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# Parallel long-term changes in body roundness index and fasting plasma glucose and their association with incident heart failure: evidence from the Kailuan study

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**Background** Body roundness index (BRI), which better captures central adiposity than the body mass index, and fasting plasma glucose (FPG) are both associated with heart failure (HF) risk, but their joint longitudinal patterns remain unclear. We aimed to identify the joint trajectories of BRI and FPG and evaluate their associations with incident HF.

**Methods** In this prospective cohort study, we enrolled 41,767 participants from the Kailuan Study with repeated measurements of BRI and FPG between 2006 and 2010. We identified joint BRI and FPG trajectories using group-based multi-trajectory modelling. We then ascertained incident HF from the 2010–2011 survey through to 31 December 2022 using medical records, administrative databases, questionnaires, and adjudication by cardiologists, and formed Cox proportional hazards models to assess the associations between joint BRI and FPG trajectories and HF risk, while adjusting for demographic, lifestyle, and clinical covariates.

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**Results** We identified four distinct dual trajectory groups: low BRI/low FPG (39.5%), moderate BRI/moderate FPG (47.3%), highest BRI/high FPG (7.8%), and high BRI/highest FPG (5.4%). A total of 747 HF cases occurred during a median follow-up of 12.1 years. Compared with the low BRI/low FPG group, multivariable-adjusted hazard ratios were 1.47 (95% confidence interval (CI) = 1.21–1.79) for the moderate BRI/moderate FPG, 2.31 (95% CI = 1.79–2.97) for the highest BRI/high FPG, and 2.90 (95% CI = 2.18–3.85) for the high BRI/highest FPG groups. Joint longitudinal trajectories of BRI and FPG group showed significant interactions with age and hypertension status in relation to incident HF. The associations were stronger associations among younger individuals and those without hypertension. Multiple sensitivity analyses confirmed the robustness of these findings.

**Conclusions** Joint longitudinal trajectories of BRI and FPG were independently and progressively associated with incident HF risk. Repeated assessment of BRI and FPG may enhance long-term HF risk stratification and support earlier targeted prevention.

**Keywords** heart failure; joint longitudinal trajectories; body roundness index; fasting plasma glucose; group-based multi-trajectory modelling

Heart failure (HF) is a major clinical and public health challenge worldwide, contributing to premature mortality, recurrent hospitalisations, and substantial healthcare costs [1]. Its burden in China has continued to rise in parallel with population ageing and rapid cardiometabolic transition [2–4]. Despite advances in medical therapy, the condition remains associated with poor long-term prognosis [5], underscoring the need to identify upstream, modifiable metabolic risk factors to aid earlier prevention. In this context, obesity and abnormal glucose metabolism were found to be closely related to HF risk [6,7]. However, overall adiposity alone may not fully capture metabolic risk relevant to HF, as fat distribution appears to be particularly important [8,9].

While it is the most used measure of adiposity, the body mass index (BMI) cannot distinguish fat mass from lean mass and does not directly assess abdominal obesity or visceral adiposity. In contrast, the body roundness index (BRI), derived from waist circumference and height, was developed as a geometric indicator of body shape and central adiposity. Unlike the BMI, which reflects overall body mass without distinguishing fat distribution, it incorporates waist circumference and may, therefore, better capture abdominal fat accumulation and its associated metabolic burden [10]. Parallel to adiposity, elevated fasting plasma glucose (FPG), even below the diagnostic threshold for diabetes, has been independently associated with incident HF [11]. Given the strong metabolic coupling between central adiposity and glucose regulation, their combined contribution to HF risk may exceed the effect of either factor alone.

Adiposity and dysglycaemia may jointly contribute to HF through insulin resistance [12], adipose tissue inflammation [13], oxidative stress [14], and neurohormonal activation [15]. Prior studies have demonstrated that single baseline or long-term trajectories of BMI, BRI, or FPG measures are more strongly associated with cardiovascular outcomes than baseline values alone [16–19]. Nevertheless, existing trajectory research has largely focused on individual risk factors in isolation. Therefore, whether joint long-term trajectories of BRI and FPG identify subgroups with different risks of incident HF remains unclear. To address this limitation, we aimed to identify joint longitudinal trajectories of BRI and FPG from 2006 to 2010 and evaluate their associations with incident HF during subsequent follow-up through 2022.

## METHODS

We used data from the Kailuan Study (ChiCTR-TNRC-11001489), a community-based prospective cohort initiated in Tangshan, China [20]. We report our findings according to the STROBE guidelines [21]. Between June 2006 and October 2007, the Kailuan Study enrolled 101,510 adults aged 18–98 years from the Kailuan community, who subsequently participated in biennial health

assessments [20]. Because the exposure of interest was the joint longitudinal trajectory of BRI and FPG, rather than a single baseline measurement, we set the period from 2006 to 2010 as the trajectory assessment window. Eligible participants were required to have attended three consecutive health examinations during the trajectory assessment period. We excluded participants who developed HF or died before the 2010–2011 examination and those with insufficient BRI or FPG measurements for trajectory modelling. Follow-up for incident HF began at the 2010–2011 survey, after completion of the 2006–2010 trajectory assessment period, to ensure that trajectories of BRI and FPG preceded outcome ascertainment, and ended at the occurrence of incident HF, death, or 31 December 2022 (the endpoint of the study), whichever came first.

