BJMB
Brazilian Journal of Motor Behavior

Special issue:
Effects of aging on locomotor patterns

Gait velocity and stability are correlated to muscle and bone mass loss in people with Parkinson's disease: a preliminary study

FABIO A. BARBIERI[1] | MURILO H. FARIA[1] | LUCAS SIMIELI[1] | TIAGO PENEDO[1] | CARLOS A. KALVA-FILHO[1] | VICTOR S. BERETTA[2]

[1] São Paulo State University (Unesp), School of Sciences, Department of Physical Education, Human Movement Research Laboratory (MOVI-LAB), Bauru, SP, Brazil.
[2] São Paulo State University (Unesp), School of Technology and Sciences, Physical Education Department, Presidente Prudente, São Paulo, Brazil.

Correspondence to: Prof. Dr. Fabio Augusto Barbieri, São Paulo State University (Unesp), School of Sciences, Department of Physical Education, Human Movement Research Laboratory (MOVI-LAB).
Av. Eng. Luiz Edmundo Carrijo Coube, 14-01-Vargem Limpa, Bauru/SP, CEP: 17033-360, Brazil.
Phone: +55 14 3103-9612
email: fabio.barbieri@unesp.br
https://doi.org/10.20338/bjmb.v16i5.346

HIGHLIGTHS

- Reduced lean and bone mass were related to gait deficits in Parkinson's disease.
- Reduced lean and bone mass were related to slower gait speed in Parkinson's disease.
- Bone mass loss may cause gait instability in people with Parkinson's disease.
- Body composition should be monitored over the gait disability in Parkinson's disease.

ABBREVIATIONS

C7	7th cervical vertebra
DXA	Dual-energy X-ray
H&Y	Hoehn & Yahr scale
MMSE	Mini-Mental State Examination
ON	One hour after the participants had taken their dopaminergic medication
PD	Parkinson's disease
UPDRS-III	Motor section of the Unified Parkinson's Disease Rating Scale

PUBLICATION DATA
Received 10 11 2022
Accepted 10 12 2022
Published 15 12 2022

BACKGROUND: Parkinson's disease (PD) exacerbates muscle and bone mass loss, which is associated with several negative outcomes such as falls and disability. Thus, muscle and bone mass loss may be one mechanism for the mediator role between gait impairments and PD.
AIM: To verify the relationship between the spatial-temporal gait parameters and the body composition of the lower limbs in people with PD.
METHOD: Thirteen people with PD were evaluated on two different days: i) clinical and gait evaluation; ii) body composition evaluation. The step length, width, duration and speed, the percentage in double support, and gait velocity during walking at self-selected velocity. Dual-energy X-ray absorptiometry technique was used to measure fat mass, lean mass, bone mass, and total mass, for the whole body, and separately for each limb. Pearson's correlation coefficients were applied between the spatial-temporal gait parameters and the variables of body composition of lower limbs.
RESULTS: Higher lean and bone mass of both legs were related to faster gait velocity ($r=0.6$, $p<0.03$ and $r=0.7$, $p<0.01$, respectively) and step speed ($r=0.5$, $p<0.05$ and $r=0.65$, $p<0.02$, respectively). Also, narrower step width was related to the higher bone mass of both legs ($r=0.6$, $p<0.03$). However, muscle and bone mass did not correlate with step length and duration, and percentage of double support.
CONCLUSION: Our findings suggest that the muscle and bone mass of the lower limbs are important body characteristics for gait impairments in people with PD and should be monitored over the disease.

KEYWORDS: Walking | Body composition | Parkinson's disease | Sarcopenia | Osteopenia

INTRODUCTION

Sarcopenia is commonly seen in older people, mainly in those with chronic degenerative diseases, such as rheumatoid arthritis, type 2 diabetes mellitus, Alzheimer's and Parkinson's disease (PD) [1]. It shows a high prevalence, becoming a serious global public health concern [2]. Sarcopenia stems from an abnormal decline in quality, quantity, and functionality (i.e., strength) of the muscle mass related to aging [3]. In addition, aging is related to a gradual loss of bone mineral density, known as osteopenia [4], which may progress to osteoporosis [5]. Both sarcopenia and osteopenia are associated with several negative outcomes such as falls, disability, poor quality of life, institutionalization, hospitalization, and death [6]. Also, they reduce the ability to perform daily living activities,

affecting gait velocity and stability [7].

