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CONSEQUENCES OF SARCOPENIA AMONG NURSING HOME RESIDENTS AT LONG-TERM FOLLOW-UP

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Long-term consequences of sarcopenia

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Abstract

The consequences of and transition into sarcopenia with long-term survival was investigated in the nursing home setting. Eligible residents from 11 nursing homes were followed-up 18-months after their assessment for sarcopenia using the European Working Group on Sarcopenia in Older People criteria, with other demographic, physical and cognitive health measures collected. Of the 102 older adults who consented at baseline, 22 had died and 58 agreed to participate at follow-up, 51.7% of whom had sarcopenic. Sarcopenia at baseline was associated with a depression ($p < .001$), but not mortality, hospitalization, falls or cognitive decline at follow-up. Age was the strongest predictor of mortality ($p = .05$) with the relative risk of death increasing 5.2% each year. The prevalence of sarcopenia is high and increases with long-term survival in end-of-life care. However, the risk of sarcopenia-related mortality is not as great as from increasing age alone.

***Key words*:** Consequences, Mortality, Nursing-home, Sarcopenia

Introduction

According to the European Working Group on Sarcopenia in Older People (EWGSOP), sarcopenia is an age related syndrome defined by a progressive and generalized loss of muscle mass and muscle function (either or both of below normal muscle strength or physical performance).[1] As a geriatric syndrome, sarcopenia affects quality of life, is associated with poor survival rates,[2] plays an important role in the etiology of frailty and is highly predictive of several adverse health events in later life.[3] In addition, it has been reported that in comparison to non-sarcopenic adults, those with sarcopenia are at greater risk of falls, are more likely to be physically disabled and have greater care needs.[4, 5]

Following the definition review in 2010,[1] a large a body of work has emerged looking to establish prevalence and risk factors of sarcopenia across varied older cohorts. However, due to variations in cohort characteristics, diagnostic and measure criteria, prevalence data are mixed.[6] Conclusive is that sarcopenia increases rapidly after the age of 65 years, with prevalence being as high as 50% in people older than 80 years.[7, 8] In addition, current research has identified male gender, low body mass index (BMI) and reduced physical activity as common risk factors to being sarcopenic.[9, 10]

Work by our group has shown that in the environment where prevalence is highest, the nursing home,>40% of adults are sarcopenic.(Removed for blinding) However, while prevalence and risk factor data grows, longitudinal analysis of the consequence of and the progression to sarcopenia is scarce. Given the economical and personal implications of being

sarcopenic, a broader understanding of the consequences may assist in informing interventions pathways, particularly in light of evidence that a number of sarcopenia risk factors are modifiable.[12, 13] The aim of this study was to report the implications of sarcopenia in nursing home residents at an 18 month follow-up, and to track progression in sarcopenia among those with no previous diagnosis.

Methods

Study design

The study employed a longitudinal follow-up of randomly selected adults with secondary data collected 18 months after the parent-study baseline assessment. A detailed account of the methodologies used in the baseline cross-sectional study and of the reported sarcopenia prevalence and risk factors can be found elsewhere, including a CONSORT diagram detailing recruitment, randomization and assessment.[(Removed for blinding)] In brief, 273 adults residing in 11 purposefully selected South East Queensland (Australia) nursing homes were randomized into the study from an eligible sample of 381 and total resident cohort of 709. The inclusion criteria were (i) ≥60 years, (ii) residing in a nursing home and (ii) could provide consent, self or by proxy given directly by the participants substitute decision maker or verbally to the facility Service Manager. Residents were excluded if they; (i) had a pacemaker; (ii) were end-stage palliative or terminal (iii) had difficult behaviors that would limit data collection; or (iv) had a medical condition or other issue that would limit data collection (eg. total uncommunicable deafness). Ninety-one individual self-consented and 11 consented by proxy to participate in the baseline study

(84.5 ± 8.2 years; > 70% women, 1204.2 ± 1220.1days in care). Consent to the baseline study included agreement to be approached at the 18-month follow-up and a secondary data set collected. Specifically, facilities were re-contacted and the follow-up study explained to the Service Manager, who was then given the list of participant per facility and a request to seek consent for participation. Consent for the follow-up study was considered appropriate given the time frame, setting and the risk of negative health change among participants. As with the baseline study, consent was obtained directly from cognitive sound participants or from the substitute decision maker of participant not able to consent themselves. The eligibility criteria were retained from the baseline study for the follow-up study.