## BRI measurements

Trained staff collected anthropometric measurements, including height, weight, and waist circumference (WC), according to standardised procedures. Height and WC were recorded to the nearest 0.1 cm, and weight to the nearest 0.1 kg, with participants wearing light clothing and no shoes during measurement. Standing height was measured using a stadiometer, and weight was assessed with calibrated platform scales. Waist circumference measurement was obtained at the midpoint between the lowest rib and iliac crest using a non-stretchable tape. Using these measures, we calculated the BRI according to the formula developed by Thomas *et al.* [22]:

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

## Determination of fasting plasma glucose

Following an eight-hour minimum overnight fast, venous blood specimens were drawn from the antecubital vein. FPG concentration was quantified *via* the hexokinase/glucose-6-phosphate dehydrogenase enzymatic method using automated analysis (Hitachi 747, Tokyo, Japan). Sample processing occurred within four hours at the Kailuan General Hospital's centralised laboratory.

## Ascertainment of outcomes

Consistent with previous studies [23], incident HF constituted the primary endpoint, identified through four integrated surveillance mechanisms: Municipal Social Insurance Institution databases, which encompassed all participants; Hospital Discharge Register networks; mortality records; and biennial participant questionnaires. HF events were identified using ICD-10 codes recorded on the hospital medical record cover sheets (I50 and all its subcodes). An independent adjudication panel consisting of experienced cardiologists blinded to the study design reviewed relevant medical records to verify suspected HF cases.

## Covariate data collection

We selected covariates (demographic characteristics, lifestyle factors, and clinical information) *a priori* based on previous literature [24], clinical relevance, and their potential roles as confounders of the associations between metabolic trajectories and HF risk. We gathered demographic and behavioural information on age, sex, education level, marital status, smoking, drinking, physical activity, and salt habits *via* standardised questionnaires during the 2010–2011 assessment [24,25].

We categorised the participants' educational status as illiterate/elementary, middle school, or vocational/high school and beyond and marital status as married/remarried or other. Participants were classified as smokers if they reported smoking cigarettes daily throughout the preceding year and as drinkers if they reported drinking alcohol at least once a month over the preceding year [26]. We categorised physical activity as none, occasional (<80 minutes/week), or frequent

(≥80 minutes/week) and salt intake as high, medium, or low based on participant reporting [27]. The BMI was computed as weight in kilograms divided by squared height in meters. Blood pressure measurements employed a calibrated mercury sphygmomanometer following a minimum five-minute seated rest period. Two readings were averaged; discrepancies exceeding 5 mm Hg prompted a third measurement with three-reading averaging. Medical history and medication usage were assessed per participants' self-reporting and medical record reviews. We defined hypertension as a systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg, antihypertensive drug usage, or self-reported history of physician diagnosed hypertension [28], and diabetes as a fasting blood glucose ≥7.0 mmol/L or self-reported history of diabetes or use of antihyperglycemic agents [29].

Blood samples were collected as described above. Low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), creatinine, and high-sensitivity C-reactive protein (hs-CRP) were measured using an automated analyser (Hitachi 747; Hitachi, Tokyo, Japan). We calculated the estimated glomerular filtration rate (eGFR) using the Chronic Kidney Disease Epidemiology Collaboration creatinine equation [30].

## Statistical analysis

We applied group-based multi-trajectory modelling to identify joint patterns of BRI and FPG across the 2006–2010 period. As seen from previous research [24,25], this method identifies subgroups of individuals exhibiting similar joint trajectories across two indicators over time. Model estimation started with a two-group solution and sequentially increased to three, four, and five groups, while we tested alternative polynomial orders (linear, quadratic, and cubic) to determine the optimal trajectory forms. We selected the optimal model based on multiple criteria [26,31]: minimum group membership exceeding 5% of total participants; the lowest Bayesian information criterion (BIC); logged Bayes factor (approximating 2ΔBIC) exceeding 6; mean posterior probability for group classification surpassing 0.7, demonstrating acceptable classification precision; and clinical interpretability and practical significance of extracted trajectories.

For the baseline description, we expressed continuous variables with a normal distribution as means and standard deviations, and those that were non-normally distributed as medians and interquartile ranges. We summarised categorical variables as frequencies and percentages. We compared trajectory groups using analysis of variance for normally distributed variables, the Kruskal–Wallis test for non-normally distributed variables, and the chi-squared test for categorical variables.

We calculated HF incidence rates per 1,000 person-years by trajectory group. Then, we used the Kaplan–Meier method to estimate cumulative incidence and the log-rank test to assess differences between groups. Additionally, we used Cox proportional hazards models to estimate hazard ratios (HRs) and corresponding 95% confidence intervals (CIs) of incident HF in each trajectory groups, using the low BRI/low FPG group as the reference. We assessed the proportional hazards assumption using Schoenfeld residuals and found no meaningful violations. We also used adjusted models, with model 1 adjusted for age, sex, education background, marital status, smoking status, drinking status, physical activities, salt intake habits; model 2 further adjusted for LDL-C, HDL-C, the natural logarithms eGFR and hs-CRP; and model 3 further adjusted for hypertension, using antihypertensive medication, using antidiabetic medication, and using lipid-lowering medication. We performed stratified analyses by age (<50, 50–59, and ≥60 years), sex, hypertension, and diabetes mellitus to assess whether the influence of BRI and FPG dual trajectories on incident HF varied across specific factors. We employed likelihood ratio tests to evaluate interactions between stratified variables and multi-trajectories.

