

Table 1 Participant characteristics by age half decade

		Overall			40–44 years			45–49 years			50–54 years			55–59		
		Men (<i>n</i> = 16 379)	Women (<i>n</i> = 21 660)		Men (<i>n</i> = 3697)	Women (<i>n</i> = 3828)		Men (<i>n</i> = 3151)	Women (<i>n</i> = 3686)		Men (<i>n</i> = 2202)	Women (<i>n</i> = 3597)		Men (<i>n</i> = 1952)	Women (<i>n</i> = 3002)	
		Mean	SD	% change over 40–44 years	Mean	SD	% change over 40–44 years	Mean	SD	% change over 40–44 years	Mean	SD	% change over 40–44 years	Mean	SD	% change over 40–44 years
Total SMI (kg/m ²)	Men	7.97	0.73	–	8.20	0.78	–	8.11	0.66	–1.0	8.11	0.67	–1.1	7.98	0.64	–2.7
	Women	6.26	0.64	–	6.41	0.67	–	6.39	0.64	–0.3	6.33	0.64	–1.3	6.23	0.59	–2.8
Arm SMI (kg/m ²)	Men	2.08	0.28	–	2.14	0.31	–	2.11	0.26	–1.4	2.11	0.26	–1.2	2.08	0.24	–3.0
	Women	1.47	0.22	–	1.49	0.24	–	1.49	0.23	–0.5	1.47	0.22	–1.4	1.46	0.21	–2.3
Leg SMI (kg/m ²)	Men	7.98	0.73	–	6.06	0.51	–	6.00	0.46	–0.9	5.99	0.46	–1.1	5.91	0.45	–2.5
	Women	6.26	0.64	–	4.92	0.48	–	4.91	0.45	–0.3	4.85	0.46	–1.3	4.77	0.42	–3.0
Visceral fat area (cm ²)	Men	100.6	29.2	–	88.4	28.8	–	91.9	27.1	4.0	98.9	28.8	11.9	103.5	25.7	17.1
	Women	84.7	27.4	–	68.0	25.3	–	72.1	23.9	6.0	79.3	23.6	16.5	89.4	23.0	31.5
		60–64 years			65–69 years			70–74 years			75–79 years			ANOVA		
		Men (<i>n</i> = 2274)	Women (<i>n</i> = 3490)		Men (<i>n</i> = 1683)	Women (<i>n</i> = 2314)		Men (<i>n</i> = 1030)	Women (<i>n</i> = 1269)		Men (<i>n</i> = 390)	Women (<i>n</i> = 474)		<i>F</i> -value	<i>P</i> -value	
		Mean	SD	% change over 40–44 years	Mean	SD	% change over 40–44 years	Mean	SD	% change over 40–44 years	Mean	SD	% change over 40–44 years			
Total SMI (kg/m ²)	Men	7.84	0.68	–4.3	7.64	0.67	–6.9	7.59	0.66	–7.4	7.32	0.62	–10.8	251.1	<0.001	
	Women	6.14	0.61	–4.2	6.08	0.60	–5.2	6.09	0.55	–5.1	6.00	0.60	–6.4	135.6	<0.001	
Arm SMI (kg/m ²)	Men	2.05	0.25	–4.4	1.99	0.25	–6.9	1.96	0.24	–8.5	1.87	0.26	–12.6	132.1	<0.001	
	Women	1.45	0.22	–3.1	1.44	0.21	–3.6	1.46	0.20	–2.5	1.43	0.21	–4.1	24.1	<0.001	
Leg SMI (kg/m ²)	Men	5.80	0.48	–4.3	5.64	0.46	–6.9	5.64	0.51	–7.0	5.45	0.45	–10.1	273.2	<0.001	
	Women	4.69	0.43	–4.6	4.64	0.44	–5.7	4.63	0.41	–5.9	4.57	0.45	–7.1	192.2	<0.001	
Visceral fat area (cm ²)	Men	108.3	26.2	22.5	113.0	25.7	27.8	122.3	25.1	38.3	126.4	25.2	42.9	376.9	<0.001	
	Women	94.0	23.3	38.2	101.6	23.0	49.4	108.5	24.1	59.5	112.4	29.3	65.3	966.7	<0.001	

Percentage change of 40–44 years = (absolute change value / 40–44 years value) × 100. SMI, skeletal muscle mass index.

Age-dependent decreases in skeletal muscle mass

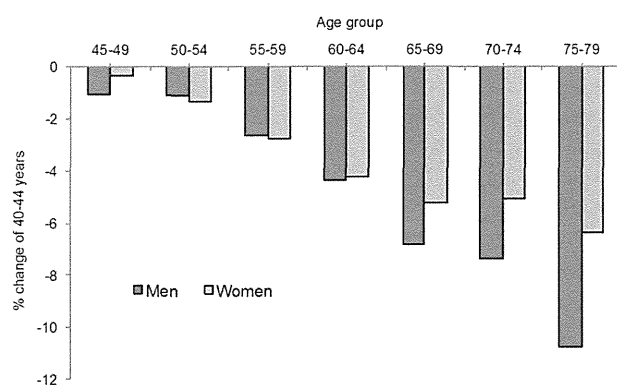


Figure 1 The percentage of change in the total skeletal muscle mass index in each sex and each age group, using 40–44 years-of-age as a reference.

Table 2 20th percentile of total skeletal muscle mass index (kg/m^2) in both sexes

Age group (years)	20th percentile of SMI	
	Men	Women
65–69	7.06	5.61
70–74	7.09	5.63
75–79	6.83	5.54
65–79	7.02	5.61

SMI, skeletal muscle mass index.

age-dependent decrease in both sexes (Fig. 2, Table 1). The age-dependent changes in the leg SMI were similar in men and women. However, the age-dependent changes in the arm SMI were greater in men than in women.

Next, we examined the age-dependent changes in visceral obesity. The visceral fat area showed an age-dependent increase in both sexes (men, $F = 376.9$, $P < 0.001$; women, $F = 966.7$, $P < 0.001$; Table 1). The percentage change from 40–44 years in the visceral fat area showed an age-dependent increase in both sexes (Fig. 3, Table 1).

To examine the association between skeletal muscle mass and visceral obesity, we carried out a multiple regression analysis using the SMI as an outcome. We found that the visceral fat area, age, and weight were significant and independent determinants of the SMI in both men ($\beta = -0.586$) and women ($\beta = -0.627$; Table 3). Therefore, the age-dependent change in the SMI was negatively associated with the visceral-fat area in both sexes.

Discussion

The current cross-sectional study was carried out to evaluate the SMI in Japanese adults aged between 40

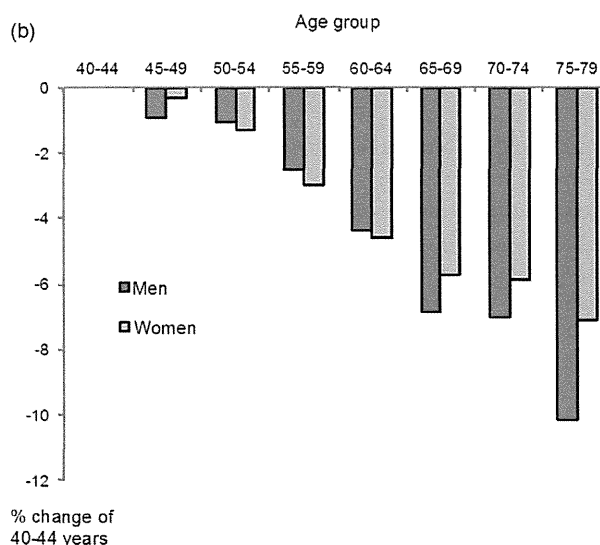
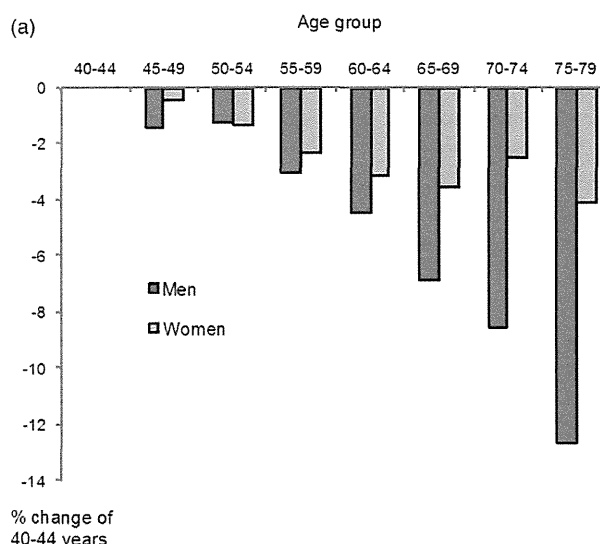


Figure 2 The percentage of change in the (a) arm and (b) leg skeletal muscle mass index (SMI) in each sex and each age group using 40–44 years-of-age as a reference.

and 79 years. Our data show that the SMI decreased age-dependently in both sexes. Notably, regarding the age-dependent decreases in the total SMI and in those aged over 65 years, the percentage change in the total SMI was greater in men than in women. From 40 to 79 years, the total SMI decreased by 10.8% in men and by 6.4% in women. Previous epidemiological studies of body composition have shown that between 40 and 79 years, the fat-free mass decreases by 6.6–23.3% in both sexes.^{19,20} The age-dependent increases in inflammatory cytokines, such as IL-6 and TNF- α , can result in increased skeletal muscle breakdown.²³ In contrast, the age-dependent decrease in anabolic hormones, such as testosterone, growth hormone, and insulin-like growth factor-1 (IGF-1), might lead to a loss of skeletal muscle

mass.^{27,28} In addition, there is also an age-dependent decrease in the amount of physical activity and energy intake. These behavioral changes can enhance the age-dependent reduction in skeletal muscle mass.

