Taylor & Francis
Taylor & Francis Group

Acta Clinica Belgica
International Journal of Clinical and Laboratory Medicine

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/yacb20

Associations between obesity, self-reported weakness and their combinations with mortality in nursing home residents

Gulistan Bahat, Birkan Ilhan, Nezahat Muge Catikkas, Asli Tufan, Savaş Ozturk, Hafize Dogan & Mehmet Akif Karan

To cite this article: Gulistan Bahat, Birkan Ilhan, Nezahat Muge Catikkas, Asli Tufan, Savaş Ozturk, Hafize Dogan & Mehmet Akif Karan (2023) Associations between obesity, self-reported weakness and their combinations with mortality in nursing home residents, Acta Clinica Belgica, 78:2, 112-121, DOI: 10.1080/17843286.2022.2075180

To link to this article: https://doi.org/10.1080/17843286.2022.2075180

ACTA CLINICA BELGICA
2023, VOL. 78, NO. 2, 112–121
https://doi.org/10.1080/17843286.2022.2075180

Taylor & Francis
Taylor & Francis Group

ORIGINAL PAPER

Associations between obesity, self-reported weakness and their combinations with mortality in nursing home residents

Gulistan Bahat[a], Birkan Ilhan[a], Nezahat Muge Catikkas[a], Asli Tufan[a], Savaş Ozturk[b], Hafize Dogan[c] and Mehmet Akif Karan[a]

[a]Department of Internal Medicine, Division of Geriatrics, Istanbul University, Istanbul Medical School, Istanbul, Turkey; [b]Department of Internal Medicine, Istanbul University, Istanbul Medical School, Istanbul, Turkey; [c]Department of General Practitioner, Istanbul Kayisdagi Nursing Home, Istanbul, Turkey

ABSTRACT

Objectives: There are studies on associations between obesity and mortality in nursing home (NH) residents, but the presence of concomitant muscle weakness has not been examined. We considered that self-reported weakness might be a low muscle strength proxy marker. We aimed to examine associations of obesity alone, self-reported muscle weakness alone, and their combination with mortality in NH residents.

Methods: This is a retrospective longitudinal follow-up study. We noted age, sex, nutritional status, functionality, number of chronic diseases, and regular medication. Obesity was assessed by the body fat-percentage method estimated by bioimpedance analysis. Weakness was identified by self-reported muscle weakness. Survival was evaluated with a univariate log-rank test and multivariate Cox regression analyses.

Results: We included 214 participants. In a median follow-up time of 46 months, mortality occurred in 37.4%. In multivariate analysis adjusted by age, sex, undernutrition, number of chronic diseases, and regular medication, functional scores; 'non-weak non-obese' participants or 'weak alone' participants or 'weak+obese' participants had higher mortality risk when compared with the 'obesity alone' participants [hazard ratio (HR) = 2.6, 95% confidence interval (CI) = 1.2–5.5, p = 0.01; HR = 2.6, 95% CI = 1.2–5.9, p = 0.02; HR = 3.0, 95% CI = 1.2–7.7, p = 0.02].

Conclusion: This is the first report showing that obesity was associated with lower mortality risk if the weakness was not present in NH residents. However, obesity with concomitant weakness was associated with mortality risk similar to non-weak non-obese or weak alone participants. Our study suggests a simple consideration of weakness that can easily be integrated into everyday practice.

KEYWORDS
Mortality; Nursing home; obesity; self-reported; muscle weakness

1. Introduction

Body composition phenotypes have received attention for decades to stratify older adults according to risks for disability and mortality [1–4]. Recognising the changes in the body composition by ageing, which indicate loss of muscle mass, strength, and increase in adiposity, researchers have been questioning whether the increased mortality and disability rates were consequences of these changes to some extent. One of the most commonly used operational definitions of sarcopenia (S) is the EWGSOP definition. It was updated in late 2018 and proposed low muscle strength as the vital characteristic of sarcopenia [5]. EWGSOP2 suggested a new term, 'probable sarcopenia', which corresponds to the presence of low muscle strength [5].

Health-related self-assessment of individuals has been suggested as having significant success in line with objective assessments of the individuals. While objective assessment is the gold standard, the self-assessment approach has a great potential for broader application, as objective assessment mostly requires instruments and increased assessment time to use the instruments. For example, self-rated health was independently associated with mortality even after adjustment for confounding factors [6,7]. Moreover, it has been recently shown that besides self-rated health, caregiver-rated health was independently associated with mortality, and this association was superior to the patient's self-rated health in dementia patients [8].

Recently, researchers suggested an analogy of this approach for evaluating handgrip strength considering the limited access to hand-dynamometer in most practice settings. It has been suggested that patient-rated subjective evaluation of grip strength can be used as a proxy marker to some extent to evaluate low muscle strength [9–12].

Obesity is another recognised body composition phenotype, common among older adults besides sarcopenia [13]. The prevalence of overweight and

CONTACT Gulistan Bahat gbahatozturk@yahoo.com Department of Internal Medicine, Istanbul University, Istanbul Medical School, Capa, 34390, Istanbul, Turkey

Current address: University of Health Sciences Istanbul, Sisli Hamidiye Etfal Training and Research Hospital
Current address: Marmara University

obesity in older adults has become a growing concern. However, a well-known concept, 'obesity-paradox', suggests the protective effect of obesity against mortality in particular circumstances, including the older population [14–16]. Yet, there are conflicting reports in this regard, so the relation between obesity and mortality in older adults is equivocal at present [16–19]. In most of these reports, the relation between obesity and mortality was not explicitly analysed, considering the presence or absence of accompanying weakness. The setting of the older adults may have a differential effect in this regard [17].

Sarcopenia, obesity, and sarcopenic obesity (SO) were all reported as prevalent conditions in the nursing home NH residents [20]. There are many studies on associations of sarcopenia with its variable definitions for mortality [21–25]. However, the presence of obesity has also not been examined in most of them. Sarcopenic obesity (SO) researches are few in comparison to S or obesity, and to date, there has been no consensus operational diagnostic definition of SO. Research examining SO identified by applying different definitions is needed to clarify the relationship between obesity and adverse outcomes in older adults [26].