PD exacerbates sarcopenia and osteopenia. People with PD have three times more chance of being sarcopenic than neurologically healthy older adults [1]. In addition, osteopenia and osteoporosis are common in people with PD, affecting around 40% of individuals [8]. The literature has suggested that few aspects are related to the exacerbated sarcopenia and osteopenia conditions in PD [9]. Malnutrition results from reduced energy intake [10]. The motor limitations caused by the PD progression [11] reduce the amount and intensity of exercises, decreasing the mechanical load that the lower limb muscles exert on the structure of the bones and that contributes to remodeling by stimulating osteoblastic and osteoclastic activation [12] and keeping muscle mass and strength [13]. Also, the loss of motor neurons that occurs in PD contributes to the genesis of sarcopenia, leading to weakness and reduction in movements [14].

The individuals with both PD and sarcopenia had greater difficulty performing activities of daily living, such as brushing their teeth, taking a shower, and getting up from bed [15]. In addition, a higher risk of falls is associated with people with PD and sarcopenia [9]. The negative effects of PD and sarcopenia, such as reduced mobility, poor balance, and reduced lower limb muscle strength, explain this association [16]. However, there is a lack of literature on the effects of muscle and bone mass loss on gait parameters in people with PD.

The step of people with PD is characterized by slower speed, shorter length, longer duration and double support time, and wider width compared to neurologically healthy older peers [17,18]. Despite gait impairments being related to motor symptoms of PD, mainly rigidity and bradykinesia and basal ganglia deficits [19], sarcopenia and osteopenia can be important aspects to explain gait deficits in PD. Quantitative and qualitative changes in muscle structure and function and bone mass are associated with slower gait speed and reduced stability of gait in older adults [20]. Thus, sarcopenia and osteopenia may be one mechanism for the mediator role between gait impairments and PD [21]. In addition, sarcopenia and PD share common pathophysiological pathways for muscle fiber and bone mass loss: inflammation, muscle autophagy, oxidative stress, and apoptosis [14].

Considering that, understanding the effects of sarcopenia and osteopenia on gait in people with PD is promising to improve interventions for walking impairments. Therefore, this study aimed to verify the relationship between the spatial-temporal gait parameters and the body composition (i.e., lean mass, fat mass, bone mass, and total mass) of the lower limbs in people with PD. We expected that higher muscle and bone mass of the lower limbs would be related to increased gait speed and step length, and reduced step duration, double support time, and step width.

METHODS

Participants

Thirteen individuals with PD were recruited from the Ativa Parkinson Group at São Paulo State University (Unesp – Bauru, SP, Brazil) to participate in this study. The diagnosis of idiopathic PD was performed by an expert neurologist according to the UK Parkinson's Disease Brain Bank criteria. The exclusion criteria comprised (a) less than 60 years old, (b) other parkinsonism syndromes and/or neurological diseases, (c) rheumatic or orthopedic impairments that affect gait, and (d) vestibular deficits and uncorrected

vision. In addition, individuals were included in the study if they were under dopaminergic medication treatment for more than four months (using the same medication), exhibited no signs of dementia, and were below four on the Hoehn & Yahr scale (H&Y) [22]. Written informed consent was obtained from all participants according to the protocol approved by the Ethical Committee of the School of Science at São Paulo State University, and it was conducted according to the Declaration of Helsinki.

Experimental Protocol

People with PD performed the evaluations during the ON-medication state (one hour after the participants had taken their dopaminergic medication [23]). The participants were evaluated on two different days (three to five days apart): i) day 1 – clinical and gait evaluation; ii) day 2 – body composition evaluation.

Clinical evaluation

A specialist evaluated the clinical characteristics of people with PD. First, an anamnesis (clinical historical, cognition, and medication status) was performed. To determine the degree and stage of disease, participants were evaluated by the motor section of the Unified Parkinson's Disease Rating Scale (UPDRS-III) [24] and the H&Y [25] scale, respectively. In addition, cognitive screening for dementia was performed using Mini-Mental State Examination [26,27].

