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Longitudinal associations between body composition, sarcopenic obesity and outcomes of frailty, disability, institutionalisation and mortality in community-dwelling older men: The Concord Health and Ageing in Men Project

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Abstract

Background: to explore the longitudinal associations between body composition measures, sarcopenic obesity and outcomes of frailty, activities of daily living (ADL) and instrumental ADL (IADL) disability, institutionalisation and mortality.
Methods: men aged ≥ 70 years (2005–07) from the Concord Health and Ageing in Men Project were assessed at baseline (n = 1,705), 2 (n = 1,366) and 5 years (n = 954). The main outcome measures were frailty (adapted Fried criteria), ADL, including personal care and mobility and IADL disability (ability to perform tasks for independent living), institutionalisation and mortality. The Foundation for the National Institutes of Health cut-points were used for low muscle mass: appendicular lean mass (ALM):Body Mass Index (BMI) ratio (ALM_{BMI}) <0.789 and obesity was defined as >30% fat. Generalised estimating equations were used to examine the longitudinal associations between the independent variables (obesity alone, low muscle mass and sarcopenic obesity) and frailty, ADL and IADL disability.
Results: in unadjusted, age adjusted and fully adjusted analysis, men with low muscle mass showed increased risk of frailty and IADL disability. In fully adjusted analysis, men with sarcopenic obesity had an increased risk of frailty (odds ratio (OR): 2.00 (95% confidence of interval (CI): 1.42, 2.82)) ADL disability (OR: 1.58 (95% CI: 1.12, 2.24)) and IADL disability (OR: 1.36 (95% CI: 1.05, 1.76)). Obesity alone was protective for institutionalisation (OR: 0.51 (95% CI: 0.31, 0.84)) but was not associated with any other outcomes.
Conclusions: low muscle mass and sarcopenic obesity were associated with poor functional outcomes, independent of confounders. This would suggest that future trials on frailty and disability prevention should be designed to intervene on both muscle mass and fat mass.

Keywords: *body composition, frailty, ADL and IADL disability, institutionalisation, mortality, older men*

Introduction

An age-related reduction in muscle mass and strength is termed sarcopenia [1] and is associated with a range of adverse health consequences among older people [2] .The interplay between low muscle mass and rising trends in obesity in an aging population [3] is emerging as an important public health problem called 'sarcopenic obesity' [4, 5]. Roubenoff postulated that declines in lean mass could contribute to further gains in fat mass, and vice-versa [6]. High fat mass has been shown to be associated with lower muscle quality, and predicts accelerated loss of lean mass [7]. Low muscle mass with obesity termed 'sarcopenic obesity', can co-occur and share common inflammatory pathways [8], and have biologically plausible synergistic effects that can result in substantially increased risk of adverse functional outcomes [5] compared to the risk from either condition alone [9]. Studies have shown that sarcopenic obesity predicts worse clinical outcomes than low muscle mass or obesity in isolation [4, 8] but a variety of indices for this have been used to define sarcopenic obesity, since there have been no standard definitions [4]. The Foundation for the National Institutes of Health (FNIH) Sarcopenia Project has recently developed a recommended set of clinically relevant criteria and identified cut-points for low lean mass [10] from cohort studies of community-dwelling, diverse and well-characterised older populations. While low muscle mass has been shown to be associated with frailty in older people [11], it is unclear whether sarcopenic obesity is associated with increased risk of frailty [4]. The studies that have evaluated associations between sarcopenic obesity with disability [4, 12] have been conflicting possibly due to a variation in criteria used to define sarcopenic obesity. There are no studies that have looked at associations between sarcopenic obesity with institutionalisation and, although studies have shown increased risk of mortality related to low muscle mass or low muscle strength [13, 14], the relationship between having both low muscle mass with obesity and mortality is unclear [15, 16].

