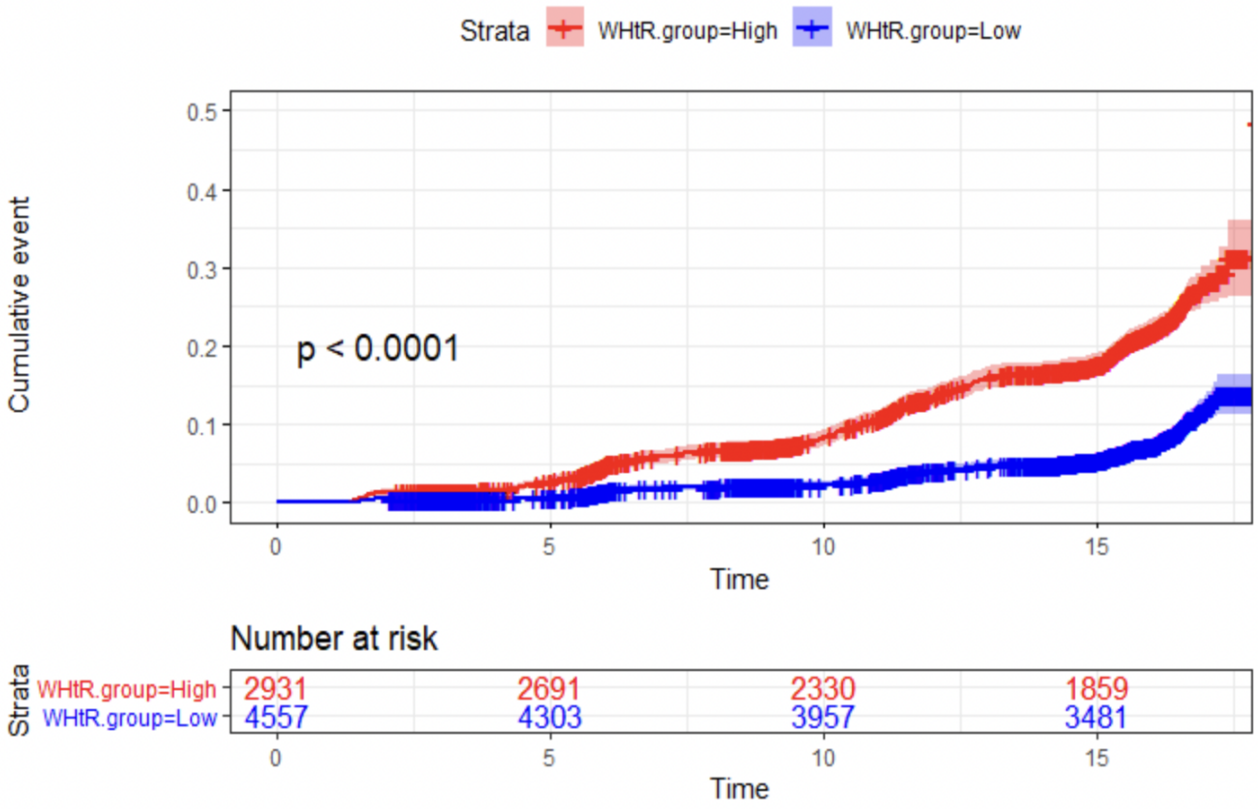
b. WC



Time: time after Phase 3 (year 0) in years;