Approval for the study was provided by the Human Ethics Committee of the (Removed for blinding) University, and the nursing care provider's internal ethics committee.

Data Collection

Participants were assessed individually and data collection was finalized at one facility before moving to the next. For low care participants, the research assistant (RA) was left to conduct the data collection without assistance. For high care and dementia participants, a facility staff member was present.

Measures

All measures were validated for use among old and very old adults and have been described in detail previously.[10, 11] Where an individual could not complete a measure due

to health or disability issues the measure was excluded, with the exceptions of the 2.4 meter walk (scored at 0 if unable) to ensure a measure of physical performance. For individuals who were unable to or would not assent to the bioelectrical impedance analysis (BIA), baseline data were carried forward.

Primary outcome: Sarcopenic

Sarcopenia was measured using the EWGSOP definition, cut-off points and assessment criteria. Specifically, a diagnosis of sarcopenia required the presence of both low muscle mass and low muscle function (muscle strength or physical performance).[1] *Muscle mass* was measured using BIA (Maltron BF-906, Maltron International Ltd, Rayleigh, UK) with the participant lying flat and the standardized electrode placement. Skeletal Muscle Mass (SMM) was calculated from the equation (SMM = [(height^2(cm) /resistance (ohms) x 0.401) + (gender x 3.825) + (age (yrs) x -0.071)] + 5.102), then divided by height^2 (m) to give the Skeletal Muscle Index (SMI). The SMI cut-off of <8.87 kg/m^2 in men and <6.42kg/m^2 in women were used to define low muscle mass. *Muscle strength* was measured by Jamar hand grip dynamometer (Sammons Preston Roylan, Bolingbrook, IL), using the individuals dominant hand with their elbow at 90^0 and locked at their side. The best of three trials was used in the analysis and cut-off points of < 30 and < 20 kg for men and women, respectively, used to define low muscle strength. *Physical performance* was measured by the Short Physical Performance Battery(SPPB) 2.4-meter walk. The best of three trials was retained for the analysis and the cut-off point of < 0.8 m/s used to define low physical performance. In addition, the remaining SPPB measures, the standing balance

and the repeated chair stands, where collected to allow the generation of the SPPB summary score.

Secondary outcomes

The RA collected height (cm) and weight (kg) by standardized methodologies. Demographics and clinical data were collected from facility records with outstanding variables collected directly from the participants. The Mini-Mental State Examination and the Geriatric Depression Scale were used to measure cognitive status and depression, respectively, and the Mini-Nutritional Assessment Instrument (MNA) to assess nutritional status.[10]

Statistical analysis

Within cohort, sex and group comparisons were made on demographic, functional and clinical variables between baseline and the follow-up by t-test (continuous data) and by Pearson's chi-square test (categorical data). Between group (Sarcopenia versus No Sarcopenia at baseline) differences were investigated by repeated measures analysis of variance (2 x 2 ANOVA). Generalized linear models were used to quantify the effects of sarcopenia (diagnosed at baseline) on functional and clinical variables whist controlling for the sarcopenic risk factors of age, gender, BMI, physical activity level and nutritional status.[10] For binary data including occurrence of death, hospitalization or a fall, prevalence was not rare and therefore modified Poisson regression models with robust estimation of SE values were used to calculate relative risk (RR).[14] Data were analyzed through a combination of SPSS version 22.0 (SPSS Inc., Chicago, IL, USA) and the geepack

statistical package for the R programming language, version 3.1.3. Statistical significance were based on two-tailed tests with $p<0.05$ considered significant.

Results

Participants

Twenty-two of the 102 baseline participants died within the 18-month follow-up period, and 58 of the surviving 80 participants consented to the follow-up analysis (85.7 ± 8.2 years; > 70% women, 1970.8 ± 1403.7 days in care, 14 by substitute decision maker consent). Those who died had been in care for 1314.7 ± 884.1 days, were 88.3 ± 8.3 years of age at death, 13 were female and 9 had a baseline diagnosis of sarcopenia. Among those who did not consent to participate, 9 were unavailable, 7 refused, 4 had been relocated and 2 were not consented by their substitute decision maker (Baseline: 82.8 ± 7.7 years, 983.2 ± 944.5 days in care, 40.9% sarcopenic (N; 9 of 22) versus 37.9% of those who did consent (N; 22 of 58)). Analysis of baseline measures revealed no difference between those who consented to the follow-up investigation and those who didn't ($p > .05$). At the follow-up assessment, women were older, had a higher percent body fat and walked faster than men, while men were taller, heavier and had a higher SMI and lean mass than women ($p < .05$). Sixteen participants were unable (pacemaker or could not transferred to laying) or would not assent (behaviors) to the BIA measured. An overview of demographic, physical and mental health data at follow-up is given in table 1.

