Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 12 May 2026
Revised 18 June 2026
Accepted 02 July 2026

Natural Resources for Human Health 2026, Vol. 6 (11s): 1321–1329

KEYWORDS:
Electric Impedance; Basal Metabolism; Blood Glucose; Lipids; Dyslipidemias; Young Adult.

Bioimpedance-Derived Metabolic Age Is Associated with an Unfavorable Lipid Profile but Not Fasting Glucose in Young Adults: A Multiethnic Cross-Sectional Study

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ABSTRACT: Bioimpedance-derived metabolic age (MA) may capture aspects of metabolic phenotype beyond chronological age, but its relationships with routine biochemical markers in young adults remain unclear. This cross-sectional study examined associations of MA with fasting glucose and lipid parameters in 345 adults aged 18–25 years from the Minahasa, Bolaang Mongondow, and Sangihe Talaud ethnic groups in North Sulawesi, Indonesia. MA was obtained using a Tanita RD-953 dual-frequency bioelectrical impedance monitor. Unadjusted associations were assessed using Spearman correlation with 10,000 bootstrap resamples, and separate multivariable linear regression models were adjusted for chronological age, sex, ethnicity, and physical activity. Higher MA was correlated with total cholesterol, LDL-C, and triglycerides, but not fasting glucose or HDL-C. After adjustment, each 1-year higher MA was associated with higher total cholesterol (B=0.999 mg/dL; 95% CI 0.464–1.534), LDL-C (B=0.816; 95% CI 0.372–1.260), and triglycerides (B=1.688; 95% CI 0.906–2.470), and lower HDL-C (B=−0.231; 95% CI −0.388 to −0.074), all p≤0.004. No association was observed with fasting glucose (B=−0.007; 95% CI −0.184 to 0.170; p=0.939). Sensitivity analyses supported the triglyceride and fasting-glucose findings. Higher BIA-derived MA was therefore associated with an unfavorable lipid pattern, but not fasting glucose, in young adults.

1. INTRODUCTION

Cardiometabolic risk can accumulate from early adulthood, well before cardiovascular disease becomes clinically apparent. Young adulthood therefore represents an important window for prevention, particularly because long-term risk may not be fully captured by short-term risk assessment [1]. Cumulative exposure to low-density lipoprotein cholesterol (LDL-C) from young adulthood through middle age has been associated with incident coronary heart disease independently of midlife LDL-C levels [2]. Dyslipidemia is already evident in younger populations and varies according to sex, body mass index (BMI), and ethnicity [3], while dyslipidemia, obesity, dysglycemia, and other cardiometabolic abnormalities frequently cluster [4].

Although BMI remains clinically useful, it does not fully characterize metabolic heterogeneity. Bioelectrical impedance analysis (BIA) provides a practical and non-invasive approach to estimating body composition, although BIA-derived body-fat measures do not consistently outperform BMI for cardiometabolic risk discrimination [5]. Metabolic age (MA), a basal metabolic rate-related metric generated by some BIA systems, has emerged as another potential metabolic phenotype. Higher MA has been associated with greater metabolic-syndrome risk [6] and with a higher waist-to-height ratio, an established marker of

cardiovascular risk [7]. Cardiometabolic differences are also detectable among young adults across BMI strata before overt cardiovascular disease develops [8].

More advanced approaches to metabolic phenotyping further suggest that metabolic characteristics may capture information not fully reflected by measured BMI or chronological age alone [9,10]. Although these molecular approaches are distinct from BIA-derived MA, they support the broader rationale for examining metabolic phenotype beyond chronological age. However, the associations of BIA-derived MA with fasting glucose and individual conventional lipid fractions remain insufficiently characterized, particularly in ethnically diverse young adults.

Therefore, this study examined the associations of metabolic age with fasting glucose, total cholesterol, high-density lipoprotein cholesterol (HDL-C), LDL-C, and triglycerides among young adults from three ethnic groups in North Sulawesi, Indonesia.