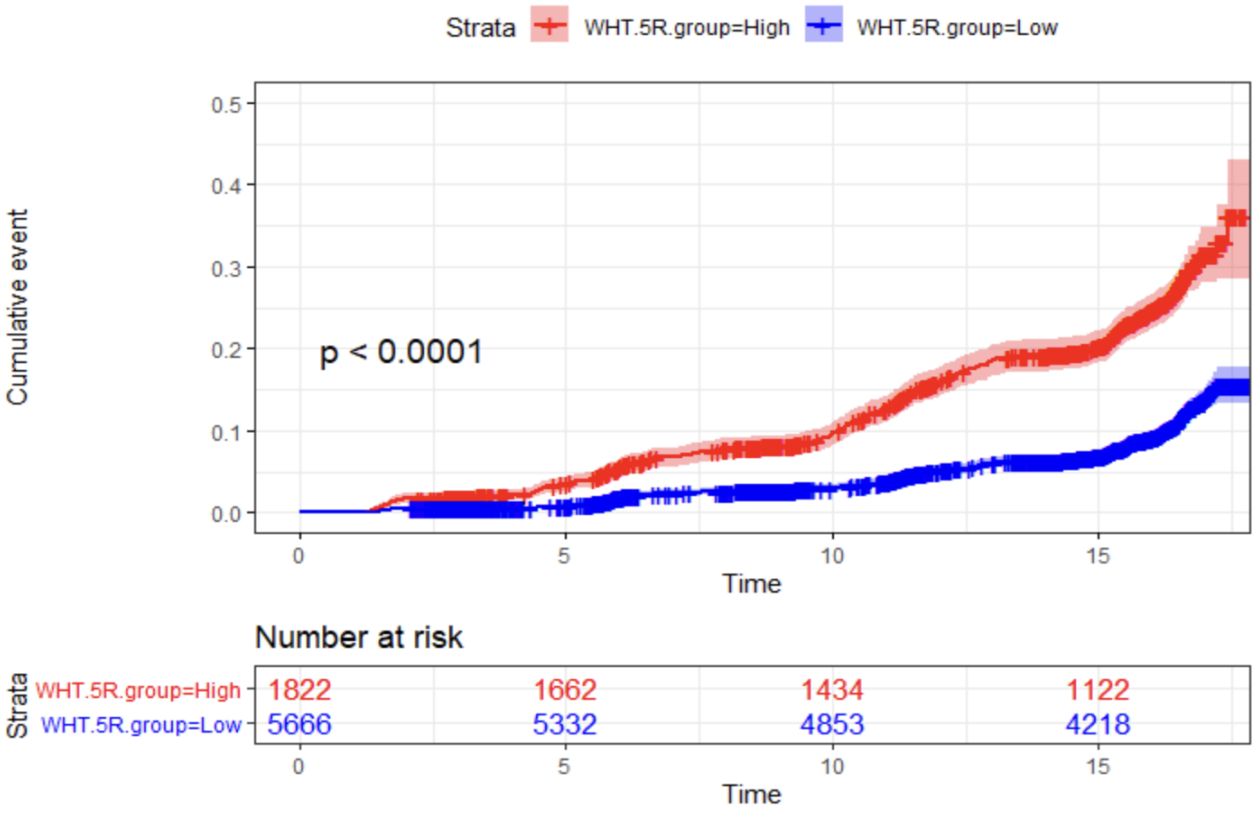
WC, waist circumference

c. WHtR



Time: time after Phase 3 (year 0) in years;
WHtR, waist-to-height ratio

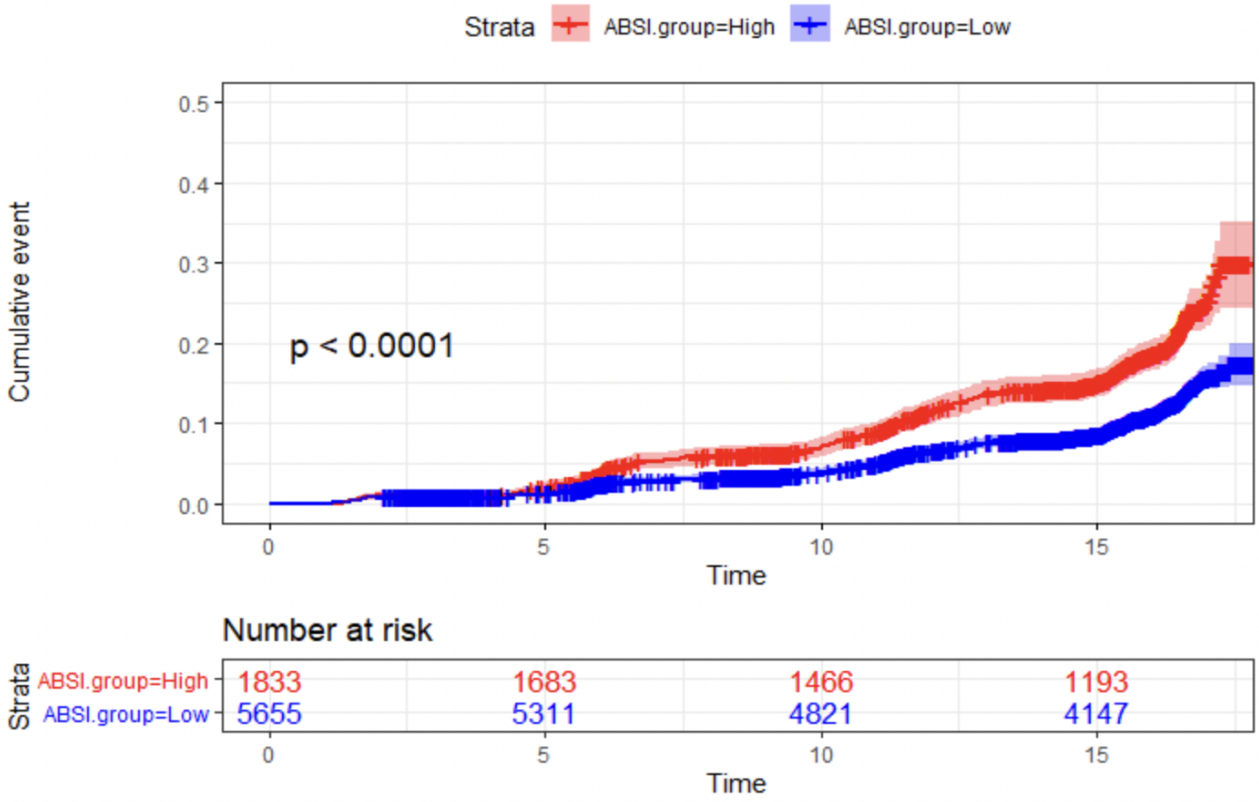
