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Dyrk1b as a potential biomarker for sarcopenia in older adults

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Abstract

Background Sarcopenia is characterized by the progressive loss of muscle mass and function due to aging. Dual-specificity tyrosine-regulated kinase 1b (Dyrk1b) plays a key role in muscle differentiation by regulating transcription, cell cycle progression, and cell survival. However, the relationship between Dyrk1b levels and sarcopenia is unclear. This study aimed to evaluate the association of serum Dyrk1b level with sarcopenia in the elderly of community-dwelling.

Methods A total of 939 community-dwelling elderly people (median age = 76.0 years) were recruited, including 524 men and 415 women. Serum Dyrk1b was measured by enzyme-linked immunosorbent assay. Appendicular skeletal muscle mass index (ASMI), grip strength, and gait speed were taken to assess sarcopenia.

Results We found that serum Dyrk1b levels in patients with sarcopenia [median (IQR) = 67.37 (55.13–82.56) pg/mL] were lower than those in elderly people without sarcopenia [70.40 (58.34–92.35) pg/mL, $P < 0.001$]. Receiver operating characteristic curve analysis indicated that the optimal cutoff value of serum Dyrk1b level for predicting sarcopenia was 44.73 pg/mL, with a sensitivity of 94.8% and a specificity of 14.7% (AUC = 0.577, 95% CI = 0.540–0.613, $P < 0.001$. Multivariate logistic regression analysis showed that high serum Dyrk1b levels (> 44.73 pg/mL) were related to decreased risk of sarcopenia (adjusted OR = 0.342, 95%CI = 0.194–0.603, $P < 0.001$). Moreover, serum Dyrk1b concentration was positively correlated with ASMI ($r = 0.169$, $P < 0.001$), grip strength ($r = 0.157$, $P < 0.001$) and gait speed ($r = 0.164$, $P < 0.001$).

Conclusions In summary, our results indicate that low serum Dyrk1b level is associated with an increased risk of sarcopenia in the elderly, suggesting that Dyrk1b may be valuable as a surrogate biomarker for screening and evaluation of sarcopenia.

Keywords Older adults, Dyrk1b, Sarcopenia, Serum

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Introduction

Sarcopenia is an age-related syndrome characterized by degenerative loss of skeletal muscle mass, muscle strength, and/or physical function [1, 2]. The overall prevalence of sarcopenia is about 10% among community-dwelling adults aged 60 years and older [3]. In China, the prevalence of sarcopenia ranges from 9.8–18.6% [4, 5]. Sarcopenia increases the risk of falls and fractures, reduces quality of life, and increases the risk of illness and even death. It has become a major threat to the health of the elderly [6–8]. The diagnosis of sarcopenia requires measuring muscle mass, muscle strength, and physical performance [9]. Considering that sarcopenia is a predictor of mortality in the elderly [10], it is a priority to explore feasible biomarkers for early identification of subjects at high risk of sarcopenia.

Dual-specificity tyrosine phosphorylation-regulated kinase 1b (Dyrk1b) is one of the best functionally characterized members of the Dyrk family of dual-specificity kinases [11]. Dyrk family kinases are highly conserved mediators of growth control and differentiation [12]. Previous studies have revealed that Dyrk1b is expressed at high levels in skeletal muscle and plays a critical role in muscle differentiation by regulatory effects on motility, transcription, cell cycle progression, autophagy, and cell survival [13–16]. Dyrk1b links skeletal muscle glycolysis metabolism with insulin resistance [17]. Also, Dyrk1b may be an anti-aging target for senescent endothelial cells, suggesting a potential role of Dyrk1b in aging-related diseases [18]. These results suggest that Dyrk1b may play an important role in the pathogenesis of sarcopenia. However, whether circulating levels of Dyrk1b are associated with the risk of sarcopenia remains unclear. Therefore, we conducted a case-control study investigating the relationship between serum Dyrk1b and sarcopenia in Chinese community-dwelling older adults.