Interestingly, in those aged over 65 years, age-dependent decreases in total SMI were greater in men than in women. Furthermore, this age-dependent sex difference was more prominent in the arm than in the leg. From 40 to 79 years, the arm SMI decreased by 12.6% in men and by 4.1% in women. This is consistent to the previous studies in Japanese older adults. Kitamura *et al.* reported that the arm lean tissue mass was 5.97 ± 0.75 and 5.01 ± 0.67 in men, and 3.56 ± 0.54 and 3.24 in women aged in their 40s and 70s, respectively.²⁹ Based on their data, the percentage change in the arm lean tissue mass in men is -16.0% and is -8.9% in women. However, there is no sex difference in the percentage change in the leg lean tissue mass. The mechanism of this sex difference in the arm and leg lean tissue mass change is not clear. In general, older Japanese women frequently use the upper limbs, such as when washing and cooking. However, older Japanese men usually do not carry out such work. Therefore, it is

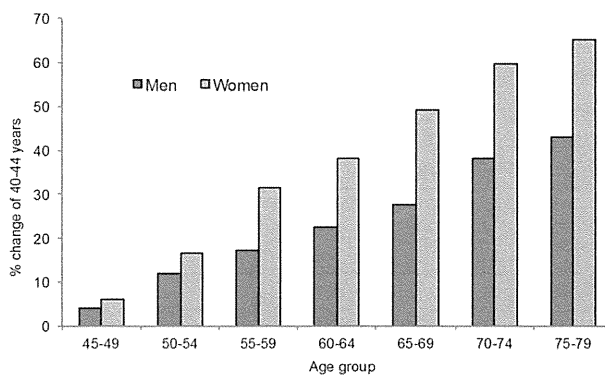


Figure 3 The percentage of change in the visceral fat area in each sex and each age group using 40–44 years-of-age as a reference.

possible that these behavioral differences lead to greater age-dependent decreases in the arm SMI in men than in women. As another possibility, Baumgartner reported that the sex hormone signal is an important factor for muscle mass in men, but not in women; however, physical activity is an important factor for muscle mass in both sexes.³⁰ Furthermore, previous studies have shown that 20% of men older than 60 years, 30% of men older than 70 years, and 50% of men older than 80 years have serum testosterone levels below the normal range.³¹ Thus, it is also possible that the sex hormone-dependent changes in muscle mass are greater in men than in women. Therefore, age-dependent gender differences in the SMI might be influenced by daily activity or alterations in sex hormone levels.

The present data show that aging is associated with a progressive increase in visceral fat area in both sexes. From 40 to 79 years of age, the visceral fat area increased by 42.9% in men and by 65.3% in women. Furthermore, the SMI was negatively associated with the visceral fat area when adjusted for age and body weight in both sexes. The visceral adipose tissue produces many catabolic factors, such as TNF- α and IL-6.²² Therefore, the age-dependent increases in both visceral adipose tissue and inflammatory cytokines might lead to a loss of skeletal muscle mass. Recently, sarcopenic obesity has been defined as both low muscle mass and high adipose tissue in older adults, and the health-related risk is higher in sarcopenic obesity than in sarcopenia.³² The current data show that the age-dependent changes in body composition can accelerate sarcopenic obesity. These results suggest that it is very important to begin prevention of sarcopenia and sarcopenic obesity as early as possible.

According to our analysis of this cohort, we found that the 20th percentile of total SMI in men and women aged 65–79 years was 7.02 kg/m^2 and 5.61 kg/m^2 , respectively. These values were slightly higher than those determined by the young adult mean in our database (men 6.75 kg/m^2 ; women 5.07 kg/m^2).¹⁸ That these values were lower than the 20th percentile of total SMI

Table 3 Multiple regression analysis for the association with skeletal muscle mass index in both sexes

Independent variables	Men Adjusted R^2 value = 0.781** standard regression value	Women Adjusted R^2 value = 0.627** standard regression value
Visceral fat area (cm^2)	-0.586^{**}	-0.627^{**}
Age (year)	0.212^{**}	0.252^{**}
Weight (kg)	1.180^{**}	1.169^{**}

** $P < 0.01$.

is probably because we did not use the data of SMI in participants aged 80 years and older. Other studies on sarcopenia in Asia also show that the cut-off of SMI is 6.08–7.27 kg/m² in men and 4.79–5.80 kg/m² in women,^{13,33–35} which is quite consistent with the present results. Thus, the 20th percentile of total SMI in men and women in our data can be used for the cut-off of SMI in Asians; however, further studies are required to address whether these cut-off points are associated with adverse health outcomes in Asian older adults.

There were several limitations to the present study that warrant mention. First, physical performance data were not measured. The European Working Group on Sarcopenia in Older People (EWGSOP) has recommended using the presence of both low muscle function (low physical performance or muscle strength) and low muscle mass to diagnose sarcopenia.³⁶ Therefore, the prevalence of sarcopenia could not be determined. Second, the study design was cross-sectional, and no outcome data are available. Further research with a longitudinal design will be required to clarify whether low muscle mass can predict adverse health outcomes in older Japanese adults. Third, the SMI measurement was estimated using BIA, which is not a method that is recommended by the EWGSOP for assessing muscle mass. However, it is very challenging to measure muscle mass in community-dwelling older adults using dual-energy X-ray absorptiometry (DXA); thus, BIA is a more practical screening method to use in large samples, especially in a community setting. However, to determine the specific effect of an intervention, a more accurate measurement, such as DXA, computed tomography, or magnetic resonance imaging, should be used in future studies. Serum outcomes were not measured. Therefore, the relationship between the SMI and hormone signals could not be determined. Finally, the participants in the present study were limited to visitors to fitness and community centers. Therefore, the participants of this study might not be a representative sample of community-dwelling adults.

In conclusion, the SMI showed an age-dependent decrease in both sexes, and the total SMI decreased by 10.8% in men and by 6.4% in women aged 40–79 years. Notably, age-dependent sex differences were more pronounced in the arm SMI; from 40 to 79 years, the arm SMI decreased by 12.6% in men and 4.1% in women. These results suggest that the age-dependent loss of skeletal muscle mass begins at approximately 40 years-of-age, and becomes prominent after 50 years-of-age in Japanese adults. Furthermore, the visceral fat area showed an age-dependent increase in both sexes, and the visceral fat area increased by 42.9% in men and by 65.3% in women of 40–79 years-of-age. Finally, the SMI was negatively associated with the visceral fat area in both sexes. Thus far, no studies have reported age-dependent changes and the association of muscle mass

and visceral fat in Asian populations. Therefore, the current data could be used as the reference value for Asian adults.

Acknowledgements

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Disclosure statement

The authors declare no conflict of interest.

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ORIGINAL ARTICLE

Arterial stiffness is associated with low skeletal muscle mass in Japanese community-dwelling older adults

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Aim: To examine whether arterial stiffness, measured by the cardio-ankle vascular index (CAVI), is associated with skeletal muscle mass index (SMI) in Japanese community-dwelling older adults.

Methods: Data were collected from 175 participants through questionnaires and specific tests; the data included demographic, lifestyle and health characteristics, body mass index (BMI), and body composition features determined by the bioelectrical impedance analysis, ankle-brachial index, the Mini-Nutritional Assessment, handgrip strength (GS), walking speed and shuttle walking tests (SW), and arterial stiffness determined by the CAVI. Absolute SMI was dichotomized according to the first quintile, which determined low ($n = 35$) and normal ($n = 140$) SMI.

Results: Participants with low SMI were older ($P = 0.01$), had more polypharmacy ($P = 0.01$), a lower BMI ($P < 0.001$), and fat mass index ($P = 0.02$), and had a greater risk of malnutrition ($P < 0.001$) than the normal group. Additionally, they showed poorer physical performance (GS and SW, $P = 0.007$ and 0.01 , respectively) than the normal group. Furthermore, CAVI was associated with SMI even after adjustments (OR 1.82, 95% CI 1.14–2.90, $P = 0.01$).

Conclusions: Our data showed that arterial stiffness is associated with low SMI in community-dwelling older adults, even when adjusting by multiple factors, showing a close interaction of vascular aging and muscle mass decline. *Geriatr Gerontol Int* 2014; 14 (Suppl. 1): 109–114.

Keywords: arterial stiffness, cardio-ankle vascular index, older adults, sarcopenia, skeletal muscle mass.

