

Body roundness index, frailty, and stroke

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Stroke remains a leading global cause of death and disability. Primary prevention efforts to reduce the burden of stroke should focus on the management of modifiable risk factors, and 10 such factors were noted to account for approximately 90% of the population-attributable risk of stroke in the INTERSTROKE study.¹ These risk factors are often inter-related, with obesity contributing to major cardiovascular risk factors including hypertension, diabetes mellitus, and dyslipidaemia. A meta-analysis of prospective cohorts found that metabolically unhealthy phenotypes carry roughly double the stroke risk at any body weight, whereas metabolically healthy obesity confers only a modest excess (risk ratio of 1.17), suggesting that much of obesity-related stroke risk is transmitted through metabolic intermediaries, with a smaller direct contribution.² In INTERSTROKE, abdominal obesity defined by waist-to-hip ratio, rather than body mass index (BMI) carried an independent population-attributable risk of 18.6%,¹ and central adiposity has been linked to greater stroke severity and poorer early functional outcome in a single-centre prospective cohort,³ which may partly explain the obesity paradox previously described for BMI. This has been attributed to the detrimental effects of visceral fat accumulation in driving systemic inflammation, insulin resistance, and cardiovascular risk. The body roundness index (BRI) was introduced by Thomas et al. in 2013 to quantify abdominal adiposity and overall body fat by integrating the geometric relationship between waist circumference and height, with subsequent research validating its relationship with visceral adiposity and metabolic syndrome.^{4,5}

As populations age globally, growing attention has focused on frailty—characterised as a clinical syndrome of excess vulnerability to morbidity and mortality, owing to progressive decline in physiological reserves. Cross-sectional analyses of prevalent stroke in 2 nationally representative datasets—National Health and Nutrition Examination Survey (NHANES) in the United States and the Health and Retirement Longitudinal Study (CHARLS) in China—showed graded associations between a deficit-accumulation frailty index and stroke (odds ratio [OR] 2.90 and 1.78 per 0.1-unit increase, respectively).⁶ Beyond its

association with stroke risk, pre-stroke frailty influences treatment tolerance, complications, rehabilitation, and recovery.

Prior NHANES analyses had separately evaluated the relationship between BRI and prevalent stroke, and between BRI and prevalent frailty.^{7,8} In their current cross-sectional analysis of 36,324 adults from NHANES 1999–2023 (1236 with self-reported stroke; weighted prevalence 2.52%), Hu and colleagues extended prior work using the NHANES dataset, examining BRI estimates under progressively expanded covariate adjustment, and the effect of including frailty as measured using a 43-item deficit-accumulation frailty index (FI) on the BRI-stroke association.⁹ In this updated analysis, BRI was positively associated with self-reported stroke (OR 1.08, 95% confidence interval (CI) 1.05–1.12) in the primary model adjusted for age, sex, smoking, poverty-income ratio, education, physical activity, and energy intake. The restricted cubic spline curve supported the non-linear association between BRI and self-reported stroke, rising across lower-to-middle BRI values, before appearing to plateau at higher values.

Unadjusted threshold analysis further suggested a BRI breakpoint of 6.34 (bootstrap 95% CI 6.14–7.65), below which higher BRI values associated with increased odds of self-reported stroke, although the authors caution interpretation of the breakpoint as a clinical threshold owing to the sparsely populated upper range and lack of validation. The BRI association attenuated to the null (OR 1.01, 95% CI 0.98–1.04) once hypertension, diabetes and dyslipidaemia were added to the model, consistent with these comorbidities lying on the causal pathway from central adiposity to stroke rather than confounding it. The subsequent inversion of the BRI coefficient after FI was added (OR 0.91, 95% CI 0.88–0.95) should not be read as a protective effect: hypertension and diabetes were simultaneously covariates and components of the FI, creating collinearity and over-adjustment that the authors appropriately label exploratory. Meanwhile, each 0.1 unit increase in FI more than doubled the odds for self-reported stroke (OR 2.38, 95% CI 2.23–2.54). BRI negligibly improved the discriminative performance of an age-and-sex only model for self-reported stroke (AUC 0.769, 95% CI 0.758–0.781 versus [vs] 0.763, 95% CI 0.752–

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0.775; Δ AUC 0.006; Brier score 0.0318 vs 0.0319), a difference that reaches statistical significance only because of the sample size, while the addition of FI provided a much larger gain (AUC 0.868, 95% CI 0.860–0.877). This gain must be interpreted with care. A deficit-accumulation FI contains mobility, activities-of-daily-living and self-rated health items that rise mechanically after a stroke, so the FI partly detects the consequences of the very outcome it is being used to classify. Because BRI, FI, and stroke were measured concurrently, and the discrimination analysis was unweighted and internally validated only, the design cannot distinguish a BRI-frailty-stroke pathway from stroke-related disability raising the FI, a limitation the authors themselves acknowledge.⁹

The formula for BRI is well established and validated, but its clinical utility remains hampered by the lack of a universally accepted cutoff unlike the BMI. Threshold values of BRI for the prediction of metabolic syndrome vary across populations and across the outcomes against which they are derived^{5,10}; sex-specific cut-offs for metabolic syndrome in Brazilian adults, for example, fall at 3.8–4.0, far below the stroke breakpoint of 6.34. In stroke risk, the cutoff of 6.34 reported by Hu et al. was lower than the inflection point of 8.489 reported by Gan et al. using data from NHANES 1999–2018.⁷ However, the 2 estimates derive from different model specifications on overlapping samples, and Hu et al.'s own segmented model was unadjusted whereas their spline was covariate-adjusted, so part of the discrepancy reflects specification instability rather than population instability. For Asian readers, a BRI of 6.34 corresponds to a waist-to-height ratio of approximately 0.64, or a waist circumference of about 109 cm in a person 1.70 m tall,⁹ well beyond the ethnic-specific waist thresholds (90 cm for men, 80 cm for women) at which cardiometabolic risk is already elevated in Asian populations. No BRI threshold has been validated in an Asian general population, and any local derivation would need to be stratified by ethnicity given differences in visceral adiposity at a given BMI among Chinese, Malay and Indian Singaporeans.

NHANES also samples only the non institutionalised population, excluding the most disabled stroke survivors. Notwithstanding the limitations of both studies being observational data and relying only on self-reported stroke diagnosis, the premature adoption of BRI cutoffs runs the risk of misclassification and distraction from other established risk factors. Prospective population-specific validation, with adjudicated incident stroke, pre-stroke frailty assessment and pre-registered thresholds, will be necessary for clinical implementation of BRI as a target for stroke prevention and management. Until then, BRI, which is a monotonic transformation of waist-to-height ratio, adds no information beyond the waistbased measures already

embedded in vascular risk assessment, and there is no evidence that monitoring its trajectory improves stroke prevention. While frailty's apparent discriminative advantage is likely inflated by reverse causation, central adiposity, frailty, and cerebrovascular disease share pathophysiological processes such as chronic inflammation and oxidative stress. Thus, instead of establishing superiority of FI over BRI, or the temporal ordering of BRI-frailty-stroke relationship, the findings of Hu et al. change nothing for practice today: waist-based measures of central adiposity remain part of vascular risk assessment, and frailty assessment remains a prognostic rather than a preventive tool.

Ethics statement

Not applicable, as no study participants were recruited.

Declaration

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