

Predicted fat mass index and incident stroke in adults aged 65 years or older: heterogeneous associations across three national ageing cohorts

Shikun Li

Tongji Hospital

Xu Chen

Tongji Hospital

Rudong Chen

Tongji Hospital

Zhuo Yang

Tongji Hospital

Yuyang Hou

Tongji Hospital

Jiasheng Yu

yujiaseng2000@tjh.tjmu.edu.cn

Tongji Hospital

Research Article

Keywords: adiposity, body composition, predicted fat mass index, stroke, older adults, ageing, CHARLS, ELSA, HRS, cohort study

Posted Date: October 11th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10403308/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Abstract

Background

Predicted fat mass index (pBFMI) can be calculated when direct body-composition measurements are unavailable, but it is a deterministic transformation of body mass index (BMI), age, and sex. We examined whether its association with incident stroke was consistent across three national ageing cohorts.

Methods

We analysed stroke-free adults aged 65 years or older in CHARLS (2011–2020), ELSA (2004–2018), and HRS (2006–2020). Because stroke onset was known only between survey waves, the primary analysis used discrete-time complementary log-log models. Model 2 adjusted for age, sex, education, marital status, smoking, and an alcohol-use indicator; hypertension, diabetes, and heart disease were added in Model 3 as potential intermediates. Sex-specific standardization was recalculated within each cohort's age-65-or-older sample. We assessed mathematical coupling with BMI, compared apparent model fit and discrimination, and treated random-effects synthesis as descriptive.

Results

Model 2 included 3,241 CHARLS participants (317 strokes), 2,494 ELSA participants (205 strokes), and 3,614 HRS participants (413 strokes). Per sex-specific standard deviation higher pBFMI, hazard ratios were 1.247 (95% CI 1.165–1.334) in CHARLS, 0.924 (0.800–1.067) in ELSA, and 0.987 (0.892–1.091) in HRS. A one-stage discrete-time model showed cohort heterogeneity (joint interaction $P < 0.001$), which persisted when all Model 2 covariate effects were allowed to vary by cohort ($P < 0.001$). Hartung–Knapp descriptive synthesis gave an HR of 1.051 (0.709–1.560), $I^2=89.8\%$, and a 95% prediction interval of 0.491–2.250. pBFMI correlated strongly with BMI ($r = 0.977\text{--}0.988$) and did not materially improve apparent AIC or C-index.

Conclusions

Among adults aged 65 years or older, pBFMI was positively associated with incident stroke in CHARLS but not in ELSA or HRS. The findings do not support a consistent cross-population association and provide no evidence of materially improved apparent model fit or discrimination over BMI.

Background

Stroke remains a leading cause of death and disability, and its absolute burden continues to rise as populations grow and age. High body mass index (BMI) accounted for an estimated 24% of stroke-

related disability-adjusted life-years in 2019 and was among the fastest-growing contributors to the global stroke burden [1]. Yet BMI is an indirect measure of adiposity: identical BMI values may represent different proportions of fat and lean tissue [2, 31], and this ambiguity becomes particularly relevant in later life, when muscle mass declines while fat mass and its distribution change [3, 39, 40].

Prospective evidence generally links higher BMI with greater stroke risk, but the association is non-linear and differs by stroke subtype and population [4, 5, 16–19]. Studies using dual-energy X-ray absorptiometry, bioelectrical impedance, or regional adiposity measures further suggest that fat distribution may carry information not captured by BMI alone [6, 7, 20–23]. Recent studies have also examined relative fat mass, another anthropometric adiposity proxy, in relation to stroke [33, 34]. In large Chinese cohorts, general and central adiposity measures were positively associated with total and ischaemic stroke, while their relationships with haemorrhagic stroke were more complex [5, 18, 19]. These observations motivate evaluation of body-composition proxies, but they also indicate that an index developed in one setting may not show consistent associations across populations.

The Deurenberg equation estimates percentage body fat from BMI, age, and sex and explained 79% of between-person variation in its original adult samples [8]. Combining this estimate with measured weight and height yields predicted fat mass and predicted fat mass index (pBFMI). The approach is inexpensive and can be applied retrospectively to ageing cohorts that lack dual-energy X-ray absorptiometry. However, the relation between BMI and body fat varies by age, sex, ethnicity, and body habitus [9, 24, 25, 32]. Equations developed primarily in European-origin samples may therefore misestimate body fat in Asian populations. Whether pBFMI shows a consistent prospective association with stroke across ageing cohorts is uncertain.

We used three national ageing cohorts to examine the association between pBFMI and incident stroke in adults aged 65 years or older. We estimated associations separately within each cohort and formally assessed between-cohort heterogeneity. We also examined whether pBFMI improved model fit or discrimination relative to BMI and quantified the mathematical coupling created by its derivation.

Methods

Study design and cohorts

This study comprised parallel secondary analyses of three prospective ageing cohorts. CHARLS is a nationally representative study of middle-aged and older Chinese adults and their spouses [10]. We used the 2011 baseline and follow-up waves in 2013, 2015, 2018, and 2020. ELSA is a representative panel of community-dwelling adults in England, with repeated interviews and nurse assessments [11]. Wave 2 (2004) was the analytic baseline because measured anthropometry was available, with stroke follow-up through wave 9 (2018). HRS is a nationally representative US panel of older adults and their spouses [12]. We used 2006 as baseline, when enhanced physical measures were collected, and biennial follow-up through 2020.