Introduction

The progressive loss of skeletal muscle mass is the primordial factor to determine sarcopenia, a syndrome that combines low skeletal muscle mass and strength, and can lead to adverse health outcomes, such as physical disability, poor quality of life and mortality.¹ In addition, the elderly might experience several adverse health outcomes as a result of vascular aging, such as increased arterial stiffness, which can contribute to the development of cardiovascular and cerebrovascular diseases.² It is known that both the loss of skeletal muscle mass and arterial stiffness worsen with age, and that some of the predisposing factors and mechanisms underlying

low muscle mass and sarcopenia; for example, oxidative stress,³ inflammation,⁴ and insulin resistance,¹ are also associated with atherosclerosis.⁵ However, only a few studies have been carried out to verify such associations.^{5–8} A study showed that arterial stiffness is associated with an increase in the loss of muscle mass index over time independent of age, body fat, peripheral arterial disease, chronic inflammation, and cardiac disease.⁶ Other studies have verified the associations of peripheral lean mass and visceral fat mass with atherosclerosis,⁷ and the relationships between regional fat and lean mass and large artery properties in young men and women.⁸

A novel measurement tool to assess arterial stiffness is the cardio-ankle vascular index (CAVI), which reflects the stiffness of the aorta, femoral artery, and tibial artery, and involves the measurement of the brachial-ankle pulse wave velocity (baPWV) and blood pressure (BP). The most important feature of CAVI is its independence from BP during examination,^{2,9,10} which shows that it is a useful tool to assess those who are

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subject to variation in blood pressure at different times of the day, suffer from masked hypertension or are taking antihypertensive medications.¹¹

Therefore, the objective of the present study was to examine whether arterial stiffness, measured by CAVI, is associated with skeletal muscle mass index (SMI) in Japanese community-dwelling older adults. We hypothesized that arterial stiffness is associated with a low SMI independent of sex, nutritional status, and physical performance in community-dwelling older adults.

Methods

The present study had a cross-sectional design.

Participants

The participants of the present study were community-dwelling Japanese older adults ($n = 175$; male = 84, female = 91) recruited through local press announcements requesting healthy volunteers. Recruited participants were 65 years-of-age or older, and were able to carry out the activities of daily living (ADL) and answer the proposed questionnaires. The exclusion criteria at the first screening were the following: (i) moderate cognitive impairment (i.e. Mini-Mental State Examination score ≤ 21 points); (ii) uncontrolled cardiovascular, pulmonary, or metabolic diseases; (iii) any orthopedic conditions that could restrain the ADL; and (iv) comorbidities associated with a greater risk of falls (e.g. Parkinson's disease and stroke). Additionally, in the present study, none of the participants had peripheral artery disease as evidenced by an ankle-brachial index (ABI) of less than 0.9. All participants were informed of the purpose and procedures of the study, and signed a written consent. The data were collected in November 2012.

The study protocol was approved by the Kyoto University Graduate School of Medicine Ethics Committee (No. E1245, E1583).

Assessments

The participants answered a self-administered questionnaire about demographic, lifestyle, and health characteristics, including age, regular physical activity engagement, alcohol consumption, smoking, current number of medications, and morbidities (i.e. diabetes, hypertension, hyperlipidemia and coronary artery disease; determined by the assumption that the prescribed medications they reported in the analysis were being used for the morbidity).

Additional relevant health indicators, such as (i) body mass index (BMI); (ii) body composition features determined by bioelectrical impedance analysis (Inbody 430;

Biospace, Seoul, Korea); (iii) ABI; and (iv) the Mini-Nutritional Assessment short-form (MNA)¹² were also collected.

The bioelectrical impedance instrument made use of octapolar tactile electrodes, two in contact with the palm and thumb of each hand, and two with the anterior and posterior aspects of the sole of each foot. The participants were instructed to stand with their soles in contact with the foot electrodes and to grasp the hand electrodes. The resistance of the arms, trunk, and legs was measured at frequencies of 5, 50, and 250 kHz. The participants' ID number, height (measured with a standard stadiometer), age, and sex were also inserted in the analyzer. Then, body mass and consequently BMI were automatically measured by the "InBody". For classification purposes, the BMI cut-offs used were those proposed by the Japan Society for Study of Obesity (i.e. underweight, BMI <18.5 kg/m², normal weight, BMI 18.5–25 kg/m², and obese, BMI ≥ 25 kg/m²).¹³ The bioelectrical impedance examination provided values for absolute skeletal muscle mass, body fat percentage, absolute fat mass, and segmental muscle mass (right and left arms/legs and trunk). From these measurements, absolute skeletal muscle mass and absolute fat mass were posteriorly adjusted by height to determine the SMI and fat mass index (FMI), respectively. The "InBody" system uses direct segmental multifrequency technology, and had previously been validated as having a strong correlation to muscle volume and fat mass as measured by dual energy X-ray absorptiometry.^{14,15}

Physical performance was investigated by the following: (i) handgrip strength (GS); (ii) walking speed (WS); and (iii) shuttle walking tests (SW).^{16,17}

GS was collected with a standard handgrip dynamometer (Smedley's Dynamo Meter; TTM, Tokyo, Japan). The participants were asked to stand up and hold the dynamometer with their arms parallel to their bodies without touching their bodies. GS was measured once for each hand, and the higher value was used to characterize his/her maximum muscle strength. GS was expressed in kilograms (kg).

In the WS test, outside marks of 12 m in length were clearly placed on the ground. Inside this distance, another 10 m long delimitation was marked. The participants were asked to walk the entire distance at their usual pace, but only the time to complete the inner 10 m distance was measured. Such measurement was intended to avoid the acceleration and deceleration stages of the participant's walking.

Finally, the SW test was carried out; two cones were placed 10 m apart. The participants were instructed to walk around the cones without stopping at a pace set by a timed signal played on a CD player. The SW test consists of 102 shuttles divided into 12 levels, each lasting approximately 1 min. The first level consists of three shuttles with a subsequent one-shuttle (i.e. 10 m)

increase at each following level. At each level, the speed is increased by 0.17 m/s, with an initial speed of 0.5 m/s rising to a maximum speed of 2.37 m/s.^{16,17} The test ended if the participant was unable to continue (due to breathlessness or any other reason) or was unable to reach the next cone before the timer sounded. If none of these mentioned factors occurred, we stopped the test at shuttle 50 (or 500 m, half of the total) to assure the participants' safety due to fatigue issues. Then, the values in meters were included in the analysis. A resting time of at least 3 min was provided between each assessment, and a longer time was provided if the participant claimed fatigue.

CAVI

CAVI was determined using VaSera1500 (Fukuda Denshi, Tokyo, Japan). The procedures started with the participants resting for 5 min in a sitting position. Afterwards, they were placed supine on a standard stretcher. Cuffs were wrapped around both arms and ankles to detect the brachial and ankle pulse waves. An electrocardiogram was carried out, and the heart sound was monitored. The pulse wave velocity (PWV) from the heart to the ankle was determined by measuring the length from the aortic valve to the ankle divided by time, according to the heart sound and the rise of the brachial and ankle pulse wave. The BP was measured at the four limbs alternately, first at the right arm and ankle, and then at the left arm and ankle. This procedure is important not only because it reduces the burden of the examinees, but also because it enables a more accurate measurement. Finally, a scale conversion was carried out using the following formula: $CAVI = a\{(2\rho/\Delta P) \times \ln(Ps/Pd)PWV^2\} + b$ (no unit), in which "ρ" is blood density, "Ps" is systolic blood pressure, "Pd" is diastolic blood pressure, "ΔP" is Ps – Pd, "PWV" is pulse wave velocity, and "a" and "b" are specific constants. This procedure has also been detailed in previous studies.^{2,10}

This measurement was carried out once for each participant, and the mean of the right and left values of CAVI for each participant was used for analytical purposes.¹⁸ The validity, reproducibility, and blood pressure-independent nature of this system have been widely documented by other researchers.^{2,9,10}

Statistical analysis

The Kolmogorov–Smirnov test was carried out to determine the normality of the data. Absolute SMI was dichotomized according to the first quintile for males (8.81 kg/m²) and females (7.57 kg/m²). Then, we arbitrarily assumed that those in the first quintile had a low SMI ($n = 35$), coded 1, and the others were considered normal SMI ($n = 140$), coded 0.

We analyzed the relationship between the two groups using the unpaired *t*-test for the age, BMI, body fat percentage, FMI, and ABI variables, and the Mann–Whitney *U*-test for the SMI, CAVI, GS, WS, and SW tests. Furthermore, the χ^2 -test was used for sex, regular physical activity engagement, alcohol consumption, smoking, number of medications, morbidities, and malnutrition. In addition, a univariate logistic regression was carried out to verify the association of each variable and the muscle mass condition, except for the number of medications as a result of missing values in the variable; then, a stepwise multivariate logistic regression was carried out to investigate whether CAVI was associated with low SMI. We assigned the status of muscle mass as the dependent variable, CAVI as the main covariate, and sex, age, BMI, MNA, GS, and SW as adjusted covariates. Differences were considered statistically significant at $P < 0.05$. All analyses were carried out using the Statistical Package for the Social Sciences software (SPSS; IBM, Chicago, IL, USA) version 20.0.

Results

A total of 175 subjects participated in the present study; we divided them into two groups: low SMI older adults ($n = 35$) and normal SMI ($n = 140$) participants. The participants in the first group were older and had more polypharmacy (four or more concurrent medications) than the normal participants. No significant differences were found for the lifestyle characteristics or morbidities (Table 1).

The results of the health indicators showed that low SMI participants had a lower BMI and FMI, and were at a higher risk of malnutrition than the normal group. Additionally, they presented with poorer physical functioning, such as low muscle strength and lower SW test scores. Regarding the CAVI results, the low SMI older adults had higher CAVI (Table 2).