d. WHT.5R



Time: time after Phase 3 (year 0) in years;

WHT.5R, waist-by-height$^{0.5}$

e. ABSI



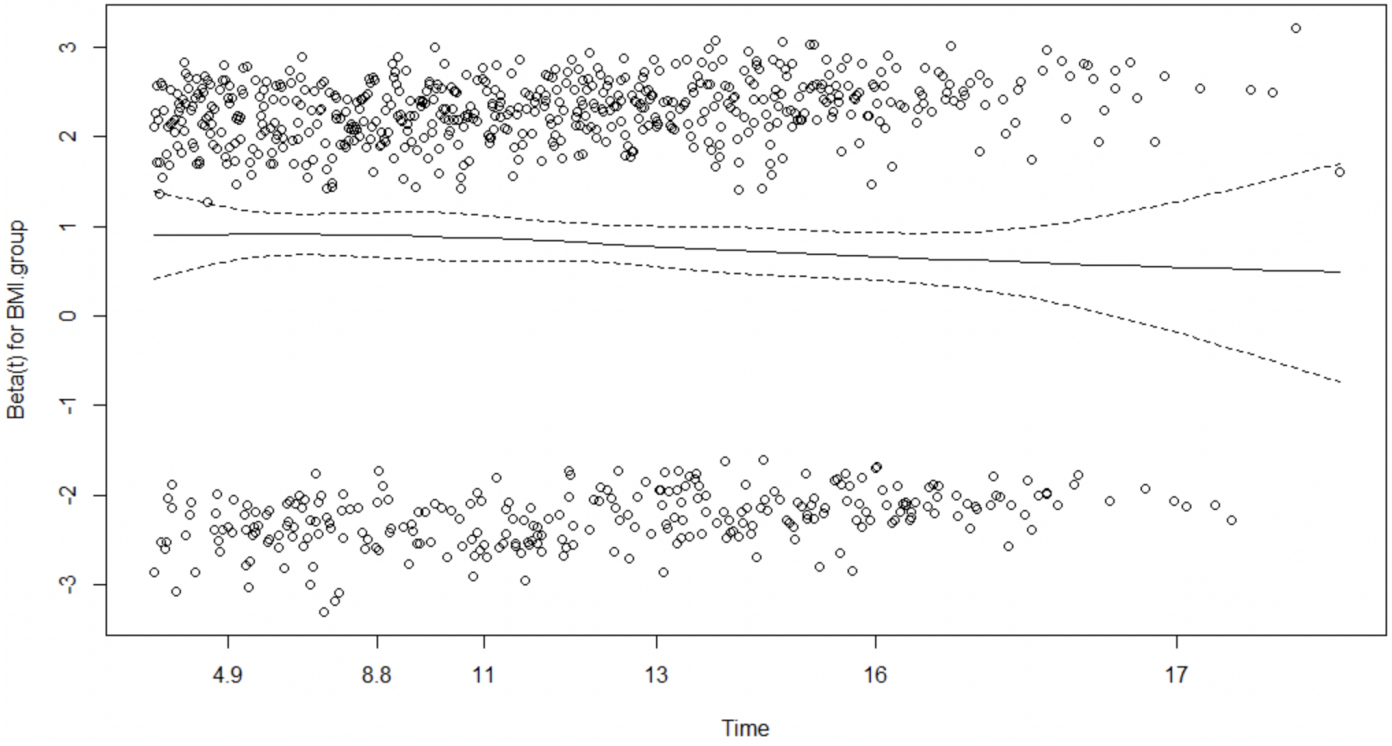
Time: time after Phase 3 (year 0) in years**;**