Participants were eligible if they were aged 65 years or older at the analytic baseline, had valid sex, height, weight, and calculated pBFMI, reported no stroke at baseline, and had at least one subsequent stroke assessment. The age threshold of 65 years was selected as a conventional definition of older adulthood and to define a clinically relevant geriatric target population consistently across cohorts. Participants younger than 65 years were excluded from all analyses. Cohort-specific complete-case samples for the common primary model were analysed. The study followed STROBE recommendations [13].

Predicted fat mass index

BMI was calculated as weight in kilograms divided by height in metres squared. Percentage body fat was estimated using the adult Deurenberg equation: $BF\% = 1.20 \times BMI + 0.23 \times \text{age} - 10.8 \times \text{sex} - 5.4$, where sex was coded 1 for men and 0 for women [8]. Predicted fat mass (kg) was $\text{weight} \times BF\%/100$, and pBFMI (kg/m^2) was predicted fat mass divided by height squared. Implausible anthropometric values and predicted body-fat percentages outside 0–75% were excluded. After restricting each cohort to participants aged 65 years or older, pBFMI was standardized to a mean of zero and standard deviation of one within sex and cohort. The primary coefficient therefore represents the hazard ratio per one sex-specific standard deviation higher pBFMI in the target older population.

Incident stroke

Baseline and follow-up stroke were defined from participant reports of a physician diagnosis in each parent study. Participants reporting stroke at baseline were excluded. Incident stroke was assigned to the interval ending at the first follow-up wave with a positive report. Participants without an event were followed to their last observed stroke assessment. Exact onset dates were not available consistently; the outcome was therefore treated as grouped interval-time data in the primary analysis.

Covariates

Potential baseline confounders were age, sex, education, marital status, current smoking, and an alcohol-use indicator. The alcohol measure was not fully equivalent across cohorts: CHARLS recorded any consumption in the previous year, whereas ELSA and HRS used harmonized ever-drinking indicators. We therefore use the generic term alcohol use rather than current alcohol use; cohort-specific definitions are reported in Additional file 1 (Table S6). Hypertension, diabetes, and heart disease were analysed separately because they may lie partly on pathways linking adiposity to stroke. Education retained cohort-specific categories to respect differences in national systems. Chronic conditions were defined according to the available parent-survey items, primarily self-reported physician diagnoses.

Statistical analysis

Discrete-time complementary log-log models were primary because stroke occurrence was observed within grouped survey intervals [36]. Cohort-specific interval indicators represented the baseline hazard. Model 1 included age and sex. Model 2, designated the primary association model, additionally included education, marital status, current smoking, and alcohol use. Model 3 further added hypertension, diabetes, and heart disease to examine attenuation after adjustment for potential metabolic or cardiovascular intermediates. To separate covariate adjustment from changes in sample composition, Models 1–3 were also fitted to the identical Model 3 complete-case sample in each cohort. Effect estimates are reported as hazard ratios (HRs) with 95% confidence intervals (CIs) per sex-specific standard deviation higher pBFMI.

We quantified mathematical coupling using correlations of pBFMI with BMI and age, the exposure variance-inflation factor in Model 2, and a condition index for pBFMI, BMI, and age. By construction, pBFMI was fully determined by BMI, age, and sex. Separate pBFMI and BMI models were fitted to identical samples. Discrete-time Akaike information criterion (AIC) and Cox-model Harrell C-index values were compared as apparent, within-sample measures; no cross-validation or optimism correction was performed. pBFMI and BMI were not entered together in outcome models.

A one-stage discrete-time complementary log-log model formally tested heterogeneity within the primary model framework. It included cohort-specific survey-interval baseline hazards, common Model 2 covariate coefficients, and cohort-by-pBFMI terms; a likelihood-ratio test jointly assessed the interaction terms. Because education, marital status, smoking, and alcohol use were not necessarily measurement-equivalent across cohorts, a sensitivity model additionally allowed every Model 2 covariate coefficient to vary by cohort and repeated the joint cohort-by-pBFMI test. Cox models using the survey-wave endpoint served as sensitivity analyses, and proportional hazards were assessed with Schoenfeld residuals and the Grambsch–Therneau test [26]. Cohort estimates were summarized descriptively using restricted maximum likelihood with Hartung–Knapp adjustment and a 95% prediction interval [27]. Given only three cohorts and different absolute pBFMI increments underlying one standardized unit, this synthesis was not interpreted as a common population effect.

Primary analyses used complete cases within each cohort. We reported covariate-specific missingness and compared baseline characteristics of participants included in versus excluded from Model 2 using standardized differences (Additional file 1, Tables S4–S5). Multiple imputation was not used because education and alcohol measures were not operationally equivalent across cohorts and a common auxiliary-variable set was unavailable; the common-sample sequential analysis directly addressed changes in sample composition across adjustment models.

Sensitivity analyses excluded events in the first two years, repeated Model 2 without the non-equivalent alcohol-use indicator, replaced pBFMI with sex-standardized BMI or waist-to-height ratio, examined sex and age strata (65–74 versus ≥ 75 years), and applied cohort-specific anthropometry weights—CHARLS bio_weight2, ELSA r2nwtresp, and HRS r8nwtresp—with normalized weights and robust standard errors [30]. Weighting remained a sensitivity analysis because compatible strata and primary sampling units

were not harmonized. Exploratory competing-risk models were restricted to cohorts with usable mortality timing [14, 37, 38]. Possible time variation in ELSA was additionally explored using a log-time interaction and a piecewise Cox model with a 6-year cut point; the cut point was exploratory. Analyses used R 4.5.1 and two-sided tests.