The findings of the multivariate logistic regression showed that females were less likely to have low SMI than males. Similar conditions were verified in the participants with higher BMI and GS. Additionally, CAVI showed an independent association with SMI, even when adjusted for age, sex, BMI, MNA, GS, and SW. Thus, a higher CAVI was associated with low SMI in older adults (Table 3).

Discussion

The present study supported the hypothesis that arterial stiffness (assessed by CAVI) is associated with low SMI in community-dwelling older adults. Other studies have been carried out to ascertain this association;^{5–8} however, none of them considered arterial stiffness as measured by CAVI, a non-invasive and BP-independent tool.

Table 1 Bivariate comparisons of the participants' demographic and lifestyle characteristics

Variables	Normal (<i>n</i> = 140)	Low SMI (<i>n</i> = 35)	<i>P</i>
Age (years)	73 [70–77]	76 [71–78]	0.01
Females	52.1 (73)	51.4 (18)	0.94
Regular physical activity [†]	65.4 (83)	63.3 (19)	0.83
Alcohol consumption [‡]	39.4 (52)	36.7 (11)	0.78
Smoking [‡]	9.1 (12)	6.7 (2)	1.00
No. medications [§]	76.6 (95)	52 (13)	0.01
	23.4 (29)	48 (12)	
Morbidities [‡]			
Diabetes	12.1 (16)	10 (3)	1.00
Hypertension	42.4 (56)	36.7 (11)	0.56
Hyperlipidemia	14.4 (19)	20 (6)	0.41
Coronary artery disease	7.6 (10)	10 (3)	0.71

[†]*n* = 157; [‡]*n* = 162; [§]*n* = 149; values are expressed as medians [interquartile range] or valid percentages (*n*).

Table 2 Bivariate comparisons of the participants' health indicators

Variables	Normal (<i>n</i> = 140)	Low SMI (<i>n</i> = 35)	<i>P</i>
BMI (kg/m ²)	23.6 ± 2.71	20.7 ± 2.61	<0.001
SMI (kg/m ²)	9.00 [8.20–9.81]	7.54 [7.39–8.43]	<0.001
Body fat percentage	29.0 ± 7.72	28.1 ± 8.80	0.56
FMI (kg/m ²)	7.06 ± 2.37	6.03 ± 2.42	0.02
ABI	1.10 ± 0.07	1.08 ± 0.07	0.10
MNA at risk	20.0 (28)	51.4 (18)	<0.001
Handgrip strength (kg)	28.7 [25–35]	24.5 [22.5–31]	0.007
Walking speed (m/s)	1.39 [1.25–1.50]	1.39 [1.22–1.48]	0.48
Shuttle walking (m)	400 [360–470]	360 [300–440]	0.01
CAVI	9.13 [8.52–9.71]	9.57 [8.93–10.4]	0.008

Values are means ± SD, medians [interquartile range] or valid percentages (*n*). ABI, ankle-brachial index; BMI, body mass index; CAVI, cardio-ankle vascular index; FMI, fat mass index; MNA, mini-nutritional assessment; SMI, skeletal muscle mass index.

A previous study investigated the occurrence of a specific association between arterial stiffening (analyzed by baPWV) and peripheral skeletal muscle mass, and concluded that arterial stiffness was associated with a higher loss of muscle mass index over time independent of age, total body fat, peripheral arterial disease, chronic inflammation, or cardiac disease. Ochi *et al.* hypothesized that age-related decline of muscle mass and atherosclerosis share common pathological processes and interact with each other. In fact, the authors verified a direct association with baPWV and thigh muscle sarcopenia in men, but that association was not confirmed in women.⁵ Furthermore, Kohara *et al.* found that men with sarcopenic obesity had higher baPWV

than normal, sarcopenic, or obese men.¹⁹ In theory, changes in arterial stiffness might mediate the association between body composition and cardiovascular risk.⁸ However, it is unclear how arterial stiffness and the loss of muscle mass relate to each other. Authors suggested that because basal limb blood flow declines with aging, in part due to arterial stiffening, dysfunction in blood vessel dynamics could have a predictive role in muscle mass decline.⁶

Some researchers have linked the higher prevalence of low muscle mass in men²⁰ to their findings of arterial stiffness in men, but not in women.^{5,19} To examine any sex effect on CAVI, we carried out further analysis and verified that men had higher CAVI than women (data

Table 3 Stepwise multivariate logistic regression considering skeletal muscle mass index (normal or low condition) as dependent variable and cardio-ankle vascular index, age, gender, body mass index, Mini-Nutritional Assessment, handgrip strength, and shuttle walking as covariates

Variables	OR (95% CI)	P
Sex		
Female	0.23 (0.61–0.90)	0.03
BMI	0.71 (0.59–0.85)	<0.001
Handgrip strength	0.83 (0.74–0.94)	0.002
CAVI	1.82 (1.14–2.90)	0.01

Values are odds ratio (95% confidence interval). BMI, body mass index; CAVI, cardio-ankle vascular index; SMI, skeletal muscle mass index.

not shown). However, in our fully adjusted analysis, we verified the association of CAVI and low SMI independent of sex.

Regarding physical performance, those with normal muscle mass presented better physical functioning, such as a higher GS, higher SW test scores, and a lower CAVI, than the group with low SMI. GS is a representative measure of strength and is an important screening tool for sarcopenia,¹ whereas SW represents aerobic capacity.^{16,17} Regarding SW, a previous study showed that endurance-trained older men demonstrated lower arterial stiffness than their sedentary age peers despite similar systolic blood pressures, suggesting that age-associated augmentation of arterial stiffness might be mitigated by regular aerobic exercise.²¹

Furthermore, polypharmacy was observed more frequently among participants in the low SMI group. Although we did not deeply investigate the classes of medications to which they were exposed, our results were in agreement with previous studies that identified the association of concomitant medications and impaired physical functioning in older adults.²²

Based on the present results, we would like to emphasize the importance of physical activity, mainly the combination of progressive resistance exercise and aerobic exercise,²³ in accordance with well-balanced nutrition in relation to low SMI and arterial stiffness, especially because the participants with low SMI in the present study had lower physical performance, lower BMI, and a higher risk for malnutrition. Nutritional status is widely known to be associated with both muscular and vascular health. Thus, aiming to reverse low muscle mass, Yamada *et al.* verified that a diet rich in proteins and vitamin D in combination with resistance exercise was more effective at improving muscle mass than resistance exercise alone.²⁴ In addition, evidence showed that lower levels of 25-hydroxyvitamin D,

an established marker of vitamin D status, are associated with abnormalities in the indices of arterial stiffness.²⁵

Although the low SMI participants had lower BMI and FMI than the normal group, both groups presented similar results for body fat percentage. This result might show that lean body mass is lost, and fat could be preserved or even increased in people with low muscle mass or sarcopenia. As intramuscular and visceral fat increase, and subcutaneous fat decreases with age,^{1,4} the association with muscle mass decline and arterial stiffness might also be perceived from the standpoint of the relationship between fat mass and cardiovascular risks.

Some limitations of the present study should be mentioned: (i) its cross-sectional design did not permit the determination of a cause–effect relationship between CAVI and the low SMI condition; and (ii) the small number of participants limited further group subdivision (i.e. to differentiate pre-sarcopenic and sarcopenic older adults) as a result of the low statistical power achieved when further dividing the groups. However, the present study showed that a relationship between CAVI and low SMI does exist, and might serve as a basis for further studies with a larger sample size, analyzing the time effect on muscle, and physical performance decline, and also investigating the role of sex on such an association.

To our knowledge, this is the first study to verify the interaction of CAVI and total SMI in Japanese older adults. The main clinical advantage of the present study was that it clearly showed the important relationship between arterial stiffness and low SMI in community-dwelling older adults as measured by CAVI, a non-invasive reliable method and blood pressure independent measure. It would be useful to perform further health analyses in older adults with arterial stiffness, including body composition features and physical performance measurements, to aid in the early detection of people with the risk of developing sarcopenia; and also to verify arterial stiffness in older adults already in a progressive muscle loss condition. We believe that a suitable intervention for the promotion of improvements in vascular and muscular parameters would be aimed at increasing physical fitness levels and improving nutrition; this combined intervention might reduce the probability of a person developing systolic hypertension and the associated risk of cardiovascular events, and could help maintain SMI and function, especially in older adults.

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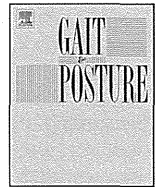
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Disclosure statement

The authors declare no conflict of interest.

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The association between fear of falling and gait variability in both leg and trunk movements



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ABSTRACT

The aim of this study was to explore whether FoF was associated with variability in both leg and trunk movements during gait in community-dwelling elderly. Ninety-three elderly people participated in this study. Each participant was categorized into either Fear or No-Fear group on the basis of having FoF. The participants walked 15 m at their preferred speed. The wireless motion recording sensor units were attached to L3 spinous process and right posterior surface of heel during gait. Gait velocity, stride time and stride length were calculated. Variability in lower limb movements was represented by coefficient of variation (CV) of stride time. Trunk variability was represented by autocorrelation coefficients (AC) in three directions (vertical: VT, mediolateral: ML and anteroposterior: AP), respectively. Gait parameters were compared between groups, and further analyses were performed using generalized linear regression models after adjustment of age, sex, fall experience, height, weight, and gait velocity. Although gait velocity, mean stride time and stride length did not differ significantly between groups, stride time CV and all ACs were significantly worse in the Fear group after adjustment for variables, even including gait velocity (stride time CV: $p = 0.003$, $\beta = -0.793$; AC-VT: $p = 0.011$, $\beta = 0.053$; AC-ML: $p = 0.044$, $\beta = 0.075$; AC-AP: $p = 0.002$, $\beta = 0.078$). Our results suggest that fear of falling is associated with variability in both leg and trunk movements during gait in community-dwelling elderly. Further studies are needed to prove a causal relationship.