Lastly, we conducted multiple sensitivity analyses to test our results for robustness. First, we performed additional adjustment for BMI to evaluate independence from general adiposity. Second, we conducted separately adjusted analyses for BRI and FPG measured at two time points, i.e. in 2006–2007 and in 2010–2011, respectively, to assess independence from single-time point meas-

urements. We handled missing covariates using multiple imputation *via* fully conditional specification, applying discriminant analysis for categorical variables and linear regression for continuous variables, with results compared against complete-case analyses. Third, we conducted analyses after excluding medication users to minimise treatment-related confounding. Fourth, we excluded HF events occurring within the first two years of follow-up to reduce potential reverse causation. Fifth, we applied cause-specific hazard models treating death as a competing event. Lastly, we excluded participants with hs-CRP>10 mg/L to mitigate potential bias from acute inflammation.

We conducted all analyses in SAS, version 9.4 (SAS Institute Inc., Cary, North Carolina, USA), with 'PROC TRAJ' used to fit group-based multi-trajectory models and identify joint BRI and FPG trajectories from 2006 to 2010. The statistical significance threshold was set at a two-tailed $P < 0.05$.

## RESULTS

We excluded 2,654 participants who developed HF or died before the 2010–2011 examination and 13,506 with insufficient BRI or FPG measurements for trajectory modelling. The final analysis included 41,767 participants with a mean age of 52.9 years (standard deviation = 11.8) who were alive and free of HF at the 2010–2011 examination (Figure 1). Among them, we identified four distinct dual trajectories from all investigated models (Figure 2): the low BRI/low FPG group

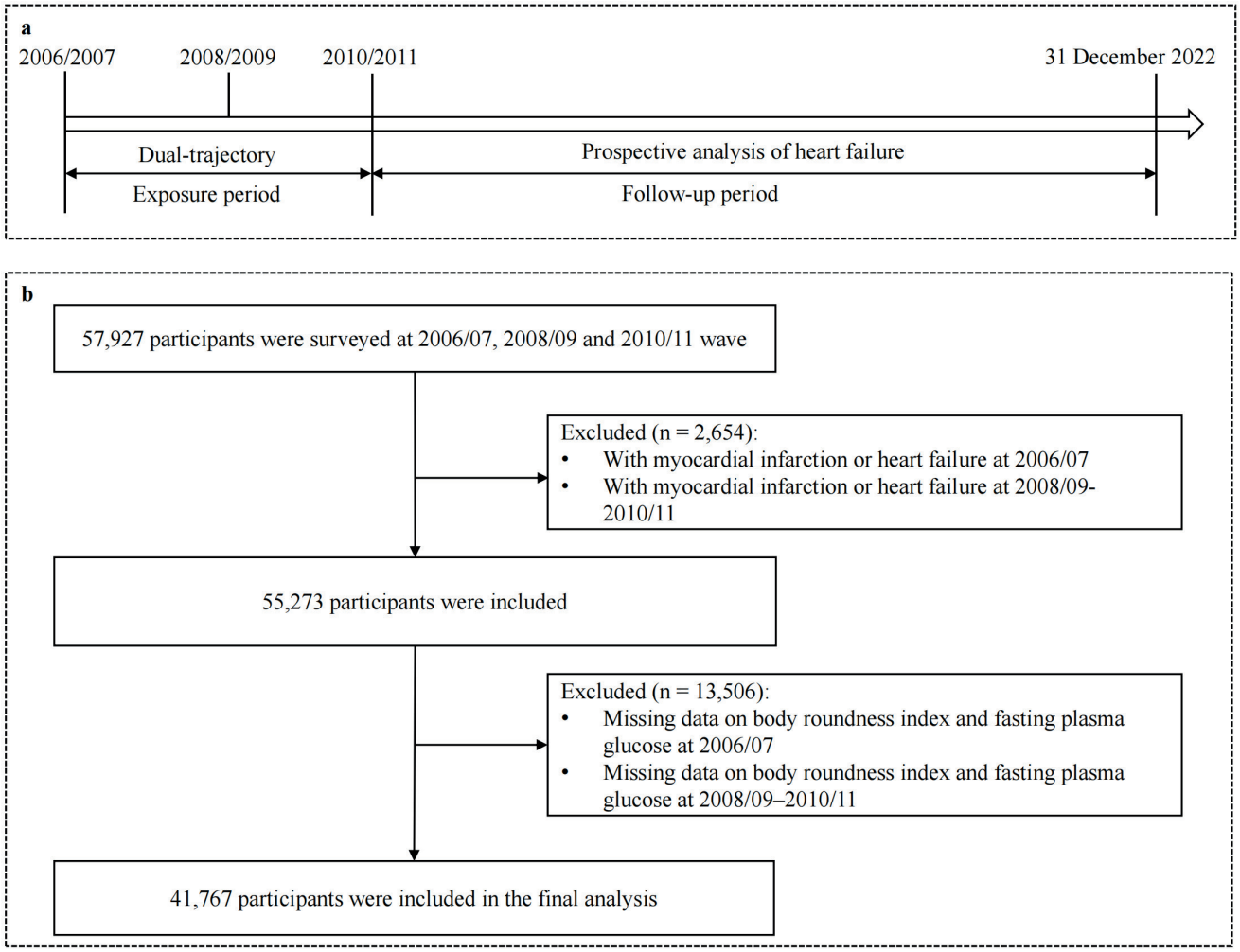



**Figure 1. Panel A.** Study design for examining the association between joint trajectories group and heart failure risk. **Panel B.** Flowchart of the participant selection process.