Results

Cohort characteristics

Model 2 included 3,241 CHARLS, 2,494 ELSA, and 3,614 HRS participants aged 65 years or older (Table 1). Mean age ranged from 71.6 years in CHARLS to 73.9 years in HRS, and women comprised 50.1–57.7% of each sample. Mean pBFMI was 7.58 kg/m² in CHARLS, 11.20 kg/m² in ELSA, and 12.11 kg/m² in HRS. Across 79,726 person-years, 935 incident strokes were reported: 317 in CHARLS, 205 in ELSA, and 413 in HRS.

Table 1
Baseline characteristics of the primary complete-case samples aged 65 years or older

Characteristic	CHARLS	ELSA	HRS
Participants, n	3,241	2,494	3,614
Incident strokes, n	317	205	413
Person-years	23,520	22,992	33,214
Age, mean (SD), years	71.6 (5.5)	73.0 (6.1)	73.9 (6.8)
Women, n (%)	1,624 (50.1)	1,327 (53.2)	2,086 (57.7)
pBFMI, mean (SD), kg/m ²	7.58 (3.07)	11.20 (3.98)	12.11 (4.43)
BMI, mean (SD), kg/m ²	22.5 (3.9)	27.7 (4.6)	28.6 (5.2)
Married/partnered, %	72.7	63.1	63.6
Current smoker, %	29.9	10.3	9.0
Alcohol use indicator, %	29.2	88.2	50.1
Hypertension, %	30.5	44.9	62.6
Diabetes, %	6.3	9.0	19.9
Heart disease, %	15.4	19.4	27.5
Median follow-up, years	9	10	10

Values are unweighted and describe participants aged 65 years or older included in Model 2. The alcohol-use indicator was cohort specific: previous-year consumption in CHARLS and ever-drinking

indicators in ELSA and HRS. Cardiometabolic conditions are shown descriptively but enter only Model 3. BMI, body mass index; pBFMI, predicted fat mass index.

Missing data and complete-case composition

Among participants aged 65 years or older with pBFMI and follow-up data, Model 2 retained 99.1% in CHARLS, 80.5% in ELSA, and 99.7% in HRS. In ELSA, 603 participants and 47 strokes were excluded from Model 2; missingness was concentrated in education (9.8%) and the alcohol-use indicator (10.5%). Model 3 retained 97.9%, 80.5%, and 96.8% of eligible participants, respectively. Included-versus-excluded comparisons and covariate-specific missingness are reported in Additional file 1 (Tables S4–S5).

Cohort-specific and pooled associations

In the primary Model 2 discrete-time analysis, pBFMI was associated with incident stroke in CHARLS (HR 1.247, 95% CI 1.165–1.334; $P < 0.001$), but not in ELSA (HR 0.924, 95% CI 0.800–1.067; $P = 0.281$) or HRS (HR 0.987, 95% CI 0.892–1.091; $P = 0.794$) (Fig. 1). A one-stage discrete-time complementary log-log model with cohort-specific interval hazards showed evidence of heterogeneity (joint cohort-by-pBFMI likelihood-ratio $P < 0.001$). Heterogeneity persisted when every Model 2 covariate coefficient was allowed to vary by cohort (joint likelihood-ratio $P < 0.001$; Additional file 1, Table S3).

Sequential models restricted to each cohort's common Model 3 complete-case sample separated adjustment from sample changes (Table 2). In CHARLS, the Model 2 estimate was 1.243 (95% CI 1.161–1.331) and attenuated to 1.191 (1.107–1.282) after hypertension, diabetes, and heart disease were added. Corresponding estimates changed from 0.924 to 0.890 in ELSA and from 0.995 to 0.936 in HRS.

The Hartung–Knapp descriptive synthesis was 1.051 (95% CI 0.709–1.560), with $I^2=89.8\%$ and a 95% prediction interval of 0.491–2.250. These statistics summarize three heterogeneous cohort estimates and were not interpreted as a common population effect.

Table 2
Discrete-time associations between pBFMI and incident stroke across sequential models restricted to a common Model 3 complete-case sample

Cohort	Model	n	Events	HR	95% CI	P value
CHARLS	Model 1	3,201	315	1.241	1.160–1.328	< 0.001
CHARLS	Model 2	3,201	315	1.243	1.161–1.331	< 0.001
CHARLS	Model 3	3,201	315	1.191	1.107–1.282	< 0.001
ELSA	Model 1	2,494	205	0.934	0.808–1.078	0.350
ELSA	Model 2	2,494	205	0.924	0.800–1.067	0.281
ELSA	Model 3	2,494	205	0.890	0.768–1.032	0.124
HRS	Model 1	3,509	397	1.000	0.904–1.106	0.999
HRS	Model 2	3,509	397	0.995	0.898–1.102	0.918
HRS	Model 3	3,509	397	0.936	0.840–1.042	0.224

All three models within a cohort used the identical Model 3 complete-case sample. Hazard ratios are per 1 sex-specific SD higher pBFMI. Model 1: age and sex. Model 2: Model 1 plus education, marital status, current smoking, and alcohol use. Model 3: Model 2 plus hypertension, diabetes, and heart disease. CI, confidence interval; HR, hazard ratio.