In this study, we considered self-reported muscle weakness as a proxy marker of muscle strength. As far as we know, there is no study investigating the associations of mortality with weakness+obesity, obesity alone, weakness alone, and non-weakness non-obesity in NH residents comparatively. Also, in the literature, there is no study evaluating muscle weakness through self-reported muscle weakness while grouping the body phenotypes, i.e. non-weak non-obese, weak alone, weak+obese and obesity alone. Considering the constantly increasing ratio of the NH population and the unelucidated association between the body phenotypes and mortality risk, we aimed to examine comparable associations of body phenotypes with mortality in NH residents.

2. Methods

We designed a retrospective longitudinal follow-up study. We complied with the guidelines in the Helsinki Declaration. We briefly informed all participants about the aims and procedures of the research study and received a signed consent form from all participants or their legal representatives in cases of cognitively impaired/not communicable. We obtained ethical approval from the Istanbul University Istanbul Medical School ethical board (No: 2021/234). We also received legal permission from the NH administration.

2.1. Population and setting

The study was conducted among residents of the most crowded NH in the city. It was the largest NH in the city and was subordinated to Metropolitan Municipality. The acceptance depended only on the lack of adequate support for the older adults to stay in their homes. The Metropolitan Municipality met all health expenses and maintenance of the NH residents. We excluded the residents who did not give informed consent or had conditions that might interfere with bioimpedance analysis (BIA) measurement (e.g. having metal implants, cardiac pacemaker, presence of the edematous state, or major disorders of fluid disturbance). We collected the baseline data in September 2009 and assessed the mortality outcome in June 2013. We recently reported longitudinal follow-up data examining the success of the Simpler Modified Fried Frailty Scale proposed by our group to predict mortality in this cohort [10]. We considered that a period would need to pass the emergence of the adverse effect of chronic disease (e.g. diabetes, hypertension, etc.). As such, we considered that a period would be needed for body phenotypes to exert any effect on mortality. Hence, we excluded the participants who died within the period less than six months of the study entry (Only ten participants had died in the first six months.) considering that an interval would be required for the effect of a recent change in fat or muscle strength to become apparent on the mortality risk. We aimed to increase the reliability of our analysis by this consideration.

2.2. Measurements, definitions of body phenotypes and weakness

Bodyweight (kg) and height (m) were measured, and body mass index (BMI) was calculated (kg/m^2). We estimated body composition for fat percentage by BIA (Tanita BC532 model) after 8-hour fasting. Tanita BC 532 is a simple bioimpedance analyser that is easy and available in everyday practice. Correlation analyses in both sexes showed that the body composition parameters, including body fat percentage, by Tanita BC 532 were closely correlated with the measurements by the standard dual-energy x-ray absorptiometry or magnetic resonance imaging [27]. Considering that the classification of obesity by BMI is problematic in older adults, Zoico et al. defined obesity as independent of BMI but based on body fat percentage. They considered obesity as a percentage of fat over 60 per cent of the study population [4]. In a study conducted by our group, aiming to investigate the fat-percentage cutoff values for obesity, the 60th percentile of fat ratio was calculated to determine the obesity threshold for different genders using the fat

percentage method. Body fat percentage cutoffs for obesity were 27.3% for men and 40.7% for women [28]. In the current study, obesity was assessed by the body fat percentage method, which defines obesity as a body fat percentage above the 60th percentile [4]. We used the population-specific cutoffs (>27% in males and >41% in females), which were derived by using the same bioimpedance analyser [28]. We evaluated self-reported muscle weakness by inquiry of the patients and nursing staff (in cognitively non-intact residents) by inquiring about the residents' handgrip strength. We asked the following question '-Do you think grip strength is decreased compared to the same-aged healthy individuals?' A positive answer was noted as self-reported muscle weakness and was considered a proxy marker of low muscle strength [9–12].

We assessed the functional status by Katz activities of daily living (ADL) and Lawton instrumental ADL (IADL) scales with their modified scoring by which the higher scores indicate better functionality [29]. We evaluated nutritional status by mini nutritional assessment short-form (MNA-SF) [30]. An MNA-SF score <12 was assessed as undernutrition (overt malnutrition or at risk of malnutrition). Patients with a clinical diagnosis and/or using a cholinesterase inhibitor and/or memantine and patients with a mini-mental state examination (MMSE) score <24 were defined as having cognitive impairment [31]. Phung et al. compared self- and caregiver-rated health for the patients with mild dementia (MMSE<24) as independent predictors of mortality, and caregiver-rated health but not patient-rated health was found to be a predictor of the mortality [8]. In this context, we used the <24 points on MMSE for the threshold at which the residents were considered unable to express their opinion (cognitively impaired, possibly demented). We derived the residents' medical information from the medical files and responsible health professionals, including the number of chronic diseases and regular medication.

2.3. Statistical analysis

We examined the continuous variables for normality by using the Kolmogorov-Smirnov test. We gave the continuous variables as mean ± standard deviation for normally distributed variables and median (minimum-maximum) for skew-distributed ones. Categorical variables were expressed as absolute numbers and percentages. We evaluated the survival in the follow-up between body phenotype groups with the Kaplan-Meier method and compared it using the log-rank test. We performed a multivariate survival analysis using Cox regression to examine whether body phenotypes, alone or in combination, were related to mortality after adjustment with potential confounders. Potential confounders were determined as covariates associated with mortality in the literature. We checked the multicollinearity among the independent variables of regression analysis with Pearson, Spearman, or Kendall's tau-b correlation analyses. Multicollinearity was not present in between the variables in the models. Hazard ratio (HR) and 95% confidence interval (CI) were computed. A p-value of less than 0.05 was accepted as significant. We used SPSS 21.0 (Statistical Package for the Social Sciences, version 21.0) for Windows was used for the analyses.

3. Results

We included 254 residents in the study entry. Thirty patients were lost to follow-up, and ten residents died within the first six months, leaving 214 residents at the 46th month of evaluation. The mean age was 75.3 ± 8.1 years, and 61.7% were male. Ninety-eight (98) patients (45.8%) were considered cognitively impaired and assessed by the nursing staff. Twenty-nine per cent of the participants had undernutrition (malnutrition or malnutrition risk).