Methods and materials

Study participants

A total of 939 older adults aged ≥ 65 were recruited consecutively from both rural and urban areas of Jiangsu Province as previously described [19]. Participants with the following conditions were excluded: (a) Bedridden or unable to move independently; (b) Unable to perform specific movements due to neurological disorders, bone and joint diseases, or cardiorespiratory insufficiency; (c) Severe renal insufficiency (creatinine clearance < 60 mL/min) or severe hepatic damage (transaminase elevated more than 2 times); (d) Malignant tumors [19]. This study was conducted under the principles outlined in the Declaration of Helsinki [20] and approved by the Ethics Committee of Sir Run Run Hospital, Nanjing Medical University (approval number 2019-SR-S041). Written informed consent was obtained from all enrolled participants.

Data collection

After overnight fasting, venous blood was collected in the early morning and separated into serum and cell components within 2 h. The serum was stored at -80 °C for further analysis. A series of blood biochemical indicators were measured, as mentioned previously [19]. Participants who smoked more than 1 cigarette per day in the past 12 months were classified as current smokers. Current drinkers were those who drank at least once a day in the past 12 months [21].

Assessment of sarcopenia

Sarcopenia was diagnosed based on the latest criteria of the Asian Working Group on Sarcopenia (AWGS) 2019 [2]. Muscle mass was measured by bioelectrical impedance analysis (BIA) using Inbody S10 (Inbody, Korea). Appendicular skeletal muscle mass index (ASMI) was calculated as ASM divided by height in square meters ($ASM/height^2$). Grip strength was measured with a dynamometer (CAMRY EH101, China). Gait speed, usually on a 6-meter track, was tested for physical performance. Briefly, elderly patients with low muscle mass (ASMI < 7.0 kg/m^2 in men and < 5.7 kg/m^2 in women) and low muscle strength (grip strength < 28 kg in men and < 18 kg in women) and/or low physical function (walking speed < 1.0 m/s) were categorized as having sarcopenia [2]. Patients with low muscle mass combined with low muscle strength or low physical performance were assumed to have moderate sarcopenia. Patients with low muscle mass, low muscle strength, and low physical performance were considered to have severe sarcopenia [2].

Serum Dyrk1b measurements

Serum Dyrk1b levels were measured using an enzyme-linked immunosorbent assay (ELISA) kit (Cat. JM-5853H1, JINGMEI, China) according to the manufacturer's protocol. The intra-assay and inter-assay coefficients of variance were 3.23% and 2.83%, respectively. The analytical sensitivity was 0.49 pg/mL.

Statistical analysis

The Kolmogorov-Smirnov test was used to test the normality of continuous variables, described as the median (interquartile range, IQR). The Mann-Whitney U test was utilized to determine differences between two groups. The Pearson χ^2 test was used to compare qualitative variables expressed as frequencies. Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cut-off value of serum Dyrk1b levels for best prediction of sarcopenia by calculating the largest Youden index. Spearman's correlation was used to

Table 1 The characteristics of the enrolled subjects

Variables	Non-Sarcopenia (*n* = 401)	Sarcopenia (*n* = 538)	*P*
Age, years	76.0 (71.0–80.0)	77.0 (70.0–82.0)	0.037
Male, n (%)	230 (57.4)	294 (54.6)	0.408
BMI, kg/m^2	25.17 (23.06–26.94)	22.35 (20.00-23.92)	< 0.001
Smokers, n (%)	60 (15.0)	71 (13.2)	0.440
Drinkers, n (%)	45 (11.2)	50 (9.3)	0.332
Hypertension, n (%)	192 (47.9)	238 (44.2)	0.268
Diabetes, n (%)	50 (12.5)	105 (19.5)	0.004
FBG, mmol/L	5.47 (5.10–6.11)	5.63 (5.19–6.53)	0.002
ALT, U/L	16.18 (12.33–21.31)	15.07 (11.06–20.18)	0.006
AST, U/L	21.38 (19.00–26.00)	22.00 (18.52-27.00)	0.183
TBil, μmol/L	13.13 (10.93–16.90)	12.28 (9.50-15.94)	< 0.001
SCr, μmol/L	72.00 (53.50-85.57)	66.53 (51.86–83.55)	0.074
BUN, mmol/L	5.63 (4.59–6.44)	5.47 (4.60–6.97)	0.083
TC, mmol/L	4.74 (4.13–5.39)	4.93 (4.27–5.48)	0.002
TG, mmol/L	1.41 (1.04–1.95)	1.12 (0.89–1.60)	< 0.001
LDL-C, mmol/L	2.40 (1.88–2.78)	2.47 (1.88–3.08)	0.022
HDL-C, mmol/L	1.37 (1.21–1.56)	1.47 (1.24–1.74)	< 0.001
Dyrk1b, pg/mL	70.40 (58.34–92.35)	67.37 (55.13–82.56)	0.008
Grip, kg	28.10 (22.50–33.90)	17.90 (15.50-24.83)	< 0.001
Gait speed, m/s	1.05 (0.96–1.14)	0.95 (0.80–1.09)	< 0.001
ASMI, kg/m^2	7.10 (6.46–7.70)	5.62 (5.13–6.50)	< 0.001