Mathematical coupling and model comparison

pBFMI was strongly correlated with sex-standardized BMI in CHARLS ($r = 0.977$), ELSA ($r = 0.987$), and HRS ($r = 0.988$). By construction, it was fully determined by BMI, age, and sex. Exposure variance-inflation factors in Model 2 were low (1.03–1.05) because BMI was not included simultaneously, whereas condition indices for pBFMI, BMI, and age ranged from 11.5 to 17.8. These findings confirm mathematical coupling without indicating unstable estimation in the pBFMI-only model.

pBFMI did not materially improve apparent model performance relative to BMI. The pBFMI model had a higher discrete-time AIC than the BMI model in CHARLS (difference 10.98) and nearly identical AIC in ELSA and HRS (differences -0.17 and -0.06). Apparent C-index differences were -0.0028 , 0.0006 , and 0.0001 , respectively. These were within-sample comparisons rather than validated prediction analyses.

Robustness and subgroup analyses

Excluding strokes in the first two years produced Model 2 estimates of 1.244 (95% CI 1.156–1.339) in CHARLS, 0.898 (0.768–1.049) in ELSA, and 1.000 (0.896–1.115) in HRS. BMI and waist-to-height ratio retained the cohort contrast: both were positively associated with stroke in CHARLS but not in ELSA or HRS. The CHARLS estimate was positive in women, men, participants aged 65–74 years, and those aged

75 years or older; corresponding ELSA and HRS estimates generally included the null (Fig. 2; Additional file 1, Table S7).

Omitting the alcohol-use indicator increased the ELSA sample from 2,494 participants and 205 strokes to 2,792 participants and 229 strokes, but did not materially change the cohort contrast: the HRs were 1.245 (95% CI 1.164–1.333) in CHARLS, 0.939 (0.821–1.075) in ELSA, and 0.993 (0.898–1.098) in HRS (Additional file 1, Table S7).

Model 2 sampling-weighted Cox estimates were 1.290 (95% CI 1.169–1.423) in CHARLS, 0.914 (0.792–1.053) in ELSA, and 0.991 (0.895–1.098) in HRS. Weighting therefore did not remove the between-cohort contrast (Additional file 1, Table S8; Additional file 2, Figure S2).

Exploratory competing-risk and time-varying analyses

Exploratory competing-risk analyses in CHARLS and ELSA did not materially alter the cohort-specific pattern (Additional file 1, Table S10); mortality ascertainment was incomplete in CHARLS and unavailable for HRS. The pBFMI-specific proportional-hazards test in ELSA was not significant ($P = 0.159$). Additional time-varying analyses were also compatible with no association: the log-time interaction P value was 0.105 and the 6-year piecewise interaction P value was 0.128 (Additional file 1, Table S11; Additional file 2, Figure S1). No late protective association was inferred.

Discussion

pBFMI was positively associated with incident stroke in CHARLS, but no corresponding association was observed in ELSA or HRS. This contrast persisted after exclusion of early events and sampling-weighted analysis. The formal cohort interaction remained significant when all Model 2 covariate effects were allowed to vary by cohort. The wide Hartung–Knapp confidence and prediction intervals further indicate that the three-cohort synthesis should not be interpreted as an overall effect.

Adding hypertension, diabetes, and heart disease attenuated the CHARLS estimate from 1.243 to 1.191 in the common-sample analysis. This attenuation is compatible with a role for cardiometabolic pathways, especially blood pressure [23], although the present analysis was not designed to estimate mediation and cannot separate pathway effects from confounding or measurement error. The positive CHARLS association is directionally consistent with large Chinese studies of general and central adiposity [5, 18, 19], but it does not establish a direct effect of predicted fat mass.

The mathematical diagnostics limit the interpretation of pBFMI as an independent body-composition measure in older adults. Its correlation with BMI exceeded 0.97 in every cohort and, by construction, it was fully determined by BMI, age, and sex. pBFMI did not materially improve apparent AIC or C-index relative to BMI. It should therefore be understood as a transformed anthropometric index rather than a direct or independently measured fat-mass phenotype. This distinction may help explain why the same

formula behaved differently across populations with different BMI distributions and BMI–adiposity relations [9, 24, 25, 32].

The absence of replication may also reflect differences in stroke subtype, survival, diagnosis, treatment, self-reporting, and selection into anthropometry samples. These issues are especially relevant after age 65, when selective survival, multimorbidity, sarcopenia, and illness-associated weight loss can weaken the correspondence between anthropometric size and metabolically adverse adiposity [3, 35, 39, 40]. One standardized pBFMI unit also represented a different absolute exposure increment in each cohort. These alternatives cannot be resolved with the present harmonized variables.

The ELSA time-varying analyses did not provide strong evidence that the pBFMI association changed over follow-up. Regression dilution from a single baseline measurement, illness-associated weight loss, selective survival, and competing morbidity nevertheless remain plausible sources of attenuation in older cohorts [35]. Because pBFMI was not updated and the 6-year cut point was not prespecified in a protocol, these analyses were treated as hypothesis-generating (Additional files 1–2).

