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Associations of body roundness index with steatotic liver disease and mortality from the UK biobank cohort study

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Abstract

Background Steatotic liver disease (SLD) is a chronic condition associated with cardiometabolic risk. The body roundness index (BRI) is a novel visceral adiposity marker. We evaluate the associations between BRI and risks of SLD, major adverse liver-related outcomes (MALO), liver-related mortality, and all-cause mortality using the UK Biobank cohort.

Methods Data from 399,115 participants (aged 37–73 years) without baseline SLD or MALO were analyzed. BRI was categorized into sex-specific quartiles. Outcomes were identified via national health records. Adjusted hazard ratios (HRs) were estimated using Fine–Gray competing risk models and Cox proportional hazards models.

Results During a median follow-up of 13.9 years, the incidence of SLD, MALO, liver-related mortality, and all-cause mortality was 1.38%, 1.25%, 0.24%, and 8.31%, respectively. Higher BRI was significantly associated with increased SLD risk (HR 6.20; 95% CI 5.28–7.28), with a more pronounced association in women (HR 9.11) than in men (HR 3.38). Significant non-linear, J-shaped associations were observed for SLD and all-cause mortality (both p for nonlinearity < 0.001). Conversely, MALO and liver-related mortality showed linear positive associations (p for nonlinearity > 0.05), with significant risks primarily observed in the highest BRI quartiles.

Conclusion Higher BRI is significantly associated with increased risks of SLD, MALO, and bothliver-related and all-cause mortality. These findings suggest that BRI is a valuable tool for identifying individuals at risk of adverse hepatic outcomes, potentially offering predictive utility beyond conventional anthropometric indices.

Keywords Liver disease, Body roundness, Mortality, UK biobank

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Introduction

Steatotic liver disease (SLD) is one of the most prevalent chronic liver conditions worldwide and is characterized by excessive fat accumulation in the liver [1]. SLD is closely associated with various adverse health outcomes, including obesity, cardiovascular disease, type 2 diabetes mellitus, cancer, chronic renal disease, and dementia [2–7]. In 2023, the term nonalcoholic fatty liver disease (NAFLD) was redefined and incorporated into the SLD spectrum through a global consensus [8]. Globally, NAFLD is estimated to affect 1.66 billion individuals, with its prevalence increasing from approximately 25% in 2010 to 38% in 2021 in the adult population [9, 10].

Obesity is a multisystem disease characterized by an abnormal excessive accumulation of body fat, contributing to a growing burden of metabolic disorders and chronic diseases [11]. Although conventional anthropometric indices such as body mass index (BMI) and waist circumference are commonly employed to evaluate obesity-related risks, these measures have inherent limitations in accurately capturing body shape and visceral adiposity [12]. The body roundness index (BRI) is a novel anthropometric measure derived from waist circumference (WC) and height and serves as a simple, non-invasive marker for early risk stratification [13]. As a composite indicator of central adiposity and fat distribution, a higher BRI is associated with an increased risk of cardiovascular disease, stroke, all-cause mortality, and mortality in individuals with diabetes mellitus [14–18].

The clinical significance of BRI in liver diseases remains unclear. A recent meta-analysis of 10 cross-sectional studies with 59,466 participants revealed an association between BRI and NAFLD [19]. However, large-scale population-based cohort studies investigating the association between BRI and the incidence of SLD and related mortality are still lacking. Major adverse liver-related outcomes (MALO), including liver cirrhosis, hepatic decompensation, chronic liver failure, hepatocellular carcinoma, and liver transplantation, represent critical clinical endpoints of liver disease [20]. If a significant association between BRI and MALO is established, BRI could potentially serve as a valuable marker for the early identification of individuals at risk of serious liver diseases.

This study aimed to investigate the association between BRI and the risk of SLD, MALO, and liver-related and all-cause mortality using large-scale population-based cohorts.

Methods

Data source

Data were obtained from the UK Biobank, a large-scale, prospective cohort comprising 502,369 community-dwelling adults aged 37–73 years who were recruited between 2006 and 2010 across 22 assessment centers throughout the United Kingdom [21]. At baseline, comprehensive information was collected from all participants, including electronic health records (death and cancer records), sociodemographic data, self-reported medical histories (such as medication use), lifestyle questionnaires, physical measurements, and blood and urine samples.

The present study is a retrospective cohort study conducted as a secondary analysis of existing UK Biobank data and was performed under UK Biobank Application Number 477,427. The UK Biobank has received ethical approval from the North West Multi-center Research Ethics Committee [22]. All participants provided written informed consent at the time of recruitment, and all study procedures were conducted in accordance with the Declaration of Helsinki and relevant institutional guidelines and regulations. For the present analysis, the institutional review board of Konkuk University Medical Center approved the exemption of ethical review and waived the additional requirement for informed consent, as the data used in this secondary analysis were fully anonymized and contained no identifiable information (KUMC 2025–10–022).

Study population

Figure 1 illustrates the participant selection process. Of the 502,369 individuals from the UK Biobank assessed at baseline, 103,254 were excluded because of pre-existing SLD and/or MALO ($n = 1492$) and missing data in the BRI calculation or covariates ($n = 101,762$). Finally, 399,115 participants without SLD or MALO at baseline who had completed at least one follow-up assessment after enrollment were included in the study. The median follow-up duration was 13.9 years (interquartile range, 13.2–14.6 years).

BRI measurements

The BRI, as proposed by Thomas et al., assessed individuals' fat distribution, with a particular focus on visceral fat, thereby offering a more precise assessment of body shape [23]. Height (m), weight (kg), and WC (cm) were measured by trained personnel following standardized protocols. BRI was calculated using the following formula:

$$\mathrm{BRI} = 364.2 - 365.5 \times \sqrt{1 - \left(\frac{WC}{2\pi}\right)^2 / (0.5 \times \ height)^2}$$

The participants were categorized into sex-specific BRI quartiles for analysis [23]. Higher BRI values indicate a rounder body shape and higher amounts of body fat.

