



Associations Between Poor Appetite, Sarcopenia, and Cognitive Function in Community-dwelling Malaysian Older Adults

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Objectives: This study aimed to explore the associations between poor appetite, sarcopenia, and cognitive function among Malaysian older adults.

Methods: This nationwide study involved 1086 community-dwelling older adults aged 55 years and above. Poor appetite was defined using a self-reported question, while cognitive function was assessed with the Montreal Cognitive Assessment. Sarcopenia was identified based on handgrip strength, 6-meter gait speed, and muscle mass, in accordance with the Asian Working Group for Sarcopenia 2019 criteria. Associations between poor appetite, sarcopenia, and cognitive function were analyzed using both univariate and multivariate analyses.

Results: Multivariate analysis revealed that handgrip strength ($\beta=0.067$, $p=0.012$) and gait speed ($\beta=1.080$, $p=0.017$) were significantly associated with cognitive function after adjusting for confounders such as age, ethnicity, marital status, education, and alcohol and smoking consumption. However, no significant association was observed between poor appetite and cognitive function, nor was any moderation effect found between poor appetite and sarcopenia-related traits on cognitive function.

Conclusions: Our study confirms that low muscle strength and reduced physical performance are significantly associated with an increased risk of cognitive impairment among community-dwelling older adults. These findings underscore the critical importance of muscle strength and physical performance in preserving cognitive function—a decline that is not inevitable with age. Routine screening and early detection of muscle health and cognitive function are essential, and should be followed by intervention strategies targeting muscle health to mitigate cognitive decline in aging populations.

Key words: Appetite loss, Sarcopenia, Cognition, Muscle strength, Aged

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INTRODUCTION

With rising life expectancy, many countries are confronting the challenges associated with rapidly aging populations. The World Health Organization (WHO) estimates that by 2050, the global population of individuals aged 60 years and older will double, increasing from 1.0 billion in 2020 to 2.1 billion in 2050, which will account for 22% of the world's population [1].

Malaysia is experiencing a similar demographic transition, with the proportion of its population aged 65 years and above expected to rise from 7% to 14% between 2020 and 2043 [2]. This global demographic shift presents significant public health challenges, particularly regarding cognitive decline, nutritional deficiencies, and sarcopenia among older adults.

Sarcopenia is characterized by the progressive decline in skeletal muscle mass, strength, and physical performance. The prevalence of sarcopenia has increased substantially due to population aging, making it an urgent concern among older adults. Rates of sarcopenia vary widely across countries and ethnicities. Previous reports indicate a higher prevalence of sarcopenia in Asians compared to Caucasians, whereas individuals with darker skin exhibit significantly lower prevalence [3]. Emerging evidence suggests that sarcopenia and its related traits (such as low muscle mass, weak handgrip strength, and slow gait speed) are independently associated with mild cognitive impairment (MCI) [4-6].

MCI represents an intermediate stage between normal cognitive aging and early dementia, characterized by declines in cognitive domains such as memory, executive function, and attention, but without significant disruption of daily activities [4]. It is a critical predictor of dementia, with an annual conversion rate to Alzheimer's disease estimated at 5% to 10% [7]. The prevalence of MCI ranges from 9.9% to 18.1% among older adults, depending on the assessment tools and demographic characteristics used [6]. Studies have emphasized that MCI is associated with multiple risk factors, including inflammatory processes, hormonal dysregulation, and nutritional deficiencies [4,6].

Recent studies have highlighted an association between poor appetite and cognitive decline, especially in individuals with MCI and Alzheimer's disease [8]. Reduced meal intake may lead to inadequate nutrition, a weakened immune system, and impaired cognitive function, posing significant challenges to maintaining independent living. Several studies have shown a positive association between sarcopenia and cognitive decline [4-6], and this relationship may be moderated by nutritional status, as inadequate dietary intake can exacerbate both sarcopenia and cognitive decline.