Table 1 shows the body phenotypes of the study population. Non-weak non-obese participants constituted 43.5% of the study population. Weakness+obesity was the least common phenotype with a prevalence of 8.4%, while weakness alone and obesity alone were at comparable rates (25.7% and 22.4%, respectively). Among participants with obesity, 72.7% were non-weak (obesity alone), and 27.2% were concomitantly weak (weak+obese). Among participants with weakness, 75.3% were non-obese (weak alone), and 24.7% were concomitantly obese (weak+obese).

n = 214, mortality occurred in 80 (37.4%) participants

Weakness was defined according to self-reported muscle weakness (low grip strength) assessed through the inquiry of patient and/or caregiver by the following

Table 1. Body phenotypes of the study population (n = 214).

Non-weak non-obese	93 (43.5%)
Weak alone	55 (25.7%)/(75.3%)*
Weak+obese	18 (8.4%)/(24.7%)**/ (27.2%)†
Obesity alone	48 (22.4%)/(72.7%)‡
Weakness [(Weak alone) + (Weak +obese)]	73 (34.1%)
Obesity [(Weak+obese) + (Obesity alone)]	66 (30.8%)

*percentage (%) among participants with weakness (i.e. among participants with weakness, 75.3% were weak alone (without concomitant obesity)

**percentage (%) among participants with weakness (i.e. among participants with weakness, 24.7% were weak+obese (with concomitant obesity)

†percentage (%) among participants with obesity (i.e. among participants with obesity, 27.2% were weak+obese (with concomitant weakness)

‡percentage (%) among participants with obesity (i.e. among participants with obesity, 72.7% were obesity alone (without concomitant weakness)

question: -Do you think grip strength is decreased compared to the same-aged healthy individuals? [9–12].

Obesity was defined as a body fat percentage above the 60th percentile according to the population-specific cutoffs (>27% in males and >41% in females) [4,28].

The median duration of the follow-up period was 46 months (range: 6–46 months). Mortality occurred in 80 of 214 residents (37.4%). The mean (standard deviation) time to mortality was 21.4 (10.5) months [median (min-max): 20.2 (6–40) months]. In univariate analysis, in the participants with obesity alone, mean survival time was significantly longer than those in the other three phenotypes, i.e. non-weak non-obese, weak alone and weak+obese (mean survival time: 41.5 months vs 36.4 months, 33.5 months and 36.5 months; log-rank, p = 0.02, 0.002 and 0.01, respectively) (Figure 1).

n = 214, mortality occurred in 80 (37.4%) participants.

Mean survival time was significantly higher in participants with obesity alone body phenotype (41.5 months) when compared to participants with non-weak non-obese (36.4 months), weak alone (33.5 months) and weak+obese (36.5 months) phenotypes (Log Rank, p = 0.02, 0.002 and 0.01, respectively).

W: weakness, WO: weakness+obesity.

There were no differences between 'non-weak non-obese vs weak alone', 'non-weak non-obese vs weak +obese', and 'weak alone vs weak+obese' phenotypes for their relation to mortality (log-rank, p = 0.30, 0.50 and 0.90, respectively). We ran a multivariate survival analysis to assess whether body phenotype was associated with mortality when adjusted for potential confounders, including age, sex, undernutrition, number of chronic diseases and regular medication, ADL and IADL scores (*Model 1*). In this analysis, when compared with the'obesity alone' phenotype, having non-weak non-obese or weak alone or weak+obese phenotypes were significantly associated with higher mortality risk (HR = 2.6, 95% CI = 1.2–5.5, p = 0.01; HR = 2.6, 95% CI = 1.2–5.9, p = 0.02; HR = 3.0, 95% CI = 1.2–7.7, p = 0.02) (Figure 2 and Table 2).

n = 214, mortality occurred in 80 (37.4%) participants.

When compared with the 'obesity alone' phenotype, having non-weak non-obese or weak alone or weak+obese phenotypes were significantly associated with higher mortality risk (HR = 2.6, 95% CI = 1.2–5.5, p = 0.01; HR = 2.6, 95% CI = 1.2–5.9, p = 0.02; HR = 3.0, 95% CI = 1.2–7.7, p = 0.02)

W: weakness, WO: weakness+obesity, ADL: activities of daily living, IADL: instrumental activities of daily

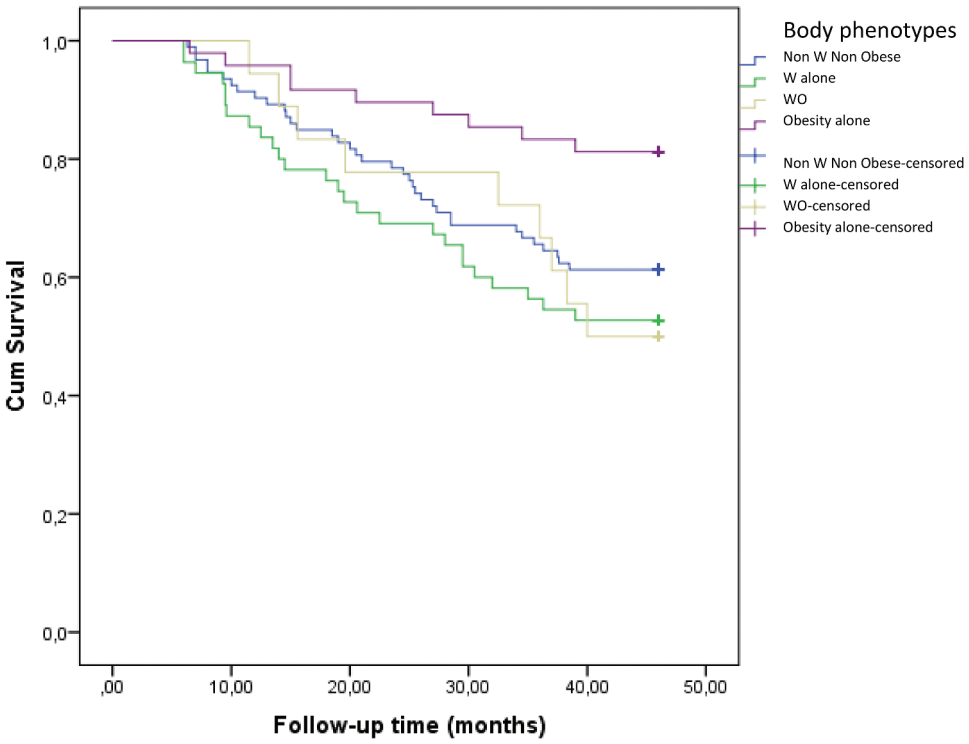

Figure 1. Kaplan Meier survival curves of the patients with different body phenotypes.