Sarcopenia

At baseline 40.2% were classified sarcopenic, with prevalence higher in males (45.2%), but not statistically different to females (36.7%) (χ^2 = .660, df = 1, p = .416).(Removed for blinding) At follow-up, sarcopenia prevalence increased to 51.7% (N = 30). Prevalence remained higher among males (70.6% versus 43.9%, males (N; 12 of 17) versus females (N; 18 of 41), respectively), and approached significance (χ^2 = 3.427, df = 1, p = .064). Among those who died, there was no difference from baseline to time of death between sarcopenic (n = 9) and non-sarcopenic individuals (342.8 ± 143.0 versus 350.4 ± 150.0 days, respectively; p =.906).

Impact of sarcopenia on health outcomes

Generalized linear models results demonstrate that depression was associated at baseline with sarcopenic status (p = .003), with a significant interaction effect (p = .002) identified between sarcopenic status and gender, highlighting that depression was highest in males with sarcopenia. In contrast, sarcopenic status was not associated with change in cognitive status or in SPPB (p ≥ .690) (Table 2). The Poisson regression approach revealed no significant association between a baseline diagnosis of sarcopenia and mortality, hospitalization or falls at follow-up in either the unadjusted or adjusted models (Table 3). Age was found to be the single best predictor of mortality (p = .05) with the relative risk of death increasing by 5.2% each year.

Sarcopenia versus non-sarcopenic group change to follow-up

Independent of sarcopenic status, a within group analysis revealed a significant decrease in walking speed, standing balance, SPPB summary score, grip strength and the

MNA ($p \leq .047$) over 18 months till follow-up. For the group diagnosed non-sarcopenic at baseline, significant decreases in the SMI was found ($p = .014$) at follow-up. Between group analysis revealed a group*time effect for SMI ($p = .041$) and a between group difference for BMI, SMI, lean mass and grip strength ($p \leq .032$) at follow-up. Data are presented in table 4.

Discussion

This study supports that the prevalence of sarcopenia in the nursing home setting is high[15] and demonstrated an increase in prevalence with long-term survival. However, for long-term surviving residents an increase in age has a stronger association to mortality than a diagnosis of sarcopenia. This study also demonstrates that among long-term survivors, being non-sarcopenic was associated to greater negative change across measures of body composition, but not muscle function, than among those with a diagnosis of sarcopenia. While it would be assumed that increasing age in the nursing home setting would be a predictor of death and that sarcopenia prevalence continues to increase if untreated, to date few studies have explored this assumption.

Sarcopenia is known to bring with it a number of negative consequences that increase with age and place the individual at an elevated risk of disability, institutionalization and declining health.[1] According to Fried et al.[16] sarcopenia has an important etiological role in the frailty process and harmful consequences, such as falls, functional decline, loss of independence, emergency room visit, hospital and nursing home admission, and mortality. Supporting the association to mortality, and using the same methodological technique

employed in this study, Landi et al.[17] reported that at a 6-monthfollow-up older institutionalized Italians with sarcopenia had greater risk of mortality. Arango- Lopera et al.[18] corroborated this associating at a three-year follow-up, but among community-dwelling Turkish adults with muscle mass defined by calf circumference.

In our study, 21.6% of the original cohort had died at follow-up, with time to death from diagnosis or risk of mortality demonstrating no difference between groups (sarcopenic and non-sarcopenic). In contrast, our study showed the risk of mortality had a stronger association to increasing age than to sarcopenia. This is in agreement with other research that reports sarcopenia a poor predictor and age as a stronger predictor of mortality in older subjects.[19] In the present study, while still having a greater absolute value at follow-up, those diagnosed non-sarcopenic at baseline experienced accelerated declines in SMI. This maybe reflective of a greater potential to decline within the non-sarcopenic group and that the sarcopenic group had reached a SMI plateau associated to survival. As an alternate predictors of mortality risk, Cheung et al.[20] recently suggested that muscle mass and gait speed may be stronger individual indicators, which is supported by Srikanthan et al.[21] who showed low muscle mass a better predictor of mortality than BMI. The presented study was focused on investigating the consequences of sarcopenia on long-term survival, but given these aforementioned associations, more work may be warranted looking at the independent components of a sarcopenic as alternate predictors of mortality in the nursing home setting.