ABSI, a body shape index

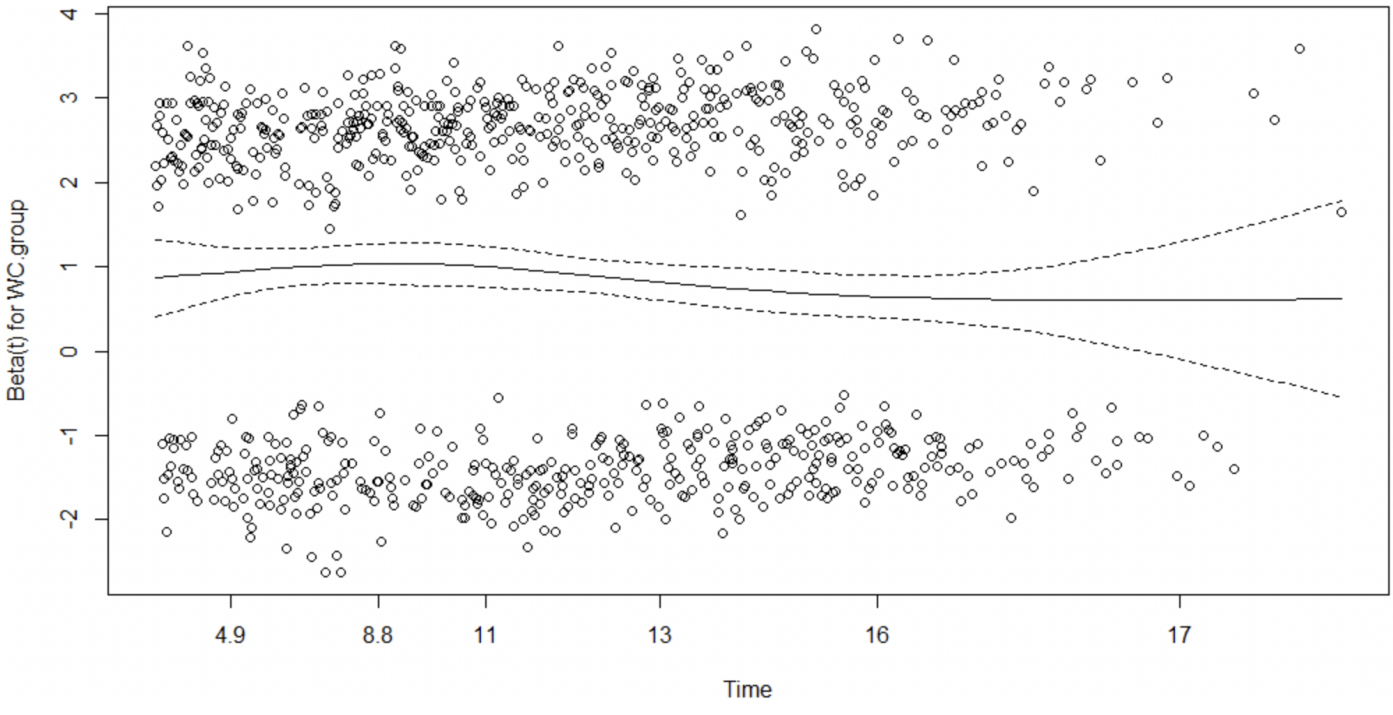
Supplementary Fig. S2 Graphs of the scaled Schoenfeld residuals against the transformed time for the five exposure variables

In the graphs, the coefficients of the exposure variables did not change significantly during the follow-up period, i.e., Beta(t) for exposure was nearly constant over time (there is also no significant tendency to rise and then fall, or to fall and then rise), which means the violation of proportionality was not extreme, and a single HR for the exposures can still be a reasonable summary of the data.

