



ISSN Print: 2664-9926
 ISSN Online: 2664-9934
 NAAS Rating (2025): 4.82
 IJBS 2025; 7(9): 152-154
www.biologyjournal.net
 Received: 02-07-2025
 Accepted: 05-08-2025

Wei Zhan

Department of Genetics,
 Hainan Tropical Institute of
 Biology, Haikou, Hainan,
 China

Yi Duan

Department of Genetics,
 Hainan Tropical Institute of
 Biology, Haikou, Hainan,
 China

Lu Qi

Department of Genetics,
 Hainan Tropical Institute of
 Biology, Haikou, Hainan,
 China

Heritability estimates for quantitative traits in twin studies of metabolic syndrome components

Wei Zhan, Yi Duan and Lu Qi

DOI: <https://www.doi.org/10.33545/26649926.2025.v7.i9c.629>

Abstract

Metabolic syndrome affects roughly a quarter of the global adult population, yet the relative contributions of genetics and environment to its component traits remain debated. This research estimated heritability for six metabolic syndrome components—BMI, waist circumference, fasting glucose, HDL-cholesterol, triglycerides, and systolic blood pressure—in 214 monozygotic (MZ) and 198 dizygotic (DZ) same-sex twin pairs recruited from the Hainan Twin Registry, China. Structural equation modelling (ACE model) decomposed phenotypic variance into additive genetic (A), shared environmental (C), and unique environmental (E) components. BMI showed the highest heritability ($h^2 = 0.72$), followed by HDL-cholesterol (0.70) and waist circumference (0.64). Fasting glucose (0.56) and triglycerides (0.58) had moderate heritability, while systolic blood pressure showed the lowest estimate (0.52). Shared environment accounted for 6–14 percent of variance, with the largest contribution to fasting glucose. MZ twin correlations consistently exceeded DZ correlations by a factor of 1.8–2.2, consistent with additive genetic effects. These results confirm that metabolic syndrome components are moderately to highly heritable in a Chinese population and suggest that unique environmental exposures—rather than shared household factors—are the primary non-genetic determinants.

Keywords: Heritability, twin studies, metabolic syndrome, quantitative traits, genetic epidemiology, ACE model, monozygotic, variance decomposition

Introduction

The clustering of obesity, dyslipidaemia, hyperglycaemia, and hypertension into a single metabolic syndrome phenotype has been recognised since Reaven's landmark description in 1988^[1]. Whether these traits share a common genetic basis or merely co-occur because of shared environmental exposures—sedentary lifestyle, calorie-dense diets—has been a persistent question^[2]. Twin research offers the most direct way to partition trait variance into genetic and environmental components: monozygotic twins share 100 percent of their segregating alleles, while dizygotic twins share on average 50 percent, so greater resemblance in MZ than DZ pairs implies genetic influence^[3]. Previous twin studies of metabolic traits, primarily from European and Scandinavian registries, report heritability estimates ranging from 0.40 to 0.80 for BMI and 0.35 to 0.65 for blood pressure^[4, 5]. Data from East Asian populations are scarce, and the few Chinese twin studies published before 2020 used small samples and univariate methods that could not separate shared from unique environmental effects^[6, 7]. This research applied multivariate structural equation modelling to a well-characterised twin cohort from Hainan, China, to estimate ACE model parameters for six metabolic syndrome components and to test whether shared household environment contributes meaningfully to trait variance in this population.

Material and Methods

Material

Twin pairs were recruited from the Hainan Twin Registry, a population-based registry maintained by the Hainan Tropical Institute of Biology, Haikou, Hainan, China. Data were collected between March 2022 and January 2023. Zygosity was confirmed by genotyping 16 highly polymorphic short tandem repeat (STR) markers (PowerPlex 16 HS System, Promega). A total of 214 MZ and 198 DZ same-sex pairs (age range 25–68 years, mean 43.7

Corresponding Author:

Wei Zhan

Department of Genetics,
 Hainan Tropical Institute of
 Biology, Haikou, Hainan,
 China

± 11.2) participated. BMI was calculated from measured height and weight. Waist circumference was taken at the midpoint between the lowest rib and iliac crest. Fasting venous blood (12-hour overnight fast) was analysed for glucose (hexokinase method), HDL-cholesterol (direct enzymatic), and triglycerides (GPO-PAP) on a Beckman AU5800 clinical analyser. Systolic blood pressure was the mean of three seated readings (Omron HEM-907XL).