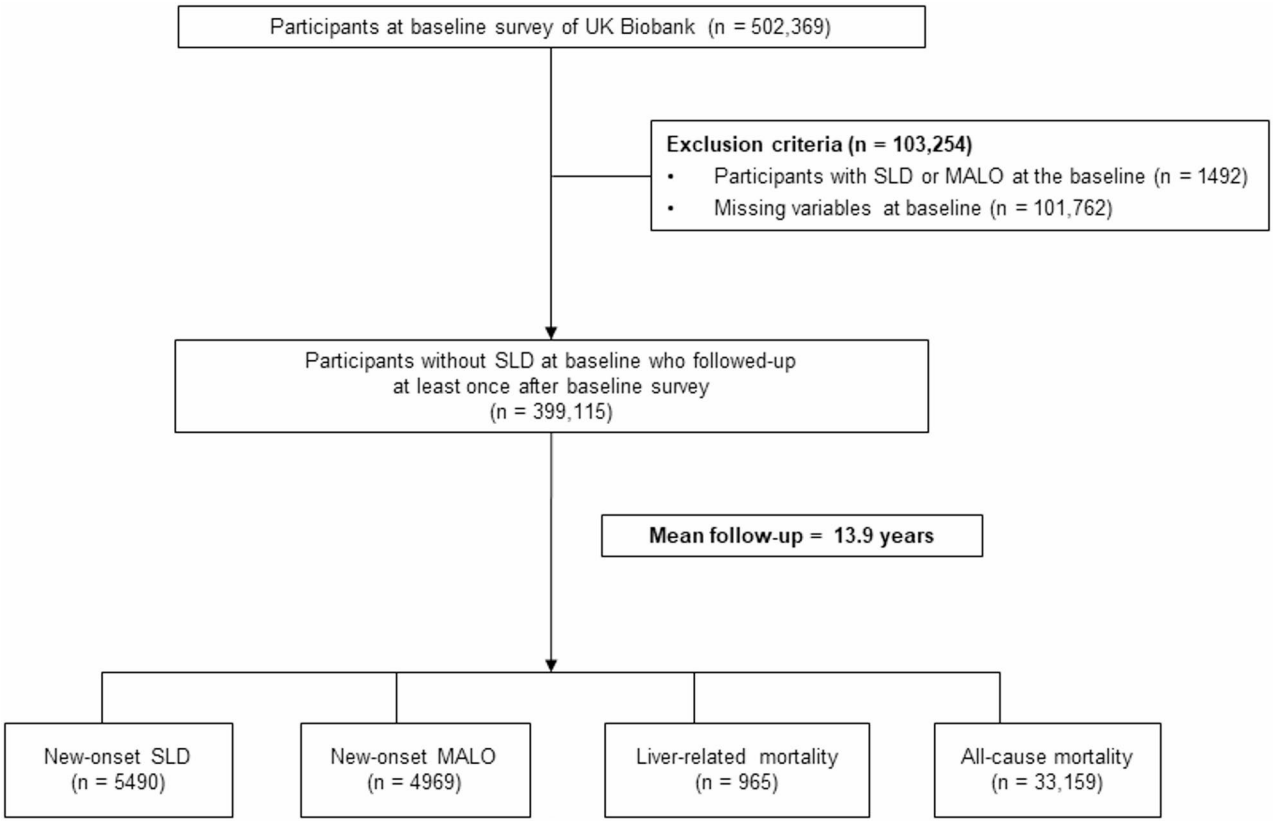

Fig. 1 Study population

SLD, MALO, liver-related mortality, and all-cause mortality

Liver-related outcomes, including SLD incidence, MALO, liver-related mortality, and all-cause mortality, were ascertained through linkage to national mortality registries and classified according to the International Classification of Diseases, 10th Revision (ICD-10) codes. In the UK Biobank dataset, new-onset SLD was defined as a diagnosis of SLD (K76.0 or K75.8) occurring after the baseline assessment [24]. MALO was defined as liver cirrhosis, hepatic decompensation (portal hypertension, esophageal varices, ascites, encephalopathy, or hepatorenal syndrome), chronic liver failure, hepatocellular carcinoma, or liver transplantation (V42.7, C22.0, I85.0, I85.9, I86.4, I98.2–I98.3, K70.3–K70.4, K71.7, K72.1, K72.9, K74.6, K76.6-K76.7, R18, T86.4, and Z94.4) [20]. Liver-related mortality included deaths due to liver disease (C22.0, C22.2–C22.9, and K70–K76) [25]. All-cause mortality was defined as death from any cause during follow-up.

Covariates

During the assessment center visit, baseline body composition was measured by trained staff using standardized procedures. The body mass index (BMI) was calculated by dividing the weight (kg) by the square of the height (m^2). Waist and hip circumferences were measured using a Wessex non-stretchable sprung tape. Peripheral venous blood samples were collected from all participants at baseline following validated procedures based on those used in the UK Biobank study [26]. Venous blood samples were collected after an overnight fast, and the levels of serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), glucose, lipid profiles, and high-sensitivity C-reactive protein (hs-CRP) were measured using a Beckman Coulter AU5800 analyzer. Baseline covariates included age, sex, smoking status (non-smoker or current smoker), alcohol consumption (non-drinker or current drinker), physical activity (measured as metabolic equivalent of task [MET] hours per week), and comorbidities such as hypertension (ICD-10 codes: I10, I11, I12, I13, I15), diabetes mellitus (E10, E11, E12, E13, E14), and dyslipidemia (E78). Trained staff measured the blood pressure twice in a seated position using a digital device.