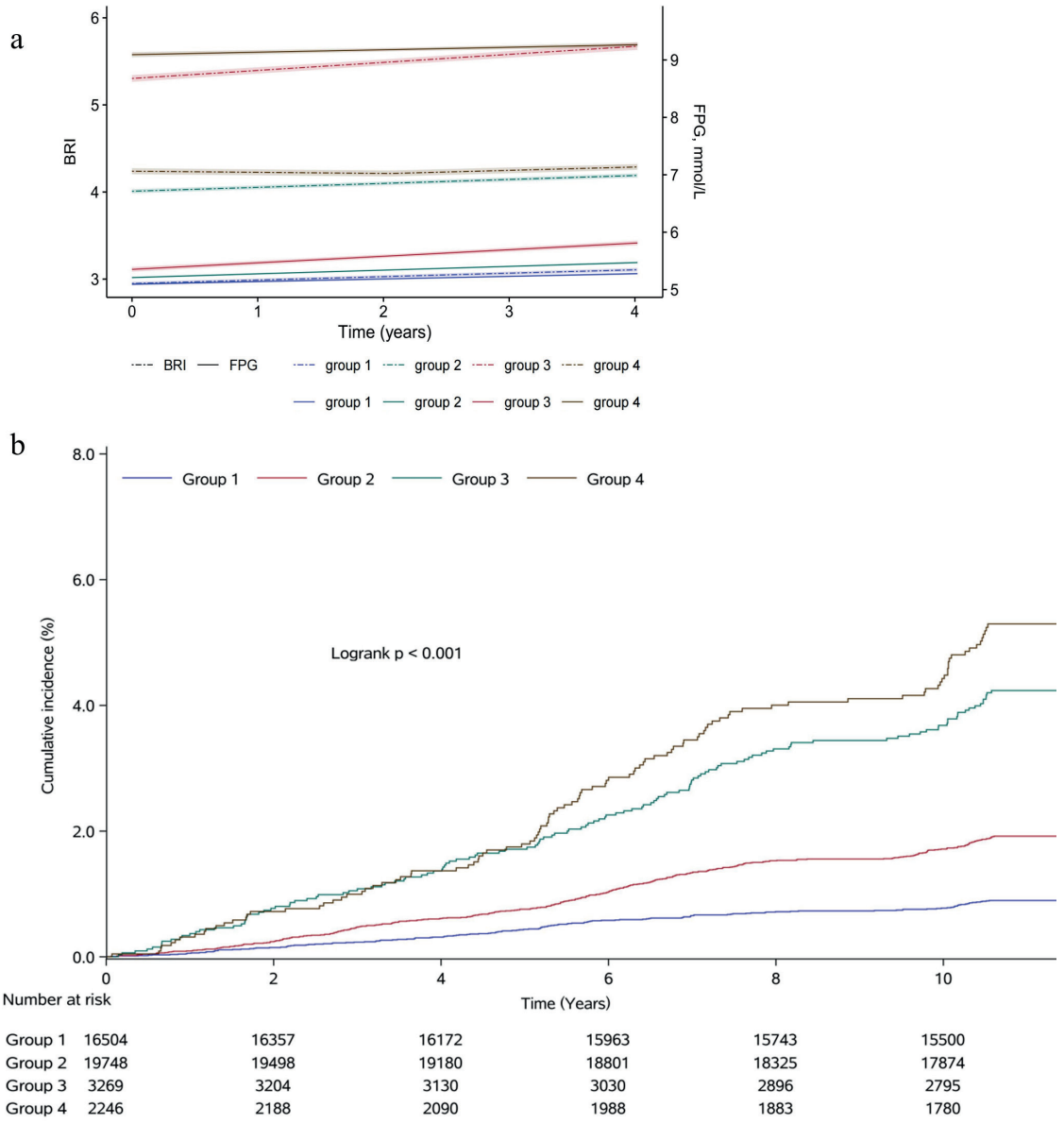



**Figure 2.** Joint trajectories of body roundness index and fasting plasma glucose and their association with HF risk. Group 1 (39.5%): low BRI/low FPG group. Group 2 (47.3%): moderate BRI/moderate FPG group. Group 3 (7.8%): highest BRI/high FPG group. Group 4 (5.4%): high BRI/highest FPG group. **Panel A.** Dual-trajectory plot illustrating the longitudinal changes in BRI (left y-axis) and FPG (right y-axis) over four years across four identified trajectory groups. Both BRI and FPG showed gradual increases over time, with higher baseline and follow-up levels observed in higher-risk trajectory groups. **Panel B.** Cumulative incidence curves of HF across the four joint trajectory groups during a 12-year follow-up. Participants in the higher trajectory BRI and FPG groups exhibited significantly increased HF risk compared with those in lower trajectory groups (log-rank $P < 0.001$). BRI – body roundness index, FPG – fasting plasma glucose, HF – heart failure.

(n = 16,504, 39.5%), moderate BRI/moderate FPG group (n = 19,748, 47.3%), highest BRI/high FPG group (n = 3,269, 7.8%), and high BRI/highest FPG group (n = 2,246, 5.4%). We compared alternative two-, three-, four-, and five-group solutions and selected the four-group model based on BIC, logged Bayes factor, minimum group size, average posterior probability, and clinical interpretability (Table S1 in the **Online Supplementary Document**). Although the five-group model had a slightly improved BIC, it generated a small group comprising fewer than 5% of participants and offered limited additional clinical interpretability. In terms of baseline characteristics (**Table 1**), participants in the high BRI/highest FPG group were older, more likely to be male, and more frequently used hypoglycaemic and lipid-lowering medications compared with those in the other groups.