Previous studies conducted in Malaysia have shown that sarcopenia is significantly associated with an increased risk of cognitive impairment [9,10]. However, existing research has primarily focused on the direct association between sarcopenia

and cognitive impairment, without adequately considering the moderating role of appetite. Poor appetite can lead not only to inadequate nutrition, but also to the onset of sarcopenia and subsequent cognitive impairment. For instance, in a Brazilian study, over 80% of older adults demonstrated nutritional inadequacy in energy, dietary fiber, and micronutrient intake [11]. Inadequate intake of energy, protein, and certain nutrients has been correlated with low muscle mass and strength among older adults [11]. Poor nutritional status affects not only physical health but also cognitive performance. Data from the Korean Brain Aging Study indicated that older individuals have lower appetite and a higher risk of malnutrition, and that the prevalence of dementia was significantly higher in the malnourished group [12].

To address these issues, the present study aimed to determine the associations between poor appetite, sarcopenia, and cognitive function among community-dwelling Malaysian older adults. Specifically, the objectives were to: (1) determine the prevalence of poor appetite, sarcopenia, and mild cognitive disorder in the Malaysian older adult population; (2) explore the relationship between poor appetite, sarcopenia traits, and cognitive decline; and (3) assess the moderating effect of poor appetite and sarcopenia-related traits on cognitive function. Based on existing literature and theoretical considerations, we hypothesize that: (1) poor appetite is negatively associated with cognitive function among older adults; (2) sarcopenia and its related traits (muscle mass, handgrip strength, and gait speed) are positively associated with cognitive function; and (3) poor appetite moderates the relationship between sarcopenia-related traits and cognitive function, such that the adverse effects of sarcopenia traits on cognitive performance are stronger in individuals with poor appetite.

METHODS

This study is a secondary data analysis of the Transforming Cognitive Frailty into Later-Life Self-Sufficiency (AGELESS) cohort, involving community-dwelling older adults aged 55 years and above recruited from both urban and rural regions via electoral rolls and sampling frames. For the purposes of this analysis, only individuals with complete data on appetite, anthropometry, sarcopenia indicators, and cognitive assessment were included. Subjects were excluded if they had missing data for any of the key variables of interest. The detailed methodology has been published elsewhere [13].

A structured questionnaire was used to obtain information on socio-demographic factors (e.g., age, sex, ethnicity, marital status, and highest education), anthropometric measurements (e.g., height, body mass index [BMI], waist, hip, and calf circumference), and lifestyle factors (e.g., past and current alcohol use and smoking). Poor appetite was defined by a self-reported question: "Have you been eating poorly because of a decreased appetite?" Cognitive function was assessed using the Montreal Cognitive Assessment (MoCA). MoCA is designed to evaluate various cognitive domains, including attention, concentration, executive function, memory, language, visuo-constructional skills, calculation, and orientation. A cutoff

score of 26 out of 30 was used to indicate normal cognitive function, with scores below 26 indicating potential cognitive impairment [14]. Although a universal MoCA cutoff of 26 was applied, it is recognized that educational attainment may influence cognitive performance; therefore, education level was included as a confounding factor in the multivariate analysis.

Sarcopenia was defined according to the Asian Working Group for Sarcopenia (AWGS), 2019 criteria [15], which include 3 main traits: low appendicular muscle mass, together with either low muscle strength or low physical performance. Appendicular muscle mass was estimated using an equation derived from

Table 1. Demographic and anthropometric characteristics of participants (n = 1086)