2. MATERIALS AND METHODS

2.1 Study Design and Participants

This analytical cross-sectional study was conducted among young adults in North Sulawesi, Indonesia, in May 2025. Participants were recruited from multiple locations in North Sulawesi using purposive sampling according to predefined eligibility criteria. The target population comprised adults aged 18–25 years from the Minahasa, Bolaang Mongondow, and Sangihe Talaud ethnic groups.

Eligible participants were aged 18–25 years based on date of birth, had resided in North Sulawesi for at least one year, were willing to undergo questionnaire assessment, anthropometric and bioelectrical impedance measurements, and laboratory testing, and provided written informed consent. Participants were excluded if they had a known chronic condition likely to substantially affect glucose or lipid metabolism, including diabetes mellitus, hypertension, renal failure, or severe infectious disease; were pregnant or breastfeeding; were using medications known to affect blood glucose or lipid concentrations; or had fasted for less than 8 h before blood sampling.

Chronological age was calculated from the recorded date of birth using 28 May 2025 as the common reference date. The initial dataset contained 348 participants. Following verification of chronological age, three individuals aged <18 years were excluded, yielding a final analytical sample of 345 participants. All participants included in the analysis had complete data for the primary exposure, biochemical outcomes, and covariates.

The minimum sample size was estimated for a correlation analysis using Fisher's z transformation. Assuming a correlation coefficient of 0.30, a two-sided α of 0.05, and 80% statistical power, approximately 85 participants were required. Allowing an additional 10–15% for potentially incomplete data resulted in a target of approximately 94–98 participants. The final sample of 345 substantially exceeded this minimum requirement.

2.2 Metabolic Age and Anthropometric Assessment

The primary exposure was absolute metabolic age (MA), expressed in years and obtained directly from a Tanita RD-953 dual-frequency body composition monitor (Tanita Corporation, Tokyo, Japan). The device applies bioelectrical impedance at 5 and 50 kHz with a measurement current of 100 μA and generates MA as a device-derived estimate related to basal metabolic rate and individual characteristics. MA was recorded directly from the device output and was not recalculated using an external equation.

Measurements were performed with participants barefoot and standing upright with appropriate contact with the device electrodes according to a standardized measurement procedure. Height and body weight were obtained using standardized procedures, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Before data collection, measurement equipment was checked for proper function, and measurements were performed by trained personnel using uniform procedures.

2.3 Biochemical Measurements and Covariates

Venous blood samples were obtained after an overnight fast of 8–10 h. Fasting glucose, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides were measured at the Regional

Health Laboratory of North Sulawesi Province, Manado, Indonesia. All biochemical variables were analyzed as continuous measures in mg/dL.

Demographic and health-related information was obtained using structured study forms and interviews. Sex was recorded as female or male. Ethnicity was recorded as Minahasa, Bolaang Mongondow, or Sangihe Talaud based on participants' reported ethnic group. Physical activity was assessed using the International Physical Activity Questionnaire–Short Form (IPAQ-SF) and classified as low, moderate, or high according to the IPAQ scoring criteria.

Chronological age, sex, ethnicity, and physical activity were treated as potential confounders because of their plausible relationships with both metabolic characteristics and biochemical outcomes. These variables were therefore included in all multivariable models. Female sex, Minahasa ethnicity, and low physical activity served as the reference categories.

2.4 Data Quality Control

Data were checked for completeness, internal consistency, and plausibility before analysis. Recorded values showing logical inconsistencies were verified against the corresponding source documents, and confirmed data-entry errors were corrected using the original records. Verified measurement values were retained without data-driven modification. Dates of birth were rechecked and chronological age was recalculated uniformly before construction of the final analytical dataset.

2.5 Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics version 30.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as median and interquartile range [25th–75th percentile], and categorical variables as frequency and percentage. Unadjusted associations between MA and fasting glucose, total cholesterol, HDL-C, LDL-C, and triglycerides were evaluated using Spearman's rank correlation coefficient (ϱ). Spearman correlation was selected because MA and several biochemical variables showed non-normal distributions and MA contained substantial tied values. Percentile 95% confidence intervals (CIs) for the correlation coefficients were estimated using 10,000 bootstrap resamples.