We have previously reported relationships between sarcopenia and incident activities of daily living (ADL) disability, institutionalisation and all-cause mortality in the Concord Health and Ageing in men project (CHAMP) population [17]. In this current study we extend that work to investigate the influence of lean mass, fat percentage and their interaction, as continuous variables, and use FNIH-defined low muscle mass to focus on investigating longitudinal associations between low muscle mass, obesity and sarcopenic obesity on these outcomes, as well as additional outcomes of frailty and instrumental ADL (IADL) disability. This study formed the basis of a working report published by CEPAR [18].

Methods

Population

CHAMP is an epidemiological study of a wide range of health issues in Australian men aged 70 years and over [19]. The selection of study subjects has been described in detail elsewhere [19]. Briefly, CHAMP involves men living in a defined urban geographical region near Concord Hospital in Sydney, Australia. The sampling frame was the New South Wales Electoral Roll. The only exclusion criterion was living in a residential aged care facility (RACF). Of the 2,815 eligible men with whom contact was made, 1,511 participated in the study (54%). An additional 194 eligible men living in the study area heard about the study from friends or the local media and were recruited after contacting the study investigators prior to being identified through electoral rolls, yielding a total of 1,705 subjects.

Data collection

Baseline data were collected between January 2005 and June 2007. Men completed a questionnaire at home before coming to the study clinic at Concord Hospital that consisted of a range of measures. Two-year follow-up assessments were conducted between January 2007 and October 2009 and 5-year follow-up was conducted between January 2012 and October 2013, using the same measures as at baseline. Of the 1,705 subjects who completed the baseline assessments, a total of 1,666 subjects were included in this study. Of these 1,666, 1,314 (79%) had 2-year follow-up assessments and 917 (55%) had 5-year follow-up assessments

Measurements

Body mass index

Height (measured using the Harpenden Portable Stadiometer) and weight (measured using Wedderburn digital scales) were measured to determine body mass index (BMI = weight/height^2, with units kg/m^2).

Appendicular lean mass and fat percentage

Whole-body dual energy x-ray absorptiometry (DXA) scans were acquired using the fan beam Discovery-W scanner (Hologic Inc., Bedford, MA, USA). Appendicular lean mass (ALM) was calculated as the sum of lean mass of arms and legs (kg). Fat percentage was calculated using bone, lean and fat mass to estimate total fat mass divided by measured weight (kg) × 100 [20]. The coefficient of variation (CV%) for scans duplicated on 30 men from the study cohort was 11.0% for lean mass and 2.5%, for body fat mass. The same DXA machine was used at baseline, 2- and 5-year follow-up.

Definitions of low muscle mass, obesity and both low muscle mass with obesity

We used the FNIH clinically relevant cut-points for low lean mass defined as ALM:BMI ratio (ALM_{BMI}) less than 0.789 for men [10].

BMI fails to differentiate between lean and fat tissue, so it has been suggested that obesity should be identified by body fat levels [12]. In our study we defined obesity as

percent fat mass more than 30% according to recent definitions [21]. A four-level variable was created: neither obese nor low muscle mass, obese only, low muscle mass only and sarcopenic obesity.

Main outcome measures

Frailty

Frailty was defined using both Fried criteria in the Cardiovascular Health Study (the CHS frailty index) [22] and the criteria proposed by Ensrud *et al.* [23] in the Study of Osteoporotic Fractures (the SOF frailty index). The CHS frailty index is comprised of five criteria: weight loss, exhaustion, low activity, slowness, and weakness; measurement in CHAMP was as previously described [24]. Subjects were considered frail if they had three or more of the frailty components. Frailty scores were calculated at baseline, at 2- and 5-year follow-up. Participants were classified as frail or not at each time point.

ADL disability

ADL disability was assessed by seven items from a modified version of the Katz ADL scale [26] and was defined as needing help with ≥1 activities on the Katz ADL scale [27]. Participants were classified as disabled or not at each time point.

IADL disability

The IADL questionnaire asks subjects how much help they need to perform ten tasks considered important for independent living [28]. IADL disability was defined as needing help to perform ≥1 of the IADL tasks [27]. Participants were classified as disabled or not at each time point.

Institutionalisation and mortality

Data on institutionalisation and mortality was regularly updated at 4-monthly intervals. Follow-up was for a median of 7 years (range: 4.0 days–9.4 years).