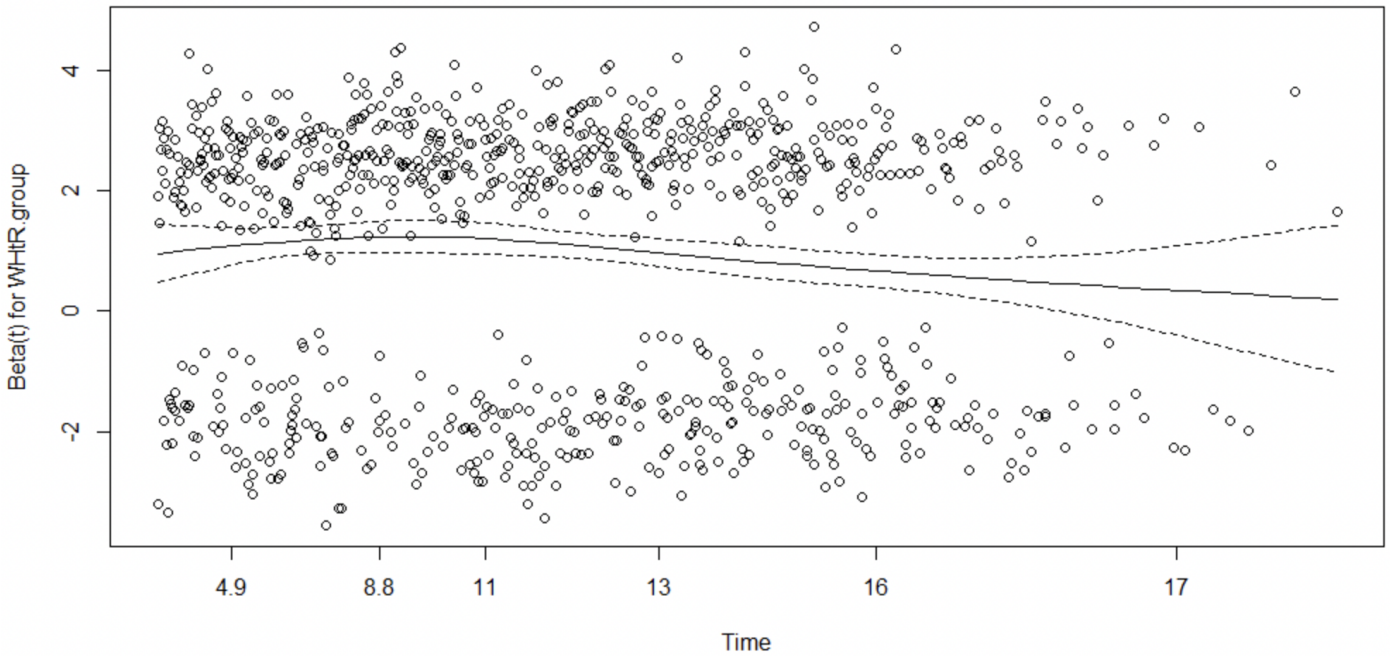
a. BMI



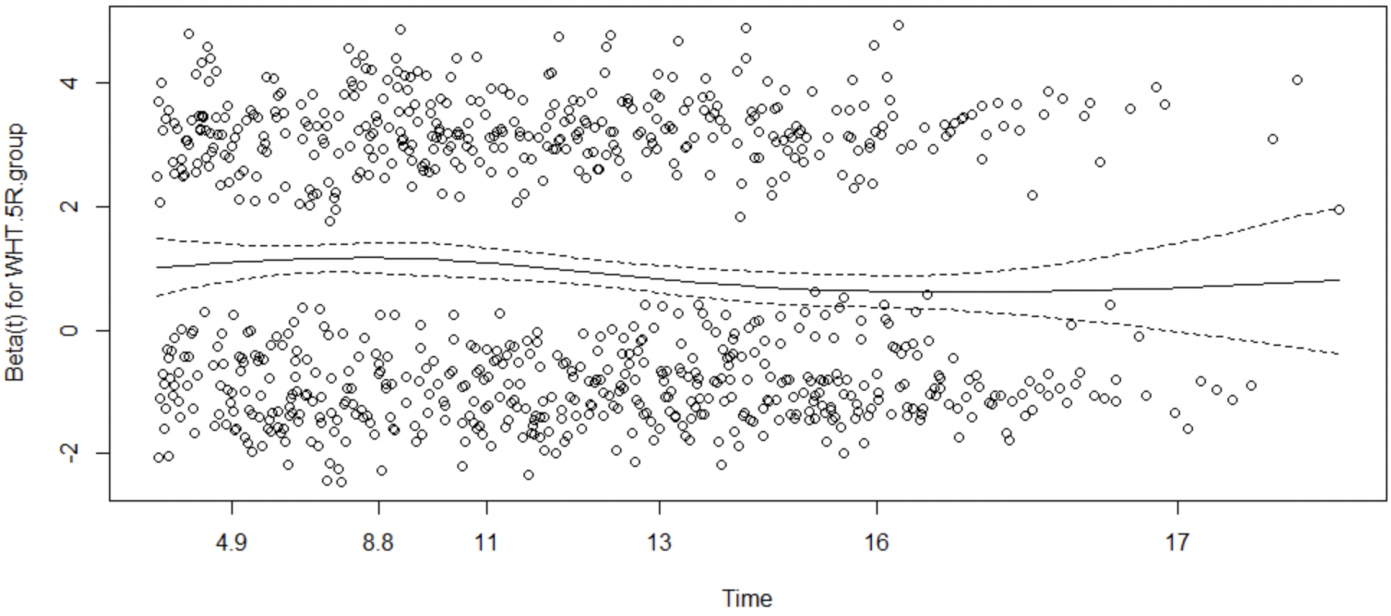