It is not surprising this study found no strong association between sarcopenia, falls risk, hospitalization and functional capacity. Specifically, in the nursing homes environment

risk of adverse events are already high, and associated to other factors such as increasing age, disease, extended sitting times, assisted transfers and the use of mobility aids.[22] As with mortality, sarcopenia may influence these markers of dependence, but be only one factor in geriatric decline that drives increased risk. In contrast, the interplay between depression and sarcopenia found here supports the association identified previously,[23] a relationship thought to be influenced by declining physical independence and poor health outcomes.[1, 24]

Moreover, it is not surprising individuals continue to decline across markers of health and wellbeing in the nursing home setting, prominently associated to extended sitting, high levels and the progression of chronic disease, and a lack of intervention to promote physical gains.[25, 26] However, in a recent review of exercise in the nursing home setting, evidence supports that appropriate physical activity, and specifically weight bearing exercise, can be effective at increasing muscle mass, muscle strength and physical performance, thereby targeting the three physical components associated to sarcopenia.[27] This is supported by recent work from our group that showed a reduced transition in sarcopenia among adults doing resistance training, accompanied by increased muscle strength and physical performance following 24 weeks of twice weekly progressive resistance plus balance training.[28]

Current research in the field of sarcopenia has seen a significant body work of appear in an abbreviated period. In the presence of this work, the current study is a reminder for clinicians that while negative consequences can follow a diagnosis of sarcopenia, in end of life care it is increasing ages that presents the greatest risk for mortality. For researchers

and care providers working in the nursing care sector and concerned about the impact of long-term survival for residents, this study and others done by our group is suggestive that when left untreated sarcopenia can continue to have negative consequences for resident health and that with an intervention of exercise, significant positive physical changes can occur.[25, 28.]

This study has several limitations. The small sample size and that some data were carried forward may have decreases the statistical power of the study.[29] Even though our exclusion criteria aimed to maximize the intake at baseline, low voluntary participation rates (common in this setting) and an 18 month follow-up period in end-of-life care has significant implication for sample numbers. Still our baseline cohort sample and death rate was not dissimilar to that reported in short-term consequence investigations previously.[17] In addition, due to the exclusion of residents with difficult behaviors or at end-of-life, data must be interpreted with caution as being representative of a higher function nursing home populations. A final consideration is that even though evidence is suggestive that low muscle mass, muscle strength and gait speed are predictive of mortality, in the interest of a focused investigation we have only analyzed the influence of sarcopenia.[19, 21] Future work should seek to overcome these limitations.

Data from the present study will hold interest for those curious about the impact of long-term survival in nursing care. Not surprisingly, individuals continue to decline across markers of health and wellbeing, associated to unmet physical activity needs and escalating chronic disease.[25, 26] However, while the prevalence of sarcopenia is high in this setting and increases with time in care, it appears that age is the strongest predictor of mortality.

In contrast, sarcopenia was associated with the consequence of and increased level of depression, but a relationship falls or hospitalization was not so apparent when compared to those with a non-sarcopenia diagnosis. Not unexpectedly, this study also demonstrated that with increased time in care individuals continue to decline across markers of health, but that change in SMI is more pronounced among non-sarcopenic residents. This work highlights the need for interventions into health and wellbeing in end-of life-care.

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Conflict of interest

The second authors (BH) was supported by the Egyptian Government support as a Post-Doctoral Fellow at the University of Queensland during the writing of this paper. All remaining Authors were employed by their primary institution during the undertaking of this study and writing of this publication. Author JK was the recipient of a small Bond University seeding grant to undertake the baseline study with authors TH and HS also list on the grant. We declare there are no conflicts of interest in this publication.

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Table 1: Nursing home aged care cohort and gender demographic, physical and mental health data 18 months' post-baseline assessment (N = 58).