**Table 1.** Participant baseline characteristics across combined trajectory groups of body roundness index and fasting plasma glucose*

| | Group 1 (n = 16,504) | Group 2 (n = 19,748) | Group 3 (n = 3,269) | Group 4 (n = 2,246) |
|---|---|---|---|---|
| **Age in years** | 49.8 (11.8) | 54.1 (11.3) | 57.9 (11.6) | 58.2 (9.8) |
| **Male, n (%)** | 12,775 (77.4) | 16,011 (81.1) | 2,331 (71.3) | 1,852 (82.5) |
| **Education background, n (%)** | | | | |
| Illiterate/elementary | 906 (5.5) | 1,536 (7.8) | 417 (12.8) | 226 (10.1) |
| Middle school | 10,662 (64.6) | 14,030 (71.0) | 2,366 (72.4) | 1,687 (75.1) |
| Vocational/high school and above | 4,936 (29.9) | 4,182 (21.2) | 486 (14.9) | 333 (14.8) |
| **Married or remarried, n (%)** | 16,238 (98.4) | 19,557 (99.0) | 3,236 (99.0) | 2,226 (99.1) |
| **Current smoking, n (%)** | 6,059 (36.7) | 6,837 (34.6) | 941 (28.8) | 727 (32.4) |
| **Current drinking, n (%)** | 5,917 (35.9) | 7,154 (36.2) | 965 (29.5) | 657 (29.3) |
| **Physical activities, n (%)** | | | | |
| No | 5,298 (32.1) | 6,020 (30.5) | 1,041 (31.8) | 626 (27.9) |
| Occasionally | 9,095 (55.1) | 10,945 (55.5) | 1,743 (53.3) | 1,278 (56.9) |
| Frequently | 2,111 (12.8) | 2,774 (14.0) | 486 (14.8) | 342 (15.2) |
| **Salt intake habits, n (%)** | | | | |
| High | 1,677 (10.2) | 2,121 (10.7) | 393 (12.0) | 237 (10.6) |
| Medium | 11,696 (70.9) | 14,348 (72.7) | 2,376 (72.7) | 1,620 (72.1) |
| Low | 3,131 (19.0) | 3,279 (16.6) | 500 (15.3) | 389 (17.3) |
| **BMI in kg/m$^2$** | 22.9 (2.4) | 26.1 (2.6) | 29.5 (3.0) | 26.0 (3.1) |
| **SBP in mm Hg** | 125.2 (17.1) | 133.2 (17.9) | 139.7 (18.2) | 139.8 (19.5) |
| **DBP in mm Hg** | 81.9 (10.0) | 85.8 (10.2) | 88.3 (10.5) | 86.6 (10.5) |
| **LDL-C in mmol/L** | 2.5 (0.7) | 2.6 (0.8) | 2.7 (0.9) | 2.7 (0.8) |
| **HDL-C in mmol/L** | 1.6 (0.4) | 1.5 (0.4) | 1.4 (0.4) | 1.5 (0.4) |
| **Hs-CRP in mg/L, Mdn (IQR)** | 0.8 (0.3–1.7) | 1.2 (0.6–2.8) | 1.9 (0.9–4.0) | 1.4 (0.7–3.2) |
| **eGFR in ml/min/1.73 m$^2$, Mdn (IQR)** | 93.4 (76.4–105.9) | 89.5 (74.2–101.9) | 88.0 (73.5–99.6) | 87.2 (71.6–99.5) |
| **Hypertension, n (%)** | 5,678 (34.4) | 10,740 (54.4) | 2,335 (71.4) | 1,501 (66.8) |
| **Medication use, n (%)** | | | | |
| Antihypertensive | 867 (5.3) | 2,490 (12.6) | 778 (23.8) | 462 (20.6) |
| Antidiabetic | 72 (0.4) | 272 (1.4) | 136 (4.2) | 807 (35.9) |
| Lipid-lowering | 65 (0.4) | 195 (1.0) | 54 (1.7) | 60 (2.7) |
| **FPG_2006 in mmol/L** | 5.1 (0.7) | 5.2 (0.8) | 5.4 (0.9) | 9.0 (2.4) |
| **FPG_2008 in mmol/L** | 5.2 (0.7) | 5.4 (0.8) | 5.6 (1.0) | 9.4 (2.2) |
| **FPG_2010 in mmol/L** | 5.2 (0.7) | 5.5 (0.8) | 5.8 (1.1) | 9.2 (2.3) |
| **BRI_2006** | 2.9 (0.7) | 4.1 (0.8) | 5.4 (0.9) | 4.2 (1.0) |
| **BRI_2008** | 3.0 (0.6) | 4.1 (0.8) | 5.5 (1.0) | 4.2 (1.1) |
| **BRI_2010** | 3.0 (0.7) | 4.3 (0.8) | 5.8 (1.0) | 4.3 (1.1) |

BMI – body mass index, BRI – body roundness index, DBP – diastolic blood pressure, eGFR – estimated glomerular filtration rate, FPG – fasting plasma glucose, HDL-C – high-density lipoprotein cholesterol, Hs-CRP – high-sensitivity C-reactive protein, IQR – interquartile range, LDL-C – low-density lipoprotein cholesterol, Mdn – median, SBP – systolic blood pressure

*Presented as mean (standard deviation) unless specified otherwise. Group 1: low BRI/low FPG group. Group 2: moderate BRI/moderate FPG group. Group 3: highest BRI/high FPG group. Group 4: high BRI/highest FPG group.