Gait evaluation

The spatial-temporal gait parameters were acquired through eight Vicon Motion System® cameras (Bonita System Cameras), with a frequency of 100 Hz. Two passive reflective markers were placed on each participant's foot (second metatarsal and calcaneus). Also, one marker was positioned in the 7th cervical vertebra (C7).

The participants were instructed to walk five times on an 8 x 3 m walkway at their self-selected velocity. For safety purposes, an evaluator accompanied the participant during the execution of the task. The data were filtered using a 5th-order low-pass digital Butterworth filter (zero-lag) with a cut-off frequency of 6 Hz. The following gait parameters were calculated: step length, step width, step duration, the percentage in double support (time in double support normalized by step duration), and step speed. The spatial-temporal parameters were calculated in four to six steps, and the average for each parameter was calculated in each trial. In addition, the mean gait velocity was calculated by displacing a marker located in C7.

Body composition evaluation

Dual-energy X-ray (DXA) absorptiometry technique (DXA Discovery, Hologic, USA) with a Hologic Discovery total body scan, fan-beam densitometer, software QDR for Windows version 12.5 (Hologic, Waltham, Massachusetts, USA) was used to measure body composition. The DXA has a low coefficient of variation (bone mineral content = 0.6%, soft tissue without fat = 0.3%, fat mass, and body fat percentage = 2.5%) [28], and the radiation exposure during the procedure is less than 0.05 mRem (0.5 Sv). Before performing the analyses with the participants, a phantom step with six acrylic and aluminum fields of different thicknesses and known absorption properties were scanned to serve as an external standard for the analysis of different tissue components following the

recommendations of the protocol described by the manufacturer, which is a validated procedure for the general use of DXA [29].

The segmental body composition was determined from the regional analysis of the whole body. The lower limbs were assessed by separating the legs from the hip by an inclined line crossing the hip joint. The participant was instructed to wear no metal objects, including undergarments with metal parts, on the day of the examination. This orientation was recalled and verified before the participant entered the room where they would carry out the examination. Participants were placed supine in a table fixed from which X-rays were emitted. A "sweep" was performed through a reader arm (cursor) that walked over the body area of interest without touching the individual. The values of the body composition of each participant were calculated by summing the values of the right and left lower limbs for each variable. The values of the body composition measured were fat mass, lean mass, bone mass, and total mass, for the whole body, and separately for each limb.

Statistical analysis

The software SPSS 21.0 for Windows® was used for statistical analysis. The level of significance was maintained at $p < 0.05$. The data showed normal distribution in the Shapiro-Wilk test. To verify the correlation between the spatial-temporal gait parameters and the variables of body composition of lower limbs, Pearson's correlation coefficients were applied. The correlation coefficients were classified as weak: $0 < r < 0.3$, moderate: $0.4 < r < 0.6$, and strong $r > 0.7$ [30].

RESULTS

General and clinical characteristics, spatial-temporal parameters, and body composition parameters are presented in Table 1.

The r-values of Pearson's correlation and significant p-values are presented in Table 2. Faster gait velocity was strong and moderate related to higher bone mass and lean mass of both legs, respectively. In addition, wider step width was moderately related to lower bone mass of both legs, while faster step speed was moderately related to higher lean mass for both legs. Step length, step duration, and the percentage of double support were not related to body composition parameters ($p > 0.05$).

Table 1. Means, standard deviations, and maximum and minimum values (in brackets) of general and clinical characteristics, body composition, and spatial-temporal gait parameters.