ALT, alanine transaminase; ASMI, appendicular skeletal muscle mass index; AST, aspartate aminotransferase; BMI, body mass index; BUN, blood urea nitrogen; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Dyrk1b, Dual specificity tyrosine-phosphorylation-regulated kinase 1B; Scr, serum creatinine; TBil, total bilirubin; TC, total cholesterol; TG, triglyceride

calculate the correlation between clinical variables. Univariate and multivariate logistic regression analyses were used to identify variables contributing to the presence of sarcopenia. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. All tests were two-sided, and $P < 0.05$ was considered statistically significant. All analyses were performed using SPSS 28.0 (IBM, Chicago, IL).

Results

Baseline characteristics of the study participants

The study population included 939 older adults with 538 sarcopenia patients (Table 1). As expected, patients with sarcopenia had lower levels of ASMI, grip strength, and gait speed compared to older adults without sarcopenia ($P < 0.001$). Patients with sarcopenia were older, and had lower levels of BMI, ALT, TBil, and TG but higher levels of FBG, TC, LDL-C, and HDL-C compared with the individuals without sarcopenia. Furthermore, a higher proportion of patients with sarcopenia were suffered from diabetes compared to non-sarcopenic patients ($P = 0.004$). There were no significant differences between sarcopenic and non-sarcopenic patients in terms of sex, smoking, drinking, hypertension, AST, and renal function. Significantly, serum Dyrk1b levels were lower in patients with sarcopenia [median (IQR) = 67.37 (55.13–82.56) pg/mL] than those without sarcopenia [70.40 (58.34–92.35) pg/mL] ($P = 0.008$, Table 1).

Table 2 Spearman's correlation between serum Dyrk1b and clinical variables

Variables	Dyrk1b	
(*n* = 939)	*r*	*P*
Age	-0.092	0.005
BMI	0.022	0.491
FBG	-0.052	0.113
ALT	0.019	0.557
AST	-0.061	0.062
TBil	0.109	0.001
Scr	0.087	0.007
BUN	-0.135	< 0.001
TC	-0.099	0.002
TG	-0.045	0.165
LDL-C	-0.005	0.885
HDL-C	-0.148	< 0.001

ALT, alanine transaminase; ASMI, appendicular skeletal muscle mass index; AST, aspartate aminotransferase; BMI, body mass index; BUN, blood urea nitrogen; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Dyrk1b, Dual specificity tyrosine-phosphorylation-regulated kinase 1B; Scr, serum creatinine; TBil, total bilirubin; TC, total cholesterol; TG, triglyceride

Association of serum Dyrk1b with the risk of sarcopenia

To analyze the association between Dyrk1b and the risk of sarcopenia, we first examined the correlation between Dyrk1b and clinical variables. As shown in Table 2, serum Dyrk1b levels were negatively correlated with age, BUN, TC, and HDL-C but positively correlated with TBil and Scr. ROC curve analysis showed that the

best cut-off value for predicting sarcopenia was 44.73 pg/mL, with a sensitivity of 94.8% and a specificity of 14.7% (AUC = 0.577, 95% CI = 0.540–0.613, $P < 0.001$) (Figure S1A). Univariate and multivariate logistic regression analyses showed that high serum Dyrk1b levels (> 44.73 pg/mL) were significantly associated with decreased risk of sarcopenia both before and after adjustment for potential confounders including BMI, diabetes, FBG, and TG (Table **S2** and Table 3). Similar results were observed when serum Dyrk1b concentration was used as a continuous variable (adjusted OR = 0.984, 95% CI = 0.978–0.989, $P < 0.001$) (Table 3).