Additional covariates included noninvasive liver fibrosis indices such as the AST to platelet ratio index (APRI), fibrosis-4 (FIB-4) index, and triglyceride-glucose (TyG) index [27, 28]. Metabolic syndrome was defined according to harmonized criteria as the presence of three or more of the following: (1) increased WC (≥ 94 cm for men and ≥ 80 cm for women), (2) elevated triglycerides (≥ 150 mg/dL) or treatment for elevated triglycerides, (3)

Table 1 Baseline characteristics of UK biobank participants

Characteristic	Overall (*n* = 399,115)	Q1 (*n* = 99,780)	Q2 (*n* = 99,780)	Q3 (*n* = 99,776)	Q4 (*n* = 99,779)	*p*-value
BRI Range	[−0.628,23.6]	[−0.628,3.02]	(3.02,3.91]	(3.91,4.95]	(4.95,23.6]	
Age, years	56.3 ± 8.1	54.2 ± 8.2	56.0 ± 8.1	57.2 ± 8.0	57.9 ± 7.7	< 0.001
Sex, n (%)						< 0.001
Men	209,308 (52.4%)	75,873 (76.0%)	50,301 (50.4%)	39,766 (39.9%)	43,368 (43.5%)	
Women	189,807 (47.6%)	23,907 (24.0%)	49,479 (49.6%)	60,010 (60.1%)	56,411 (56.5%)	
BMI, kg/m^2	27.3 ± 4.7	22.8 ± 2.1	25.6 ± 2.1	28.0 ± 2.3	32.9 ± 4.4	< 0.001
WC, cm	90.2 ± 13.4	74.7 ± 6.0	85.8 ± 5.4	93.9 ± 5.7	106.3 ± 9.5	< 0.001
SBP, mmHg	137.5 ± 18.6	130.5 ± 18.2	136.8 ± 18.1	140.4 ± 17.9	142.6 ± 17.8	< 0.001
DBP, mmHg	82.2 ± 10.1	77.8 ± 9.5	81.5 ± 9.7	83.9 ± 9.7	85.6 ± 9.9	< 0.001
Alcohol consumption, n (%)	369,272 (92.5%)	93,039 (93.2%)	93,483 (93.7%)	92,866 (93.1%)	89,884 (90.1%)	< 0.001
Smoking history, n (%)	40,614 (10.2%)	9557 (9.6%)	10,197 (10.2%)	10,399 (10.4%)	10,461 (10.5%)	< 0.001
MET-hour/week	44.2 ± 45.2	48.0 ± 44.8	46.2 ± 45.7	44.4 ± 46.0	38.4 ± 43.8	< 0.001
Fasting glucose, mmol/L	92.1 ± 21.9	88.3 ± 15.0	90.0 ± 16.8	92.1 ± 20.1	97.9 ± 30.8	< 0.001
HbA1c, %	5.44 ± 0.61	5.28 ± 0.39	5.35 ± 0.47	5.44 ± 0.56	5.69 ± 0.82	< 0.001
hs-CRP, mg/dL	0.25 ± 0.42	0.15 ± 0.33	0.20 ± 0.38	0.26 ± 0.41	0.39 ± 0.50	< 0.001
Platelet count, ×10^9/L	252.1 ± 59.5	251.8 ± 58.6	252.1 ± 58.7	250.9 ± 59.1	253.3 ± 61.7	< 0.001
AST, U/L	26.2 ± 10.4	24.5 ± 9.3	25.6 ± 9.4	26.8 ± 10.2	28.1 ± 12.2	< 0.001
ALT, U/L	23.6 ± 14.1	18.1 ± 9.9	21.8 ± 12.0	25.4 ± 14.3	28.9 ± 16.9	< 0.001
Total Cholesterol, mg/dL	219.9 ± 44.0	218.7 ± 40.1	223.2 ± 42.7	222.6 ± 45.0	215.2 ± 47.3	< 0.001
TG, mg/dL	154.2 ± 90.9	106.4 ± 52.8	143.8 ± 78.1	172.3 ± 94.3	194.6 ± 104.6	< 0.001
HDL-C, mg/dL	56.0 ± 14.8	65.1 ± 15.0	57.2 ± 13.9	52.7 ± 12.9	49.0 ± 12.0	< 0.001
LDL-C mg/dL	137.4 ± 33.4	132.3 ± 30.4	139.9 ± 32.6	141.1 ± 34.1	136.3 ± 35.6	< 0.001
APRI	0.28 ± 0.54	0.26 ± 0.16	0.27 ± 0.16	0.29 ± 0.44	0.31 ± 0.96	< 0.001
FIB-4	1.35 ± 2.09	1.35 ± 0.73	1.34 ± 0.71	1.35 ± 2.16	1.35 ± 3.45	0.700
TyG index	8.71 ± 0.57	8.35 ± 0.45	8.64 ± 0.51	8.84 ± 0.53	9.01 ± 0.56	< 0.001
DM, n (%)	19,765 (5.0%)	1029 (1.0%)	2233 (2.2%)	4521 (4.5%)	11,982(12.0%)	< 0.001
HTN, n (%)	104,615 (26.2%)	11,651 (11.7%)	19,752 (19.8%)	29,053 (29.1%)	44,159 (44.3%)	< 0.001
Dyslipidemia, n (%)	67,616 (16.9%)	5818 (5.8%)	12,354 (12.4%)	19,668 (19.7%)	29,776 (29.8%)	< 0.001
Metabolic syndrome, n (%)	114,684 (33.3%)	2089 (2.4%)	14,280 (16.6%)	39,266 (45.4%)	59,049 (68.5%)	< 0.001

BRI body roundness index, *BMI* body mass index, *WC* waist circumference, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *MET* metabolic equivalent of task, *hs-CRP* high-sensitivity C-reactive protein, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *TG* triglyceride, *HDL* high density lipoprotein cholesterol, *LDL-C* low density lipoprotein cholesterol, *APRI* AST to platelet ration index, *FIB-4* fibrosis – 4 index, *TyG* triglyceride-glucose index, *DM* diabetes mellitus, *HTN* Hypertension

reduced high-density lipoprotein (HDL) cholesterol (< 40 mg/dL for men and < 50 mg/dL for women) or treatment for low HDL, (4) elevated blood pressure (≥ 130/85 mmHg) or treatment for hypertension, and (5) elevated fasting glucose (≥ 100 mg/dL) or treatment for hyperglycemia [29].