Characteristics	Overall (n = 1086)	Sarcopenia		p-value ¹
		With (n = 199)	Without (n = 887)	
Age (y)	69.29 ± 6.03	71.98 ± 6.77	68.69 ± 5.69	<0.001
Female sex	596 (54.9)	92 (46.2)	504 (56.8)	0.007
Ethnicity				0.036
Malay	91 (8.4)	11 (5.5)	80 (9.0)	
Chinese	832 (76.6)	150 (75.4)	682 (76.9)	
Indian	147 (13.5)	37 (18.6)	110 (12.4)	
Others	16 (1.5)	1 (0.5)	15 (1.7)	
Marital status				0.553
Single	121 (11.1)	22 (11.1)	99 (11.2)	
Married	802 (73.8)	152 (76.4)	650 (73.3)	
Divorced/Separated/Widowed/Other relationship	163 (15.0)	25 (12.6)	138 (15.6)	
Higher education (above high school)	707 (65.1)	115 (57.8)	592 (66.7)	0.017
Past and current alcohol use	544 (50.1)	92 (46.2)	452 (51.0)	0.228
Smoker	58 (5.3)	7 (3.5)	51 (5.7)	0.206
Height (cm)	160.28 ± 8.26	159.21 ± 8.14	160.52 ± 8.27	0.042
BMI (kg/m ²)	24.02 ± 3.86	20.39 ± 2.34	24.83 ± 3.66	<0.001
Obesity	388 (35.7)	5 (2.5)	383 (43.2)	<0.001
Waist circumference (cm)	87.18 ± 11.39	81.87 ± 10.21	88.37 ± 11.31	<0.001
Hip circumference (cm)	98.40 ± 8.89	91.61 ± 6.63	99.92 ± 8.62	<0.001
Calf circumference (cm)	34.75 ± 3.56	31.22 ± 2.32	35.55 ± 3.30	<0.001
ASMI (kg/m ²)	6.77 ± 1.17	5.76 ± 0.93	6.99 ± 1.10	<0.001
Gait speed (m/s)	0.96 ± 0.32	0.95 ± 0.36	0.97 ± 0.32	0.477
Handgrip strength (kg)	23.23 ± 7.88	20.42 ± 6.75	23.86 ± 7.99	<0.001
Poor appetite	73 (6.7)	26 (13.1)	47 (5.3)	<0.001
Total MoCA scores	27.50 ± 2.79	27.03 ± 3.12	27.60 ± 2.70	0.018
Cognitive function (MoCA)				0.021
Normal	901 (83.0)	154 (77.4)	747 (84.2)	
Mild cognitive impairment	185 (17.0)	45 (22.6)	140 (15.8)	
Sarcopenia (AWGS)	199 (18.3)			

Values are means ± standard deviation or number (%).

BMI, body mass index; ASMI, appendicular skeletal muscle mass index; MoCA, Montreal Cognitive Assessment; AWGS, Asian Working Group for Sarcopenia.

¹From the chi-square test for categorical data and independent *t*-test for continuous data.

the Kawakami muscle mass model, which is based on age, sex, and anthropometric measures such as weight, height, waist, and calf circumference. The equation has been validated against dual-energy X-ray absorptiometry measurements, demonstrating reliability for field-based assessments [16]. Low muscle mass was defined as appendicular skeletal muscle mass index (ASMI) $<7.0 \text{ kg/m}^2$ for male and $<5.4 \text{ kg/m}^2$ for female [16]. Handgrip strength was measured using a JamarTM analogue handheld dynamometer (Samson Preston, Bolingbrook, IL, USA), with the elbow adducted and flexed at a right angle. Participants were instructed to grip the dynamometer as hard as possible. Three attempts were allowed per hand, and the highest value from 6 trials was analyzed. Low muscle strength was defined as $<28 \text{ kg}$ for male and $<18 \text{ kg}$ for female. Physical performance was evaluated using 6-meter gait speed. Participants were instructed to walk at their usual pace, and the time taken to complete the distance was recorded to calculate gait speed in meters per second. Slow gait speed was defined as $\leq 1.0 \text{ m/s}$ for both male and female.

Statistical analysis was conducted using SPSS version 29.0 (IBM Corp., Armonk, NY, USA) with statistical significance set at $p\text{-value} < 0.05$. Univariate associations between demographic factors, anthropometric measures, lifestyle variables, poor appetite, sarcopenia, and cognitive function were analyzed using the chi-square test, binary correlations, and the independent-sample t -test. A generalized linear model was used to examine the relationship between poor appetite, sarcopenia, and cognitive function, adjusting for age, education level, marital status, ethnicity, alcohol consumption, and smoking status. Prior to applying the generalized linear model, multicollinearity was assessed using variance inflation factor (VIF) and tolerance values. All VIF values were below 2.0 and tolerance values above 0.5, indicating no concerns regarding multicollinearity among the independent variables. Moderation analysis was performed by including interaction terms between poor appetite and sarcopenia-related traits (e.g., ASMI $\times$ appetite, gait speed $\times$ appetite, handgrip strength $\times$ appetite) in the regression model.