b. WC



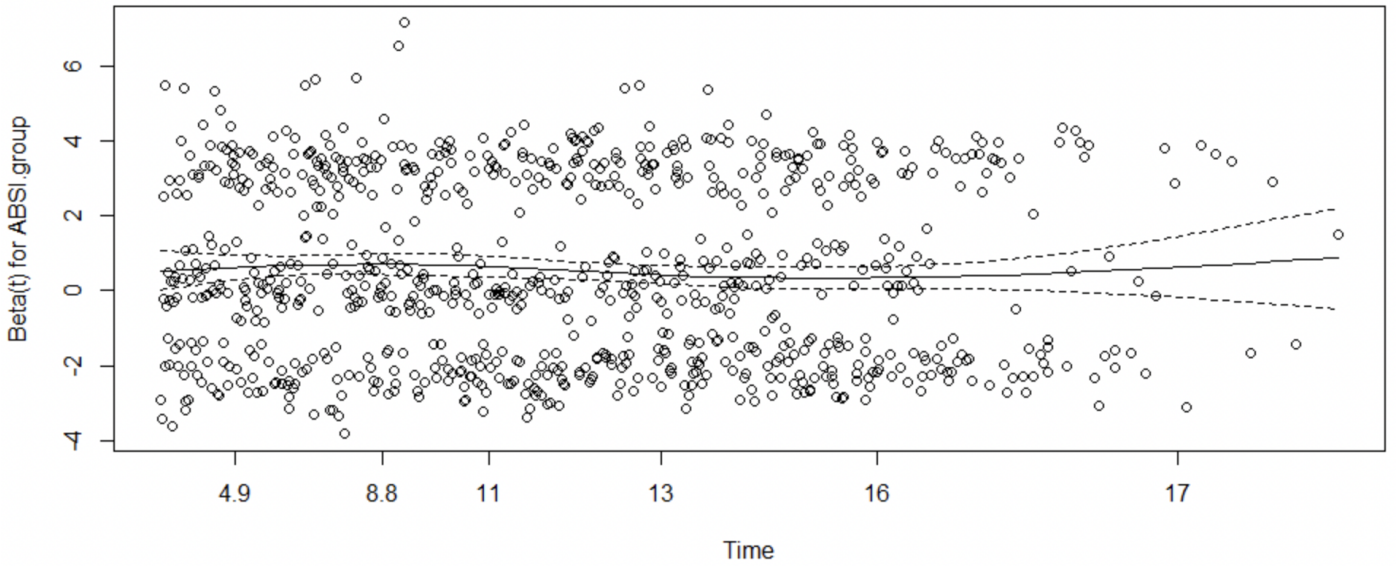
c. WHtR



d. WHT.5R