## Dual trajectory patterns of BRI combined with FPG and HF

We documented 747 cases of incident HF during a median follow-up of 12.1 years. The proportions of participants who developed HF across the low BRI/low FPG, moderate BRI/moderate FPG, highest BRI/high FPG, and high BRI/highest FPG groups were 0.9%, 1.8%, 4.0%, and 4.8%, respectively, corresponding to incidence rates of 0.75, 1.62, 3.63, and 4.54 per 1,000 person-years (**Table 2**). The Kaplan–Meier analysis further showed a significantly increased risk of HF in high BRI/highest FPG group compared with all other groups (log-rank $P < 0.001$) (**Figure 2**). After adjustment for potential confounders in model 3, all other groups had significantly higher risks of HF compared with low BRI/low FPG group, with adjusted HRs being 1.47 (95% CI = 1.21–1.79) for the moderate BRI/moderate FPG group, 2.31 (95% CI = 1.79–2.97) for the highest BRI/high FPG group, and 2.90 (95% CI = 2.18–3.85) for the high BRI/highest FPG group.

**Table 2.** Association of joint trajectory groups of body roundness index and fasting plasma glucose and incident heart failure*

| | Group 1 (n/N = 144/16,504) | Group 2 (n/N = 364/19,748) | Group 3 (n/N = 131/3,269) | Group 4 (n/N = 108/2,246) |
|---|---|---|---|---|
| **Incidence rate per 1,000 person-years, HR (95% CI)** | 0.75 (0.64–0.88) | 1.62 (1.46–1.79) | 3.63 (3.06–4.31) | 4.54 (3.76–5.48) |
| **Model 1** | ref | 1.64 (1.35–1.99) | 2.84 (2.23–3.61) | 3.70 (2.88–4.76) |
| **Model 2** | ref | 1.59 (1.30–1.93) | 2.68 (2.09–3.44) | 3.52 (2.73–4.54) |
| **Model 3** | ref | 1.47 (1.21–1.79) | 2.31 (1.79–2.97) | 2.90 (2.18–3.85) |

BRI – body roundness index, CI – confidence interval, FPG – fasting plasma glucose, HR – hazard ratio, ref – reference
*Group 1: low BRI/low FPG group. Group 2: moderate BRI/moderate FPG group. Group 3: highest BRI/high FPG group. Group 4: high BRI/highest FPG group. Model 1 was adjusted for age, sex, education background, marital status, smoking status, drinking status, physical activities, salt intake habits. Model 2 included covariates in model 1 plus LDL-C, HDL-C, ln eGFR, and ln hs-CRP. Model 3 included covariates in model 2 plus hypertension, using antihypertensive medication, using antidiabetic medication, and using lipid-lowering medication.

## Subgroup and sensitivity analysis

We examined potential effect modification by age, sex, hypertension status, and diabetes status (**Table 3**) and found a significant interaction between dual trajectory groups and age (*P*-value for interaction = 0.006), suggesting that the association between BRI combined with FPG of dual trajectories and HF risk varied across age strata. Specifically, among participants aged <50 years, the high BRI/highest FPG group had a higher risk of HF compared with the low BRI/low FPG group (HR = 5.27; 95% CI = 1.99–13.94). We observed similar patterns among participants aged 50–59 and ≥60 years, with the corresponding HRs of 4.09 (95% CI = 2.49–6.71) and 1.97 (95% CI = 1.35–2.86), respectively. The association between joint BRI and FPG trajectory group and incident HF differed by hypertension status (*P*-value for interaction = 0.007), with stronger associations among individuals without hypertension. In contrast, we observed no significant interactions for sex (*P*-value for interaction = 0.258) or diabetes status (*P*-value for interaction = 0.426), indicating that the associations were consistent across these subgroups. The results of the primary analysis remained consistent across all sensitivity analyses (Tables S2–10 in the **Online Supplementary Document**).

# DISCUSSION

In this large prospective community-based study, distinct joint longitudinal trajectories of BRI and FPG were associated with incident HF risk over more than a decade of follow-up. Participants with persistently higher BRI and FPG exhibited progressively greater HF incidence rates and adjusted HRs compared with participants with low BRI/low FPG. These associations were stronger among younger participants and those without hypertension, suggesting that adverse joint metabolic trajectories may identify high-risk subgroups at earlier stages of risk accumulation of established cardiovascular risk conditions. The findings were robust across multiple sensitivity analyses, supporting the relevance of long-term combined metabolic patterns as markers of HF risk.