Institutionalisation was defined as entry into a RACF at any time during follow-up. There were 191 RACF admissions during follow-up that ended in June 2014.

If men withdrew from the study but agreed to passive follow up, the New South Wales Registry of Births, Deaths and Marriages was contacted to ascertain death status. Mortality follow-up ended on the date of death, date of withdrawal or 26 June 2014. There were 535 deaths and 61 men lost to follow-up.

Other measures

Sociodemographic and economic measures

Sociodemographic variables included age and income categorised as reliant on a government pension only versus other sources of income.

Lifestyle factors

Smoking status (never smoker, ex-smoker and current smoker) was assessed. Physical activity was measured using the Physical Activity Scale for the Elderly (PASE) [25].

Health status

Data on doctor diagnosed medical conditions were obtained from a self-reported questionnaire where participants reported having any of the following diseases: diabetes, thyroid dysfunction, osteoporosis, Paget's disease, stroke, Parkinson's disease, epilepsy, hypertension, heart attack, angina, congestive heart failure, intermittent claudication, chronic obstructive lung disease, liver disease, cancer (excluding non-melanoma skin cancers), osteoarthritis and gout. Participants with a total of five or more depressive symptoms, evaluated by the Geriatric Depression Scale (GDS) [29] were considered to have possible depression.

All participants were screened for cognitive impairment using the mini–mental state examination (MMSE) and the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE) [29, 30]. Men who scored ≤26 on the MMSE or ≥3.6 on the IQCODE were invited to have a detailed clinical assessment. Using all the available information at a consensus meeting, participants were categorised as having no cognitive impairment, mild cognitive impairment or dementia.

Blood tests

Blood tests were performed at the Diagnostic Pathology Unit of Concord RG Hospital, which is a NATA (National Australian Testing Authority) accredited pathology service, using a MODULAR Analytics system (Roche Diagnostics, Castle Hill, Australia). Haemoglobin measured by absorption spectrophotometry and white blood cell analysis performed by laser flow cytometry were used as a continuous measure in the analyses.

Medication assessment

Polypharmacy was defined as the regular use of ≥5 prescription medicines [31]. The full Methods section is available online.

Statistical analyses

Analysis was carried out using STATA v12 (Stata Corp., College Station, TX). Descriptive characteristics are expressed as means (SD) and percentages. The goodness of fit of all the final adjusted models was assessed using the Hosmer–Lemeshow statistic.

To study the longitudinal associations between measures of body composition (continuous and categorical) and outcomes between baseline, 2-year follow-up and 5-year follow-up, we used generalised estimating equation (GEE) analyses [32]. GEE takes into account the time- varying nature of both the outcome and the exposure. With GEE analysis, the

association between two longitudinally measured variables can be studied using all longitudinal data simultaneously and adjusting for within person correlations caused by repeated measurement on each participant using robust estimation of the variances of the regression coefficients. We used the GEE model in continuous analysis for ALM_{BMI}, fat percentage and their interaction. GEE analysis was also used in categorical analysis (neither obese nor low muscle mass (referent variable)), obese only, low muscle mass only and sarcopenic obesity. Separate models were run for each outcome variable: frailty, ADL or IADL disability as the dependent variables.

Univariate Cox regressions were conducted to determine the unadjusted hazard ratios (HR) for mortality and institutionalisation in separate analysis for continuous variables (ALM_{BMI}, fat percentage and their interaction and categorical variables.

For Cox regression and GEE analysis, variables that had a $P < 0.1$ in univariate analyses were included in the model as covariates. Backward stepwise elimination was used to eliminate non-significant variables from the multivariate model. Models were initially unadjusted, then age adjusted and then further adjusted by potential confounders including sociodemographic, lifestyle factors, health conditions and measures of clinical significance. Variables of clinical significance were also included as independent variables for the adjusted GEE and Cox regression analysis.

Ethics approval and informed consent

All participants gave written informed consent. The study was approved by the Sydney South West Area Health Service Human Research Ethics Committee, Concord Repatriation General Hospital, Sydney, Australia.