General and clinical characteristics		
Age (years)		67 ± 9 [56 – 84]
Height (m)		1.60 ± 0.08 [1.44 – 1.70]
Time of disease (years)		5.3 ± 3.7 [1 – 13]
H&Y (pts)		2.2 ± 0.4 [2 – 3]
UPDRS-III (pts)		25.7 ± 10.4 [15 – 50]
MMSE (pts)		27.2 ± 2.8 [21 – 30]
Body composition		
Body mass (kg)		63.8 ± 11.6 [46.7 – 80.2]
Body fat mass (kg)		23.5 ± 8.6 [11.5 – 40.7]
Body lean mass (kg)		40.2 ± 6.6 [29.5 – 51.7]
Body bone mass (kg)		1.1 ± 0.24 [1.58 – 0.69]
Fat mass (kg)	**Left limb**	4.1 ± 1.3 [1.9 – 6.3]
	Right limb	4.3 ± 1.3 [2.1 – 7.1]
Lean mass (kg)	**Left limb**	6.4 ± 1.1 [4.6 – 8.9]
	Right limb	6.4 ± 1.3 [4.2 – 9.2]
Bone mass (kg)	**Left limb**	0.4 ± 0.1 [0.2 – 0.6]
	Right limb	0.4 ± 0.2 [0.2 – 0.8]
Total mass (kg)	**Left limb**	10.5 ± 1.7 [8.4 – 12.9]
	Right limb	10.7 ± 1.9 [8.2 – 13.6]
Spatial-temporal gait parameters		
Mean gait velocity (m/s)		0.92 ± 0.19 [0.61 – 1.19]
Step length (cm)		48.11 ± 8.20 [31.32 – 57.25]
Step duration (s)		0.51 ± 0.02 [0.47 – 0.57]
Step speed (cm/s)		94.08 ± 18.96 [62.71 – 122.89]
Step width (cm)		13.30 ± 3.54 [8.55 – 19.56]
Double support time (%)		36.77 ± 4.62 [28.22 – 39.84]

H&Y - Hoehn & Yahr scale; UPDRS III – motor part of Unified Parkinson's Disease Rating Scale; MMSE - Mini-Mental State Examination.

Table 2. Relationship between spatial-temporal gait parameters and lower limbs composition. r and p-values are presented in each column. (*) statistically significant.

	Lower limb	Mean velocity	Step length	Step width	Step duration	Step speed	Percentage in double support
Bone mass	**Left**	*r = 0.70 (p < 0.01)**	r = 0.55	*r = - 0.59 (p < 0.04)**	r = - 0.47	*r = 0.65 (p < 0.02)**	r = - 0.05
	Right	*r = 0.71 (p < 0.01)**	r = 0.52	*r = - 0.62 (p < 0.03)**	r = - 0.52	*r = 0.65 (p < 0.02)**	r = - 0.07
Fat mass	**Left**	r = 0.042	r = 0.26	r = - 0.06	r = 0.52	r = 0.01	r = 0.29
	Right	r = 0.004	r = 0.22	r = - 0.06	r = 0.54	r = - 0.02	r = 0.31
Lean mass	**Left**	*r = 0.64 (p < 0.03)**	r = 0.48	r = - 0.55	r = - 0.43	*r = 0.58 (p < 0.05)**	r = 0.09
	Right	*r = 0.62 (p < 0.03)**	r = 0.49	r = - 0.55	r = - 0.35	*r = 0.56 (p < 0.05)**	r = 0.14
Total mass	**Left**	r = 0.52	r = 0.54	r = - 0.46	r = 0.02	r = 0.45	r = 0.25
	Right	r = 0.49	r = 0.52	r = - 0.47	r = 0.04	r = 0.42	r = 0.28

DISCUSSION

This is a preliminary study that first time directly investigated the relationship between lean and bone mass in the lower limbs and gait in people with PD. We demonstrated that reduced lean and bone mass were related to slower gait speed and reduced stability in people with PD (i.e., greater step width), which corroborated our hypothesis. Specifically, faster gait velocity and step speed were related to higher bone mass and lean mass of both legs, while narrower step width was related to the higher bone mass of both legs. However, muscle and bone mass did not correlate with step length and duration, and percentage of double support. Our findings suggest that muscle and bone mass of the lower limbs are important body characteristics for gait impairments in people with PD and should be monitored over the disease.

Reduced bone and muscle mass change the spatial-temporal gait parameters in people with PD. It is well established in the literature that gait impairments in PD reflect dysfunctions of cortico-basal ganglia-brainstem circuits 19. However, our findings seem to show that changes in bone and muscle mass are also related to gait impairments in PD. A previous study also reported that the decline in bone and muscle mass are related to the stage of disease and reduced mobility in people with PD. Walking involves muscle activity from the lower limbs, and other parts of the body, such as the trunk and upper limbs muscles. Reduced muscular strength is known to be significantly and independently associated with functional impairment, walking speed, balance, mobility tasks, physical performance, and all-cause mortality [31]. Gait speed and stability performance are related to muscle mass quality, which is important for independence [21]. Landi et al. [32] reported that a reduction in lower limb muscle mass is directly related to lower strength of the legs, which is correlated to slower gait speed and longer double support time in older people. People with PD also reduce the strength of the lower limb [33]. Thus, the combination of inadequate control of dorsiflexors [34] and muscle weakness [14] caused by PD can contribute to changes in gait speed and stability.