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1. Introduction

Fear of falling (FoF) refers to a lack of self-confidence that normal activities can be performed without falling [1]. The prevalence of FoF ranges up to 60% in the community-dwelling elderly [2–4] and is even higher in given populations—especially in women or men with a previous history of falls [3]. Factors associated with FoF are psychological problems [5] and poor

physical performance [6,7]. Moreover, FoF results in limitations in activities of daily living (ADL) and decreased quality of life [8].

Most falls among older adults occur during movement, such as walking, and it is therefore important to assess the relationship between FoF and gait. Changes in gait that are associated with FoF in the elderly and have been reported consistently in previous studies are reduction in gait velocity [9–11], shortening of stride length [10–12], and increase in step width and prolongation of double-support time [10,11]. Gait variability, a measure of the consistency of movement [13], may provide a more sensitive measure of the risk of falls [14], functional decline, and various adverse health outcomes than do routine spatiotemporal measures such as gait velocity [15]. Gait variability is therefore used as a clinical index of gait stability [16]. The results of studies of the

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relationship between FoF and gait variability have been inconsistent. Reelick et al. [9] found that gait variability did not differ significantly between those with and without FoF. On the other hand, Rochat et al. [17] reported that FoF was associated with gait variability. The former conducted an analysis adjusted for gait velocity, whereas the latter did not. Gait variability was linked with gait velocity [18]. Beauchet et al. showed that walking at slow velocity increases stride-time variability [18]. The variability in these findings indicates that there is a need to clarify the association between FoF and gait variability, with adjustment for gait velocity.

To assess gait variability in the clinical setting, the body can be divided functionally into two units, namely “passenger” (head, neck, trunk, and arms) and “locomotor” (the two lower limbs and the pelvis) [19]. The trunk—a component of the passenger unit—sits upon the locomotor unit and acts mainly to help to maintain body equilibrium spatially during gait [20]. Propulsion of the body during gait is the primary role of the locomotor unit. Because the locomotor unit shifts constantly during gait, the trunk must maintain body equilibrium in these relatively unstable positions; therefore, the trunk movement during gait should be assessed. Moreover, the trunk, being the largest segment of the body, is easily influenced by inertial force from the movement of the locomotor unit and is itself unstable during gait. For these reasons, when gait variability is evaluated it is important to assess not only leg movements but also trunk movement during gait. However, few studies have explored the association between FoF and trunk movement during gait [9].

The aim of this study was to explore the cross-sectional association between FoF and gait variability, including both the temporal and spatial aspects of trunk movement, during gait in the community-dwelling elderly. Our hypothesis was that both lower leg and trunk movements during gait would be associated with FoF, independent of gait velocity.

2. Methods

2.1. Participants

We recruited elderly subjects who were community-dwelling and independent in ADL ($n = 120$). Inclusion criteria were age ≥ 65 years and the ability to walk independently without an assistive device; 119 participants met these criteria. Participants were excluded if they had a history of neuromuscular disease that affected gait or scored less than 8 on the Rapid Dementia Screening Test (RDST) [21]. In addition, participants who did not complete our assessment were excluded. There were 93 participants (38 men and 55 women) in the final analyzed sample (mean age [standard deviation: SD]; 73.1 [4.1] years; height, 155.2 [8.8] cm; weight, 56.5 [11.0] kg). Ethical approval for the study was given by the Ethics Committee of the Kobe University Graduate School of Health Sciences. All participants were properly informed about the study and signed written consent forms, in accordance with the Declaration of Helsinki, before their participation.

2.2. Fear of falling and other measures

FoF was assessed through the question “Are you afraid of falling? Yes – No”. Participants who responded “Yes” were assigned to the Fear group, and those who responded “No” were assigned to the No-Fear group. This format has the advantages of being straightforward and making it easy to generate prevalence estimates [22]. Fall events during the past 12 months were checked. We also assessed the following background characteristics by using a questionnaire: age, sex, number of years of education, self-reported medical history (arthritis, hypertension,

diabetes mellitus, heart disease, cardiovascular disease, respiratory disease), and number of medications. The Geriatric Depression Scale (GDS) [23], a 15-item yes/no questionnaire, was used to evaluate depression. Scores can range from 0 to 15, with higher scores indicating more depressive symptoms. Lower extremity performance was measured by using timed repeated chair stands (5-chair-stand test, 5CS) [24]. Participants were asked to stand up and sit down five times from a chair as quickly as possible, keeping their arms folded across their chests.

2.3. Gait measurement

Participants were instructed to walk at preferred speed along a 15-m smooth, horizontal walkway. A 10-m section of the walkway was marked off by two lines, one positioned 2.5 m from each end, to allow space and time for acceleration and deceleration. Walking time in the middle 10 m was measured with a stopwatch, and gait velocity was expressed in meters per second. Trunk and lower limb movement during gait was measured by using two wireless motion-recording-sensor units (MVP-RF8, MicroStone Co., Ltd., Nagano, Japan), one fixed to a belt at the level of the L3 spinous process and one attached to the posterior surface of the right heel with surgical tape. Acceleration and angular velocity could thus be measured without restricting the subject’s movement. We considered it likely that the accelerometers attached to the body would be in variable states of inclination caused the body’s curvature. To correct for any potential effects of this inclination, we calibrated the accelerometer before each walking trial to take into account the static gravity component. All signals were sampled at 200 Hz and synchronously wirelessly transferred to a personal computer via a bluetooth personal area network.

2.4. Data analysis

Signal processing was performed with MATLAB (The Math-Works Co., Release 2008, Cybernet Systems Co., Ltd., Tokyo, Japan). Before the analysis, all acceleration and angular velocity data were high-pass filtered with a cutoff frequency of 1 Hz and then low-pass filtered with a cutoff frequency of 20 Hz. To compute temporal gait parameters, we analyzed heel acceleration and heel angular velocity data. On the basis of pilot testing to determine temporal parameters by using heel acceleration data, a heel contact event was identified as a vertical acceleration peak. These events were used to calculate each stride time and to compute the mean stride time and the coefficient of variation (CV) of stride time. We used the CV of stride time to estimate the variability of lower limb movement as only a temporal parameter. The CV was calculated by using the formula: $CV = (\text{standard deviation}/\text{mean}) \times 100$. Stride length was computed by multiplying mean stride time by gait velocity. Because the CV of stride time was a measure of variability based on only a temporal parameter, we analyzed other measures of variability by using trunk acceleration to add a spatial element. Trunk acceleration data for each direction, namely vertical (VT), mediolateral (ML), and anteroposterior (AP), were analyzed to evaluate the variability of trunk movement, as computed by using an unbiased autocorrelation procedure [25]. An unbiased autocorrelation coefficient (AC) is an estimate of the regularity of a time series by cross-correlation with itself at a given time shift; it is independent of the amount of data managed. A perfect replication of the gait cycle signal between neighboring strides will return an AC of 1, and no association will give a coefficient of 0.

2.5. Statistical analysis

Characteristics of participants were compared between groups (No-Fear and Fear) by using a chi-squared test for categorical

Table 1
Characteristics of participants in each group.

	No-Fear (n = 72)	Fear (n = 21)	p-Value
Age (years)	72.5 [65–88]	73 [67–83]	0.394
Sex, female (%)	51.4	85.7	0.005
Height (m)	1.56 ± 0.09	1.51 ± 0.08	0.022
Weight (kg)	58.0 ± 11.4	51.2 ± 8.1	0.012
Number of comorbidities	1.4 ± 1.2	1.6 ± 1.0	0.454
Number of medications	2.2 ± 1.8	1.7 ± 1.4	0.267
Falling in past, fall ≥1 (%)	16.7	33.3	0.098
Education (years)	12.2 ± 2.7	11.2 ± 1.4	0.118
RDST	11 [8–12]	10 [8–12]	0.702
GDS	2.4 ± 2.4	2.7 ± 2.3	0.603
5CS (s)	8.5 ± 2.0	9.5 ± 3.2	0.088

Values are shown as mean ± SD for continuous variables except for age and RDST (these variables presented as median [minimum–maximum]); % for categorical variables.

RDST, Rapid Dementia Screening Test; GDS, Geriatric Depression Scale; 5CS, timed repeated chair stands.

Significant *p* values are <0.05.

variables, an unpaired *t*-test for parametric variables, and the Mann–Whitney *U* test for nonparametric variables. All gait parameters were explored for normal distribution. Gait parameters, except for stride time and stride length, could not have a normal distribution even after logarithmic transformation and were therefore treated as nonparametric parameters. Comparisons between groups were conducted by using an unpaired *t*-test for stride length and the Mann–Whitney *U* test for other gait parameters. Age, sex, and fall events in the past have been suggested as risk factors for the development of FoF [3], and gait parameters are influenced by gait velocity [18,25]. Hence, further analyses were performed by using generalized linear regression models after adjustment for age, sex, at least one fall in the past 12 months, gait velocity, and other variables that differed significantly between groups. FoF was taken as the main explanatory variable, whereas gait parameters were the outcome variables. The level of significance for all analyses was set at $p < 0.05$. All analyses were performed with SPSS 20.0.0 J for Mac (SPSS Japan Inc., Tokyo, Japan).