Results

Descriptive data at the three CHAMP study points of baseline, 2- and 5-year follow-up studies are shown in Table 1. The mean age of the participants was 76.9 ± 5.5 years at baseline, 78.6 ± 5.2 years at 2-year follow-up and 81.4 ± 4.6 years at 5-year follow-up. There were significant differences in body composition measures (neither obese nor low muscle mass, obese only, low muscle mass only and sarcopenic obesity. as categorical variables and the outcomes between baseline and 2- and 5-year follow-up. Of 1,705 men at baseline, 191 (11.4%) men were institutionalised and 535 (31.8%) died during follow-up from January 2006 to June 2014.

Longitudinal analyses: frailty, ADL and IADL disability

As shown in Table 2, there were significant longitudinal associations between low muscle mass and sarcopenic obesity with frailty in unadjusted, age adjusted and fully adjusted analysis. Sarcopenic obesity was significantly associated with ADL disability: OR 1.58 (95% confidence of interval (CI): 1.12, 2.24, $P = 0.01$) in the fully adjusted analysis. Low muscle mass and sarcopenic obesity were also significantly associated with IADL disability in unadjusted, age adjusted and fully adjusted analysis. Obesity was not associated with frailty, ADL or IADL disability in unadjusted, age adjusted analysis or with full adjustment for covariates.

Further GEE analysis (Supplementary Table 1A) of the continuous body composition variables showed that in unadjusted, age adjusted and fully adjusted analysis, ALM_{BMI} was inversely associated with frailty and ADL disability and fat percentage was positively associated with frailty and ADL disability. GEE analysis of ALM_{BMI}–fat percentage interaction showed significant inverse associations with frailty and ADL disability. ALM_{BMI} was inversely associated and fat percentage was positively associated with IADL disability but the interaction between these two variables was not significantly associated with IADL disability.

Institutionalisation

In the unadjusted, age adjusted model, and fully adjusted categorical analysis there was a significant inverse association

Table 1. Selected general characteristics of men aged 70 and older by CHAMP study period

	Baseline mean (SD) or *N* (%), *n* = 1,685	2-Year mean (SD) or *N* (%), *n* = 1,347	5-Year mean (SD) or *N* (%), *n* = 950	*P* value
Age (years)	76.9 (5.5)	78.6 (5.2)	81.4 (4.6)	<0.0001
BMI (kg/m^2)	27.8 (4.0)	27.8 (3.9)	27.6 (3.9)	<0.0001
No. of comorbidities	2.5 (1.8)	2.5 (1.7)	2.5 (1.6)	0.02
Physical activity (PASE)	124.5 (62.2)	119.8 (59.7)	117.4 (63.2)	<0.0001
Current smoker	100 (6%)	49 (4%)	33 (4%)	0.03
Haemoglobin (g/dl)	14.3 (1.4)	14.2 (1.3)	14.1 (1.4)	0.03
White cell count	6.5 (2.4)	6.6 (3.4)	6.7 (3.7)	0.003
Neither obesity or low muscle mass	691 (41.2%)	517 (38.5%)	308 (33.9%)	<0.0001
Obesity alone	574 (34.2%)	574 (37.2%)	348 (38.2%)	<0.0001
Low muscle mass alone	247 (14.7%)	184 (13.7%)	143 (15.7%)	<0.0001
Sarcopenic obesity	166 (9.9%)	143 (10.6%)	111 (12.2%)	<0.0001
Frailty	158 (9.5%)	129 (9.7%)	93 (9.9)	0.003
ADL disability	140 (8.2%)	138 (10.1%)	121 (12.7%)	<0.0001
IADL disability	550 (33.5%)	488 (37.0%)	395 (41.2%)	<0.0001

Data are means ± SD unless otherwise indicated.