Table 3 Associations of serum Dyrk1b with the risk of sarcopenia

	Continuous		Categorical	
	OR (95% CI)	***P***	**OR (95% CI)**	***P***
Crude model	0.990 (0.984–0.995)	< 0.001	0.497 (0.286–0.866)	0.014
Adjusted model	0.984 (0.978–0.989)	< 0.001	0.342 (0.194–0.603)	< 0.001

The adjusted model included BMI, Diabetes, FBG, and TG

BMI, body mass index; CI, confidence interval; FBG, fasting blood glucose; Dyrk1b, Dual specificity tyrosine-phosphorylation-regulated kinase 1B; OR, odds ratio; TG, triglyceride

Stratification analyses for the association of serum Dyrk1b with the risk of sarcopenia

Stratified analyses were further conducted according to age, sex, diabetes, and BMI (Table 4). The association of high levels of Dyrk1b with decreased risk of sarcopenia remained significant in overweight (adjusted OR = 0.174, 95% CI = 0.075-0.400, $P < 0.001$) participants, as well as in males (adjusted OR = 0.158, 95% CI = 0.057–0.441, $P < 0.001$) and in females (adjusted OR = 0.566, 95% CI = 0.282–0.835, $P = 0.026$). By contrast, high serum Dyrk1b levels were associated with decreased risk of sarcopenia only in the older adults <80 years old (adjusted OR = 0.163, 95% CI = 0.076–0.348, $P < 0.001$) and those without diabetes (adjusted OR = 0.392, 95% CI = 0.217–0.710, $P < 0.01$).

Association of serum Dyrk1b with the severity of sarcopenia

Severe sarcopenia is recognized when there is low muscle strength, low muscle mass, and poor physical performance. We showed that serum Dyrk1b levels were positively correlated with ASMI, grip strength, and gait speed (Fig. 1A-C). Although there was no significant difference in the serum Dyrk1b levels between patients with moderate sarcopenia and control participants [70.40 (58.34–92.35) vs. 70.21 (55.10–90.40) pg/mL, $P = 0.122$], the serum Dyrk1b levels were much lower in patients

Table 4 Stratification analyses for the association of serum Dyrk1b with the risk of sarcopenia

	Continuous				Categorical			
Variables	**Crude OR (95% CI)**	***P***	**Adjusted OR (95% CI)**	***P***	**Crude OR (95% CI)**	***P***	**Adjusted OR (95% CI)**	***P***
Age								
< 80	0.984 (0.978–0.990)	< 0.001	0.979† (0.973–0.986)	< 0.001	0.182 (0.094–0.352)	< 0.001	0.163† (0.076–0.348)	< 0.001
≥ 80	0.999 (0.998–1.011)	0.905	0.999† (0.986–1.011)	0.845	1.004 (0.440–2.291)	0.004	1.266† (0.507–3.158)	0.614
Sex								
Male	0.985 (0.979–0.991)	< 0.001	0.979† (0.972–0.987)	< 0.001	0.150 (0.058–0.387)	< 0.001	0.158† (0.057–0.441)	< 0.001
Female	0.990 (0.981–0.998)	0.020	0.989† (0.979–0.999)	0.025	0.511 (0.276–0.945)	< 0.001	0.566† (0.282–0.835)	0.026
Diabetes								
With	0.995 (0.982–1.007)	0.375	0.997‡ (0.983–1.011)	0.656	1.248 (0.225–2.389)	0.493	1.022‡ (0.362–3.188)	0.836
Without	0.984 (0.979–0.990)	< 0.001	0.980‡ (0.974–0.987)	< 0.001	0.396 (0.236–0.664)	< 0.001	0.392‡ (0.217–0.710)	0.002
BMI								
< 24.0	0.983 (0.976–0.990)	< 0.001	0.983§ (0.976–0.990)	< 0.001	0.523 (0.273–0.904)	0.041	0.533§ (0.272–0.943)	0.046
≥ 24.0	0.990 (0.981–0.998)	0.014	0.986§ (0.977–0.995)	< 0.001	0.201 (0.089–0.456)	< 0.001	0.174§ (0.075-0.400)	< 0.001

BMI, body mass index; BUN, blood urea nitrogen; CI, confidence interval; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; hs-CRP, hypersensitive C-reactive protein; Metrnl, Meteorin-like; OR, odds ratio; TG, triglyceride