Statistical analysis

Continuous variables are presented as mean ± standard deviation (SD), and categorical variables are reported as counts (percentages). Baseline characteristics across the BRI quartiles were compared using analysis of variance (ANOVA) for continuous variables and chi-square tests for categorical variables. The cumulative incidence of new-onset SLD, MALO, liver-related mortality, and all-cause mortality across the BRI quartiles was estimated using cumulative incidence functions. For new-onset SLD, MALO, and liver-related mortality, differences between groups were evaluated using Gray's test, considering death as a competing event, whereas differences in all-cause mortality were assessed using the log-rank test. For new-onset SLD, MALO, and liver-related mortality, subdistribution hazard ratios (HRs) and 95% confidence intervals (CIs) were estimated using Fine–Gray competing risk models, treating death as a competing event. For all-cause mortality, hazard ratios and 95% confidence intervals were estimated using Cox proportional hazards models according to the BRI quartiles. Three models were constructed for this study. Model 1 was adjusted for age and sex. Model 2 was further adjusted for BMI, smoking status, alcohol intake, physical activity, and metabolic syndrome, whereas Model 3 was additionally adjusted for hypertension, diabetes mellitus, and dyslipidemia. The proportional hazards assumption was assessed using Schoenfeld residuals, and no significant violations were observed. Subgroup analyses were performed according to sex. The BRI ranges for men were Q1 (−0.628, 3.47), Q2 (3.47, 4.24), Q3 (4.24, 5.16), and Q4 (5.16, 23.6), while those for women were Q1 (−0.148, 2.65), Q2 (2.65, 3.48), Q3 (3.48, 4.66), and Q4 (4.66, 19.0). A restricted cubic

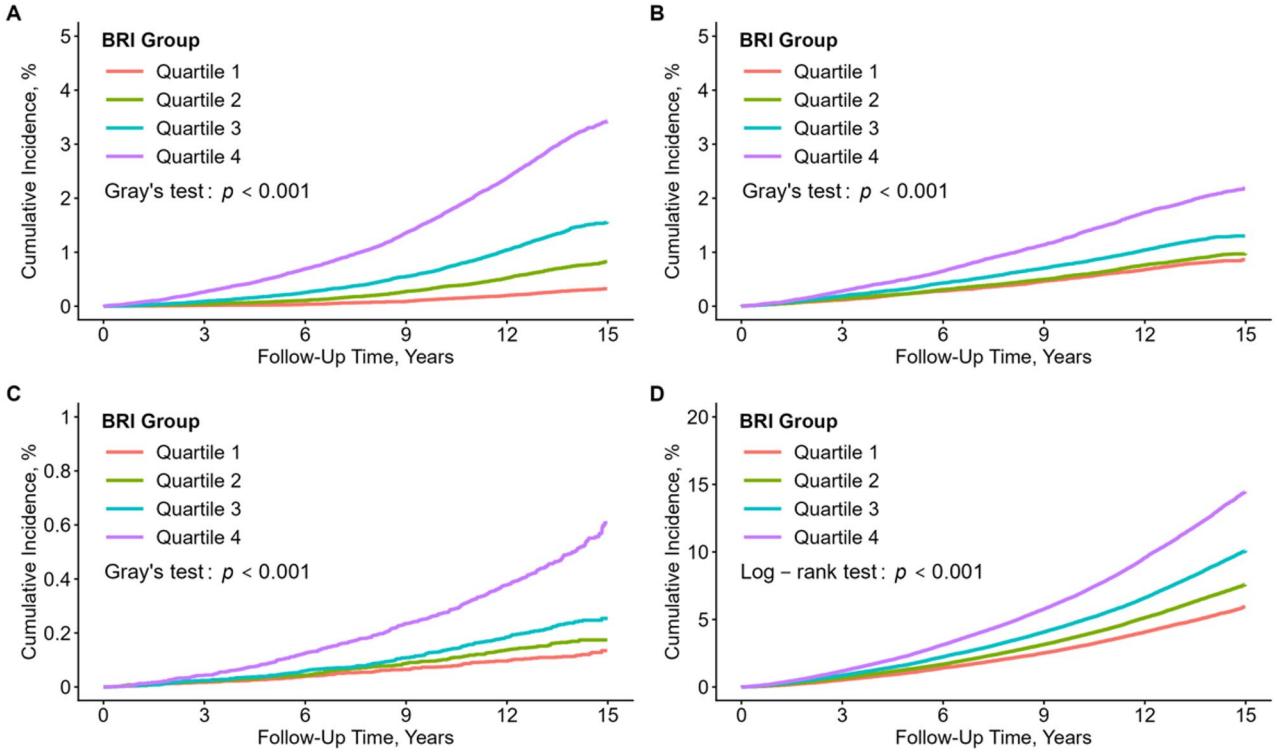

Fig. 2 Kaplan–Meier curves for new-onset steatotic liver disease (SLD), major adverse liver-related outcomes (MALO), liver-related mortality, and all-cause mortality across the body roundness index (BRI) quartiles. **A** SLD, **B** MALO, **C** liver-related mortality, and **D** all-cause mortality

spline curve was used to determine the optimal cutoff point for the BRI. Additional analyses were conducted to compare the predictive performance of BRI with traditional anthropometric measures (BMI, WC, and waist-to-height ratio [WHtR]). Nested model comparisons and performance metrics, including likelihood ratio tests, Akaike information criterion, and C-statistics, were used to assess the incremental predictive value of BRI, with detailed results provided in the Supplementary Table. All statistical analyses were conducted using R software version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria), and a two-sided p-value < 0.05 was considered statistically significant.