Adjusted associations were examined using separate multivariable linear regression models for each biochemical outcome: fasting glucose, total cholesterol, HDL-C, LDL-C, and triglycerides. MA was entered as a continuous exposure, with chronological age entered as a continuous covariate and sex, ethnicity, and physical activity entered as categorical covariates. Ethnicity was represented using two indicator variables with Minahasa as the reference category, and physical activity using two indicator variables with low activity as the reference category. No data-driven variable-selection procedure was applied. Associations are reported as unstandardized regression coefficients (B) with 95% CIs, representing the estimated difference in each biochemical outcome, in mg/dL, associated with a 1-year higher MA. Model fit was summarized using adjusted R^2. Collinearity was assessed using tolerance and variance inflation factor statistics, and model diagnostics included examination of standardized residuals and Cook's distance.

Because triglyceride concentrations were markedly right-skewed, robustness of the triglyceride association was evaluated in a sensitivity analysis using natural-log-transformed triglycerides as the dependent variable. For the fasting-glucose model, an additional sensitivity analysis re-estimated the association after excluding observations with an absolute standardized residual >3. All eligible observations were retained in the primary analyses. Statistical tests were two-sided, with $p<0.05$ considered statistically significant. Interpretation emphasized effect estimates and their 95% CIs in addition to p values.

3. RESULTS AND DISCUSSION

3.1 Participant characteristics

A total of 345 young adults were included in the analysis. As shown in Table 1, the median age was 20 [19–21] years, with a nearly balanced sex distribution (50.4% male). Participants were similarly distributed across the Minahasa (34.2%), Bolaang Mongondow (32.5%), and Sangihe Talaud (33.3%) ethnic groups. The median metabolic age was 21 [18–30] years, and 138 participants (40.0%) had a metabolic age of 18 years, the lowest observed value. Median fasting glucose was 74 [68–81] mg/dL, while median total cholesterol, HDL-C, LDL-C, and triglyceride levels were 152 [132–177.5], 37 [31–45], 92 [77–110], and 74 [59–99] mg/dL, respectively.

Natural Resources for Human Health
eISSN: 2583-1194

Table 1. Characteristics of the study participants

Characteristic	Overall (n = 345)
Age, years	20 [19–21]
Sex, n (%)	
Female	171 (49.6)
Male	174 (50.4)
Ethnicity, n (%)	
Minahasa	118 (34.2)
Bolaang Mongondow	112 (32.5)
Sangihe Talaud	115 (33.3)
Physical activity, n (%)	
Low	164 (47.5)
Moderate	102 (29.6)
High	79 (22.9)
BMI, kg/m²	22.34 [20.08–25.16]
Metabolic age, years	21 [18–30]
Fasting glucose, mg/dL	74 [68–81]
Total cholesterol, mg/dL	152 [132–177.5]
HDL-C, mg/dL	37 [31–45]
LDL-C, mg/dL	92 [77–110]
Triglycerides, mg/dL	74 [59–99]

Note: Data are presented as median [25th–75th percentile] or n (%), as appropriate. Physical activity was classified using the International Physical Activity Questionnaire–Short Form (IPAQ-SF). BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

3.2 Unadjusted associations of metabolic age with fasting glucose and lipid parameters

Metabolic age was not associated with fasting glucose (ϱ=0.017; 95% CI −0.085 to 0.122; p=0.748). In contrast, higher metabolic age showed weak positive correlations with total cholesterol (ϱ=0.191; 95% CI 0.090–0.295; p<0.001), LDL-C (ϱ=0.210; 95% CI 0.108–0.312; p<0.001), and triglycerides (ϱ=0.176; 95% CI 0.071–0.278; p=0.001). The correlation with HDL-C was negative but not statistically significant (ϱ=−0.074; 95% CI −0.174 to 0.031; p=0.171) (Table 2).