†The adjusted model included BMI, Diabetes, FBG, and TG

‡The adjusted model included BMI, FBG, and TG

§The adjusted model included Diabetes, FBG, and TG

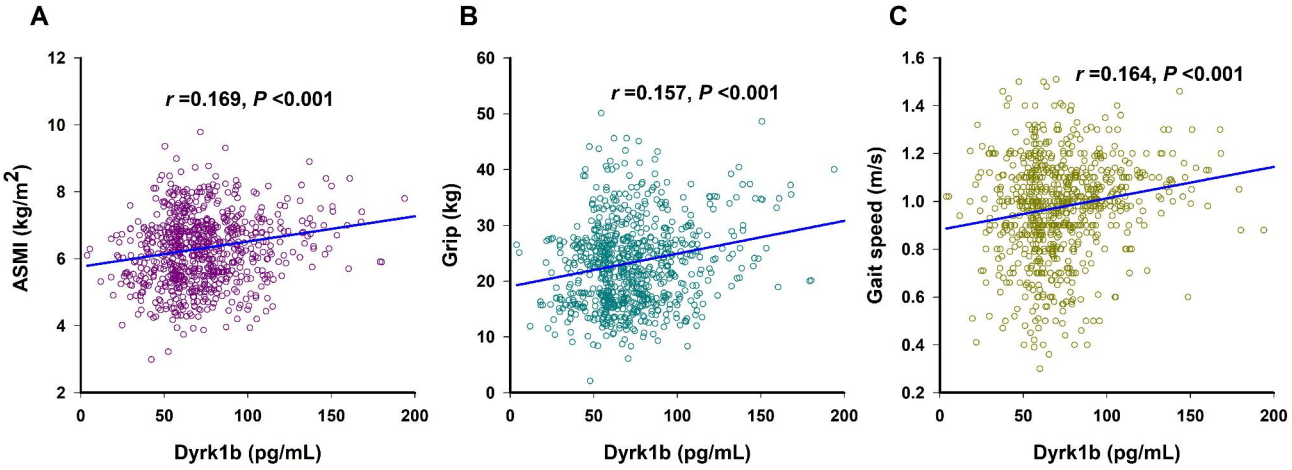

Fig. 1 Correlation of serum Dyrk1b level with ASMI (**A**), grip strength (**B**), and gait speed (**C**)

with severe sarcopenia when compared to those moderate sarcopenia [66.95 (55.12–79.09) pg/mL vs. 70.21 (55.10–90.40) pg/mL, $P = 0.008$, Figure S1B].

Discussion

This study found that lower serum Dyrk1b levels were related to an increased risk of sarcopenia in the elderly. In 939 community-dwelling older adults, serum Dyrk1b levels were positively associated with skeletal muscle mass, grip strength, and gait speed, components of sarcopenia. High serum Dyrk1b levels (> 44.73 pg/mL) were associated with decreased risk of sarcopenia in older adults.

Plasma Dyrk1b was previously detected to have no difference between patients with Alzheimer's disease compared to controls [22]. However, we showed here that serum Dyrk1b levels were lower in patients with sarcopenia compared to non-sarcopenic subjects. Furthermore, low serum Dyrk1b levels were positively correlated with decreased muscle mass, grip strength, and gait speed, suggesting a predictive role for Dyrk1b concentrations in muscle dysfunction. Indeed, despite the low specificity, the high sensitivity would provide a potential application by using serum level of Dyrk1b < 44.73 pg/mL as a cut-off value for the screening of sarcopenia in the elderly. Our data suggest that low serum Dyrk1b levels were associated with an increased risk of sarcopenia in older adults. Consistently, an in vitro study demonstrated increased Dyrk1b expression in endothelial cells after induction of senescence and suggested Dyrk1b as a possible senescence target in aging endothelial cells [18]. More prospective studies with larger sample sizes from different regions are required to confirm the predictive role of circulating Dyrk1b levels in the risk of sarcopenia.