This study used three large longitudinal cohorts of adults aged 65 years or older, excluded prevalent stroke, and analysed grouped event time directly. Sequential adjustment distinguished the primary association model from a model containing potential intermediates. Parallel cohort estimation, mathematical-coupling diagnostics, direct comparison with BMI, sampling weights, early-event exclusion, competing-risk models, and age-stratified analyses addressed several recurrent concerns in geriatric epidemiology.

pBFMI was predicted rather than measured by dual-energy X-ray absorptiometry, bioelectrical impedance, or imaging, and the original equation was not developed as a cross-ethnic clinical risk marker. Stroke was self-reported and subtype was unavailable consistently; event dates were approximated by survey wave. Covariates and outcome definitions were harmonized conceptually but not necessarily measurement-equivalent, particularly for alcohol use. Attrition and survival could bias longitudinal associations. Primary models used complete cases; although multiple imputation can reduce bias and loss of precision under appropriate assumptions [28, 29], cohort-equivalent auxiliary variables were not available for a common imputation strategy. Missingness was appreciable in ELSA, and included participants differed from excluded participants on several characteristics; common-sample sequential models address model comparability but not selection bias. Individual sample weights were available, but full design-based variance estimation was not harmonized. CHARLS mortality ascertainment was incomplete, and HRS competing-risk analysis could not be performed without the Tracker or linked mortality file. Multiple testing in subgroup and time-varying analyses increases the chance of false-positive findings. Finally, residual and unmeasured confounding preclude causal interpretation.

Among adults aged 65 years or older, the findings do not support a consistent association between pBFMI and stroke across the three cohorts. They also provide no evidence that pBFMI materially improves model fit or discrimination relative to BMI. Future cross-population work should validate

anthropometric equations against direct body-composition measurements and retain information on fat distribution, muscle mass, and metabolic health [3, 15, 39, 40].

Conclusions

Among adults aged 65 years or older, pBFMI was positively associated with incident stroke in CHARLS but not in ELSA or HRS. pBFMI was strongly coupled with BMI and did not materially improve apparent model fit or discrimination. Substantial between-cohort heterogeneity indicates that pBFMI did not provide a consistent measure of stroke risk across the three cohorts. Its use across older populations requires validation against direct body-composition measures.

Abbreviations

BF%

predicted percentage body fat

BMI

body mass index

CHARLS

China Health and Retirement Longitudinal Study

CI

confidence interval

ELSA

English Longitudinal Study of Ageing

HR

hazard ratio

HRS

Health and Retirement Study

pBFMI

predicted fat mass index

REML

restricted maximum likelihood

SD

standard deviation

STROBE

Strengthening the Reporting of Observational Studies in Epidemiology

WHtR

waist-to-height ratio.

Declarations

Ethics approval and consent to participate

CHARLS was approved by the Biomedical Ethics Review Committee of Peking University (IRB00001052-11015). ELSA received approval from the London Multicentre Research Ethics Committee (MREC/01/2/91). HRS was approved by the University of Michigan Health Sciences and Behavioral Sciences Institutional Review Board (HUM00061128). Participants or authorized representatives provided informed consent in the parent studies. The present study used de-identified secondary data and involved no new participant contact.

Consent for publication

Not applicable.

Clinical trial number: not applicable.

Availability of data and materials

CHARLS data are available to registered researchers through the CHARLS portal (<https://charls.pku.edu.cn/>). ELSA data are available through the UK Data Service (<https://ukdataservice.ac.uk/>). HRS data are available through the HRS portal (<https://hrs.isr.umich.edu/>). Access is governed by each study's registration and data-use conditions, and participant-level data cannot be redistributed by the authors. Supplementary tables, figures, reproducible analysis scripts, and non-restricted aggregate results accompany this manuscript as Additional files 1–3.

Competing interests

The authors declare that they have no competing interests.

Funding

This research received no external funding.

Authors' contributions

SL conceived the study, curated the data, conducted the formal analysis, developed the methodology, and drafted the manuscript. XC and RC contributed to data curation, investigation, and validation. ZY contributed to data curation, methodology, and validation. YH contributed to formal analysis, software, and visualisation. JY conceived and supervised the study and critically revised the manuscript. All authors read and approved the final manuscript.

Acknowledgements

The authors thank the participants and research teams of CHARLS, ELSA, and HRS for making these longitudinal resources available.