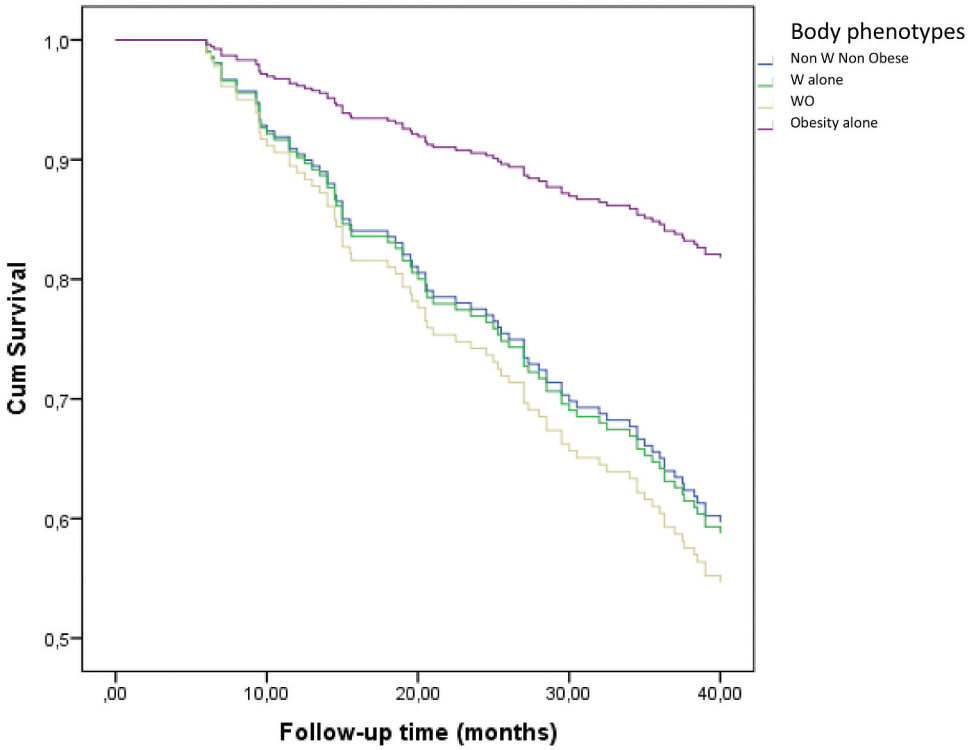

Figure 2. Survival plot curves, based on different body phenotypes (Cox regression analysis) (adjusted by age, sex, undernutrition, number of chronic diseases and regular medication, ADL and IADL scores).

Table 2. Multivariate survival analysis using cox regression for correlates of mortality after 46 months (*model 1*).

	Hazard Ratio	95% Confidence Interval	p
Age	1.03	1.004–1.06	0.03*
Sex (female vs male)	0.80	0.4–1.4	0.40
Undernutrition (present vs absent)	1.70	1.01–2.7	0.04*
Number of chronic diseases	0.98	0.8–1.2	0.90
Number of regular medications	1.10	0.98–1.2	0.10
ADL score	0.96	0.8–1.1	0.60
IADL score	0.99	0.93–1.06	0.90
Body phenotypes (*Obesity alone, Reference)*			
Non-weak non-obese	2.6	1.2–5.5	0.01*
Weak alone	2.6	1.2–5.9	0.02*
Weak+obese	3.0	1.2–7.7	0.02*

*: significantly associated with mortality

Table 3. Multivariate survival analysis using cox regression for correlates of mortality after 46 months (*model 2*).

	Hazard Ratio	95% Confidence Interval	p
Age	1.03	1.0–1.06	0.05
Sex (female vs male)	0.80	0.4–1.4	0.40
Undernutrition (present vs absent)	1.70	1.02–2.7	0.04*
Number of chronic diseases	0.98	0.8–1.2	0.80
Number of regular medications	1.07	0.98–1.2	0.10
ADL score	0.97	0.8–1.1	0.60
IADL score	1.00	0.9–1.08	0.90
Dementia (possibly)$^{\Phi}$	1.20	0.7–2.2	0.50
Body phenotypes (*Obesity alone, Reference)*			
Non-weak non-obese	2.6	1.2–5.4	0.01*
Weak alone	2.6	1.2–5.8	0.02*
Weak+obese	3.0	1.2–7.9	0.02*

*: significantly associated with mortality
$^{\Phi}$: Possibly dementia (cognitive impairment) corresponds to caregiver-rated grip strength and the <24 points on MMSE were used for the threshold at which the residents were considered unable to express their opinion.

living, HR: hazard ratio, CI: confidence interval

n = 214, mortality occurred in 80 (37.4%) participants.

Weakness was defined according to self-reported muscle weakness (low grip strength) assessed through the inquiry of patient and/or caregiver by the following question: -Do you think grip strength is decreased compared to the same-aged healthy individuals? [9–12].

Obesity was defined as a body fat percentage above the 60th percentile according to the population-specific cutoffs (> 27% in males and >41% in females) [4,28].