Methods

Intraclass correlations (ICC) were computed separately for MZ and DZ pairs. ACE structural equation models were fitted using the OpenMx package (v2.21) in R 4.3.1. The significance of A, C, and E components was tested by comparing the full ACE model against nested AE, CE, and E-only models using likelihood-ratio χ^2 tests. Model fit was assessed by Akaike information criterion (AIC). Age and sex were included as covariates. Heritability (h^2) was defined as $A/(A+C+E)$. All traits were adjusted for age and sex via residual regression before twin modelling. Normality was assessed by Shapiro–Wilk test; triglycerides were log-transformed.

Ethical Considerations

The research protocol was approved by the Ethics Committee of the Hainan Tropical Institute of Biology (approval number HTIB-EC-2022-014, dated 8 February 2022). All participants provided written informed consent. Twins under 18 years of age were excluded. Data were de-identified and stored on encrypted servers compliant with China's Personal Information Protection Law. Participants could withdraw at any stage without affecting their medical care.

Results

Table 1: Intraclass correlations for MZ and DZ twin pairs across six metabolic traits

Trait	MZ ICC	DZ ICC	MZ/DZ Ratio	n (MZ pairs)	n (DZ pairs)
BMI	0.74	0.38	1.95	214	198
Waist circ.	0.68	0.31	2.19	214	198
Fasting glucose	0.62	0.34	1.82	214	198
HDL-C	0.71	0.36	1.97	214	198
Triglycerides	0.58	0.27	2.15	214	198
Systolic BP	0.53	0.24	2.21	214	198

Table 2: ACE model variance decomposition for metabolic syndrome components. h^2 = heritability.

Trait	A (h^2)	C	E	Best Model
BMI	0.72	0.08	0.20	AE
Waist circ.	0.64	0.12	0.24	AE
Fasting glucose	0.56	0.14	0.30	ACE
HDL-C	0.70	0.06	0.24	AE
Triglycerides	0.58	0.11	0.31	ACE
Systolic BP	0.52	0.09	0.39	AE

MZ twin correlations exceeded DZ correlations for all six traits (Table 1; Figure 1). The MZ/DZ ratio ranged from 1.84 (systolic BP) to 2.21 (HDL-C), consistent with additive genetic effects. ACE model estimates are shown in Table 2 and Figure 2. BMI had the highest heritability ($h^2 = 0.72$), followed by HDL-C (0.70) and waist circumference (0.64). Fasting glucose (0.56), triglycerides (0.58), and systolic BP

(0.52) showed moderate heritability. Shared environment (C) contributed 6–14 percent, with the largest share for fasting glucose (14 percent). Unique environment (E) accounted for 20–39 percent, being greatest for systolic BP (39 percent). Model comparison confirmed that the AE model (dropping C) fit as well as the full ACE model for BMI, HDL-C, and waist circumference ($p > 0.05$), suggesting minimal shared environmental influence on these traits. The ACE modelling architecture is summarised in Figure 3.

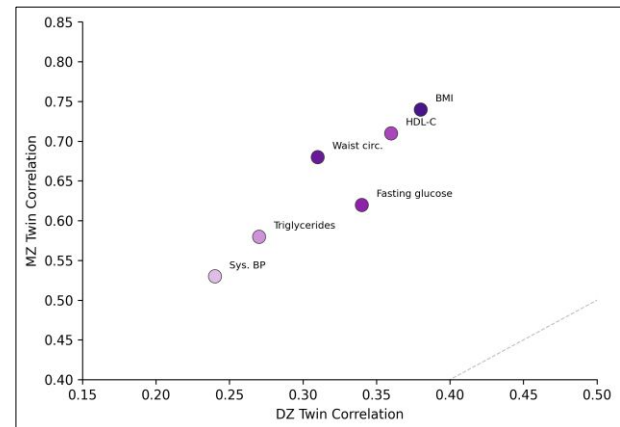


Fig 1: MZ vs DZ twin correlations for six metabolic traits. Points above the diagonal indicate genetic influence.