References

1. GBD 2019 Stroke Collaborators. Global, regional, and national burden of stroke and its risk factors, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet Neurol.* 2021;20:795–820. 10.1016/S1474-4422(21)00252-0.
2. Romero-Corral A, Somers VK, Sierra-Johnson J, Thomas RJ, Collazo-Clavell ML, Korinek J, et al. Accuracy of body mass index in diagnosing obesity in the adult general population. *Int J Obes.* 2008;32:959–66. 10.1038/ijo.2008.11.
3. Donini LM, Busetto L, Bischoff SC, Cederholm T, Ballesteros-Pomar MD, Batsis JA, et al. Definition and diagnostic criteria for sarcopenic obesity: ESPEN and EASO consensus statement. *Obes Facts.* 2022;15:321–35. 10.1159/000521241.
4. Kroll ME, Green J, Beral V, Sudlow CLM, Brown A, Kirichek O, et al. Adiposity and ischemic and hemorrhagic stroke: prospective study in women and meta-analysis. *Neurology.* 2016;87:1473–81. 10.1212/WNL.0000000000003171.
5. Pang Y, Kartsonaki C, Turnbull I, Guo Y, Clarke R, Chen Y, et al. Adiposity and risk of ischaemic and haemorrhagic stroke in 0.5 million Chinese men and women: a prospective cohort study. *Lancet Glob Health.* 2018;6:e630–40. 10.1016/S2214-109X(18)30216-X.
6. Toss F, Wiklund P, Franks PW, Eriksson M, Gustafson Y, Hallmans G, et al. Abdominal and gynoid adiposity and the risk of stroke. *Int J Obes.* 2011;35:1427–32. 10.1038/ijo.2011.9.
7. Winter Y, Rohrmann S, Linseisen J, Lanczik O, Ringleb PA, Hebebrand J, et al. Contribution of obesity and abdominal fat mass to risk of stroke and transient ischemic attacks. *Stroke.* 2008;39:3145–51. 10.1161/STROKEAHA.108.523001.
8. Deurenberg P, Weststrate JA, Seidell JC. Body mass index as a measure of body fatness: age- and sex-specific prediction formulas. *Br J Nutr.* 1991;65:105–14. 10.1079/BJN19910073.
9. Deurenberg P, Deurenberg-Yap M, Wang J, Lin FP, Schmidt G. Prediction of percentage body fat from anthropometry and bioelectrical impedance in Singaporean and Beijing Chinese. *Asia Pac J Clin Nutr.* 2000;9:93–8. 10.1046/j.1440-6047.2000.00149.x.
10. Zhao Y, Hu Y, Smith JP, Strauss J, Yang G. Cohort profile: the China Health and Retirement Longitudinal Study (CHARLS). *Int J Epidemiol.* 2014;43:61–8. 10.1093/ije/dys203.
11. Steptoe A, Breeze E, Banks J, Nazroo J. Cohort profile: the English Longitudinal Study of Ageing. *Int J Epidemiol.* 2013;42:1640–8. 10.1093/ije/dys168.
12. Sonnega A, Faul JD, Ofstedal MB, Langa KM, Phillips JWR, Weir DR. Cohort profile: the Health and Retirement Study (HRS). *Int J Epidemiol.* 2014;43:576–85. 10.1093/ije/dyu067.

13. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement. *Lancet*. 2007;370:1453–7. 10.1016/S0140-6736(07)61602-X.
14. Fine JP, Gray RJ. A proportional hazards model for the subdistribution of a competing risk. *J Am Stat Assoc*. 1999;94:496–509. 10.1080/01621459.1999.10474144.
15. Busetto L, Dicker D, Frühbeck G, Halford JCG, Sbraccia P, Yumuk V, et al. A new framework for the diagnosis, staging and management of obesity in adults. *Nat Med*. 2024;30:2395–9. 10.1038/s41591-024-03095-3.
16. Liu X, Zhang D, Liu Y, Sun X, Hou Y, Wang B, et al. A J-shaped relation of BMI and stroke: systematic review and dose-response meta-analysis of 4.43 million participants. *Nutr Metab Cardiovasc Dis*. 2018;28:1092–9. 10.1016/j.numecd.2018.07.004.
17. Hu G, Tuomilehto J, Silventoinen K, Sarti C, Männistö S, Jousilahti P. Body mass index, waist circumference, and waist-hip ratio on the risk of total and type-specific stroke. *Arch Intern Med*. 2007;167:1420–7. 10.1001/archinte.167.13.1420.
18. Bazzano LA, Gu D, Whelton MR, Wu X, Chen CS, Duan X, et al. Body mass index and risk of stroke among Chinese men and women. *Ann Neurol*. 2010;67:11–20. 10.1002/ana.21950.
19. Wang A, Wu J, Zhou Y, Guo X, Luo Y, Wu S, et al. Measures of adiposity and risk of stroke in China: a result from the Kailuan study. *PLoS ONE*. 2013;8:e61665. 10.1371/journal.pone.0061665.
20. Zahn K, Linseisen J, Heier M, Peters A, Thorand B, Nairz F, et al. Body fat distribution and risk of incident ischemic stroke in men and women aged 50 to 74 years from the general population: the KORA Augsburg cohort study. *PLoS ONE*. 2018;13:e0191630. 10.1371/journal.pone.0191630.
21. Gillum RF, Mussolino ME, Madans JH. Body fat distribution, obesity, overweight and stroke incidence in women and men—the NHANES I Epidemiologic Follow-up Study. *Int J Obes Relat Metab Disord*. 2001;25:628–38. 10.1038/sj.ijo.0801590.
22. Savva SC, Lamnisos D, Kafatos AG. Predicting cardiometabolic risk: waist-to-height ratio or BMI. A meta-analysis. *Diabetes Metab Syndr Obes*. 2013;6:403–19. 10.2147/DMSO.S34220.
23. Global Burden of Metabolic Risk Factors for Chronic Diseases Collaboration. Metabolic mediators of the effects of body-mass index, overweight, and obesity on coronary heart disease and stroke: a pooled analysis of 97 prospective cohorts with 1.8 million participants. *Lancet*. 2014;383:970–83. 10.1016/S0140-6736(13)61836-X.
24. Gallagher D, Heymsfield SB, Heo M, Jebb SA, Murgatroyd PR, Sakamoto Y. Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. *Am J Clin Nutr*. 2000;72:694–701. 10.1093/ajcn/72.3.694.
25. Jackson AS, Stanforth PR, Gagnon J, Rankinen T, Leon AS, Rao DC, et al. The effect of sex, age and race on estimating percentage body fat from body mass index: the Heritage Family Study. *Int J Obes Relat Metab Disord*. 2002;26:789–96. 10.1038/sj.ijo.0802006.
26. Grambsch PM, Therneau TM. Proportional hazards tests and diagnostics based on weighted residuals. *Biometrika*. 1994;81:515–26. 10.1093/biomet/81.3.515.