		Females (N = 41)	Males (N=17)
Age (y)	85.6 ± 8.2	87.2 ± 7.5	82.0 ± 8.9
< 75 y (N)	8	3	5
75 – 85 y (N)	12	8	6
85 – 95 y (N)	33	21	4
> 95 y (N)	5	9	2
Length of Stay (days)	1970.1 ± 1403.7	2070.7 ± 1537.6	1729.9 ± 1010.9
Height (cm)	159.5 ± 9.7	156.1 ± 8.1	167.9 ± 8.4
Weight (kg)	68.5 ± 17.9	65.5 ± 16.6	75.7 ± 19.5
BMI (kg/m2)	26.9 ± 6.2	26.8 ± 6.4	27.1 ± 6.1
Fat mass (%)	36.0 ± 11.5	39.2 ± 10.7	29.8 ± 12.7
SMI (kg/m^2)	7.2 ± 1.8	6.9 ± 1.7	8.0 ± 1.9
Lean mass (kg)	42.6 ± 9.0	40.1 ± 6.9	48.7 ± 10.6
2.4-meter walk (m/s)	.2 ± .2	.3 ± .2	.1 ± .2
Standing balance (s)	6.9 ± 10.2	8.0 ± 10.9	4.4 ± 7.9
Chair standing (N)	5.2 ± 11.4	7.0 ± 12.9	1.3 ± 5.2
SPPB summary score	2.5 ± 2.2	2.8 ± 2.4	1.6 ± 1.3
Grip strength (kg)	12.7 ± 7.6	12.2 ± 7.1	14.2 ± 9.1
Chronic diseases	7.8 ± 3.3	7.9 ± 3.4	7.4 ± 3.4
< 5 (N)	6	4	2
5 – 10 (N)	42	30	12
> 10 (N)	10	7	3
Fallen in Previous 6 mths (N)	24	16	8
Bone Fracture in previous 2 y (N)	12	11	1
MMSE	16.7 ± 9.3	17.2 ± 10.2	15.7 ± 7.2
Severe (N)	16	9	7
Moderate (N)	16	11	5
Mild (N)	8	5	3
Normal (N)	18	16	2
GDS	4.6 ± 3.8	4.4 ± 4.0	5.2 ± 3.4
Severe (N)	6	4	2
Moderate (N)	6	5	1
Mild (N)	16	6	10
Normal (N)	30	26	4
MNA	8.2 ± 3.1	8.3 ± 3.2	7.9 ± 2.9
Malnourished (N)	20	14	6
At Risk (N)	30	21	9
Normal (N)	8	6	2

Where appropriate data are expressed as mean ± standard deviation
y = years, % = percent, kg = kilograms, m = metres, s = seconds, N = number, mths = months
BMI = body mass index, SMI = skeletal muscle index, SPPB = Short physical performance battery, MMSE= Mini Mental State Examination, GDS= Geriatric Depression Scale, MNA= Mini Nutritional Assessment
MMSE - normal (25-30), mild (21-24), moderate (14-20) or severe (< 13) impairment,
GDS - normal (0-4), mild (5-8), moderate (9-11) or severe (12-15).
MNA - malnourished (0-7), at risk of malnutrition (8-11) and normal (12 – 14)

Table 2. Multivariate analyses for continuous variables from baseline diagnoses to follow up, controlled for age, gender, BMI, physical activity level and nutritional status.

Variable	Sarcopenia			Non Sarcopenia			*p*
	Group	Males	Females	Group	Males	Females	
Depression	6.5 ± 3.8	8.8 ±3. 1	5.1 ± 3.6	4.5 ± 3.5	4.7 ± 4.3	4.4 ± 3.3	.003
N	36	14	22	60	16	44	
Cognitive change	-2.7 ± 7.0	-4.6 ± 8.0	-1.2 ± 6.1	-2.2 ± 6.8	-.8 ± 4.7	-2.6 ± 7.3	.940
N	18	8	10	35	8	27	
Change SPPB	.7 ± 1.2	.6 ± 1.0	.9 ± 1.3	1.3 ± 2.4	1.0 ± 1.8	1.3 ± 2.5	.690
N	22	9	13	36	8	28	

Data are mean ± Standard Deviation, N = number of individual included, SPPB = Short Physical Performance Battery.
Cognition is measured by the Mini Mental State Exam and Depression by the Geriatric Depression Scale. Data are in arbitrary units.