Table 2. Unadjusted associations of metabolic age with fasting glucose and lipid parameters

Outcome	Spearman ϱ	95% CI	p-value
Fasting glucose	0.017	−0.085 to 0.122	0.748
Total cholesterol	0.191	0.090 to 0.295	<0.001
HDL-C	−0.074	−0.174 to 0.031	0.171
LDL-C	0.210	0.108 to 0.312	<0.001
Triglycerides	0.176	0.071 to 0.278	0.001

Note: Spearman rank correlation was used because of the non-normal distribution and substantial ties in metabolic age. Confidence intervals were estimated using percentile bootstrap with 10,000 resamples. All tests were two-sided. CI, confidence interval; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

3.3 Adjusted Associations of Metabolic Age with Fasting Glucose and Lipid Parameters

After adjustment for chronological age, sex, ethnicity, and physical activity, metabolic age remained unrelated to fasting glucose (B = −0.007 mg/dL per 1-year increase; 95% CI −0.184 to 0.170; p = 0.939). In contrast, each 1-year increase in metabolic age was independently associated with a 0.999 mg/dL increase in total cholesterol (95% CI 0.464–1.534; p < 0.001), a 0.816 mg/dL increase in LDL-C (95% CI 0.372–1.260; p < 0.001), and a 1.688 mg/dL increase in triglycerides (95% CI 0.906–2.470; p < 0.001). Higher metabolic age was also independently associated with lower HDL-C, with a reduction of 0.231 mg/dL per 1-year increase in metabolic age (95% CI −0.388 to −0.074; p = 0.004) (Table 3).

Sensitivity analyses supported the robustness of these findings. The association between metabolic age and triglycerides remained significant after logarithmic transformation of triglycerides (B = 0.016; 95% CI 0.009–0.024; p < 0.001). Likewise, exclusion of four observations with large standardized residuals from the fasting glucose model did not materially alter the null association between metabolic age and fasting glucose (B = −0.018; 95% CI −0.179 to 0.142; p = 0.822; n = 341).

Table 3. Adjusted associations of metabolic age with fasting glucose and lipid parameters

Outcome	Adjusted B per 1-year increase in metabolic age, mg/dL	95% CI	p-value	Adjusted R^2
Fasting glucose	−0.007	−0.184 to 0.170	0.939	0.081
Total cholesterol	0.999	0.464 to 1.534	<0.001	0.227
HDL-C	−0.231	−0.388 to −0.074	0.004	0.150
LDL-C	0.816	0.372 to 1.260	<0.001	0.144
Triglycerides	1.688	0.906 to 2.470	<0.001	0.053

Note: Separate multivariable linear regression models were fitted for each outcome. B represents the unstandardized regression coefficient for each 1-year increase in metabolic age. All models were adjusted for chronological age, sex, ethnicity, and physical activity. Reference categories were female sex, Minahasa ethnicity, and low physical activity. CI, confidence interval; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

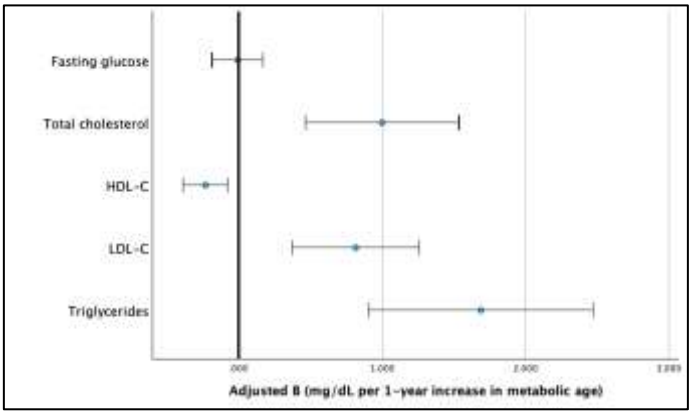

Figure 1. Adjusted associations of metabolic age with fasting glucose and lipid parameters. Points represent unstandardized regression coefficients (B) for each 1-year increase in metabolic age, and horizontal bars indicate 95% confidence intervals. Separate multivariable linear regression models were adjusted for chronological age, sex, ethnicity, and physical activity. The vertical line at B = 0 represents the null value.