Our findings extend prior work that has largely evaluated BRI indices or glycaemic status in isolation, often using single baseline measurements [11,18,19]. Although longitudinal trajectories of BRI or FPG have each been linked to increased risks of HF or other cardiovascular outcomes [32,33] studies examining their joint longitudinal patterns in relation to HF remain limited. Here, we examined the associations between these combined trajectories and HF risk in a large prospective cohort, allowing for a more integrated characterisation of metabolic patterns associated with HF risk. However, unlike in our previous Kailuan analysis of adiposity–glucose trajectories and overall cardiovascular diseases [26], here we focused specifically on incident HF, a distinct outcome involving myocardial remodelling, metabolic dysfunction, renal–cardiac interactions, and neurohormonal activation. This provides HF-specific evidence on the joint contribution of long-term BRI and FPG trajectories to HF risk. The observed associations are consistent with evidence showing that adiposity and dysglycaemia are associated with metabolic and inflammatory processes relevant to HF [6]. Both conditions have been associated with systemic inflammation,

**Table 3.** Stratified analysis of the association between joint trajectory groups of body roundness index and fasting plasma glucose and incident heart failure, HR (95% CI)*

| | Group 1 | Group 2 | Group 3 | Group 4 |
|---|---|---|---|---|
| **Age in years** | | | | |
| <50 | | | | |
| *n/N* | 15/8,487 | 44/6,958 | 11/770 | 10/453 |
| *Incidence rate per 1000 person-years* | 0.15 (0.09–0.24) | 0.53 (0.39–0.71) | 1.21 (0.67–2.18) | 1.91 (1.03–3.54) |
| *Model* | ref | 2.57 (1.40–4.70) | 4.68 (2.06–10.63) | 5.27 (1.99–13.94) |
| 50–59 | | | | |
| *n/N* | 43/5,091 | 108/7,252 | 35/1,118 | 38/900 |
| *Incidence rate per 1000 person-years* | 0.73 (0.54–0.98) | 1.28 (1.06–1.55) | 2.71 (1.95–3.78) | 3.79 (2.76–5.21) |
| *Model* | ref | 1.62 (1.13–2.32) | 3.03 (1.89–4.86) | 4.09 (2.49–6.71) |
| ≥60 | | | | |
| *n/N* | 86/2,926 | 212/5,538 | 85/1,381 | 60/893 |
| *Incidence rate per 1000 person-years* | 2.78 (2.25–3.44) | 3.67 (3.20–4.19) | 6.03 (4.88–7.46) | 7.04 (5.47–9.07) |
| *Model* | ref | 1.21 (0.93–1.56) | 1.83 (1.33–2.51) | 1.97 (1.35–2.86) |
| *P*-value for interaction | | 0.006 | | |
| **Sex** | | | | |
| Male | | | | |
| *n/N* | 124/12,775 | 314/16,011 | 92/2,331 | 85/1,852 |
| *Incidence rate per 1000 person-years* | 0.84 (0.70–1.00) | 1.73 (1.55–1.93) | 3.61 (2.94–4.43) | 4.37 (3.53–5.40) |
| *Model* | ref | 1.50 (1.21–1.85) | 2.20 (1.65–2.92) | 2.83 (2.07–3.86) |
| Female | | | | |
| *n/N* | 20/3,729 | 50/3,737 | 39/938 | 23/394 |
| *Incidence rate per 1000 person-years* | 0.45 (0.29–0.70) | 1.15 (0.87–1.51) | 3.67 (2.68–5.03) | 5.32 (3.53–8.00) |
| *Model* | ref | 1.26 (0.73–2.17) | 2.54 (1.39–4.62) | 3.24 (1.58–6.65) |
| *P*-value for interaction | | 0.258 | | |
| **Hypertension** | | | | |
| Yes | | | | |
| *n/N* | 98/5,678 | 251/10,740 | 111/2,335 | 78/1,501 |
| *Incidence rate per 1000 person-years* | 1.52 (1.25–1.86) | 2.08 (1.84–2.36) | 4.39 (3.64–5.28) | 4.99 (3.99–6.23) |
| *Model* | ref | 1.17 (0.92–1.48) | 1.94 (1.46–2.59) | 2.37 (1.70–3.30) |
| No | | | | |
| *n/N* | 46/10,826 | 113/9,008 | 20/934 | 30/745 |
| *Incidence rate per 1000 person-years* | 0.36 (0.27–0.48) | 1.08 (0.90–1.30) | 1.85 (1.20–2.87) | 3.68 (2.57–5.26) |
| *Model* | ref | 2.13 (1.50–3.03) | 2.67 (1.55–4.61) | 3.57 (2.02–6.30) |
| *P*-value for interaction | | 0.007 | | |
| **Diabetes** | | | | |
| Yes | | | | |
| *n/N* | 9/464 | 47/1,421 | 30/580 | 107/2,116 |
| *Incidence rate per 1000 person-years* | 1.73 (0.90–3.32) | 3.01 (2.26–4.01) | 4.85 (3.39–6.94) | 4.78 (3.96–5.78) |
| *Model* | ref | 1.52 (0.74–3.11) | 2.05 (0.96–4.41) | 2.21 (1.11–4.41) |
| No | | | | |
| *n/N* | 135/16,040 | 317/18,327 | 101/2,689 | 1/130 |
| *Incidence rate per 1000 person-years* | 0.72 (0.61–0.86) | 1.51 (1.36–1.69) | 3.38 (2.78–4.10) | 0.71 (0.10–5.02) |
| *Model* | ref | 1.39 (1.13–1.71) | 2.16 (1.64–2.85) | 0.58 (0.08–4.13) |
| *P*-value for interaction | | 0.426 | | |

BRI – body roundness index, CI – confidence interval, FPG – fasting plasma glucose, HR – hazard ratio
*Group 1: low BRI and low FPG group. Group 2: moderate BRI and moderate FPG group. Group 3: highest BRI and high FPG group. Group 4: high BRI and highest FPG group. Model 1 was adjusted for age, sex, education background, marital status, smoking status, drinking status, physical activities, salt intake habits. Model 2 included covariates in model 1 plus LDL-C, HDL-C, ln eGFR, and ln hs-CRP. Model 3 included covariates in model 2 plus hypertension, using antihypertensive medication, using antidiabetic medication, and using lipid-lowering medication. Here, n denotes the number of events and N the total number of participants in the corresponding group.

metabolic stress, and adverse cardiac remodelling [34]. The coexistence and persistence of these exposures over time may, therefore, be relevant to HF susceptibility [35].