ADL: activities of daily living, IADL: instrumental activities of daily living

Older age and the presence of undernutrition were also associated with higher mortality risk (HR = 1.03, 95% CI = 1.004–1.06, p = 0.03 and HR = 1.70, 95% CI = 1.01–2.7, p = 0.04, respectively) (Table 2). Similar to the univariate analyses, there were no differences between 'non-weak non-obese vs weak alone', 'non-weak non-obese vs weak+obese', and 'weak alone vs weak+obese' in their relation to mortality in multivariate analysis (Figure 2). When dementia was added to the model (*Model 2*) as a potential confounder, the

association of the other three phenotypes with higher mortality risk, compared to obesity alone phenotype, did not change (Table 3).

n = 214, mortality occurred in 80 (37.4%) participant.

Weakness was defined according to self-reported muscle weakness (low grip strength) assessed through the inquiry of patient and/or caregiver by the following question: -Do you think grip strength is decreased compared to the same-aged healthy individuals? [9–12].

Obesity was defined as a body fat percentage above the 60th percentile according to the population-specific cutoffs (> 27% in males and >41% in females) [4,28].

ADL: activities of daily living, IADL: instrumental activities of daily living

In univariate analysis, both weakness and obesity were related to mortality risk when they were not stratified by the presence of obesity or weakness, respectively. In the participants with weakness, the mean survival time was significantly shorter than the participants without weakness (31.0 months vs 36.0 months, log-rank, p = 0.02), whereas in the participants with obesity mean survival time was significantly longer than the participants without obesity (37.4 months vs 33.0 months, log-rank, p = 0.03). However, when adjusted for confounders (age, sex, undernutrition, number of chronic diseases and regular medication, ADL, IADL scores), weakness was not related to mortality risk (HR = 1.4, 95% CI = 0.9–2.2, p = 0.20); obesity was marginally related to lower mortality risk (HR = 0.6, 95% CI = 0.3–1.01, p = 0.05). The results did not change when dementia was added to the models as a potential confounder (results not detailed). Also, there was no significant difference between the ten participants excluded from the study and 214 participants included in the study in terms of age, sex, undernutrition, number of chronic diseases, number of regular medications, IADL score, dementia, and body phenotypes (p = 0.438, 0.196, 0.500, 0.394, 0.404, 0.056, 0.050, and 0.232, respectively).

4. Discussion

In this study, composed of 214 NH residents, we considered the self-rated muscle weakness as a proxy marker of low muscle strength (probable sarcopenia). We found that obesity was marginally related to lower mortality risk when the presence of concomitant self-reported muscle weakness was not considered. However, when both obesity and weakness stratified body phenotypes, it became apparent that obesity was associated with lower mortality risk when not accompanied by concomitant weakness (i.e. obesity alone phenotype). Obesity with concomitant weakness (i.e. weak+obese) was associated with mortality risk similar to non-weak non-obese or weak alone phenotypes and higher than the obesity alone phenotype. This latter finding is new and has not been analysed and reported in the literature yet.

The relationship between obesity and mortality in older individuals is not straightforward [17]. Among studies with a general focus on community-dwelling older adults, some studies reported higher mortality with obesity [3,32], some reported a mitigated effect [33] or no relationship [34], and some others reported a negative association, thus, the protective effect of obesity on mortality [3,17,19,35]. Although there are some recent reports on NH populations, there is still limited available information [18,36]. Most of them reported a protective role of obesity in this more frail population [18,37–40], while some others noted no effect [17]. As far as we know, there is no study investigating the associations of mortality with obesity alone, weakness+obesity, weakness alone, and normal phenotype in NH residents comparatively. In a study involving 5,899 NH residents, Grabowski et al. reported that mortality was not different between the obese (BMI> 28 kg/m^2) and normal weight residents (BMI: 19–28 kg/m^2) [17]. In that study, the presence of concomitant weakness/sarcopenia (i.e. as weakness/sarcopenia+obesity or obesity alone) was not specifically examined [17]. It is possible that if the presence of weakness/sarcopenia were considered in those with obesity, the association of 'obesity alone' with lower mortality risk might have been detected. Because our study showed that obesity was not associated with a protective effect on survival when concomitant weakness was present but associated with a protective effect when it was alone (i.e. weakness was not present), we speculate that it is possible if the obese participants were stratified according to the presence and absence of concomitant weakness in the study of Grabowski et al., the association of 'obesity alone' with lower mortality risk might have been detected. This is probably due to the revealed association of sarcopenia and its components, i.e. low muscle mass/low muscle strength, with increased mortality in institutionalised older adults [21,41,42]. Thus, when obesity is associated with sarcopenia or its compoınents, its association with lower mortality risk may disappear. However, this speculation should be tested in future studies. In a study involving community-dwelling older adults, when compared with the non-sarcopenic non-obese phenotype, the 'obesity alone' phenotype was associated with lower mortality risk in the oldest-old male adults (HR = 0.6, 95% CI = 0.3–0.9) but with similar mortality risk in those aged 75 [43]. Together with our study, these results suggest that the 'obesity only' phenotype, compared to the non-weak/sarcopenic non-obese phenotype, may relate to the lower mortality risk in the individuals

with limited capacity (e.g. oldest-old or NH residents). However, weakness/sarcopenia+obesity have a similar mortality risk. Also, we found that among participants with obesity, about 1/4 had a concomitant weakness, revealing a considerable prevalence of weakness+obesity among obese NH residents. Hence, these findings suggest that, especially in older adults with limited capacity, including the NH residents, obesity should be evaluated by assessing weakness simultaneously.

We found that weakness+obesity was not different from non-weak non-obese participants or weak alone participants concerning mortality. There is not any study on NH residents in this regard. However, a few studies have reported a community-dwelling older population with conflicting results [44]. In some studies, weakness/sarcopenia+obesity was related to higher mortality risk [45–47], while not in others [43,48,49]. In accordance with our study, Hamer et al. reported that when defined by high BMI>30 kg/m^2 + objectively evaluated low muscle strength, weakness+obesity did not confer any greater risk than weakness alone [48] in those with a mean age of 66. Similarly, Berens et al. reported that sarcopenic obesity was not different from the non-sarcopenic non-obese phenotype for mortality risk in those aged 75 or 87 when obesity was evaluated with the body fat percentage method and sarcopenia by EWGSOP2 definition [43].