Results

Among 399,115 participants in the UK Biobank, new-onset SLD was identified in 5490 individuals (1.38%). The rates of MALO, liver-related mortality, and all-cause mortality were 1.25% ($n = 4969$), 0.24% ($n = 965$), and 8.31% ($n = 33,159$), respectively (Fig. 1). Participants in the higher BRI quartiles (Q2–Q4) were significantly older and had higher obesity rates than those in the reference group (Q1) (Table 1). Systolic and diastolic blood pressure, fasting glucose, hs-CRP, AST, ALT, and triglyceride levels progressively increased across the BRI quartiles. Conversely, physical activity (measured in MET-hours/week) and HDL-C levels decreased with increasing BRI quartiles. A higher BRI score is also associated with an increased prevalence of cardiometabolic comorbidities.

The incidence of SLD increased significantly across the BRI quartiles (Q2–Q4), with a clear dose-response relationship (Fig. 2). Kaplan-Meier survival curves revealed that participants with a higher BRI experienced a greater cumulative incidence of SLD (Fig. 2A) (log-rank test, $P < 0.001$). Similarly, MALO, liver-related mortality, and all-cause mortality increased with increasing BRI quartiles (Fig. 2B and D), reinforcing the association between elevated BRI levels and adverse health outcomes.

Table 2 presents the associations between the BRI quartiles and the risk of new-onset SLD, MALO, liver-related mortality, and all-cause mortality. Compared with the reference group (Q1), the incidence of SLD increased progressively across higher BRI quartiles (Q2–Q4), maintaining statistical significance after adjusting for covariates. For MALO, while the unadjusted HRs showed a progressive increase, the multivariable-adjusted risk was significantly elevated only in the highest quartile (Q4). Liver-related mortality showed a progressive increase, remaining significant in both Q3 and Q4. Regarding all-cause mortality, the adjusted HR showed a decrease in Q2 compared to the reference group (Q1), followed by a significant increase in Q4.

A significant non-linear association was observed between BRI and the risks of SLD and all-cause mortality

Table 2 Hazard ratios of study outcomes for UK participants according to BRI quartiles

Outcomes	Group	Event	Unadjusted		Model 1		Model 2		Model 3	
			HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
SLD (*n* = 5490)	Q1	281	reference		reference		reference		reference	
	Q2	723	2.67 (2.30–3.10)	< 0.001	2.99 (2.57–3.48)	< 0.001	2.48 (2.12–2.88)	< 0.001	2.52 (2.17–2.94)	< 0.001
	Q3	1,398	5.13 (4.46–5.90)	< 0.001	6.08 (5.27–7.01)	< 0.001	4.30 (3.71–4.99)	< 0.001	4.33 (3.73–5.03)	< 0.001
	Q4	3,088	10.71 (9.37–12.24)	< 0.001	12.70 (11.08–14.55)	< 0.001	6.51 (5.56–7.64)	< 0.001	6.20 (5.28–7.28)	< 0.001
MALO (*n* = 4969)	Q1	805	reference		reference		reference		reference	
	Q2	907	1.14 (0.97–1.32)	0.102	1.07 (0.91–1.25)	0.398	0.98 (0.84–1.15)	0.822	1.00 (0.85–1.18)	0.992
	Q3	1,233	1.49 (1.29–1.72)	< 0.001	1.36 (1.17–1.58)	< 0.001	1.16 (0.98–1.37)	0.085	1.17 (0.99–1.39)	0.068
	Q4	2,024	2.60 (2.28–2.96)	< 0.001	2.36 (2.05–2.71)	< 0.001	1.73 (1.42–2.10)	< 0.001	1.65 (1.36–2.01)	< 0.001
Liver-related mortality (*n* = 965)	Q1	114	reference		reference		reference		reference	
	Q2	159	1.39 (1.09–1.77)	0.007	1.27 (1.00–1.62.00.62)	0.051	0.99 (0.77–1.28)	0.937	1.21 (0.95–1.56)	0.125
	Q3	222	1.94 (1.55–2.44)	< 0.001	1.68 (1.34–2.11)	< 0.001	1.13 (0.88–1.46)	0.341	1.51 (1.18–1.93)	0.001
	Q4	470	4.12 (3.36–5.06)	< 0.001	3.45 (2.81–4.24)	< 0.001	2.02 (1.52–2.70)	< 0.001	2.67 (2.02–3.54)	< 0.001
All-cause mortality (*n* = 33,159)	Q1	5,198	reference		reference		reference		reference	
	Q2	6,651	1.29 (1.24–1.33)	< 0.001	0.97 (0.93–1.00.93.00)	0.089	0.92 (0.88–0.95)	< 0.001	0.92 (0.89–0.96)	< 0.001
	Q3	8,753	1.71 (1.65–1.77)	< 0.001	1.11 (1.07–1.15)	< 0.001	1.01 (0.97–1.05)	0.74	1.00 (0.96–1.04)	0.948
	Q4	12,557	2.51 (2.43–2.59)	< 0.001	1.57 (1.52–1.62)	< 0.001	1.29 (1.23–1.36)	< 0.001	1.23 (1.17–1.29)	< 0.001

SLD, MALO, and liver-related mortality were analyzed using Fine–Gray subdistribution hazard models considering death as a competing event, whereas all-cause mortality was analyzed using Cox proportional hazards models

HR hazard ratio, *CI* confidence intervals, *SLD* steatotic liver disease, *MALO* major adverse liver outcomes

Model 1: adjusted for age and sex, Model 2: adjusted for age, sex, BMI, smoking, alcohol consumption, physical activity, and metabolic syndrome, and Model 3: age, sex, BMI, smoking, alcohol consumption, physical activity, metabolic syndrome, hypertension, diabetes mellitus, and dyslipidemia

(p for nonlinearity < 0.001, Fig. 3). while the risk increased significantly beyond certain BRI thresholds, all-cause mortality exhibited a U-shaped pattern with an initial decrease at lower BRI levels. In contrast, BRI showed a linear association with MALO and liver-related mortality, as the test for non-linearity was not statistically significant ($P = 0.813$ and $P = 0.538$).