The stronger relative associations observed in younger participants (<50 years) may reflect the lower baseline incidence of HF at younger ages, allowing differences related to metabolic trajectories to be more easily observed [36]. In addition, an adverse joint metabolic trajectory emerg-

ing earlier in adulthood may indicate a longer duration of cumulative cardiometabolic exposure, which has been associated with higher cardiovascular risk [37,38]. Similarly, the stronger associations observed among individuals without hypertension may indicate that joint metabolic trajectories capture metabolic risk relevant to HF that is not fully reflected by blood pressure status alone [39,40]. Our findings suggest that combined adiposity–glycaemia trajectories may be particularly informative for risk stratification among younger individuals and those without established hypertension, at earlier stages before the development of overt HF.

The joint association of BRI and FPG trajectories with HF risk may be explained by several interrelated biological pathways. Higher BRI reflects greater central adiposity, which is metabolically active and associated with insulin resistance [12], adipose tissue inflammation [13], oxidative stress [14], and neurohormonal activation [15]. These processes may promote endothelial dysfunction, renal sodium retention, increased circulating volume, myocardial fibrosis, and ventricular remodelling. Elevated FPG reflects impaired glucose regulation and may further contribute to HF through glucotoxicity, microvascular dysfunction, mitochondrial injury, and adverse myocardial substrate utilisation [41]. When central adiposity and dysglycaemia coexist and persist over time, they may reinforce each other through a vicious cycle of insulin resistance, chronic inflammation, and metabolic stress, thereby accelerating structural and functional myocardial changes that predispose to HF [42]. This may explain why participants with persistently higher BRI and FPG had the greatest HF risk in our study.

From a public health perspective, repeated assessment of BRI and FPG helps identify long-term adverse metabolic profiles in routine health examinations, because waist circumference, height, and FPG are simple and widely available measures [43]. Although these findings do not establish the superiority of trajectory-based monitoring over conventional risk indicators, they suggest that persistently adverse profiles of BRI and FPG may help flag individuals who warrant closer metabolic risk assessment. This may be particularly relevant for younger adults and those without hypertension, in whom long-term HF risk may be less apparent using traditional clinical markers alone. This trajectory-based approach may ultimately help shift the focus from reactive late-stage treatment to proactive prevention [5,44].

This study has several strengths. First, the large sample size, long-term follow-up and prospective cohort design allowed for longitudinal analyses of joint metabolic trajectories. Second, we examined joint longitudinal trajectories of BRI and FPG, allowing us to assess their combined associations with HF risk. Third, the person-oriented approach allowed identification of latent trajectory groups based on longitudinal data. This approach characterises individual-level patterns over time that are not captured by traditional variable-based analyses.

However, several limitations should also be acknowledged. First, trajectory modelling is data-driven; the number and shape of groups depend on model selection and cohort-specific distributions, and trajectories may not be directly generalisable to populations with different demographic or clinical characteristics. Second, although we adjusted for baseline cardiometabolic factors and medication use and performed sensitivity analyses excluding baseline medication users, time-varying medication use during follow-up was not consistently available. Therefore, treatment changes and time-varying confounding could not be fully accounted for. Third, we modelled BRI continuously in data-driven trajectory analyses, rather than classifying it by predefined cutoff points, minimising misclassification from external thresholds. Nevertheless, population-specific validation of BRI thresholds and their clinical interpretation in Chinese adults remains warranted. Fourth, BRI is a surrogate of fat distribution, rather than a direct measure of visceral adipose tissue, and HF subtypes (*e.g.* HF with preserved ejection fraction *vs*. HF with reduced ejection fraction) may not be fully differentiated depending on outcome ascertainment. Fifth, we focused on risk association, rather than prediction model development; therefore, we did not assess whether BRI and FPG trajectories improve discrimination, calibration, or reclassification beyond established indicators such as BMI, waist circumference, diabetes status, or baseline FPG. Finally, the predominantly male sample drawn from an industrial community in

northern China limits the generalisability of our findings, while the lack of consistent echocardiographic data precluded distinction between HF phenotypes. Further studies in more diverse populations with HF subtype information are needed to address this issue.

## CONCLUSIONS

Distinct joint longitudinal trajectories of BRI and FPG were associated with substantially different long-term risks of incident HF in a large Chinese cohort. Persistently elevated BRI and FPG trajectories were associated with the greatest HF risk, with particularly strong relative associations in younger adults and those without hypertension. These findings underscore the value of longitudinal, integrated metabolic surveillance and support earlier, multifaceted prevention strategies targeting both central adiposity and glycaemic dysregulation across midlife.

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**Ethics statement:** The protocol for this study was approved by the Ethics Committee of Kailuan General Hospital (no. 2006-5). All procedures followed the Declaration of Helsinki.

**Data availability:** The original contributions generated for the study are included in the article/supplementary materials. Further data will be made available by the corresponding author upon request.

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**Additional material**
Online Supplementary Document

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