3.4 Discussion

In this multiethnic sample of young adults, higher bioelectrical impedance analysis (BIA)-derived metabolic age (MA) was associated with a less favorable lipid profile, characterized by higher total cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglycerides and lower high-density lipoprotein cholesterol (HDL-C), whereas no association was observed with fasting glucose. These associations persisted after adjustment for chronological age, sex, ethnicity, and physical activity. The triglyceride association remained after logarithmic transformation, and exclusion of observations with large fasting-glucose residuals did not materially alter the null glucose finding. Previous studies have linked BIA-derived MA with metabolic syndrome, waist-to-height ratio, and insulin-resistance-related phenotypes [6,7,11,12]. The present study extends this evidence by examining conventional lipid fractions individually in young adults.

The overall direction of the lipid findings is consistent with previous evidence linking higher MA with adverse cardiometabolic phenotypes. Vásquez-Alvarez et al. [6] reported substantially higher MA among individuals at greater risk of metabolic syndrome, while Elguezabal-Rodelo et al. [7] demonstrated a strong association between Tanita-derived MA and waist-to-height ratio. In 8,590 Spanish workers, Ramírez-Gallegos et al. [11] found strong associations between BIA-derived MA and metabolic score for insulin resistance, single-point insulin sensitivity estimator, and triglyceride-glucose index. A subsequent analysis also demonstrated associations with metabolic syndrome and hypertriglyceridemic- and hypertensive-waist phenotypes [12]. Because these studies evaluated composite metabolic outcomes and used somewhat different MA definitions, they provide convergent rather than directly replicative evidence.

The lipid findings are particularly relevant in early adulthood because cardiovascular risk reflects cumulative exposure over time. Cumulative LDL-C exposure from young adulthood through middle age has been associated with incident coronary heart disease independently of midlife LDL-C concentration [2]. In the Bogalusa Heart Study, age-specific lipid concentrations from childhood onward were associated with adult carotid intima-media thickness, while adverse trajectories of total cholesterol, non-HDL-C, LDL-C, and triglycerides were associated with greater midlife subclinical atherosclerosis [13]. Dyslipidemia is already evident in large young-adult populations and varies according to sex, adiposity, and ethnicity [3]. Nevertheless, the present triglyceride association should not be interpreted as direct evidence of greater atherogenic remnant burden because plasma triglycerides, triglyceride-rich lipoproteins, and remnant cholesterol are related but non-equivalent measures [14–16].

The absence of an association with fasting glucose does not exclude early abnormalities in insulin sensitivity. Insulin resistance may precede sustained fasting hyperglycemia because compensatory hyperinsulinemia can initially maintain glucose concentrations within a relatively normal range [17]. Insulin also regulates endogenous glucose production through coordinated hepatic and extrahepatic mechanisms [18]. Consistent with this distinction, Parcha et al. [19] found insulin resistance in approximately four in ten nondiabetic young US adults, accompanied by clustering of obesity, hypercholesterolemia, hypertension, and other cardiometabolic risk factors. The null fasting-glucose finding in the present study is therefore not inconsistent with previous associations between MA and composite insulin-resistance indices [12], because those indices integrate glucose with triglycerides, anthropometric characteristics, or other metabolic information.

The inverse association between MA and HDL-C became evident only after multivariable adjustment. This pattern is plausible because lipid distributions vary across demographic and metabolic strata. Liu et al. [3], for example, documented substantial differences in dyslipidemia according to sex, BMI, and ethnicity among young adults. Adjustment for these factors may therefore have reduced confounding that obscured the unadjusted relationship. However, the change in statistical significance should not be interpreted as evidence of a specific biological mediation or suppression mechanism.

These findings do not imply that MA should replace direct lipid measurements or established cardiovascular risk assessment. Contemporary prevention frameworks remain centered on measured risk factors including LDL-C, blood pressure, diabetes, smoking, and adiposity [1,20]. BIA-derived MA may instead serve as an accessible phenotypic signal that prompts closer cardiometabolic assessment in otherwise young individuals. Molecular metabolic-age research similarly suggests that metabolically derived age measures may contain information beyond chronological age [10]. However, lipidomic metabolic age and BIA-derived MA are methodologically distinct constructs and should not be considered interchangeable.