Several factors were suggested to associate obesity with a survival advantage in individuals with limited capacity. Body fat is an energy buffer that might extend life expectancy in individuals with chronic or long-term diseases [3,50], which would be advantageous in case of life-threatening illnesses. Another explanation is that obesity may merely reflect an absence of chronic disease [43]. Obese individuals who survive old age may have characteristics that protect them from the adverse effects of being obese. This is known as the 'survival effect'. Individuals who are susceptible to the complications of obesity may have already died, leaving behind those who are more resistant [16]. Hence, individuals with obesity would have fewer comorbid diseases. However, in this study, chronic disease burden was considered a potential confounder. Also, it is suggested that the association of obesity with lower mortality risk may be due to the presence of lower rates of malnutrition in obese subjects [51,52]. However, nutritional status was also considered a potential confounder in this study, and obesity alone revealed a survival advantage independent of its association with normal nutritional status. Another suggested explanation is the 'time discrepancy of competing risk factors' theory. Namely, the higher the age, the shorter the remaining life span.

Since obesity-related adverse consequences take years to develop, those who are obese may die due to other conditions before the emergence of obesity-related adverse effects. Meanwhile, in these individuals, obesity would have possible beneficial effects such as protection from bone mineral density loss and osteoporotic fractures, which may be a cause of mortality in a shorter period. Another thing is that when the individuals become fatter, they also gain muscle. The muscle tissue, which acts as a reservoir in the stressed state, maybe one of the responsible factors that obesity exerts its protective effect. This latter suggestion can explain why obesity was protective when it was not associated with a marker of muscle loss (i.e. weakness).

In this study, we considered self-reported grip strength as a proxy marker of muscle strength, taking some studies into account in which the authors used self-rated grip strength as a substitute or a marker of objective grip strength measurement [9–12]. Namely, in the study conducted by Simard et al., they aimed to identify questions (including two questions regarding self-rated handgrip strength) that could estimate handgrip strength measured by a Martin vigorimeter. Of note, the Martin vigorimeter has a very high correlation with Jamar hydraulic hand dynamometer [9]. Here, self-rated grip strength was found most associated with objective handgrip strength in men and the answer to the question about the difficulty of opening a jar, which is an indirect assessment for self-rated grip strength, was found most associated with handgrip strength in women [9]. In another study by our group, we aimed to investigate whether a simpler modified Fried frailty scale could estimate mortality among NH residents in this same study cohort. We modified the Fried Frailty scale across its most time consuming three items including assessing low handgrip strength (weakness). We asked the participants or their caregivers (when the resident was cognitively impaired/ not communicable) to subjectively assess the resident's handgrip strength compared to the same-aged healthy individuals (self-rating), instead of objective measurements. We found that this new modified Fried frailty scale was an independent predictor of mortality in NH residents [10]. We suggest that these results provide some data that self-reported grip strength can be used as a proxy marker of muscle strength. On the other hand, there have been some doubts about the self-rated health method, such as it may be influenced by sociocultural background and

education level [53]. This consideration is also valid for using self-reported muscle weakness as a proxy marker of muscle weakness and should be considered a potential limitation of this method. Patient-rated subjective grip strength evaluation should also be evaluated in light of these findings.

This study has its limitations and strengths. We included residents who could stand still for a while to assess weight and body composition by the bioimpedance analyser; therefore, this study did not include more frail NH residents. The study findings cannot be generalised to all NH populations. On the other hand, included subgroup is expected to have a better survival expectation than the excluded subgroup so that the associations of mortality with the study parameters would be more helpful for mortality stratification. We considered only the baseline evaluation of the participants. We have no data on the body composition changes that might have occurred in the follow-up. 45.8% of the participants were considered cognitively impaired (possibly demented) and assessed by the nursing staff. Using the caregiver's assessment instead of the self-reported evaluation in these participants could be considered another limitation. However, when we included possible dementia (cognitive impairment), which corresponds to caregiver-rated grip strength in multivariate survival analysis (Model 2), the results did not change, indicating that this subgroup did not change the results of this analysis.Some potential confounders, such as laboratory parameters or the presence of specific chronic diseases, were not evaluated except dementia. We followed the participants for enough period to meet a significant mortality incidence. However, the event rate was low in some subgroups because the study participants were grouped according to four body phenotype groups. Nevertheless, the analysis revealed significant association despite the limited events prone to Type-2 error in statistical analysis. Another factor is that we evaluated weakness with a subjective method which is prone to error. We assessed self-rated muscle weakness as subjectively evaluated low muscle strength to account as a proxy marker for low muscle strength (probable sarcopenia). However, this may, in a way, be considered a potential strength of the study. It is because objective evaluation requires, at minimum, a hand dynamometer, trained staff, dedicated time, and preserved cognition which can preclude assessment of weakness. On the other hand, in a review of twenty-seven community studies, self-rated health was found as an independent predictor of mortality [6,7], and analogously subjective evaluation of grip strength has been suggested as a proxy marker to evaluate low muscle strength and showed success [6–9]. This study considered the old saying that 'patients know the best' [54]. This simple subjective approach can allow us to estimate weakness/low muscle strength in a few minutes practically in centres where there is no chance to measure handgrip strength objectively, including most of the NHs. It also can help clinicians predict patient outcomes and muscle weakness and obesity-related adverse outcomes. Another strength is, as we evaluated self-reported muscle weakness by caregiver opinion in demented participants, this approach has the potential to evaluate weakness/muscle strength in those with impaired cognition as well. Our analyser for body composition was a bioimpedance analyser (Tanita BC 532), which is very simple to use with acceptable cost and has the advantage of validated use in research. Moreover, there were no missing data at baseline. We followed the participants for enough period to meet a significant mortality incidence, and we considered a wide range of well-documented predictors of mortality as confounders. Evaluating obesity with the fat percentage method rather than the World Health Organization (WHO) definition is another strength, as the fat percentage method is suggested as a better way to identify obesity in older adults [4]. Also, we used the population-specific cutoffs (>27% in males and >41% in females). When available, it is recommended to use population-specific cutoffs on body compositions [55].