Table 2. Unadjusted age adjusted and multi-variable adjusted odds ratios (ORs) for GEE analyses for the association between obesity, low muscle mass and sarcopenic obesity status at three time points and frailty, ADL and IADL disability: the CHAMP Study

	Odds ratio (95% CI, *P* value)		
	Model 1 unadjusted	Model 2 adjusted	Model 3 adjusted
Frailty			
No obesity nor low muscle mass, low lean mass = ALM_{BMI} > 0.789 and fat mass < 30.0%) OR = 1 (reference)	1	1	1
Obesity alone (fat mass > 30.0%)	0.73 (0.45,1.20), $P = 0.21$	0.83 (0.50,1.39), $P = 0.48$	1.00 (0.56,1.76), $P = 0.94$
Low lean mass alone = ALM_{BMI} < 0.789	2.35 (1.67,3.29), $P < 0.0001$	2.24 (1.57,3.19), $P < 0.0001$	2.12 (1.42,3.18), $P < 0.0001$
Sarcopenic obesity (low lean mass = ALM_{BMI} < 0.789 and fat mass > 30.0%)	2.21 (1.65,2.96), $P < 0.0001$	2.24 (1.65,3.04), $P < 0.0001$	2.00 (1.42,2.82), $P < 0.0001$
ADL disability			
No obesity nor low muscle mass, OR = 1 (reference)	1	1	1
Obesity alone	1.13 (0.66,1.92), $P = 0.65$	1.00 (0.61,1.66), $P = 0.97$	1.03 (0.60,1.78), $P = 0.92$
Low muscle mass alone	1.84 (1.60,3.27), $P = 0.004$	2.12 (1.47,3.06), $P < 0.0001$	1.30 (0.84,1.99), $P = 0.24$
Sarcopenic obesity	2.24 (1.60,3.12), $P < 0.0001$	2.73 (2.01,3.69), $P < 0.0001$	1.58 (1.12,2.24), $P = 0.01$
IADL disability			
No obesity nor low muscle mass, OR = 1 (reference)	1	1	1
Obesity alone	1.03 (0.82,1.30), $P = 0.80$	1.11 (0.86,1.42), $P = 0.42$	0.99 (0.74,1.30), $P = 0.92$
Low muscle mass alone	1.76 (1.42, 2.17), $P < 0.0001$	1.73 (1.38,2.17), $P < 0.0001$	1.36 (1.05,1.76), $P = 0.02$
Sarcopenic obesity	1.76 (1.47,2.11), $P < 0.0001$	1.77 (1.45,2.14), $P < 0.0001$	1.32 (1.06,1.64), $P = 0.01$

Model 1—unadjusted; Model 2—adjusted for age Model 3—adjusted for: age, income, smoking status, physical activity, no of comorbidities, myocardial infarction, dementia, depressive symptoms, low haemoglobin, polypharmacy and white cell count.

between obesity and institutionalisation. Low muscle mass and sarcopenic obesity were not associated with institutionalisation (Table 3).

Further GEE analysis (Supplementary Table 2A) shows the results of the Cox proportional hazard models for institutionalisation and ALM_{BMI} and fat percentage as continuous variables. ALM_{BMI} was inversely associated with institutionalisation, in unadjusted, age adjusted and fully adjusted analysis but fat percentage and the interaction between ALM_{BMI} and fat percentage were not associated with institutionalisation.

Mortality

Table 3 shows results for the categorical analysis with mortality. There was a significant association between low muscle mass and sarcopenic obesity with increased mortality in unadjusted analysis but significance was lost with adjustment by age. Obesity was not associated with mortality.

Further GEE analysis (Supplementary Table 2A) shows that there was a significant inverse association between ALM_{BMI} and mortality in unadjusted, age adjusted and fully adjusted analysis. There was also a statistically significant inverse association between ALM_{BMI}–fat percentage interaction and mortality in unadjusted and age adjusted analysis but the significance was lost with full adjustment by covariates. Fat percentage alone was not associated with mortality.

Discussion

Our study is the first population-based longitudinal study that uses FNIH criteria for lean muscle mass to assess longitudinal associations between combined low muscle mass and obesity measures or 'sarcopenic obesity' with outcomes frailty, ADL and IADL disability, institutionalisation and all-cause mortality. We also used continuous measures of body composition, regarded as a more sensitive approach for detecting associations than categorical measures.