Reduction in bone mass is also correlated to walking changes. Older women with reduced bone mass in the hip, spine, and forearm walk with slow gait speed and large step time and stance time [35]. Also, low bone mass is correlated to less power generation at the hip and ankle as well as, less power absorption at the hip and knee, and stability during walking [36]. Frailty older individuals show reduced bone mass, increasing the risk of falls [37]. Thus, the slower gait speed and wider step width are possible gait adaptations used by people with PD to deal with lower bone mass. However, despite the wider step width being an efficient strategy to increase stability [38,39], reduced gait speed does not cope with an increased margin of stability, and thus higher stability [40]. Therefore, bone mass loss can be related to gait instability in people with PD.

An important limitation of the present study is the small sample of individuals with PD, which could impact the power of our statistical analysis. However, we found a moderate correlation and a significant association (regression) with a small sample, which is very promising. Also, we did not include neurologically older adults which did not allow us to assume that the effects are related to PD and not to aging. Thus, future studies should compare the associations of bone and muscle mass with gait parameters considering older adults with and without PD. Finally, lean mass is all mass without fat (fat-free mass), which involves not only muscles but also bones (data shown), tendons and

ligaments (data not shown). So, it should be considered when our findings are analyzed.

Despite such limitations, our preliminary study extends the current literature by providing an understanding of the influence of bone and muscle mass on gait parameters in people with PD. A practical application of our study is related to the gait rehabilitation program. It must be incorporated exercises that keep muscle and bone mass and the lower limb to improve walking in people with PD. Exercise, especially with a mechanical load that contributes to remodeling [9] fosters a vicious cycle related to an increase in gait speed, and functional independence [31]. Also, good nutrition should be considered for keeping muscle and bone mass and improving gait in PD [10].

CONCLUSION

The body composition is related to spatial-temporal parameters in people with PD. Faster gait speed and reduced stability (i.e., wider step width) are correlated to higher bone and muscle mass in PD. These findings reinforce the need to exercise in PD to preserve muscle and bone mass for improving walking behavior.

REFERENCES

1. Ponsoni A, Sardeli AV, Costa FP, Mourão LF. Prevalence of sarcopenia in Parkinson's disease: A systematic review and meta-analysis. *Geriatr Nurs*. 2022;49:44-49. doi:10.1016/j.gerinurse.2022.11.006
2. Shafiee G, Keshtkar A, Soltani A, Ahadi Z, Larijani B, Heshmat R. Prevalence of sarcopenia in the world: a systematic review and meta- analysis of general population studies. *J Diabetes Metab Disord*. 2017;16:21. doi:10.1186/s40200-017-0302-x
3. Cruz-Jentoft AJ, Bahat G, Bauer J, et al. Sarcopenia: revised European consensus on definition and diagnosis. *Age Ageing*. 2019;48(1):16-31. doi:10.1093/ageing/afy169
4. Zhang J, Morgan SL, Saag KG. Osteopenia: debates and dilemmas. *Curr Rheumatol Rep*. 2013;15(12):384. doi:10.1007/s11926-013-0384-5
5. Zeng Q, Li N, Wang Q, et al. The Prevalence of Osteoporosis in China, a Nationwide, Multicenter DXA Survey. *J Bone Miner Res*. 2019;34(10):1789-1797. doi:10.1002/jbmr.3757
6. Bahat G, Tufan A, Tufan F, et al. Cut-off points to identify sarcopenia according to European Working Group on Sarcopenia in Older People (EWGSOP) definition. *Clin Nutr*. 2016;35(6):1557-1563. doi:10.1016/j.clnu.2016.02.002
7. Barichella M, Pinelli G, Iorio L, et al. Sarcopenia and Dynapenia in Patients With Parkinsonism. *J Am Med Dir Assoc*. 2016;17(7):640-646. doi:10.1016/j.jamda.2016.03.016
8. Wood B, Walker R. Osteoporosis in Parkinson's disease. *Mov Disord*. 2005;20(12):1636-1640. doi:https://doi.org/10.1002/mds.20643
9. Lima DP, de Almeida SB, Bonfadini J de C, et al. Clinical correlates of sarcopenia and falls in Parkinson's disease. *PLoS One*. 2020;15(3):e0227238. doi:10.1371/journal.pone.0227238