5. Conclusion

In conclusion, to our knowledge, this is the first study examining the associations of obesity alone, self-reported muscle weakness alone, and their combination with mortality in NH residents. It is also the first study investigating the associations of mortality with obesity alone, weakness+obesity, weakness alone, and normal phenotype in NH residents. This study signifies the importance of assessing body phenotypes in NH residents considering obesity and weakness concomitantly. 'Obesity alone' (without concomitant weakness) phenotype came front with its relation to lower mortality rate when adjusted by potential confounders, but obesity accompanied by weakness (obesity+weakness) did not show a survival advantage. Future studies with higher NH participants must shed more light on this research area. Another thing is that our study suggests a simple consideration of weakness and weakness+obesity that can easily be integrated into everyday practice. This approach can enhance routine consideration of body phenotypes, albeit including some subjective evaluation components.

List of Abbreviations

ADL: Activities of daily living
BIA: Bioimpedance analysis

CI: Confidence interval
EWGSOP: The European Working Group on Sarcopenia in Older People
FFM: Fat-free mass
HR: Hazard ratio
IADL: Instrumental activities of daily living
MMSE: Mini-Mental State Examination
MNA-SF: Mini nutritional assessment short-form
NH: Nursing home
S: Sarcopenia
SMM: Skeletal muscle mass
SO: Sarcopenic obesity
SPSS: Statistical package for social sciences
WHO: World Health Organization

Acknowledgments

The authors certify that they comply with ethical guidelines for authorship and publishing of the **Acta Clinica Belgica**.

Availability of data and material

The data is available from the authors upon reasonable request.

Consent for publication

We received informed consent from all participants.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethics approval

We obtained ethical approval from the Istanbul University Istanbul Medical School's ethical
Board.

References

[1] Santanasto AJ, Goodpaster BH, Kritchevsky SB, et al. Body composition remodeling and mortality: the health aging and body composition study. J Gerontol A Biol Sci Med Sci. 2017;72(4):513–519.
[2] Lee DH, Giovannucci EL. Body composition and mortality in the general population: a review of epidemiologic studies. Exp biol med. 2018;243(17–18):1275–1285.
[3] Toss F, Wiklund P, Nordstrom P, et al. Body composition and mortality risk in later life. Age Ageing. 2012;41(5):677–681.
[4] Zoico E, Di Francesco V, Guralnik JM, et al. Physical disability and muscular strength in relation to obesity and different body composition indexes in a sample of healthy elderly women. Int J Obes Relat Metab Disord. 2004;28(2):234–241.
[5] Cruz-Jentoft AJ, Bahat G, Bauer J, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing. 2019;48(1):16–31.
[6] Idler EL, Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. J Health Soc Behav. 1997;38(1):21–37.
[7] Fayers PM, Sprangers MA. Understanding self-rated health. Lancet. 2002;359(9302):187–188.
[8] Phung TKT, Siersma V, Vogel A, et al. Self-rated versus caregiver-rated health for patients with mild dementia as predictors of patient mortality. Am J Geriatr Psychiatry. 2018;26(3):375–385.
[9] Simard J, Chalifoux M, Fortin V, et al. Could question on activities of daily living estimate grip strength of older adults living independently in the community? J Aging Res. 2012;2012:427109.
[10] Bahat G, Ilhan B, Tufan A, et al. Success of simpler modified fried frailty scale to predict mortality among nursing home residents. J Nutr Health Aging. 2021;25(5):606–610.
[11] het Veld Lp O, van Rossum E, Kempen GI, et al. Fried phenotype of frailty: a cross-sectional comparison of three frailty stages on various health domains. BMC Geriatr. 2015;15:77.
[12] Het Veld LPM O, de Vet HCW, van Rossum E, et al. Substitution of fried's performance-based physical frailty criteria with self-report questions. Arch Gerontol Geriatr. 2018;75:91–95.
[13] Jura M, Kozak LP. Obesity and related consequences to ageing. Age (Dordr). 2016;38(1):23.
[14] Kalantar-Zadeh K, Streja E, Molnar MZ, et al. Mortality prediction by surrogates of body composition: an examination of the obesity paradox in hemodialysis patients using composite ranking score analysis. Am J Epidemiol. 2012;175(8):793–803.
[15] Hainer V, Aldhoon-Hainerová I. Obesity paradox does exist. Diabetes Care. 2013;36(Suppl 2):S276–281.
[16] Oreopoulos A, Kalantar-Zadeh K, Sharma AM, et al. The obesity paradox in the elderly: potential mechanisms and clinical implications. Clin Geriatr Med. 2009;25(4):643–659, viii.
[17] Grabowski DC, Campbell CM, Ellis JE. Obesity and mortality in elderly nursing home residents. J Gerontol A Biol Sci Med Sci. 2005;60(9):1184–1189.
[18] Veronese N, Cereda E, Solmi M, et al. Inverse relationship between body mass index and mortality in older nursing home residents: a meta-analysis of 19,538 elderly subjects. Obes Rev. 2015;16(11):1001–1015.
[19] Grabowski DC, Ellis JE. High body mass index does not predict mortality in older people: analysis of the longitudinal study of aging. J Am Geriatr Soc. 2001;49(7):968–979.
[20] Halil M, Ulger Z, Varlı M, et al. Sarcopenia assessment project in the nursing homes in Turkey. Eur J Clin Nutr. 2014;68(6):690–694.
[21] Landi F, Liperoti R, Fusco D, et al. Sarcopenia and mortality among older nursing home residents. J Am Med Dir Assoc. 2012;13(2):121–126.
[22] Arango-Lopera VE, Arroyo P, Gutiérrez-Robledo LM, et al. Mortality as an adverse outcome of sarcopenia. J Nutr Health Aging. 2013;17(3):259–262.