Table 3 presents the associations between the BRI quartiles and the risk of SLD, MALO, liver-related mortality, and all-cause mortality stratified by sex. The risk of SLD increased across BRI quartiles in both men and women. Compared with Q1, the adjusted HR for SLD in Q4 was 3.38 (95% CI: 2.77–4.13) in men and 9.11 (95% CI: 7.16–11.59) in women. For MALO, a significant increase in risk with higher BRI was observed in men in Q4 and in women in Q3 and Q4. Liver-related mortality was significantly increased only in Q4 in both men and women. For all-cause mortality, significant associations were observed in Q3 and Q4 among men, whereas in women, a significant association was observed only in Q4, with a lower hazard ratio observed in Q2 compared with Q1.

Discussion

In this large population-based prospective cohort study using UK Biobank data, we demonstrate that the BRI is a robust anthropometric marker associated with the risks of incident SLD, MALO, and liver-related and all-cause mortality. Beyond confirming the adverse prognostic impact of higher BRI, our findings provide important insight into the heterogeneous nature of these associations. Specifically, we observed distinct non-linear relationships for SLD and all-cause mortality, suggesting that both low and high extremes of body roundness may confer elevated risk, whereas the associations with MALO and liver-related mortality were predominantly linear. These differential patterns underscore the complexity of body shape–related risk in liver disease progression and

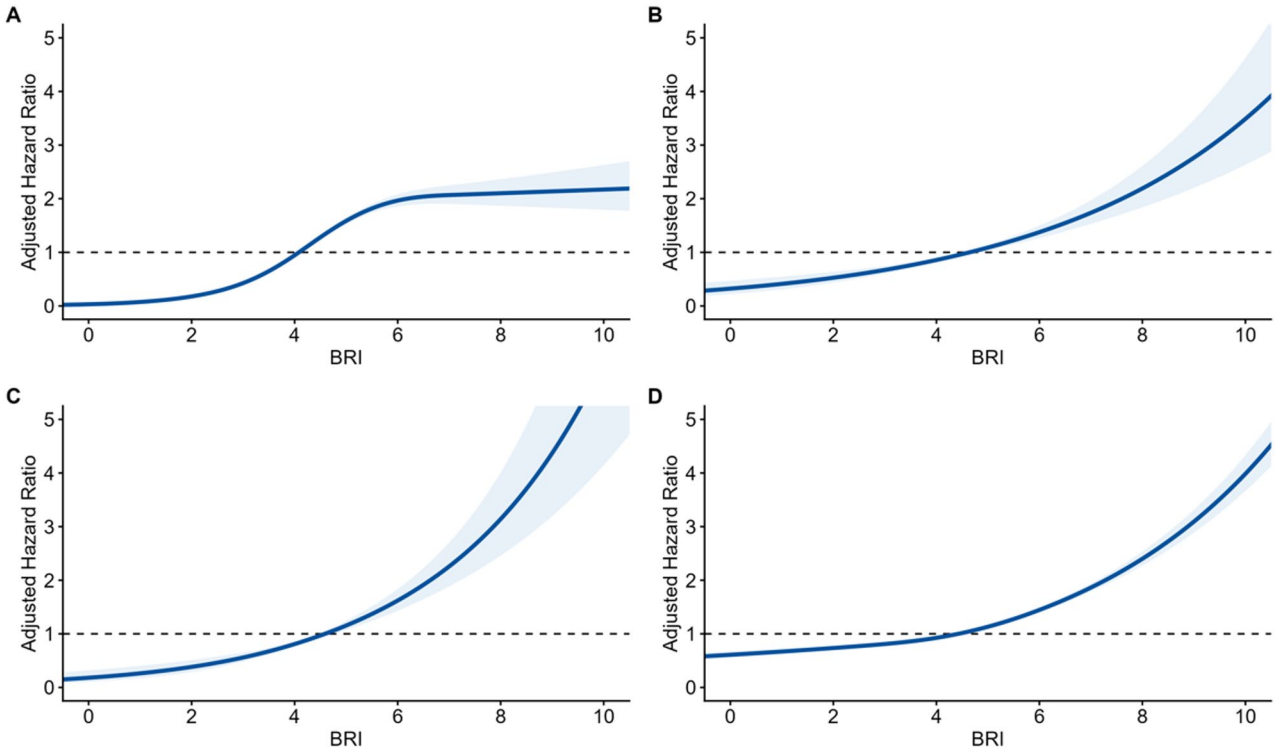

Fig. 3 Restricted cubic spline curve of outcomes according to the body roundness index (BRI). **A** Steatotic liver disease (SLD), **B** Major adverse liver-related outcomes (MALO), **C** liver-related mortality, and **D** all-cause mortality

mortality and suggest that BRI captures clinically relevant aspects of body composition not fully reflected by conventional anthropometric measures (such as BMI, WC, and WHtR) [30]. These measurements are widely used to assess cardiometabolic risk, but they do not account for height and may inadequately reflect overall body composition [31]. The BRI has therefore been proposed as an alternative indicator of body shape and visceral adiposity. A key distinction of the present study is its prospective longitudinal design using the UK Biobank cohort. While much of the existing evidence regarding BRI and metabolic health has relied on cross-sectional data [19], our 13.9-year follow-up provides robust evidence of temporal precedence, demonstrating that baseline BRI can predict the long-term incidence of serious hepatic outcomes and mortality.