Ethics Statement

Ethical approval was obtained from the Medical Research Ethics-Committee of the University Malaya Medical Centre (MREC ID No. 20191231-8121), and written informed consent was obtained from all participants prior to their inclusion.

RESULTS

General Characteristics of Study Participants and Correlates of Sarcopenia Risk

Table 1 presents the demographic and anthropometric characteristics of the participants. Data from 1086 older adults were included, with a mean age of 69.29 ± 6.03 years. Participants with sarcopenia were significantly older than those without sarcopenia ($p < 0.001$). The overall prevalence of sarcopenia was 18.3%, with a higher prevalence among males (21.8%) than among females (15.4%, $p = 0.007$) (Table 2). Sarcopenia was also associated with lower BMI, ASMI, waist, hip, and calf circumference (all $p < 0.001$), as well as shorter height ($p = 0.042$) and reduced handgrip strength ($p < 0.001$). Educational attainment differed significantly, with a higher percentage of participants without sarcopenia having attained education beyond high school (66.7 vs. 57.8%, $p = 0.017$). Poor appetite was more common among participants with sarcopenia (13.1%) than among those without (5.3%, $p < 0.001$). Table 2 further shows that male participants had significantly higher ASMI and handgrip strength (both $p < 0.001$). However, there were no sex differences in gait speed, appetite-related questions, or cognitive function, including MoCA scores and the prevalence of MCI. These findings highlight potential sex disparities in the preva-

Table 2. Distribution of participants according to appetite, sarcopenia components, and cognitive function (n = 1086)

Variables	Male (n=490)	Female (n=596)	p-value
Have you recently lost weight without trying?			0.543
Yes	49 (10.0)	59 (9.9)	
Unsure	13 (2.7)	23 (3.9)	
No	428 (87.3)	514 (86.2)	
Have you been eating poorly because of a decreased appetite?			0.988
Yes	33 (6.7)	40 (6.7)	
No	457 (93.3)	556 (93.3)	
ASMI (kg/m^2)	7.54 ± 0.88	6.14 ± 1.02	< 0.001
Gait speed (m/s)	0.96 ± 0.32	0.97 ± 0.32	0.678
Handgrip strength (kg)	28.52 ± 7.18	18.89 ± 5.40	< 0.001
Sarcopenia (AWGS)	107 (21.8)	92 (15.4)	0.007
Total MoCA scores	27.39 ± 2.64	27.59 ± 2.90	0.245
Cognitive function (according to MoCA)			0.463
Normal	402 (82.0)	499 (83.7)	
Mild cognitive impairment	88 (18.0)	97 (16.3)	

Values are presented as number (%) or mean $\pm$ standard deviation. ASMI, appendicular skeletal muscle mass index; AWGS, Asian Working Group for Sarcopenia; MoCA, Montreal Cognitive Assessment.

lence of sarcopenia and sarcopenia-related traits, but not in overall cognitive outcomes.

Cognitive Function and Correlates

Bivariate correlation analysis (Table 3) showed that cognitive function (assessed by MoCA scores) was inversely associated with age ($r=-0.266, p<0.001$), with younger participants demonstrating higher cognitive function. In contrast, cognitive function was positively correlated with gait speed ($r=0.203, p<0.001$), handgrip strength ($r=0.158, p<0.001$), calf circumference ($r=0.092, p=0.002$), and height ($r=0.099, p=0.001$). No significant associations were observed between BMI, waist circumference, hip circumference, or ASMI and cognitive function.

Table 3. Correlations between study variables and MoCA scores (n = 1086)

Variables	Cognitive function (MoCA)	
	r	p-value
Age (y)	-0.266	<0.001
Height (cm)	0.099	0.001
BMI (kg/m ²)	-0.014	0.65
Waist circumference (cm)	-0.029	0.336
Hip circumference (cm)	-0.016	0.588
Calf circumference (cm)	0.092	0.002
ASMI (kg/m ²)	0.034	0.264
Gait speed (m/s)	0.203	<0.001
Handgrip strength (kg)	0.158	<0.001

MoCA, Montreal Cognitive Assessment; BMI, body mass index; ASMI, appendicular skeletal muscle mass index.