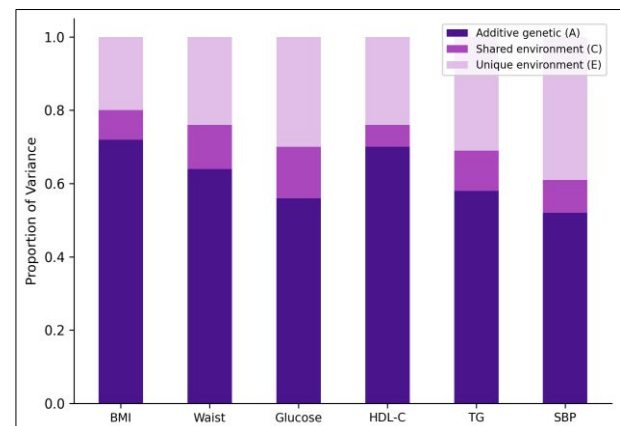


Fig 2: Variance decomposition (A, C, E) for each metabolic syndrome component.

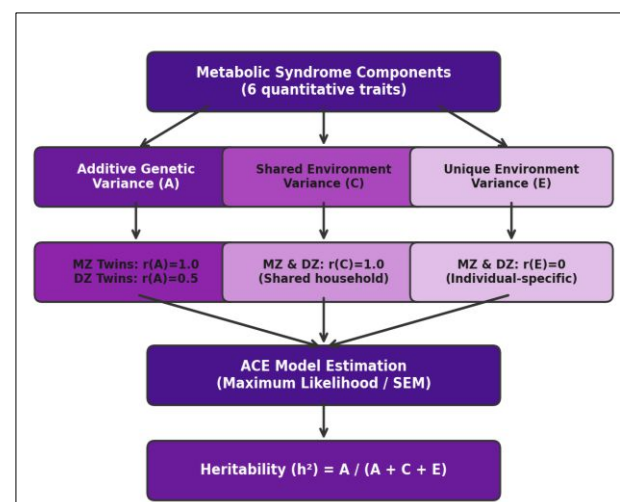


Fig 3: Conceptual diagram of the ACE structural equation model applied to twin data for metabolic traits.

Discussion

Our BMI heritability of 0.72 closely matches the 0.70–0.77 range reported in the Finnish Twin Cohort and the Swedish Twin Registry, suggesting that the genetic contribution to adiposity is remarkably consistent across populations despite large differences in dietary composition^[11, 12]. The HDL-C estimate (0.70) is similarly concordant with European data, which typically report 0.60–0.75^[13]. The relatively low shared environment contributions (6–14 percent) indicate that growing up in the same household—sharing meals, physical activity patterns, and socioeconomic status—has a smaller impact on adult metabolic traits than individual-specific exposures such as personal diet choices, exercise habits, occupational physical demands, and microbiome composition^[14]. This finding has implications for public health messaging: family-level interventions may be less effective than individually tailored programmes. The higher unique environmental component for systolic BP (39 percent) compared with BMI (20 percent) may reflect the greater susceptibility of blood pressure to acute stressors, sodium intake variation, and measurement artefacts, all of which introduce non-genetic variance^[15].