[23] Sobestiansky S, Michaelsson K, Cederholm T. Sarcopenia prevalence and associations with mortality and hospitalisation by various sarcopenia definitions in 85–89-year-old community-dwelling men: a report from the ULSAM study. BMC Geriatr. 2019;19(1):318.
[24] Wang H, Hai S, Liu Y, et al. Skeletal muscle mass as a mortality predictor among nonagenarians and centenarians: a prospective cohort study. Sci Rep. 2019;9 (1):2420.
[25] Bae EJ, Park NJ, Sohn HS, et al. Handgrip strength and all-cause mortality in middle-aged and older Koreans. Int J Environ Res Public Health. 2019;16 (5):740.
[26] Zamboni M, Rubele S, Rossi AP. Sarcopenia and obesity. Curr Opin Clin Nutr Metab Care. 2019;22 (1):13–19.
[27] Wang JG, Zhang Y, Chen HE, et al. Comparison of two bioelectrical impedance analysis devices with dual-energy X-ray absorptiometry and magnetic resonance imaging in the estimation of body composition. J Strength Cond Res. 2013;27(1):236–243.
[28] Bahat G, Kilic C, Topcu Y, et al. Fat percentage cutoff values to define obesity and prevalence of sarcopenic obesity in community-dwelling older adults in Turkey. Ageing Male. 2018 23 (5):477–482.
[29] Cho CY, Alessi CA, Cho M, et al. The association between chronic illness and functional change among participants in a comprehensive geriatric assessment program. J Am Geriatr Soc. 1998;46 (6):677–682.
[30] Isautier JMJ, Bosnić M, Yeung SSY, et al. Validity of nutritional screening tools for community-dwelling older adults: a systematic review and meta-analysis. J Am Med Dir Assoc. 2019;20(10):1351.e1313–1351. e1325.
[31] Tsoi KK, Chan JY, Hirai HW, et al. Cognitive tests to detect dementia: a systematic review and meta-analysis. JAMA Intern Med. 2015;175(9):1450 –1458.
[32] Calle EE, Thun MJ, Petrelli JM, et al. Body-mass index and mortality in a prospective cohort of U.S. adults. N Engl J Med. 1999;341(15):1097–1105.
[33] Bender R, Jöckel KH, Trautner C, et al. Effect of age on excess mortality in obesity. Jama. 1999;281 (16):1498–1504.
[34] Takala JK, Mattila KJ, Ryynänen OP. Overweight, underweight and mortality among the aged. Scand J Prim Health Care. 1994;12(4):244–248.
[35] DeVore PA. Assessment of nutritional status and obesity in elderly patients as seen in general medical practice. South Med J. 1993;86(9):1008–1010.
[36] Zanandrea V, Barreto de Souto P, Cesari M, et al. Obesity and the nursing home: a review and an update. Clin Nutr. 2013;32(5):679–685.
[37] Vetrano DL, Collamati A, Magnavita N, et al. Health determinants and survival in nursing home residents in Europe: results from the SHELTER study. Maturitas. 2018;107:19–25.
[38] de Souto Barreto P, Cadroy Y, Kelaiditi E, et al. The prognostic value of the body mass index on mortality in older adults with dementia living in nursing homes. Clin Nutr. 2017;36(2):423–428.
[39] Lee JS, Auyeung TW, Chau PP, et al. Obesity can benefit survival-a 9-year prospective study in 1614 Chinese nursing home residents. J Am Med Dir Assoc. 2014;15 (5):342–348.
[40] Wirth R, Streicher M, Smoliner C, et al. The impact of weight loss and low BMI on mortality of nursing home residents - Results from the nutritionDay in nursing homes. Clin Nutr. 2016;35(4):900–906.
[41] Perkisas S, De Cock AM, Vandewoude M, et al. Prevalence of sarcopenia and 9-year mortality in nursing home residents. Aging Clin Exp Res. 2019;31 (7):951–959.
[42] Yalcin A, Aras S, Atmis V, et al. Sarcopenia and mortality in older people living in a nursing home in Turkey. Geriatr Gerontol Int. 2017;17(7):1118–1124.
[43] von Berens A, Obling SR, Nydahl M, et al. Sarcopenic obesity and associations with mortality in older women and men - a prospective observational study. BMC Geriatr. 2020;20(1):199.
[44] Batsis JA, Villareal DT. Sarcopenic obesity in older adults: aetiology, epidemiology and treatment strategies. Nat Rev Endocrinol. 2018;14(9):513–537.
[45] Tian S, Xu Y. Association of sarcopenic obesity with the risk of all-cause mortality: a meta-analysis of prospective cohort studies. Geriatr Gerontol Int. 2016;16 (2):155–166.
[46] Sanada K, Chen R, Willcox B, et al. Association of sarcopenic obesity predicted by anthropometric measurements and 24-y all-cause mortality in elderly men: the kuakini honolulu heart program. Nutr. 2018;46:97–102.
[47] Batsis JA, Mackenzie TA, Emeny RT, et al. Low lean mass with and without obesity, and mortality: results from the 1999-2004 national health and nutrition examination survey. J Gerontol A Biol Sci Med Sci. 2017;72(10):1445–1451.
[48] Hamer M, O'Donovan G. Sarcopenic obesity, weight loss, and mortality: the english longitudinal study of ageing. Am J Clin Nutr. 2017;106(1):125–129.
[49] Liu L-KCL-Y, Yeh K-P, Lin M-H, et al. Sarcopenia, but not sarcopenic obesity, predicts mortality for older old men: a 3-year prospective cohort study. J Clin Gerontol Geriatrics. 2014;5(2):42–46.
[50] Romero-Corral A, Montori VM, Somers VK, et al. Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. Lancet. 2006;368 (9536):666–678.
[51] Naseer M, Forssell H, Fagerström C. Malnutrition, functional ability and mortality among older people aged > 60 years: a 7-year longitudinal study. Eur J Clin Nutr. 2016;70(3):399–404.
[52] Lee SH, Kim DH, Park JH, et al. Association between body mass index and mortality in the Korean elderly: a nationwide cohort study. PLoS One. 2018;13(11): e0207508.
[53] Fernandes SGG, Pirkle CM, Sentell T, et al. Association between self-rated health and physical performance in middle-aged and older women from Northeast Brazil. PeerJ. 2020 Apr 10;8:e8876.
[54] Danenberg H. The patient knows best. Isr Med Assoc J. 2015;17(4):245–246.
[55] Bahat G, Cruz-Jentoft A. Putting sarcopenia at the forefront of clinical practice. Eur J Geriatric Gerontol. 2019;1:43–45.