Table 5. Predictors of cognitive function (n = 1086)

Predictors	df	β	MS	F	p-value
Age	1	-0.082	234.39	35.92	<0.001
Ethnicity	1	0.341	32.93	5.05	0.025
Marital status	1	-0.277	20.93	3.21	0.074
Education level	1	1.314	394.40	60.44	<0.001
Alcohol consumption	1	0.127	4.10	0.63	0.428
Smoking	1	-0.409	8.65	1.33	0.250
Poor appetite	1	-0.601	0.71	0.11	0.742
ASMI (kg/m ²)	1	-0.241	12.19	1.87	0.172
Gait speed (m/s)	1	1.080	37.15	5.69	0.017
Handgrip strength (kg)	1	0.067	41.72	6.39	0.012
Appetite*ASMI	1	-0.094	0.71	0.11	0.741
Appetite*Gait speed	1	-0.039	0.12	<0.01	0.966
Appetite*Handgrip strength	1	0.032	4.12	0.63	0.427

df, degrees of freedom; MS, mean square; ASMI, appendicular skeletal muscle mass index.

Table 4 shows that participants with higher education levels had significantly better cognitive performance (28.10 ± 2.16 vs. 26.36 ± 3.41 ; $p<0.001$). Participants with sarcopenia had lower MoCA scores compared to non-sarcopenic participants (27.03 ± 3.12 vs. 27.60 ± 2.70 ; $p=0.018$), which corresponded to a high-

Table 4. Comparison of study variables by MoCA scores (n = 1086)

Variables	n	Mean $\pm$ SD	t-value	p-value
Sex			-1.163	0.198
Male	490	27.39 $\pm$ 2.64		
Female	596	27.59 $\pm$ 2.90		
Education level			-9.013	<0.001
Below high school	379	26.36 $\pm$ 3.41		
Above high school	707	28.10 $\pm$ 2.16		
Past and current alcohol use			-2.596	0.010
No	542	27.28 $\pm$ 2.98		
Yes	544	27.72 $\pm$ 2.56		
Current smoking			0.328	0.743
No	1028	27.50 $\pm$ 2.81		
Yes	58	27.38 $\pm$ 2.43		
Obesity			0.671	0.502
No	698	27.54 $\pm$ 2.82		
Yes	388	27.42 $\pm$ 2.73		
Poor appetite			1.869	0.065
No	1013	27.55 $\pm$ 2.73		
Yes	73	26.77 $\pm$ 3.50		
Sarcopenia			2.390	0.018
No	887	27.60 $\pm$ 2.70		
Yes	199	27.03 $\pm$ 3.12		

MoCA, Montreal Cognitive Assessment; SD, standard deviation.

er prevalence of cognitive impairment among those with sarcopenia (22.6 vs. 15.8%, $p=0.021$), as shown in Table 1. Poor appetite was associated with lower mean MoCA scores, though this difference did not reach statistical significance (26.77 ± 3.50 vs. 27.55 ± 2.73 ; $p=0.065$) (Table 4).

Predictors of Cognitive Function

Table 5 summarizes the results of the general logistic regression analysis identifying factors associated with cognitive function. Age was a significant predictor, with older age associated with poorer cognitive performance ($\beta=-0.082$, $p<0.001$). Higher educational attainment contributed strongly to better cognitive outcomes ($\beta=1.314$, $p<0.001$), highlighting its protective effect against cognitive decline. Among sarcopenia-related factors, gait speed ($\beta=1.080$, $p=0.017$) and handgrip strength ($\beta=0.067$, $p=0.012$) were both positively associated with cognitive function, underscoring the impact of physical fitness on cognition. Other variables, such as ethnicity ($\beta=0.341$, $p=0.025$), also demonstrated significant associations, suggesting that cultural or genetic factors may influence cognitive outcomes. However, the interactions between poor appetite and sarcopenia-related traits (ASMI, gait speed, and handgrip strength) were not significant, indicating that appetite alone may not directly predict cognitive function when adjusting for other factors.