We found that both sarcopenic obesity and ALM_{BMI}–fat percentage interaction were associated with increased risk of frailty after full adjustment by confounders and covariates of clinical significance. The majority of research on functional outcomes related to muscle has focused on decline in mobility [33, 34], not on frailty *per se*. Two recent reviews have stated that sarcopenia and frailty are related and sarcopenia is a key component of frailty in older populations [35, 36], but there have not been any previous longitudinal studies showing the temporal relationship between continuous measures of body compositions or sarcopenic obesity and frailty.

Our findings on ADL disability with sarcopenic obesity and ALM_{BMI}–fat percentage interaction are similar to a cross-sectional study among older adults aged 60 years and over that showed sarcopenic obesity to be associated with a higher risk of having three or more physical disabilities [5] This in contrast to NHANES III study [37], which reported that sarcopenic obesity was not associated with functional limitations, including walking one quarter mile, walking up 10 steps without resting. These differences may be due to NHANES III using percentage body fat and muscle mass based on published anthropometric prediction equations [2]. A longitudinal study, the InCHIANTI study among participants aged 65 years and over, found that sarcopenic obesity was associated with an increased risk of decline in walking speed and developing mobility disability at 6-year follow-up. The InCHIANTI study used BMI to assess obesity, not DEXA fat mass [36].

Table 3. Unadjusted age adjusted and multi-variable adjusted hazard ratios (HRs) for the association between obesity, low muscle mass and sarcopenic obesity status and institutionalisation and mortality: the CHAMP Study

	Hazard ratio (95% CI, *P* value)		
	Model 1 unadjusted	Model 2 adjusted	Model 3 adjusted
Institutionalisation			
No obesity nor low muscle mass, OR = 1 (reference)	1	1	1
Obesity alone	0.51 (0.33,0.82), *P* = 0.01	0.53 (0.33,0.85), *P* = 0.01	0.51 (0.31,0.84), *P* = 0.01
Low muscle mass alone	0.99 (0.61,1.61), *P* = 0.99	1.08 (0.66,1.76), *P* = 0.76	0.86 (0.50,1.49), *P* = 0.59
Sarcopenic obesity	0.87 (0.58,1.29), *P* = 0.49	0.92 (0.62,1.38), *P* = 0.71	0.80 (0.52,1.23), *P* = 0.30
Mortality			
No obesity nor low muscle mass, OR = 1 (reference)			
Obesity alone	0.81 (0.63,1.03), *P* = 0.10	0.82 (0.64,1.05), *P* = 0.11	0.81 (0.60,1.04), *P* = 0.10
Low muscle mass alone	1.50 (1.10,2.02), *P* = 0.01	1.29 (0.95,1.75), *P* = 0.11	0.98 (0.70,1.38), *P* = 0.92
Sarcopenic obesity	1.30 (1.05,1.60), *P* = 0.02	1.14 (0.92,1.42), *P* = 0.22	0.88 (0.70,1.11), *P* = 0.29

Model 1 unadjusted; Model 2 adjusted for age; Institutionalisation: Model 3 age, income, physical activity, no of comorbidities, dementia, ADL disability; Mortality: Model 3 adjusted for age, income, smoking status, physical activity, no of comorbidities, dementia, myocardial infarction, ADL disability, polypharmacy, white cell count and haemoglobin levels.

We found that there were no significant longitudinal associations between ALM_{BMI} and fat percentage interaction term and IADL disability. Furthermore, while we found that sarcopenic obesity was associated with IADL disability, the magnitude of increased risk was the same as for low muscle mass. These findings are in contrast to The New Mexico Aging Process Study, where sarcopenic obesity at baseline was associated with a 2 to 3-fold increase in risk of developing IADL disability during an 8-year follow-up period compared to lean sarcopenic or non-sarcopenic obese subjects [12]. In this study, sarcopenic obesity in men was defined as <7.26 kg/m^2 and percentage body fat greater than 28% body fat in men. These contrasting findings may be due to use of different definitions for IADL disability and sarcopenic obesity.