3. Results

3.1. Characteristics of participants

There were 21 participants (23%) in the Fear group and 72 (77%) in the No-Fear group. Participant characteristics for the two groups are summarized in Table 1. Age, number of comorbidities, number of medications, number of years of education, experience of at least one fall in the past 12 months, RDST score, GDS score, and 5CS score had no significant difference. The Fear group had a significantly higher proportion of females and was characterized by shorter height and lower weight than the No-Fear group.

3.2. Gait parameters in the No-Fear and Fear groups

Table 2 provides basic gait parameters and the CV of stride time for each group and compares them between groups. No significant differences were found in gait velocity and stride time between the groups ($p = 0.268$ and 0.268 , respectively). Participants with FoF had significantly shorter stride length and higher CV of stride

Table 2
Differences in gait parameters between groups (No Fear vs. Fear).

Gait parameters	Group		p-Value	Adjusted p-value [†]
	No Fear	Fear		
Gait velocity (m/s)	1.46 [0.91–1.95]	1.40 [0.94–1.79]	0.268	–
Stride time (s)	0.96 ± 0.08	0.94 ± 0.08	0.268	–
Stride length (m)	1.37 ± 0.13	1.29 ± 0.15	0.044	0.169
Stride time CV (%)	1.82 [0.25–4.88]	2.90 [0.84–5.09]	0.003	0.003

Values are shown as mean ± SD for parametrical parameters or as median [minimum–maximum] for non-parametrical parameters.

CV, coefficient of variation.

[†] Adjusted for age, sex, falls in the last 12 months, height, weight and gait velocity using generalized linear regression models.

Significant *p* values are <0.05.

time than those without FoF ($p = 0.044$ and 0.003 , respectively). After adjustment for age, sex, fall experiences, height, weight, and gait velocity, the difference in stride length between groups was not significant. However, the CV of stride time in the Fear group was significantly higher than that in the No-Fear group ($\beta = -0.793$; 95% CI [-1.312 to -0.273]). AC-VT, AC-ML, and AC-AP were also significantly worse in the Fear group than in the No-Fear group (median [minimum–maximum] AC-VT: 0.90 [0.63–0.97] and 0.83 [0.52–0.97], respectively, for the No-Fear and Fear groups, $p = 0.019$; AC-ML: 0.72 [0.34–0.93] and 0.60 [0.24–0.89], $p = 0.020$; AC-AP: 0.87 [0.62–0.98] and 0.77 [0.45–0.96], $p = 0.017$). The differences in ACs between the groups were significant even after adjustment for age, sex, fall experiences, height, weight, and gait velocity (AC-VT: $\beta = 0.053$, CI 0.012–0.093; AC-ML: $\beta = 0.075$, CI 0.002–0.147; AC-AP: $\beta = 0.078$, CI 0.028–0.129) (Fig. 1).

4. Discussion

The prevalence rate of FoF in this study was 22.3%. The Fear group had a higher proportion of females, who were shorter in stature and weighed less than those in the No-Fear group. There were no significant differences in age, number of comorbidities, number of medications, experience of at least one fall in the past 12 months, years of education, RDST score, GDS score, and 5CS score. Participants in the Fear group had significantly shorter stride length, and all parameters representing variability were worse than in the No-Fear group. Even after adjusting for gait velocity and several confounding factors such as age, sex, fall experience, height and weight, gait variability in both leg and trunk movement was still significantly worse in the Fear group.

Gait velocity did not differ significantly between groups. Previous studies, in contrast to our finding, have found that people with FoF walk slowly [9–11]. Moreover, it has been suggested that FoF prolongs the double-support time, as subjects attempt to increase their step width and shorten their stride length [9–12]. These changes are considered to arise for two reasons. The first is because of the fear itself [10], and the second is because of adaptation to the unstable gait induced by the FoF [9]. The difference in stride length between our groups agreed with the results of some previous studies. However, the difference in stride length between the groups was no longer significant after adjustment for variables, including anthropometric data and gait velocity. Stride length differs between genders, and this difference likely partly reflects the anthropometric disparities between the sexes [26]. We found here that mean height and weight differed significantly between men and women (height: 1.64 [0.06] m vs. 1.49 [0.05] m, $p < 0.001$; weight: 63.8 [10.6] kg vs. 51.4 [8.2] kg, $p < 0.001$). In accordance with the findings of other studies, the Fear group had a higher percentage of women than the No-Fear group; thus the difference in stride length between groups would have resulted from the difference between the two groups in the male to female ratio.

Variability in stride time was significantly greater in the Fear group than in the No-fear group. Reelick et al. [9] have reported similar results and have suggested that the increase in stride-time variability in people with FoF is largely a result of their reduced gait velocity. A relationship between gait velocity and gait variability has been reported elsewhere, namely that walking at slow velocity

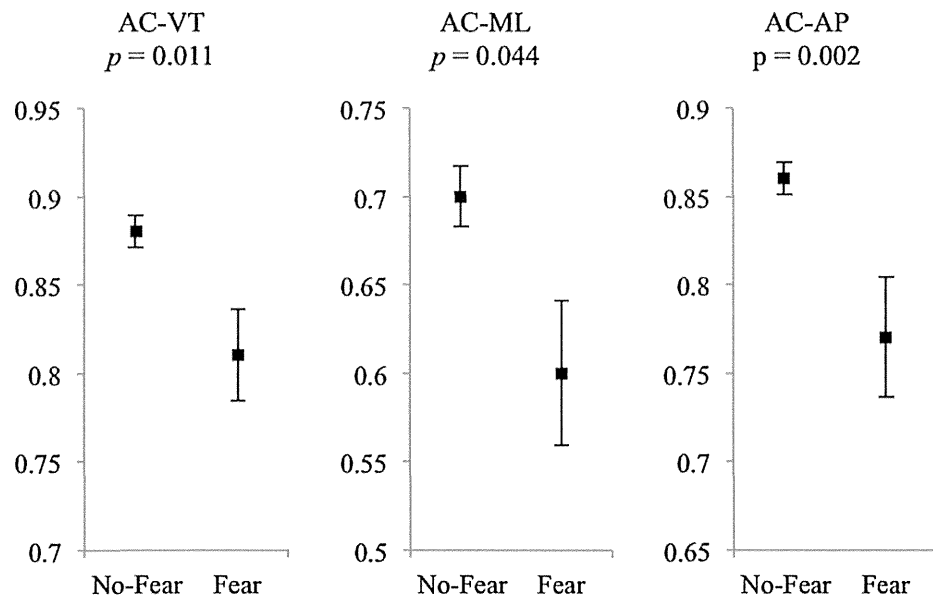


Fig. 1. The comparison of ACs in three directions between groups (No-Fear vs. Fear) adjusted by age, sex, fall experiences in past 12 months, height, weight and gait velocity using generalized linear regression models.

Squares represent mean values; error bars represent SE. AC, autocorrelation coefficient; VT, vertical; ML, mediolateral; AP, anteroposterior. Significant p values are <0.05 .

increases stride time variability [18]; in that study the between-group difference in gait variability was not significant after adjustment for gait velocity. However, we found no significant between-group difference in gait velocity. Additionally, stride-time variability in the Fear group was significantly greater than that in the No-Fear group, even after adjustment for gait velocity. This suggested that FoF influenced the variability of stride time independently of gait velocity. Although our participants were younger (mean age of all participants was 73.1 [4.1] years) and walked faster (mean gait velocity of all participants was 1.42 [0.19] m/s) than those in the study by Reelick et al. [9] (mean age 80.5 years; mean gait velocity, 0.98 m/s)—that is, our subjects might have had functioned better physically than the subjects in the other study—FoF still affected gait. Therefore, leg movement variability appeared more useful than gait velocity for measuring decreased gait performance due to FoF in our population of healthy elderly subjects.

FoF worsened trunk movement variability significantly in all directions, even after adjustment for variables, including gait velocity. The trunk includes 60% of the total body mass; with its high position relative to the feet it has a center of mass that is close to a point on the back posterior to the L3 segment. For successful locomotion it is therefore crucial to maintain trunk equilibrium through rhythmic leg movement. Although previous studies have investigated the influence of FoF on standing balance [22,27], few studies, to our knowledge, have explored whether FoF influences trunk movement during gait. One study reported that FoF reduced gait velocity but had no effect on trunk sway during gait [9]. However, that study investigated only the amplitude of trunk sway and only in the ML direction during gait. Three different deviations occur in the trunk during walking; we therefore examined trunk movement in all directions (VT, ML, and AP) by using AC to represent the variability in trunk movement with one stride. FoF decreased all ACs, even after adjustment for variables; that is, variability in trunk movement was independently increased by FoF. Increased rhythmicity in trunk movement during gait is associated with high AC values, whereas low AC values are associated with fall risk [28]. Our results build on the findings of Maki et al. [22] and suggest that FoF contributes to postural control of not only standing balance but also gait.