DISCUSSION

The current study found a prevalence of sarcopenia of 18.3%, which is consistent with other Asian studies, including Singapore (18.0%) [17], Korea (27.8%) [18], West China (19.31%) [19] and Thailand (16.1%) [20]. The higher prevalence among male also aligns with previous findings [5,19], likely due to age-related declines in testosterone and insulin-like growth factor-1. While female experience earlier muscle loss due to estrogen decline, male's later but more rapid hormonal reduction may contribute to accelerated muscle deterioration and increased risk of sarcopenia [21]. The prevalence of cognitive impairment (17.0%) aligns with global estimates, which report a range from 5.1% to 41.0%, with a median of 19.0% across 80 studies [22]. Additionally, our findings are consistent with data from 35 studies conducted in Asia, where the prevalence ranges from 6.5% to 37.0%, with a median of 19.4% [23]. These results suggest that our findings are within the expected range and comparable to both global and regional prevalence of MCI in older adults.

Consistent with our second objective, we observed that sarcopenia-related traits—specifically, low muscle strength and slow gait speed, were significantly associated with cognitive decline. These findings align with a meta-analysis of 15 cross-sectional studies, which reported a 2.25-fold increased risk of MCI among sarcopenic individuals [24]. The association may be underpinned by shared risk factors, including pathogenic mechanisms involving myokines, endocrine and inflammatory markers, as well as lifestyle factors. Age-related chronic inflammation, such as elevated levels of cytokines including IL-6 and TNF- α , has been linked to both muscle degradation and neurodegeneration [25]. However, there is regional variability, as some cross-sectional studies in Korea [26] and France [27], have reported inconsistent findings, suggesting that differences in region, study design, or population characteristics may influence results. These observations highlight the importance of considering sarcopenia in the prevention and management of cognitive decline in older adults. Nonetheless, regional discrepancies underscore the need for further research to better understand the underlying mechanisms and reasons for these differences, ultimately informing tailored interventions.

Our findings emphasize the greater importance of muscle strength and physical performance over muscle mass in predicting cognitive function. This aligns with previous studies indicating that muscle function, rather than muscle mass, is a more reliable indicator of cognitive health. For example, Chou et al. [28] demonstrated that lower handgrip strength was significantly associated with poorer cognitive performance, particularly in executive function tasks. Similarly, a systematic review reported a significant association between slower gait speed and worse cognitive function in community-dwelling older adults [29]. Declines in muscle strength may share common neurological mechanisms with slower reaction times, potentially occurring before measurable cognitive decline. Furthermore, mobility limitations can negatively impact cognitive function due to reduced social interactions and diminished participation in mentally stimulating activities [30]. These relationships are especially relevant in the Malaysian population, where many older adults experience reduced physical activity due to urbanization, sedentary lifestyles, and limited access to age-friendly infrastructure.

Sarcopenia is a complex condition. The AWGS 2019 and the European Working Group on Sarcopenia in Older People (EWGSOP) 2019 are the 2 principal guidelines shaping the understanding and diagnosis of sarcopenia. It is important to note

that, while the updated EWGSOP 2019 emphasizes reduced muscle strength rather than muscle mass, AWGS 2019 continues to prioritize low muscle mass as the primary and mandatory criterion for sarcopenia diagnosis. As both our findings and those of other studies have shown, muscle strength and physical performance, rather than muscle mass, are more strongly associated with cognitive health. The overemphasis on muscle mass in the AWGS framework may thus limit its clinical applicability, as it does not fully capture the broader and more dynamic nature of sarcopenia.