In our study, we found significant negative associations between ALM_{BMI} and institutionalisation but no significant associations were found between ALM_{BMI} and fat percentage interaction term or sarcopenic obesity and institutionalisation. However, we did find that obesity was protective of institutionalisation. In contrast to our findings a longitudinal study has found an association between obesity (BMI > 35kg/m^2) among adults aged 45 years and over and increased risk of nursing home admission [38]. These differences in findings may be due to younger age and the variation in the obesity measure in this study. There do not appear to be any previous studies of the relationship between body composition and risk of institutionalisation. A recent review concluded that more research is required in this area [39].

The interaction between ALM_{BMI} and fat percentage as continuous variables was significantly associated with mortality in our study but using categorical measures was not. A recent meta-analysis of prospective cohort studies [40] investigating the association between sarcopenic obesity and risk of all-cause mortality showed that sarcopenic obesity was significantly associated with increased risk of mortality. Another study [41], showed that sarcopenic obesity was not a predictor of mortality. These differences in findings may be due to the differences in the definition of sarcopenic obesity. Sarcopenia in men was defined as ≤5.75 kg/m^2 and obesity was based on % body fat ≥27%.

Research on the combined effect of muscle mass and obesity is hindered by the lack of a widely agreed operational definition [33]. We used the FNIH sarcopenia cut-offs derived from pooled data sets from nine large studies among Caucasian community-dwelling older people. The advantages of using the FNIH data sets are that the cut-offs are derived from studies including a population that are readily generalisable to our study population, where the majority were also Caucasian, and because the data set has a broad representation of community-dwelling older adults. Other definitions such as the European Working Group on Sarcopenia in Older People (EWGSOP) use data from young adults [1].

The main strengths of our study are that it involves a large, representative sample of community-dwelling older Australian men aged 70 years and over with longitudinal data. We have measurements of body composition and outcome measures over a relatively long follow-up period. We used DEXA to measure body composition which has an advantage in the ability to estimate total and sub compartments of lean and fat mass in comparison to BMI and equation based estimates.

Our study has some limitations. The CHAMP study had a baseline participation rate of about 50%, which is an acceptable response rate for a longitudinal study in men of this age and for epidemiological studies of this nature. Despite this response rate, the age distribution of the men in the CHAMP study is similar to that of the target census population [19] and the prevalence of self-reported disease in CHAMP participants is very similar to that found in an Australian national telephone survey of men's health [42].We acknowledge that functional components of sarcopenia are important to include in the definition of sarcopenia, however due to small numbers in the sample when including these measures we had to limit our analysis to categories: neither obese nor low muscle mass, obese only, low muscle mass

only and sarcopenic obesity. Another limitation is the relatively high loss to follow-up, mainly due to death or poor health, which are inevitable in studies of older people. However, the GEE analysis methodology is robust with regard to data missing at random in longitudinal analyses. We did not have clinical data for the men who refused to participate in the study so we are unable to provide a direct comparison between participants and non-participants.

We conclude that in community-dwelling older men, we find longitudinal associations between low muscle mass, sarcopenic obesity and increased risk of poor functional outcomes. This would suggest that future trials on frailty and disability prevention should be designed to intervene on both muscle mass and fat mass.

Key points

- Men with low muscle mass (The Foundation for the National Institutes of Health (FNIH) Sarcopenia Project definition: appendicular lean mass (ALM): Body Mass Index (BMI) ratio (ALM_{BMI}) < 0.789), showed increased risk of frailty and IADL disability.
- Men with sarcopenic obesity (ALM_{BMI} < 0.789 and fat percentage >30%) show an increased risk of frailty and disability.
- Obesity alone was protective for institutionalisation but was not associated with any other outcomes.
- Further research to investigate and identify interventions aimed at prevention of sarcopenic obesity are needed.

Supplementary data

Supplementary data mentioned in the text are available to subscribers in *Age and Ageing* online.

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Conflicts of interest

The authors declare that they have no conflict of interest.

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