10. Kwan P. Sarcopenia, a neurogenic syndrome? *J Aging Res*. 2013;2013:791679. doi:10.1155/2013/791679
11. van Nimwegen M, Speelman AD, Hofman-van Rossum EJM, et al. Physical inactivity in Parkinson's disease. *J Neurol*. 2011;258(12):2214-2221. doi:10.1007/s00415-011-6097-7
12. Bellido T. Osteocyte-driven bone remodeling. *Calcif Tissue Int*. 2014;94(1):25-34. doi:10.1007/s00223-013-9774-y
13. Kamide N, Fukuda M, Miura H. The relationship between bone density and the physical performance of ambulatory patients with Parkinson's disease. *J Physiol Anthropol*. 2008;27(1):7-10. doi:10.2114/jpa2.27.7
14. Vetrano DL, Pisciotta MS, Laudisio A, et al. Sarcopenia in Parkinson Disease: Comparison of Different Criteria and Association With Disease Severity. *J Am Med Dir Assoc*. 2018;19(6):523-527. doi:10.1016/j.jamda.2017.12.005
15. da Luz MCL, Bezerra GKA, Asano AGC, Chaves de Lemos M da C, Cabral PC. Determinant factors of sarcopenia in individuals with Parkinson's disease. *Neurol Sci*. 2021;42(3):979-985. doi:10.1007/s10072-020-04601-4
16. Canning CG, Paul SS, Nieuwboer A. Prevention of falls in Parkinson's disease: a review of fall risk factors and the role of physical interventions. *Neurodegener Dis Manag*. 2014;4(3):203-221. doi:10.2217/nmt.14.22
17. Simieli L, Gobbi LTB, Orcioli-Silva D, et al. The variability of the steps preceding obstacle avoidance (approach phase) is dependent on the height of the obstacle in people with Parkinson's disease. Grahn JA, ed. *PLoS One*. 2017;12(9):e0184134. doi:10.1371/journal.pone.0184134
18. Mirelman A, Bonato P, Camicioli R, et al. Gait impairments in Parkinson's disease. *Lancet Neurol*. 2019;18(7):697-708. doi:10.1016/S1474-4422(19)30044-4
19. Takakusaki K. Functional Neuroanatomy for Posture and Gait Control. *J Mov Disord*. 2017;10(1):1-17. doi:10.14802/jmd.16062
20. Larsson L, Degens H, Li M, et al. Sarcopenia: Aging-Related Loss of Muscle Mass and Function. *Physiol Rev*. 2019;99(1):427-511. doi:10.1152/physrev.00061.2017
21. Perez-Sousa MA, Venegas-Sanabria LC, Chavarro-Carvajal DA, et al. Gait speed as a mediator of the effect of sarcopenia on dependency in activities of daily living. *J Cachexia Sarcopenia Muscle*. 2019;10(5):1009-1015. doi:10.1002/jcsm.12444
22. Hoehn MM, Yahr MD. Parkinsonism: onset, progression, and mortality. *Neurology*. 1967;17(5):427-427. doi:10.1212/WNL.17.5.427
23. Araújo-Silva F, Santinelli FB, Felipe I. Imaizumi L, et al. Temporal dynamics of cortical activity and postural control in response to the first levodopa dose of the day in people with Parkinson's disease. *Brain Res*. 2022;1775(July 2021):147727. doi:10.1016/j.brainres.2021.147727
24. Martínez-Martín P, Gil-Nagel A, Gracia LM, Gómez JB, Martínez-Sarriés J, Bermejo F. Unified Parkinson's disease rating scale characteristics and structure. *Mov Disord*. 1994;9(1):76-83. doi:10.1002/mds.870090112