Table 3. Association between Sarcopenia and death, falls and hospitalisation (18 Months Follow-up), unadjusted and after adjustment for various risk factors. Data are presented as risk ratios (95% Confidence Interval)

	Death Unadjusted 1.03 (0.49 – 2.19)		**Fall** Unadjusted 1.02 (0.53 – 1.97)		**Hospitalization** Unadjusted 1.25 (0.73 – 2.14)	
	Model 2	Model 3	Model 2	Model 3	Model 2	Model 3
Sarcopenia	0.85 (0.39 – 1.86)	0.81 (0.33 – 1.98)	0.98 (0.39 – 1.86)	0.74 (0.34 – 1.63)	1.27 (0.74 – 2.19)	1.27 (0.73 – 2.22)
Age	1.06 (0.99 – 1.13)	1.06 (1.00 – 1.13)	1.03 (0.97 – 1.08)	1.04 (0.99 – 1.01)	0.96 (0.96 – 1.03)	1.00 (0.97 – 1.03)
Gender (Female)	0.54 (0.24 – 1.11)	0.50 (0.23 – 1.09)	1.37 (0.60 – 3.14)	1.34 (0.59 – 3.03)	0.99 (0.56 – 1.78)	0.95 (0.54 – 1.66)
Physical Activity (Active)		1.37 (0.61 – 3.01)		0.58 (0.17 – 1.11)		0.64 (0.36 – 1.13)
Nutritional Status (Normal)		0.73 (0.29 – 1.87)		0.43 (0.29 – 1.15)		0.91 (0.49 – 1.70)
Body Mass Index		0.98 (0.90 – 1.07)		0.99 (0.93 – 1.06)		1.03 (0.98 – 1.07)

Model 1 for each variable is the unadjusted model. Model 2: adjusted for age and gender; Model 3: adjusted for age, gender, physical activity, nutritional status and body mass index.

Table 4. Eighteen-month follow-up of residential aged care adults diagnosed with or without sarcopenia at baseline (N = 58)

	Sarcopenic		Non sarcopenic		F	p^*
	Baseline	Follow-up	Baseline	Follow-up		
Fat mass (%)	33.8 ± 13.9	32.9 ± 12.3	38.1 ± 10.6	37.9 ± 10.7	.061	.805
Body mass index (kg/m^2) †	23.9 ± 5.9	24.1 ± 6.2	29.9 ± 5.2	28.6 ± 5.7	1.228	.272
SMI (kg/m^2)†	5.9 ± 1.5	6.1 ± 1.2	8.7 ± 1.9	7.8 ± 1.8	4.389	.041
Lean mass (kg)‡	39.9 ± 9.2	37.9 ± 11.7	46.0 ± 11.5	44.4 ± 8.9	.023	.881
Walking speed (m/s)	.3 ± .2	.2 ± .2	.4 ± .2	.2 ± .2	1.721	.195
Standing balance (s)	11.8 ± 9.5	6.6 ± 10.0	14.4 ± 10.5	7.1 ± 10.4	.859	.358
Chair standing (s)	22.8 ± 5.4	10.1 ± 16.7	20.3 ± 6.1	11.1 ± 12.8	.156	.699
SPPB summary	3.0 ± 2.2	2.2 ± 2.0	3.8 ± 2.8	2.5 ± 2.3	.945	.355
Grip strength (kg) ‡	14.4 ± 7.0	9.8 ± 6.6	17.9 ± 7.5	14.6 ± 7.7	973	.328
Falls in previous 6 months (n)	.5 ± 1.0	1.0 ± 1.7	.3 ± .7	.9 ± 1.6	.000	.986
Hospital admission in previous year (n)	1.8 ± 1.1	1.5 ± 1.7	1.6 ± 1.4	1.9 ± 2.4	.736	.404
MMSE	18.6 ± 6.8	15.8 ± 8.1	21.0 ± 6.1	18.9 ± 8.9	.077	.782
GDS	5.4 ± 3.6	4.9 ± 2.7	4.3 ± 3.3	3.7 ± 3.4	.022	.883
MNA	9.4 ± 3.1	7.9 ± 3.4	10.9 ± 2.2	8.6 ± 2.5	.755	.389

Data are expressed as mean ± standard deviation.

* groups x time ANOVA

Between group ANOVA: †< .001; ‡ <.05.

% = percent, kg = kilograms, m = metres, s = seconds

SMI = skeletal muscle index, SPPB = Short physical performance battery, MMSE= Mini Mental State Examination, GDS= Geriatric Depression Scale, MNA= Mini Nutritional Assessment