Our study had some limitations. First, FoF was assessed by using a simple closed-ended question. Other, more complex, FoF measurement tools have been developed in the past, and most consist of no fewer than 10 questions for assessing the degree of FoF [29]. Our study was unable to detect variability in the degree of FoF. However, the advantages of the simple question format we used were the direct nature of the question, which enabled us to rapidly evaluate FoF prevalence [22]. Our participants had to answer various other various questionnaires, which may have made them too tired to answer a long FoF questionnaire correctly. Hence, we needed simplicity to measure the prevalence of FoF (although this case could have been argued for any of our other questionnaires.) From this perspective, we consider the format that we used here to be accurate. Second, because our participants were mentally and physically healthy, it may not be possible to extrapolate our results to the elderly as a whole group. Further studies should be conducted on a wide range of participants, from the healthy elderly to the frail elderly, to determine whether our results can be generalized.

In conclusion, we demonstrated that participants with FoF have significantly greater variability, not only in leg movements but also in trunk movement, during gait than those without FoF. The most notable finding of our study is that FoF is associated with deterioration in variability in trunk movement during gait, even if other functions such as physical and cognitive functions are relatively unchanged. Further studies are needed to clarify the reciprocal relationship between FoF, gait variability, and potential for falls in the community-dwelling elderly.

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Conflict of interest statement

There is no conflict of interest.

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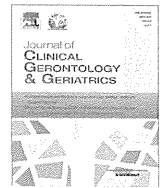
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Original article

Effect of physical activity at midlife on skeletal muscle mass in old age in community-dwelling older women: A cross-sectional study



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ABSTRACT

Background/Purpose: Measures to prevent the development of muscle mass decline should be initiated from midlife. However, the impact of physical activity at midlife on muscle mass in old age remains uncertain. The aim of this cross-sectional study was to determine whether physical activity at midlife influences muscle mass and physical performance in old age.

Methods: A total of 272 Japanese women aged 65 years and older were enrolled in the study. Information about physical activity levels at midlife and in old age were collected using a retrospective questionnaire. We calculated the skeletal muscle mass index in old age and recorded the participants' walking speed and hand grip strength in old age. We then classified the participants into four groups according to their physical activity levels at midlife and in old age and conducted multiple linear regression analysis to determine whether the physical activity levels at midlife and in old age were associated with skeletal muscle mass index and physical performance in old age.

Results: The participants in the groups that were physically inactive at midlife had a significantly lower skeletal muscle mass index in old age than those who were physically active at midlife ($p < 0.01$). Participants in the groups that were physically inactive in old age also had significantly slower walking speeds at old age than those who were physically active ($p < 0.01$). These associations remained significant after adjustment for age and body mass index.

Conclusion: Physical activity at midlife may be associated with a higher muscle mass in old age and physical activity in old age may be associated with higher walking speeds in old age.

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1. Introduction

Muscle mass declines at approximately 1–2% per year after the age of 50 years.¹ Longitudinal studies have shown a clear decline in muscle mass, strength, and power beginning at approximately 35 years of age.² The age-related loss of skeletal muscle mass induces an increased risk of falls and fractures, physical disability, mobility

disorders, and mortality.^{3,4} To promote healthy aging, it is therefore important to develop ways of preventing muscle mass decline.

The beneficial effect of physical activity in preventing adverse health outcomes is widely endorsed. There is growing evidence that older adults who engage in physical activity are more likely to experience better physical function and have a longer active life expectancy than sedentary older adults.^{5–7} Physical activity also has a positive impact on preventing muscle mass decline.⁸ Physical activity is one of the most important modifiable factors associated with the risk of chronic morbidity and high mortality in the general population.

Recent studies have shown an association between physical activity at midlife and functional and health status in old age. The level of physical activity at midlife was related to better physical

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health and functioning and lower mortality risk.^{9–12} Previous studies have also investigated the effects of midlife physical activity on different components of mobility^{13–15} and the risk of institutionalization.¹⁶ The benefits of physical activity at midlife appear to result from the maintenance of muscle strength,¹³ cognitive function,¹⁷ and other functions in old age. Furthermore, muscle mass in old age also appears to benefit from physical activity at midlife. Although it is important to prevent the development of muscle mass decline in old age and midlife, the effect of physical activity at midlife on muscle mass in old age remains uncertain.

The aim of this cross-sectional observational study was to determine whether physical activity at midlife was associated with muscle mass and physical performance in old age. We hypothesized that physical activity at midlife might prevent the decrease in muscle mass in old age.

2. Methods

2.1. Participants

Participants were recruited through a local press release requesting healthy community-dwelling volunteers. A total of 272 Japanese women aged 65 years and older (mean \pm SD age 73.6 \pm 5.5 years) living in the city of Kyoto enrolled in the study. Participants were interviewed and excluded if they met any of the following criteria: severe cognitive impairment; severe cardiac, pulmonary, or musculoskeletal disorders; and comorbidities associated with a greater risk of falls, such as Parkinson's disease and stroke. Written informed consent was obtained from each participant in accordance with the guidelines approved by the Kyoto University Graduate School of Medicine.

2.2. Assessment of physical activity

A questionnaire¹³ was used to collect retrospective information about physical activity levels during midlife and old age. In the present study, we defined midlife as the period between the ages of 40 and 65 years. The questions were: 'How much physical activity did you have during midlife?' and 'How much physical activity do you have these days?' Similar to the approach used in the previous study, there were three response categories: no regular physical activity (0); regular physical activity (1); and regular sports (2). Regular physical activity/sports were defined based on a previous study¹⁸ as activities/sports engaged in at a frequency of more than once a week. We defined light walking or moderate exercise (equivalent to less than approximately 4.0 metabolic equivalents) as physical activity and moderate or vigorous physical activities (equivalent to more than approximately 4.0 metabolic equivalents) as sports; these definitions were based on the International Physical Activity Questionnaire.¹⁹ For each of the midlife and old age physical activity levels, Category 0 was defined as 'inactive' and Categories 1 and 2 (combined) were defined as 'active' in the analyses.

2.3. Skeletal muscle mass index

A bioelectrical impedance data acquisition system (Inbody 430; Biospace Co. Ltd, Seoul, Korea) was used to perform bioelectrical impedance analysis.²⁰ This system also uses an electrical current at multiple frequencies (5, 50, 250, 500, and 1000 kHz) to directly measure the amount of extracellular and intracellular water. The participants stood on two metallic electrodes and held metallic grip electrodes. Using segmental body composition, muscle mass was determined and used for further analysis. The skeletal muscle mass index (SMI) was calculated by dividing the muscle mass by height

squared in meters (kg/m^2). This index has been used in several epidemiological studies.⁴

2.4. Measurements of physical performance

The following two measurements for the assessment of mobility and physical strength were made for each participant in the presence of experienced physiotherapists: (1) 10 m or 4 m walking test²¹; and (2) the hand grip strength (HGS) test.²²

In the walking test, participants were asked to walk 10 m or 4 m at their normal walking speed. Walking time was calculated using a stopwatch to record the time taken to cover the central 10 m or 4 m of the walkway (2 m at the start and finish were used for acceleration and deceleration). Using the better walking time of two trials, the participants' walking speed (m/s) was calculated to obtain values for analyses.

In the HGS test, participants used a hand-held dynamometer with the arm held to the side of the body. The participants squeezed the dynamometer with maximum isometric effort. No other body movement was allowed. The HGS score was defined as the better performance of two trials.

2.5. Assessment of sarcopenia

For the present study we adopted the criteria of the European Working Group on Sarcopenia in Older People (EWGSOP).²³ The EWGSOP recommended defining sarcopenia as the presence of both low muscle function (slow walking speed equal to or less than 0.8 m/s; or low HGS equal to or less than 20 kg) and low muscle mass. For assessing low appendicular muscle mass, we divided the SMIs of the participants into quartiles and defined the first quartile as the cutoff for low appendicular muscle mass (SMI 5.55 kg/m^2).

2.6. Statistical analysis

Before analysis, we classified the participants into four groups according to physical activity levels in midlife and old age: Group I, physically inactive at both midlife and old age; Group II, physically active at midlife, but not at old age; Group III, physically inactive at midlife, but active at old age; and Group IV, physically active at both midlife and old age (Fig. 1).

Differences in the demographic variables among the four groups were examined using analysis of variance (ANOVA). When a significant effect was found, differences were determined with the Tukey–Kramer's post-hoc test. In addition, we entered four

		At midlife	
		Inactive	Active
At old age	Inactive	Group I	Group II
	Active	Group III	Group IV

Fig. 1. Classification of participants in the four groups according to the midlife and old age physical activity levels: (Group I = physically inactive at both midlife and old age; Group II = physically active at midlife, but not at old age; Group III = physically inactive at midlife, but active at old age; Group IV = physically active at both midlife and old age).

dummy-coded groups, with Group IV as the reference group in models with independent variables; unadjusted and adjusted multiple linear regression analysis were conducted to determine whether physical activity levels in midlife and old age were associated with SMI and physical performance in old age. In the adjusted analyses, age and body mass index were entered as control variables.

Statistical analyses were carried out using the SPSS version 20.0 software package (SPSS, Chicago, IL, USA), with $p < 0.05$ accepted as significant.