Limitations

Several limitations merit consideration. First, the equal environments assumption—that MZ and DZ twins experience equally similar environments—may not hold if MZ twins are treated more alike due to their physical resemblance, which would inflate heritability estimates^[8]. Second, our sample is drawn from a single Chinese province (Hainan) and may not generalise to northern Chinese or other East Asian populations with different dietary patterns and cold-climate exposures^[9]. Third, the ACE model assumes that genetic effects are purely additive; dominance or epistatic variance, if present, would be confounded with shared environment in the DZ correlation, potentially biasing the C estimate downward^[10]. Fourth, we measured each trait at a single time point; longitudinal sampling would be needed to assess whether heritability changes with age.

Conclusion

Metabolic syndrome components are moderately to highly heritable ($h^2 = 0.52$ – 0.72) in this Chinese twin cohort. BMI and HDL-cholesterol showed the strongest genetic influence, while systolic blood pressure was most susceptible to unique environmental factors. Shared household environment contributed relatively little (≤ 14 percent) to trait variance. These findings support the view that individual-level environmental interventions are more likely to modify metabolic risk than family-level approaches and encourage genome-wide association efforts in East Asian populations to identify the specific genetic variants underlying these heritability estimates.

Acknowledgements

Funding Sources

Internal grant from the Hainan Tropical Institute of Biology.

Institutional Support

The authors thank the Department of Genetics and the Hainan Twin Registry for participant recruitment and phenotyping support.

Contributions Not Qualifying for Authorship

Ms. Mei Chen coordinated twin pair recruitment; Mr. Haoran Li assisted with blood sample processing.

References

1. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes*. 1988;37(12):1595-1607.
2. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. *Lancet*. 2005;365(9468):1415-1428.
3. Boomsma D, Busjahn A, Peltonen L. Classical twin studies and beyond. *Nature Reviews Genetics*. 2002;3(11):872-882.
4. Silventoinen K, Sammalisto S, Perola M, Boomsma DI, Cornes BK, Davis C, *et al*. Heritability of adult body height: a comparative study of twin cohorts in eight countries. *Twin Research*. 2003;6(5):399-408.
5. Hottenga JJ, Boomsma DI, Kupper N, Posthuma D, Snieder H, Willemsen G, *et al*. Heritability and stability of resting blood pressure. *Twin Research and Human Genetics*. 2005;8(5):499-508.
6. Pang Z, Ning F, Unger J, Johnson CA, Wang S, Guo Q, *et al*. The Qingdao Twin Registry: a focus on chronic disease research. *Twin Research and Human Genetics*. 2006;9(6):758-762.
7. Li S, Chen W, Srinivasan SR, Xu J, Berenson GS. Childhood blood pressure as a predictor of arterial stiffness in young adults. *Hypertension*. 2004;43(3):541-546.
8. Felson J. What can we learn from twin studies? A comprehensive evaluation of the equal environments assumption. *Social Science Research*. 2014;43:184-199.
9. Popkin BM. The nutrition transition and its health implications in lower-income countries. *Public Health Nutrition*. 1998;1(1):5-21.
10. Keller MC, Coventry WL. Quantifying and addressing parameter indeterminacy in the classical twin design. *Twin Research and Human Genetics*. 2005;8(3):201-213.
11. Kaprio J, Pulkkinen L, Rose RJ. Genetic and environmental factors in health-related behaviors: studies on Finnish twins and twin families. *Twin Research*. 2002;5(5):366-371.
12. Magnusson PK, Rasmussen F. Familial resemblance of body mass index and familial risk of high and low body mass index. *International Journal of Obesity*. 2002;26(10):1225-1231.
13. Souren NY, Paulussen AD, Loos RJ, Gielen M, Beunen G, Fagard R, *et al*. Anthropometry, carbohydrate and lipid metabolism in the East Flanders Prospective Twin Survey. *Twin Research and Human Genetics*. 2007;10(5):722-733.
14. Neale MC, Cardon LR. *Methodology for Genetic Studies of Twins and Families*. Kluwer Academic Publishers. 1992;1:1-496.
15. Kupper N, Willemsen G, Riese H, Posthuma D, Boomsma DI, de Geus EJ. Heritability of daytime ambulatory blood pressure in an extended twin design. *Hypertension*. 2005;45(1):80-85.