In this study, we examined both the potential mediating and moderating roles of poor appetite in the relationship between sarcopenia-related traits and cognitive function. Although poor appetite was more prevalent among participants with sarcopenia, its association with cognitive function (MoCA scores) did not reach statistical significance. Thus, the basic conditions for mediation were not met, and our findings do not support a mediating role of appetite in this relationship. We also tested moderation effects by including interaction terms between poor appetite and sarcopenia-related traits (e.g., ASMI, gait speed, and handgrip strength) in the regression models. However, none of the interaction terms were statistically significant. These results suggest that poor appetite does not significantly change the strength or direction of the relationship between sarcopenia-related traits and cognitive function in this sample.

This contrasts with previous findings that indicated nutritional status mediates the association between cognitive decline and sarcopenia [31,32]. This discrepancy may be attributed to the low prevalence of poor appetite in our study population (6.7%), which could limit the statistical power to detect a meaningful association. While the mechanisms remain to be elucidated, previous studies suggest that appetite changes with the progression of dementia, with appetite loss potentially being a consequence of dementia rather than a cause [33]. Sun et al. [33] postulated that appetite changes typically become more severe as dementia progresses, with appetite loss acting more as a consequence than a cause of cognitive decline, which is supported by another study [34]. It is important to note that in our study, individuals diagnosed with dementia were excluded. As the correlation between appetite and cognitive function is less likely to manifest in the earlier stages of cognitive decline, our focus on individuals with MCI or those at risk of cognitive decline may have reduced the ability to detect an association between appetite loss and cognitive decline. Fu-

ture studies should consider including a broader spectrum of participants, including those with advanced dementia, and should employ larger sample sizes and more sensitive measures of appetite and dietary intake to better elucidate the intricate relationships between poor appetite, sarcopenia, and cognitive decline.

Our study found that older adults with higher education levels and younger age exhibited better cognitive function. The results are consistent with previous studies [35,36]. More years of formal education are associated with a reduced risk of cognitive impairment, as individuals with higher educational attainment often have better health awareness, greater wealth, and more opportunities. Langa et al. [35] suggested that education directly affects brain development and function through multiple causal pathways, referred to as “cognitive reserve”. Aging results in structural and functional changes in the brain, including overall atrophy, particularly in the hippocampus, a key region for memory. These changes involve an imbalance in amyloid- β production and degradation, as well as activation of neuroinflammation, which exacerbates neuronal frailty in memory-related regions [36,37]. Ethnic differences in this study were also significantly associated with cognitive function, likely reflecting a combination of lifestyle factors, cultural practices, socioeconomic conditions, and genetic variations [38].

This study has several notable strengths. First, the large sample size ($n=1092$), combining 3 longitudinal studies of ageing in Malaysia, enhances the statistical power and generalizability of the findings to Malaysian older adults. Second, the comprehensive dataset included objective measurements of sarcopenia-related traits and standardized cognitive function assessments, allowing for robust analysis of their associations. Third, the adjustment for multiple confounders strengthened the validity of our findings. However, some limitations must be acknowledged. As a cross-sectional study, causal relationships between sarcopenia, appetite, and cognitive function cannot be established. Additionally, reliance on self-reported appetite data may introduce recall bias. The relatively low prevalence of poor appetite in the study population may have further reduced the statistical power to detect significant associations with cognitive outcomes. Finally, our analyses did not adjust for other important potential confounders, such as depression, functional status (activities of daily living), and nutritional status, which may be associated with both appetite and cognitive function. These issues should be addressed in future research.

In conclusion, this study found that sarcopenia-related factors, particularly reduced handgrip strength and slower gait speed, were significantly associated with cognitive function, whereas ASMI and poor appetite were not independent predictors. These findings highlight the importance of maintaining muscle strength and physical function for preserving cognitive health in older adults. Policymakers and healthcare practitioners should consider incorporating routine assessments of handgrip strength and gait speed into standard geriatric screenings. Additionally, community-based programs such as resistance training and balance exercises should be promoted to reduce the risk of cognitive decline, including dementia and Alzheimer's disease, among older adults. Although poor appetite was not independently associated with cognitive function in this study, it remains a critical clinical issue in aging populations. Regular appetite screening and timely nutritional support should continue to be integrated into geriatric care to support overall well-being.

NOTES

Conflict of Interest

The authors have no conflicts of interest associated with the material presented in this paper.

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