27. Viechtbauer W. Conducting meta-analyses in R with the metafor package. *J Stat Softw.* 2010;36:1–48. 10.18637/jss.v036.i03.
28. White IR, Royston P, Wood AM. Multiple imputation using chained equations: issues and guidance for practice. *Stat Med.* 2011;30:377–99. 10.1002/sim.4067.
29. Sterne JAC, White IR, Carlin JB, Spratt M, Royston P, Kenward MG, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. *BMJ.* 2009;338:b2393. 10.1136/bmj.b2393.
30. Lumley T. Analysis of complex survey samples. *J Stat Softw.* 2004;9:1–19. 10.18637/jss.v009.i08.
31. Rothman KJ. BMI-related errors in the measurement of obesity. *Int J Obes.* 2008;32(Suppl 3):S56–9. 10.1038/ijo.2008.87.
32. Heymsfield SB, Peterson CM, Thomas DM, Heo M, Schuna JM Jr.. Why are there race/ethnic differences in adult body mass index-adiposity relationships? A quantitative critical review. *Obes Rev.* 2016;17:262–75. 10.1111/obr.12358.
33. Suthahar N, Yang Y, Gansevoort RT, Damman K, Rienstra M, Bergman RN, et al. Relative fat mass as a predictor of incident stroke: evidence from the PREVEND cohort. *Nutr Metab Cardiovasc Dis.* 2026;36:104702. 10.1016/j.numecd.2026.104702.
34. Zheng Y, Huang C, Jin J, Zhao Y, Cui H, Wei C. Association between stroke and relative fat mass: a cross-sectional study based on NHANES. *Lipids Health Dis.* 2024;23:354. 10.1186/s12944-024-02351-2.
35. Prospective Studies Collaboration. Body-mass index and cause-specific mortality in 900,000 adults: collaborative analyses of 57 prospective studies. *Lancet.* 2009;373:1083–96. 10.1016/S0140-6736(09)60318-4.
36. Prentice RL, Gloeckler LA. Regression analysis of grouped survival data with application to breast cancer data. *Biometrics.* 1978;34:57–67. 10.2307/2529588.
37. Austin PC, Lee DS, Fine JP. Introduction to the analysis of survival data in the presence of competing risks. *Circulation.* 2016;133:601–9. 10.1161/CIRCULATIONAHA.115.017719.
38. Lau B, Cole SR, Gange SJ. Competing risk regression models for epidemiologic data. *Am J Epidemiol.* 2009;170:244–56. 10.1093/aje/kwp107.
39. Molino S, Dossena M, Buonocore D, Verri M. Sarcopenic obesity: an appraisal of the current status of knowledge and management in elderly people. *J Nutr Health Aging.* 2016;20:780–8. 10.1007/s12603-015-0631-8.
40. Batsis JA, Villareal DT. Sarcopenic obesity in older adults: aetiology, epidemiology and treatment strategies. *Nat Rev Endocrinol.* 2018;14:513–37. 10.1038/s41574-018-0062-9.