Previous study has shown that Dyrk1b is highly expressed in skeletal muscle cells and plays a critical role in the muscle differentiation by regulating the motility, transcription, cell cycle progression, autophagy, and cell survival in skeletal muscles cells, thereby may participate in the development of sarcopenia [23]. Dyrk1b was induced within the first 24 h during the differentiation of C2C12 myoblasts [13]. Moreover, Dyrk1b could activate the MEF2C transcription factor to induce myogenin transcription by reducing the nuclear localization of class II histone deacetylases and promote muscle differentiation by activating the cell cycle inhibitors p21 and p27 in myogenic precursor cells in vitro [24, 25]. In addition, Dyrk1b was found to block G0 quiescence in cycling myoblasts by modifying cell cycle regulators [14] and inhibit apoptosis in fused myoblasts [14] but upregulate 4e-bp1 mediated autophagy [16] to enhance cell differentiation. Furthermore, skeletal muscle is the main organ for glucose uptake and metabolism. In the present study, we found that serum Dyrk1b levels were higher in patients with diabetes when compared to those non-diabetic individuals (median: 71.74 vs. 68.13 pg/mL, $P = 0.022$). Indeed, Dyrk1b could promote glycogen synthesis by promoting Ser640 phosphorylation in the skeletal muscle [26]. Hepatic Dyrk1b overexpression impaired systemic glucose homeostasis in mice by decreasing the Wbp2 expression in a kinase activity-dependent manner [27]. Therefore, Dyrk1b may link the skeletal muscle glycolytic metabolism with insulin resistance and consequently leading to metabolic syndrome [28–31]. In addition, Dyrk1b promoted astrocyte activation through interaction with STAT3 and acted as a key positive regulator in neuroinflammation [32]. Inhibition of Dyrk1b suppressed inflammatory responses and Th1/Th17 immune responses in allergic contact dermatitis models [33]. Considering sarcopenia is closely associated with chronic inflammation, insulin resistance, and metabolic syndrome [34], further in-depth studies will be needed to explore the exact mechanism by which Dyrk1b participates in the development of sarcopenia

through regulating skeletal muscle glucose metabolism and inflammatory response.

Our stratified analyses showed that the correlation between low Dyrk1b levels and the risk of sarcopenia was prominent among participants without diabetes and younger than 80 years. A recent clinical study identified the pathogenic, complete loss-of-function Dyrk1b variant as causing monogenic obesity associated with type 2 diabetes [35]. Indeed, diabetes itself may influence the association of Dyrk1b with sarcopenia, as insulin resistance is a common mechanism for both diabetes and sarcopenia [36]. This may partly explain why the correlation between serum Dyrk1b and the risk of sarcopenia was only observed in participants without diabetes. Furthermore, in our present study, Dyrk1b expression declined with age, which may therefore reduce the causal role of low Dyrk1b in the risk of sarcopenia in adults over 80 years old. However, the exact correlation between Dyrk1b levels and the risk of sarcopenia in older humans of different status remains to be further confirmed.

Study limitations

First, this observational study could not determine the causal relationship between the variables. Second, our study subjects are only Chinese, and we cannot therefore be certain that our results apply to other populations. Third, we did not test the correlation between serum Dyrk1b levels and inflammatory factors to test the hypothesis that low Dyrk1b concentrations may represent a chronic inflammatory state. Finally, although our findings hint that serum Dyrk1b may serve as a potential biomarker for sarcopenia, the AUC was only 0.577 (<0.7), with a sensitivity of 94.8% and a specificity of 14.7%, which has a relatively low predictive value.

Conclusions

In conclusion, our findings suggest that lower serum Dyrk1b levels are associated with the risk of sarcopenia in older populations. Future prospective studies are needed to confirm these findings and investigate potential mechanisms, including the role of Dyrk1b in muscle differentiation, inflammation, and glucose metabolism. In addition, given the low predictive value of Dyrk1b alone, future studies should explore the potential of combining Dyrk1b with other biomarkers to improve screening for sarcopenia.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12877-025-05942-5.

Supplementary Material 1

Supplementary Material 2

Author contributions

W.G., JQ.W. and L.L. contributed to the conception and design of the study. XF.J., GJ.M., C.Z. and WY.Z. contributed to data acquisition. W.G., X.L., and R.N. analyzed the data. XF.J., GJ.M., and WY.Z. drafted the manuscript. W.G. and L.L. revised the manuscript. All authors read and approved the final submission.

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Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Human ethics and consent to participate

This study was performed in accordance with the principles outlined in the Declaration of Helsinki and approved by the Ethics Committee of Sir Run Run Hospital, Nanjing Medical University (2019-SR-S041). Written informed consent was obtained from each participant.

Disclosure of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Clinical trial number

Not applicable.

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