e. ABSI

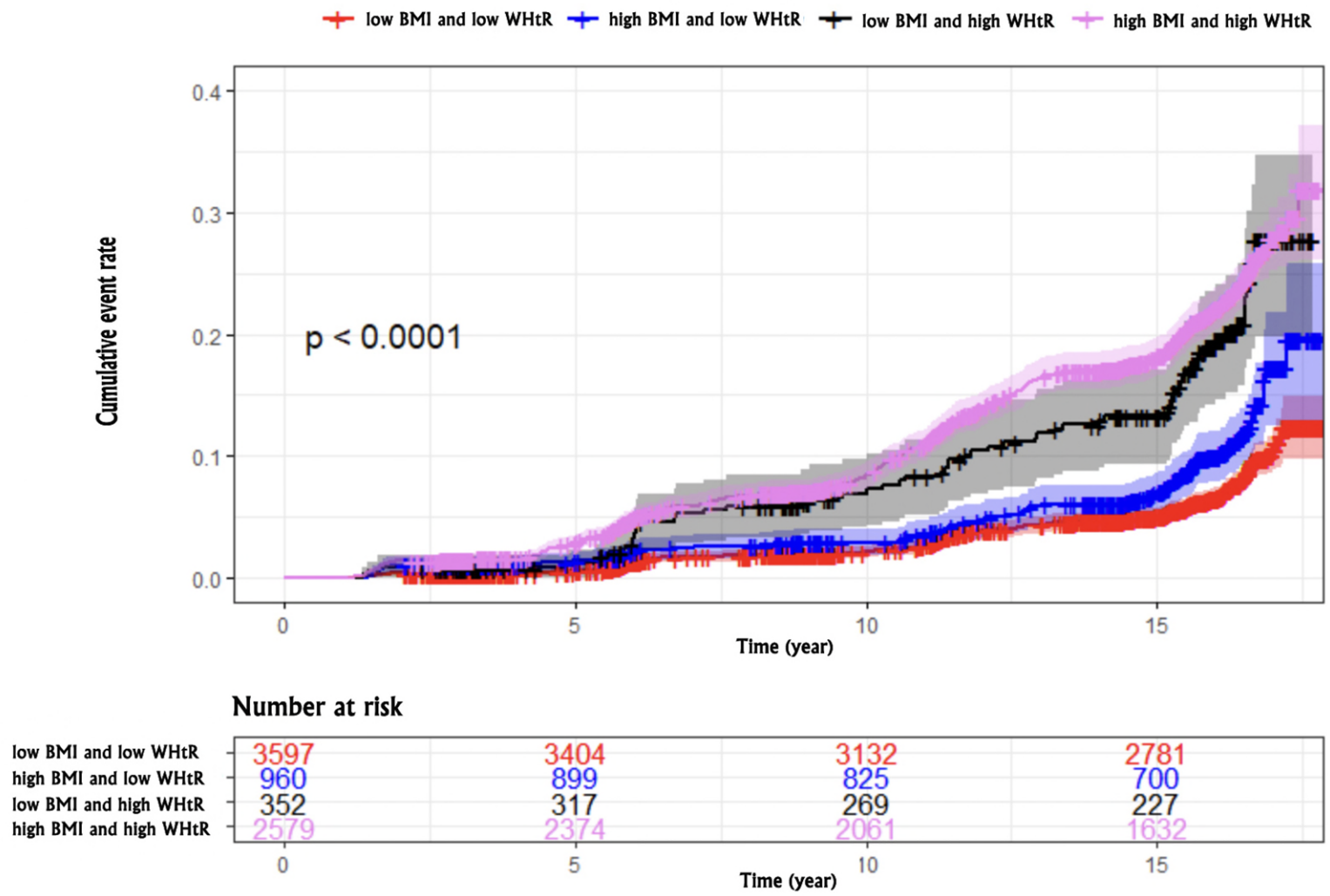


Time: time after Phase 3 (year 0);