This study has several strengths, including a relatively large young-adult sample encompassing three ethnic groups, complete primary-model data, fasting biochemical measurements, prespecified covariate adjustment, bootstrap confidence intervals, and sensitivity analyses addressing triglyceride skewness and influential fasting-glucose observations. Several limitations should

nevertheless be considered. The cross-sectional design precludes temporal or causal inference, while purposive recruitment limits population-level generalizability. BIA-derived MA is generated from basal metabolic rate- and body-composition-related information; consequently, its associations may partly reflect metabolic and adiposity characteristics incorporated into the device-derived estimate rather than a distinct biological-aging process. Bioimpedance-derived measures also depend on underlying biophysical assumptions and standardized measurement procedures, warranting caution when comparing values across devices [21]. Clustering of observations at the lower end of the MA distribution may further reduce discrimination among participants with the lowest estimated MA values. Residual confounding from diet, smoking, familial risk, and other unmeasured factors also remains possible.

Overall, higher BIA-derived MA was associated with an unfavorable lipid pattern but not with fasting glucose among young adults after adjustment for chronological age and selected demographic and behavioral factors. Prospective studies incorporating insulin-based measures, more detailed lipoprotein phenotyping, repeated MA measurements, and cardiovascular follow-up are needed to determine whether BIA-derived MA provides incremental information beyond established cardiometabolic markers [10,11].

4. CONCLUSION

Higher BIA-derived metabolic age was associated with a less favorable lipid profile among young adults from three ethnic groups in North Sulawesi, with higher total cholesterol, LDL-C, and triglycerides and lower HDL-C after adjustment for chronological age, sex, ethnicity, and physical activity. In contrast, metabolic age was not associated with fasting glucose. These findings indicate that BIA-derived metabolic age may reflect aspects of lipid-related metabolic phenotype in early adulthood, but should not be considered a substitute for direct biochemical assessment or an established measure of biological aging. Given the cross-sectional design and the device-derived nature of metabolic age, causal and prognostic interpretations are not warranted. Prospective studies incorporating insulin-based measures, repeated metabolic-age assessment, detailed lipoprotein profiling, and broader population sampling are needed to determine whether metabolic age provides incremental information beyond established cardiometabolic markers.

ACKNOWLEDGEMENTS

The authors thank all participants from Minahasa, Bolaang Mongondow, and Sangihe Talaud who participated in this study. The authors also acknowledge the staff of the Regional Health Laboratory of North Sulawesi Province, Manado, Indonesia, for their assistance with biochemical measurements, and the research personnel who supported participant recruitment, anthropometric assessment, and bioelectrical impedance measurements. The authors are grateful to the Faculty of Medicine, Universitas Hasanuddin, Makassar, Indonesia, for academic and institutional support.

AUTHOR CONTRIBUTIONS

NT: Conceptualization, Investigation, Data Curation, Formal Analysis, Visualization, and Writing – Original Draft.

AYS: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

NAT: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

AB: Methodology, Supervision, Validation, and Writing – Review & Editing.

A: Methodology, Validation, Formal Analysis, and Writing – Review & Editing.

M: Methodology, Validation, Supervision, and Writing – Review & Editing.

All authors critically reviewed the manuscript, approved the final version, and agreed to be accountable for the accuracy and integrity of the work.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

This research received no external funding.

ETHICS STATEMENT

The study protocol was approved by the Health Research Ethics Committee of Hasanuddin University, Makassar, Indonesia (Approval No. 263/UN4.6.4.5.31/PP36/2025; Protocol No. UH25040288). All participants received information regarding the study procedures and provided written informed consent before participation. Participation was voluntary, participant confidentiality was maintained throughout the study, and personally identifying information was excluded from the analytical dataset.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional restrictions.

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