The potential mechanisms underlying elevated BRI in liver diseases and mortality may involve adipocyte hypertrophy and proliferation, increased oxidative stress, and excessive free fatty acids [19]. These interrelated processes contribute to hepatic lipid accumulation and insulin resistance, ultimately leading to hepatic steatosis [1, 32]. Previous studies have suggested that the association between increased mortality risk and elevated BRI may be mediated by cardiovascular and metabolic disorders [33]. In contrast, our study was adjusted for key cardiometabolic comorbidities, including hypertension, diabetes mellitus, dyslipidemia, and metabolic syndrome. Therefore, the observed associations may reflect the independent role of BRI beyond that of traditional metabolic risk factors.

Interestingly, a lower risk of all-cause mortality was observed in women in Q2 compared with Q1. This finding is in line with previous evidence suggesting a non-linear association between BRI and mortality. This pattern likely reflects the physiological limitations of BRI in individuals at the lower extreme of the adiposity. A very low BRI may not necessarily indicate optimal metabolic health but could instead serve as a surrogate for malnutrition, frailty, or sarcopenia. In 15,157 participants over 40 years old from the National Health and Nutrition Examination Survey (NHANES, 2003–2018), low BRI is significantly associated with an increased prevalence of frailty, which in turn acts as a critical mediator for all-cause mortality [34]. Regardless of hepatic dysfunction, sarcopenia is —the loss of muscle mass and function—is also a recognized independent prognostic factor in liver diseases, associated with increased mortality [35]. Physiologically, individuals with low BRI and concurrent frailty lack the metabolic protection afforded by adequate muscle mass, which is essential for glucose disposal and systemic resilience [36].

In a cohort of 32,995 individuals from the NHANES (1999–2018), a U-shaped association between BRI and

Table 3 Sex-stratified hazard ratios for SLD, MALO, and mortality by BRI quartiles

Outcome	Group	Men						Women					
		Unadjusted		Model 1		Model 2		Unadjusted		Model 1		Model 2	
		HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
SLD	Q1	reference		reference		reference		reference		reference		reference	
	Q2	1.99 (1.65–2.39)	< 0.001	2.07(1.72–2.49)	< 0.001	1.74(1.44–2.09)	< 0.001	2.85 (2.24–3.63)	< 0.001	2.95 (2.32–3.76)	< 0.001	2.60 (2.04–3.31)	< 0.001
	Q3	3.35 (2.82–3.98)	< 0.001	3.58(3.01–4.26)	< 0.001	2.54(2.12–3.04)	< 0.001	6.87 (5.50–8.58)	< 0.001	7.26 (5.81–9.08)	< 0.001	5.46 (4.35–6.87)	< 0.001
	Q4	6.41 (5.46–7.54)	< 0.001	6.99(5.93–8.24)	< 0.001	3.38(2.77–4.13)	< 0.001	16.70(13.48–20.68)	< 0.001	17.77(14.33–22.05)	< 0.001	9.11 (7.16–11.59)	< 0.001
MALO	Q1	reference		reference		reference		reference		reference		reference	
	Q2	0.97 (0.79–1.20)	0.802	0.95(0.77–1.18)	0.659	0.88(0.71–1.09)	0.241	1.02 (0.82–1.28)	0.835	0.99 (0.79–1.24)	0.922	0.95 (0.75–1.20)	0.668
	Q3	1.31 (1.08–1.59)	0.007	1.27(1.04–1.54)	0.019	1.06(0.86–1.32)	0.574	1.49 (1.22–1.83)	< 0.001	1.41 (1.15–1.74)	0.001	1.27(1.00–1.60.00.60)	0.045
	Q4	2.56 (2.15–3.04)	< 0.001	2.45(2.05–2.92)	< 0.001	1.67(1.30–2.14)	< 0.001	2.22 (1.83–2.68)	< 0.001	2.09 (1.71–2.54)	< 0.001	1.53 (1.15–2.03)	0.004
Liver-related mortality	Q1	Ref		reference		reference		reference		reference		reference	
	Q2	0.96 (0.72–1.27)	0.758	0.89(0.67–1.18)	0.406	0.86(0.64–1.14)	0.295	1.37 (0.92–2.03)	0.116	1.22 (0.82–1.81)	0.331	1.10 (0.73–1.65)	0.640
	Q3	1.58 (1.22–2.03)	< 0.001	1.39(1.08–1.79)	0.01	1.27(0.97–1.67)	0.086	1.60 (1.09–2.35)	0.015	1.33 (0.90–1.95)	0.151	1.08 (0.70–1.65)	0.737
	Q4	3.01 (2.39–3.78)	< 0.001	2.57(2.04–3.23)	< 0.001	2.01(1.46–2.77)	< 0.001	3.53 (2.52–4.96)	< 0.001	2.87 (2.04–4.05)	< 0.001	1.74 (1.07–2.85)	0.026
All-cause mortality	Q1	reference		reference		reference		reference		reference		reference	
	Q2	1.20 (1.15–1.26)	< 0.001	1.03(0.99–1.08)	0.144	1.00(0.96–1.05)	0.968	1.18 (1.11–1.25)	< 0.001	0.98 (0.93–1.04)	0.569	0.94 (0.88–0.99)	0.024
	Q3	1.52 (1.45–1.58)	< 0.001	1.19(1.14–1.25)	< 0.001	1.11(1.05–1.16)	< 0.001	1.46 (1.38–1.54)	< 0.001	1.10 (1.04–1.16)	< 0.001	0.98 (0.92–1.04)	0.438
	Q4	2.22 (2.14–2.32)	< 0.001	1.66(1.59–1.73)	< 0.001	1.39(1.31–1.48)	< 0.001	2.10 (1.99–2.21)	< 0.001	1.55 (1.47–1.63)	< 0.001	1.17 (1.08–1.26)	< 0.001

SLD, MALO, and liver-related mortality were analyzed using Fine–Gray subdistribution hazard models considering death as a competing event, whereas all-cause mortality was analyzed using Cox proportional hazards models

HR hazard ratio, *CI* confidence intervals, *SLD* steatotic liver disease, *MALO* major adverse liver outcomes

Model 1: age and Model 2: age, BMI, smoking, alcohol consumption, metabolic syndrome, hypertension, diabetes mellitus, and dyslipidemia

all-cause mortality was observed over a median follow-up of 9.98 years, with the lowest mortality observed at a BRI range of 4.45–5.46 [33]. Although this nadir was higher than the Q2 range in our study (3.02,3.91], the optimal BRI range associated with the lowest mortality may differ across populations. In particular, visceral adiposity distribution has been reported to vary by race and ethnicity, with higher visceral fat deposition among Hispanic individuals, which may shift the reference range associated with the lowest risk [33].