3. Results

Table 1 shows the characteristics of the study population. The number (%) of participants in Groups I, II, III, and IV was 57 (21.0), 25 (9.2), 84 (30.9), and 106 (38.9), respectively. Participants in Group IV (SMI 6.35 ± 0.87 kg/m², walking speed 1.41 ± 0.26 m/s) (physically active at both midlife and old age) had significantly higher SMIs than those in Groups I (5.85 ± 0.92 kg/m², $p < 0.01$) and III (6.00 ± 1.08 kg/m², $p < 0.05$) (physically inactive at midlife) and faster walking speeds than those in Groups I (1.30 ± 0.25 m/s, $p < 0.05$) and II (1.27 ± 0.27 m/s, $p < 0.05$) (physically inactive at old age) (Table 1). There was no other significant difference among the four groups. A total of 38 (14.0%) participants had sarcopenia: 10 of 57 (17.5%), 3 of 25 (12.0%), 16 of 84 (16.7%), and 9 of 106 (8.5%) participants in Groups I, II, III, and IV, respectively.

In the unadjusted multiple linear regression analysis with Group IV as the reference, older adults within Groups I and III showed a significantly lower SMI ($p < 0.01$) and older adults in Groups I and II showed a significantly slower walking speed ($p < 0.01$) (Table 2). Thus participants who were physically inactive at midlife (Groups I and III) had a significantly lower SMI and participants who were physically inactive in old age (Groups I and II) had a significantly slower walking speed. These associations remained significant after adjustment for age and body mass index ($p < 0.05$) (Table 2). However, no group showed significant associations with HGS in the unadjusted and adjusted analysis.

4. Discussion

This is the first cross-sectional study to attempt to clarify the relationship between physical activity levels at midlife and skeletal muscle mass in old age. This study showed that older adults who were physically active at midlife might have a higher skeletal muscle mass in old age than those that were not physically active at

midlife. A previous study reported that the rate of lean mass loss was about three times less than the rate of decline in leg strength.²⁴ Our results for the relationship between physical activity at midlife and skeletal muscle mass appear to be consistent with the previous study. In addition, the previous study reported that the exercise-induced increase in muscle mass was typically less than that expected for the concomitant increase in strength.²⁵ Therefore physical activity at midlife may be important and beneficial for preventing muscle mass decline in old age.

Muscle mass is controlled by catabolic and anabolic factors. A previous cohort study showed that regular physical activity was associated with low levels of catabolic markers such as interleukin-6.²⁶ In addition to its effects on catabolic factors, an increase in physical activity was associated with a high level of insulin-like growth factor-1, one of the most important factors linked to intensifying muscle mass in premenopausal women.²⁷ These results suggest that continuous regular physical activity prevents catabolic effects and promotes anabolic effects. However, there are no longitudinal reports that have reported an association between these factors and muscle mass from midlife to old age. On the basis of our preliminary results regarding the relationship between physical activity at midlife and skeletal muscle mass, further studies are required to confirm the benefits of physical activity from midlife for the prevention of muscle mass decline.

Our study also showed that adults physically active in old age might have a faster walking speed than those who were not physically active in old age. In addition, physical activity at midlife and in old age was not associated with grip strength in old age. Hughes et al.²⁸ reported longitudinal changes in muscle mass, physical activity, and muscle strength and found that muscle mass decline explained only 5% of the decline in strength. Further, the changes in strength were no different between people of middle and old age who reported taking regular exercise in the past compared with those who had not exercised regularly in the past. These are the reasons why the relationship between physical activity and physical performance has different trends from that between physical activity and skeletal muscle mass. Furthermore, we observed significantly lower SMIs in Group III participants and slower walking speeds in Group II participants compared with Group IV, although there was no difference in muscle mass and physical performance between Groups II and III. These results seem to indicate that physical activity at midlife and old age may affect skeletal muscle mass and physical performance in old age. However, a previous longitudinal prospective study of the association between physical activity at midlife and walking speed²⁹ reported

Table 1
Demographic differences according to physical activity levels at midlife and old age.

	Total (n = 272)	Physical activity levels at midlife and old age				p	Post-hoc
		Group I (n = 57)	Group II (n = 25)	Group III (n = 84)	Group IV (n = 106)		
Age (y), mean ± SD	73.6 ± 5.5	74.1 ± 6.2	75.0 ± 5.2	74.0 ± 5.5	72.7 ± 4.9	0.146	—
Height (cm), mean ± SD	151.2 ± 5.4	151.1 ± 5.2	153.9 ± 5.4	150.9 ± 4.8	150.7 ± 5.9	0.088	—
Weight (kg), mean ± SD	49.7 ± 7.5	48.8 ± 6.9	51.7 ± 8.4	49.5 ± 7.6	49.9 ± 7.3	0.459	—
BMI (kg/m ²), mean ± SD	21.7 ± 2.9	21.4 ± 2.7	21.7 ± 2.8	21.7 ± 3.0	22.0 ± 2.8	0.653	—
SMI (kg/m ²), mean ± SD	6.11 ± 0.92	5.85 ± 0.92	6.14 ± 0.82	6.00 ± 1.08	6.35 ± 0.87	0.004	***
Walking speed (m/s), mean ± SD	1.35 ± 0.25	1.30 ± 0.25	1.27 ± 0.27	1.34 ± 0.23	1.41 ± 0.26	0.010	††
HGS (kg), mean ± SD	22.1 ± 6.7	21.3 ± 3.5	21.4 ± 7.5	22.2 ± 10.2	22.5 ± 6.8	0.672	—
Sarcopenia, n (%)	38 (14.0)	10 (17.5)	3 (12.0)	16 (16.7)	9 (8.5)		

Group I = physically inactive at both midlife and old age; Group II = physically active at midlife, but not at old age; Group III = physically inactive at midlife, but active at old age; Group IV = physically active at both midlife and old age; BMI = body mass index; HGS = hand grip strength; SMI = skeletal muscle mass index.

*Significant difference between Group IV and Group I ($p < 0.01$).

**Significant difference between Group IV and Group III ($p < 0.05$).

†Significant difference between Group IV and Group I ($p < 0.05$).

††Significant difference between Group IV and Group II ($p < 0.05$).

Table 2
Association of physical activity status with skeletal muscle index and physical performance in old age.

Dependent variable	Unadjusted model			Adjusted model		
	β	95% CI	Adjusted R^2 value	β	95% CI	Adjusted R^2 value
SMI			0.05			0.35
Group I	-0.22	-0.80 to -0.21**		-0.16	-0.61 to -0.11**	
Group II	-0.06	-0.62 to 0.21		-0.03	-0.48 to 0.24	
Group III	-0.18	-0.61 to -0.09**		-0.15	-0.50 to -0.07**	
Group IV	Reference			Reference		
Walking speed			0.05			0.17
Group I	-0.18	-0.19 to -0.03**		-0.17	-0.19 to -0.03**	
Group II	-0.18	-0.28 to -0.05**		-0.14	-0.24 to -0.02*	
Group III	-0.12	-0.14 to 0.01		-0.09	-0.12 to 0.02	
Group IV	Reference			Reference		
HGS			0.01			0.07
Group I	-0.08	-3.47 to 0.93		-0.06	-3.16 to 1.30	
Group II	-0.06	-4.51 to 1.66		-0.03	-3.93 to 2.30	
Group III	-0.02	-2.23 to 1.69		-0.01	-1.84 to 2.05	
Group IV	Reference			Reference		

Note: In the adjusted analysis, age and BMI were entered as control variables. Group I = physically inactive at both midlife and old age; Group II = physically active at midlife, but not at old age; Group III = physically inactive at midlife, but active at old age; Group IV = physically active at both midlife and old age; β = standard regression coefficient; CI = confidence interval; HGS = hand grip strength; SMI = skeletal muscle mass index.

* $p < 0.05$.

** $p < 0.01$.

results which were different from the present study. This may in part be because: (1) our assessment of physical activity was retrospective; (2) our questionnaire was not a particularly detailed assessment of physical activity as it did not contain items addressing the continuance and intensity of physical activity; and (3) the present study was cross-sectional. These may be the main reasons why our results differ from previous studies. In future studies, details regarding the level of physical activity at midlife and in old age must be collected to better understand how physical activity at midlife affects physical performance.

Many research groups have recently defined sarcopenia as the coexistence of low muscle mass and low physical performance.^{23,30,31} The evidence-based clinical effect of physical activity on the prevention of sarcopenia has also been reported from multiple points of view.⁸ The present study showed the relationship between physical activity at midlife and skeletal muscle mass as well as between physical activity in old age and physical performance, and suggested that continued physical activity from midlife to old age might be one of the important factors for the prevention of sarcopenia in old age. The benefits of constant physical activity for various health improvements are well known. Additional studies are required to determine the benefits of physical activity over the life course, not only in terms of various health improvements, but also for the prevention of sarcopenia.

There were several limitations to the present study. Firstly, this study was cross-sectional and we included no information on the effect of continuous regular physical activity from midlife to old age in the questionnaire. A longitudinal prospective study is therefore needed to confirm these results and extend the present study. Secondly, our assessment of physical activity at midlife and old age was conducted using a very simple questionnaire and was based on the participants' ability to recall information. Thirdly, the findings in the present study should be considered as preliminary due to the relatively small sample size, which may introduce some error of inference, reduce the power of the analysis, and limit generalization. Finally, we did not collect any information about comorbidity or current treatment with drugs for our participants.

In conclusion, the results of our study suggest that physical activity at midlife may be associated with high muscle mass in old age and that physical activity in old age may be associated with a fast walking speed in old age. The present study seems to be a fundamental study to determine the benefits of physical activity over the life course for the prevention of sarcopenia.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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