Figures

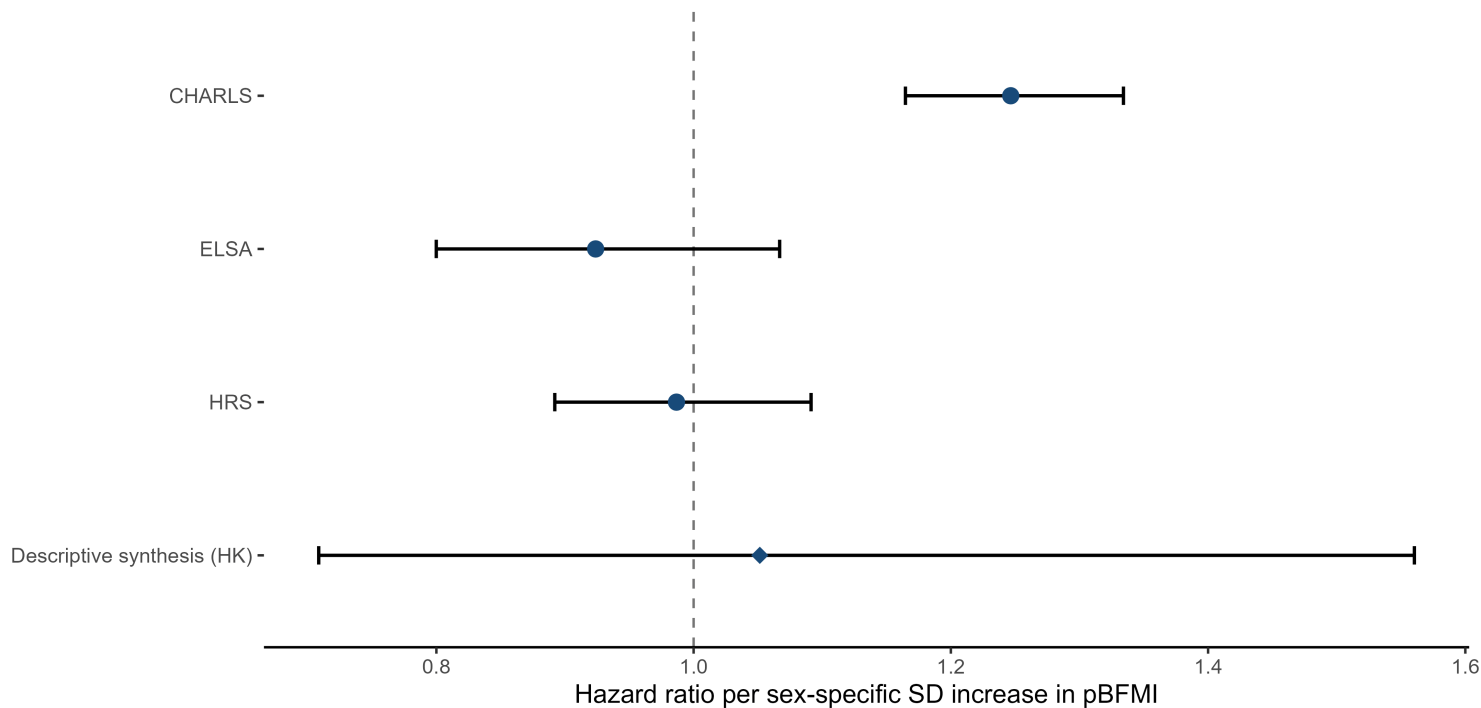


Figure 1

Cohort-specific Model 2 discrete-time associations between predicted fat mass index and incident stroke. The Hartung–Knapp estimate is a descriptive synthesis, not a common population effect. Horizontal lines indicate 95% confidence intervals.

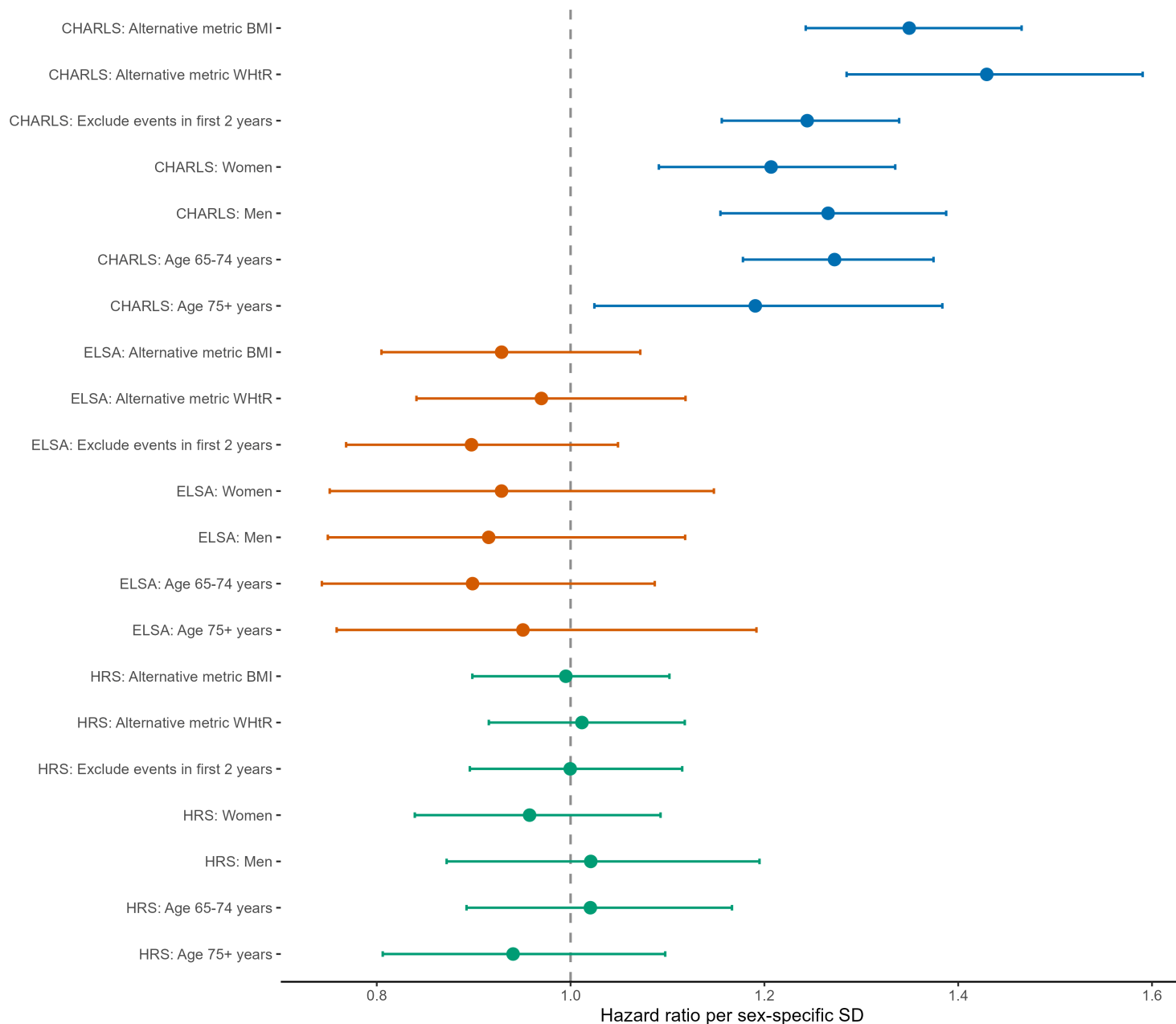


Figure 2

Sensitivity and subgroup analyses of predicted fat mass index and incident stroke. Points are cohort-specific adjusted hazard ratios; horizontal lines are 95% confidence intervals.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Additionalfile1supplementarytablesage65.xlsx](#)
- [Additionalfile2supplementaryfiguresage65.pdf](#)
- [Additionalfile3reproducibilitypackageage65.zip](#)