Previous studies have shown that the BRI tends to be higher in women than in men, increases with age, and is more elevated among Mexican American individuals, followed by non-Hispanic Black and non-Hispanic White populations [33]. Populations with higher BRI values, which often include a greater proportion of women, have also been reported to exhibit lower rates of current smoking and alcohol consumption [37]. In contrast, in our study, the proportions of men and women were comparable across BRI quartiles, resulting in largely overlapping BRI ranges between sexes. Despite this similarity in distribution, higher BRI was associated with an increased risk of SLD in both men and women, with a substantially greater magnitude of risk observed in women (HR = 9.11 in Q4) than in men (HR = 3.38 in Q4). These findings suggest that increasing BRI may confer a disproportionately higher liver-related risk in women, highlighting the potential influence of sex on the relationship between body shape and liver disease outcome.

Supporting the heterogeneity of these associations, a subset analysis of participants with metabolic dysfunction–associated steatotic liver disease (MASLD) from the same NHANES cohort demonstrated a J-shaped association between BRI and all-cause mortality [37]. This pattern has been attributed to the coexistence of obesity and cardiometabolic comorbidities among individuals with established liver disease. Notably, the reference category in that study (Q1: BRI < 5.62) would encompass a wide range of BRI values, including those corresponding to the highest quartile in our study, underscoring the need for caution when directly comparing BRI thresholds across studies with differing population distributions. Additional evidence comes from the NAGALA cohort, which included 15,299 Japanese participants without obesity or diabetes, and demonstrated a nonlinear association between BRI and MASLD prevalence, similar to our findings [38]. In that study, the risk of MASLD increased with BRI up to a certain threshold, beyond which the magnitude of risk increases attenuated. Taken together, these findings suggest that baseline metabolic health, population characteristics, and sex may modify the association between BRI and liver-related outcomes, emphasizing the importance of contextual interpretation when applying BRI in clinical and epidemiological settings.

This study had several limitations that should be considered. First, the identification of SLD and MALO was based on ICD-codes, which may have led to underdiagnosis or misclassification. This is particularly relevant in the UK Biobank cohort, where the incidence of SLD was relatively low (1.37%), possibly reflecting under-detection or under-reporting [9]. Additionally, cause-specific mortality and serious liver disease outcomes may have been subject to misclassification bias in this study. Second, potential confounding factors, such as smoking, alcohol intake, and physical activity, were derived from self-reported data and may have been affected by recall bias or underreporting. Socioeconomic factors, including educational attainment and household income, are known to correlate with the BRI and metabolic risk but were not included in the models, which may have introduced residual confounding [33]. Finally, the BRI was assessed only at baseline, precluding the evaluation of longitudinal changes and limiting causal inference regarding the relationship between the BRI and liver-related outcomes [14].

Conclusions

A higher BRI was significantly associated with an increased risk of incident SLD, MALO, liver-related mortality, and all-cause mortality. These findings suggest the clinical relevance of BRI as a simple, non-invasive surrogate marker of visceral adiposity that may serve as a valuable predictor of adverse hepatic and systemic outcomes. When considering liver disease screening and risk stratification strategies, a standardized BRI threshold value is needed. Further longitudinal and mechanistic studies across diverse populations are warranted to confirm the predictive value of BRI and guide its integration into clinical practice.

Abbreviations

BRI	Body roundness index
SLD	Steatotic liver disease
MALO	Major adverse liver-related outcomes
HRs	Hazard ratios
CI	Confidence intervals
NAFLD	Nonalcoholic fatty liver disease
WC	Waist circumference
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
Hs-CRP	High-sensitivity C-reactive protein
ICD-10	International Classification of Diseases, 10th Revision
BMI	Body mass index
APRI	AST to platelet ratio index
TyG	Triglyceride-glucose
HDL	High-density lipoprotein
SD	Standard deviation

Supplementary Information

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Supplementary Material 1.

Authors' contributions

Conceptualization: JS, SJH, YJL, YJK and JWL; methodology: JS, SJH, YJL, YJK and JWL; formal analysis: SJH and YJL; writing—original draft preparation: JS and SJH; writing—review and editing: YJL, YJK and JWL; funding acquisition: JWL. All authors have read and agreed to the published version of the manuscript.

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Data availability

The datasets generated during and/or analyzed during the current study are available in the UK Biobank Application Management System (https://www.ukbiobank.ac.uk, Application ID: 477427).

Declarations

Ethics approval and consent to participate

The UK Biobank study received ethical approval from the North West Multi-Centre Research Ethics Committee, and informed consent was obtained from all participants at the time of enrollment (https://www.ukbiobank.ac.uk/about-us/how-we-work/ethics/). For the current study, the institutional review board of Konkuk University Medical Center approved the exemption of ethical review and waived the requirement for informed consent (KUMC 2025-10-022), as the data used in this secondary analysis were fully anonymized and contained no identifiable information. All study procedures were conducted in accordance with the Declaration of Helsinki and relevant institutional guidelines and regulations.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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