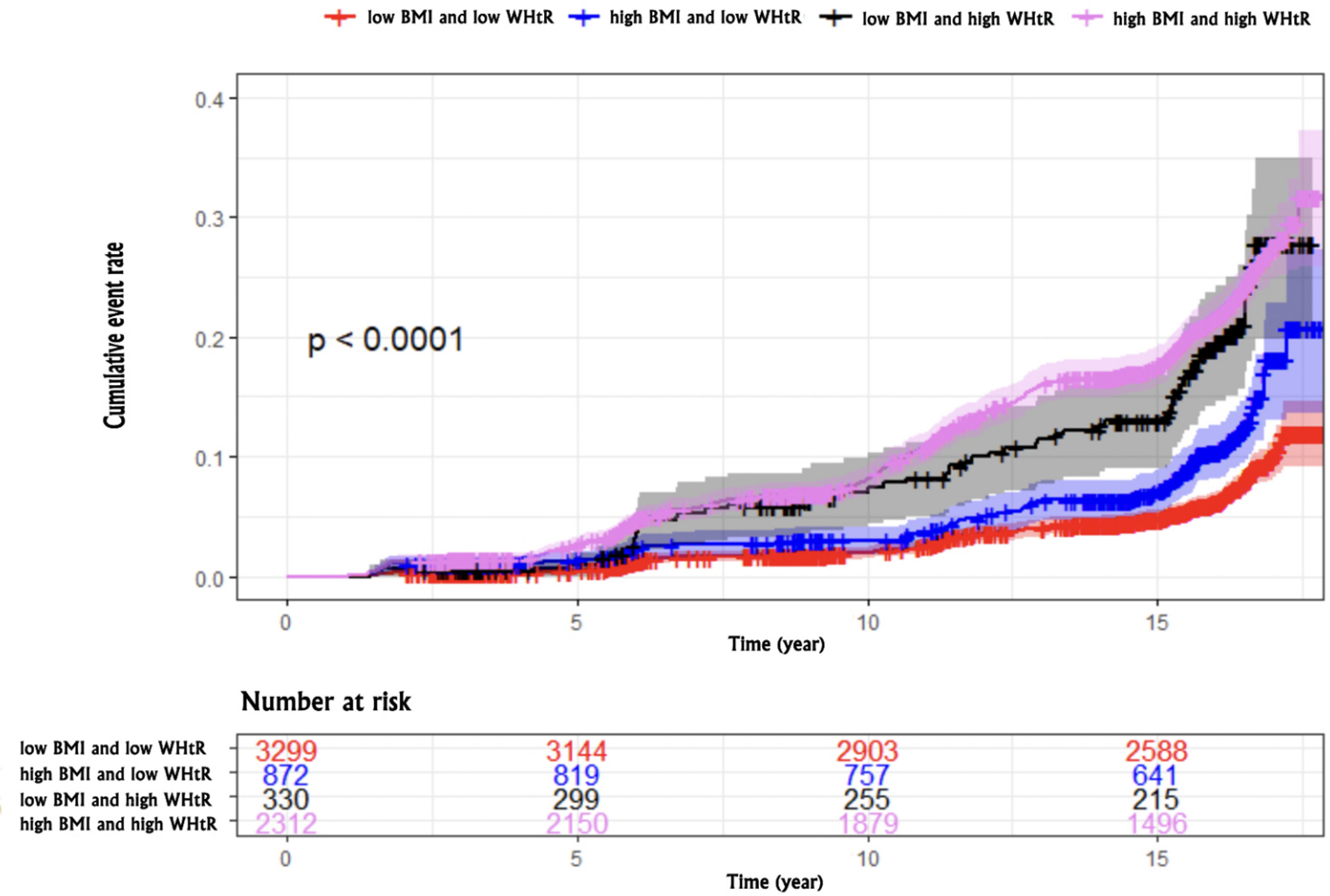
BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; WHT.5R, waist-by-height$^{0.5}$; ABSI, a body shape index

Supplementary Fig. S3 Kaplan-Meier curves for incident diabetes in relation to different levels of BMI and WHtR combinations

a. Among Sample II (N=7488)



b. Among the persons with complete data of Sample II (N=6813)



Time: time after Phase 3 (year 0)

BMI, body mass index; WHtR, waist-to-height ratio.