25. Schenkman M, Wei Zhu C, Cutson TM, Whetten-Goldstein K. Longitudinal evaluation of economic and physical impact of Parkinson's disease. *Park Relat Disord*. 2001;8(1):41-50. doi:10.1016/S1353-8020(00)00079-1
26. Almeida OP. The Mini-Mental State Examination and the Diagnosis of Dementia in Brazil. *Arq Neuropsiquiatr*. 1998;56(3 B):605-612. doi:10.1590/S0004-282X1998000400014
27. Brucki SMD, Nitrin R, Caramelli P, Bertolucci PHF, Okamoto IH. Suggestions for utilization of the mini-mental state examination in Brazil. *Arq Neuropsiquiatr*. 2003;61(3 B):777-781. doi:10.1590/s0004-282x2003000500014
28. Bilsborough JC, Greenway K, Opar D, Livingstone S, Cordy J, Coutts AJ. The accuracy and precision of DXA for assessing body composition in team sport athletes. *J Sports Sci*. 2014;32(19):1821-1828. doi:10.1080/02640414.2014.926380
29. Tataranni PA, Ravussin E. Use of dual-energy X-ray absorptiometry in obese individuals. *Am J Clin Nutr*. 1995;62(4):730-734. doi:10.1093/ajcn/62.4.730
30. Cohen J. *Statistical Power Analysis for the Behavioral Sciences (2nd Ed.).* (Routledge, ed.). Routledge; 1988. doi:10.4324/9780203771587
31. Wilson D, Jackson T, Sapey E, Lord JM. Frailty and sarcopenia: The potential role of an aged immune system. *Ageing Res Rev*. 2017;36:1-10. doi:10.1016/j.arr.2017.01.006
32. Landi F, Liperoti R, Fusco D, et al. Prevalence and risk factors of sarcopenia among nursing home older residents. *J Gerontol A Biol Sci Med Sci*. 2012;67(1):48-55. doi:10.1093/gerona/glr035
33. Pereira VAI, Barbieri FA, Zagatto AM, et al. Muscle Fatigue Does Not Change the Effects on Lower Limbs Strength Caused by Aging and Parkinson's Disease. *Aging Dis*. 2018;9(6):988. doi:10.14336/AD.2018.0203
34. Pistacchi M, Gioulis M, Sanson F, et al. Gait analysis and clinical correlations in early Parkinson's disease. *Funct Neurol*. 2017;32(1):28-34. doi:10.11138/FNeur/2017.32.1.028
35. Palombaro KM, Hack LM, Mangione KK, et al. Gait variability detects women in early postmenopause with low bone mineral density. *Phys Ther*. 2009;89(12):1315-1326. doi:10.2522/ptj.20080401
36. Eldeeb AM, Khodair AS. Three-dimensional analysis of gait in postmenopausal women with low bone mineral density. *J Neuroeng Rehabil*. 2014;11:55. doi:10.1186/1743-0003-11-55
37. Cheng M-H, Chang S-F. Frailty as a Risk Factor for Falls Among Community Dwelling People: Evidence From a Meta-Analysis. *J Nurs Scholarsh*. 2017;49(5):529-536. doi:10.1111/jnu.12322
38. Barbieri FA, Beretta SS, Pereira VAI, et al. Recovery of gait after quadriceps muscle fatigue. *Gait Posture*. 2016;43:270-274. doi:10.1016/j.gaitpost.2015.10.015
39. Santos PCR, Lamoth C, Vuillerme N, Barbieri FA. Faster walking under muscle fatigability: a strategy to improve stability or a warm-up effect? *Brazilian J Mot Behav*. 2021;15(3):149-152. doi:10.20338/bjmb.v15i3.241

40. Hak L, Houdijk H, Steenbrink F, et al. Speeding up or slowing down?: Gait adaptations to preserve gait stability in response to balance perturbations. *Gait Posture*. 2012;36(2):260-264. doi:10.1016/J.GAITPOST.2012.03.005

Citation: Barbieri FA, Faria MH, Simieli L, Penedo T, Kalva-Filho CA, Beretta VS. (2022). Gait velocity and stability are correlated to muscle and bone mass loss in people with Parkinson's disease: a preliminary study. *Brazilian Journal of Motor Behavior*, 16(5):362-371.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.
Competing interests: The authors have declared that no competing interests exist.
DOI: https://doi.org/10.20338/bjmb.v16i5.346