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Incident Impaired Cognitive Function in Sarcopenic Obesity: Data From the National Health and Aging Trends Survey

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Abstract

Objectives: The prevalence of obesity with sarcopenia is increasing in adults aged ≥65 years. This geriatric syndrome places individuals at risk for synergistic complications that leads to long-term functional decline. We ascertained the relationship between sarcopenic obesity and incident long-term impaired global cognitive function in a representative US population.

Design: A longitudinal, secondary data set analysis using the National Health and Aging Trends Survey.

Setting: Community-based older adults in the United States.

Participants: Participants without baseline impaired cognitive function aged ≥65 years with grip strength and body mass index measures.

Methods: Sarcopenia was defined using the Foundation for the National Institutes of Health Sarcopenia Project grip strength cut points (men <35.5 kg; women <20 kg), and obesity was defined using standard body mass index (BMI) categories. Impaired global cognition was identified as impairment in the Alzheimer's Disease-8 score or immediate/delayed recall, orientation, clock-draw test, date/person recall. Proportional hazard models ascertained the risk of impaired cognitive function over 8 years (referent = neither obesity or sarcopenia).

Results: Of the 5822 participants (55.7% women), median age category was 75 to 80, and mean grip strength and BMI were 26.4 kg and 27.5 kg/m^2, respectively. Baseline prevalence of

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sarcopenic obesity was 12.9%, with an observed subset of 21.2% participants having impaired cognitive function at follow-up. Compared with those without sarcopenia or obesity, the risk of impaired cognitive function was no different in obesity alone [hazard ratio (HR) 0.98; 95% confidence interval (CI) 0.82–1.16]), but was significantly higher in sarcopenia (HR 1.60; 95% CI 1.42–1.80) and sarcopenic obesity (HR 1.20; 95% CI 1.03–1.40). There was no significant interaction term between sarcopenia and obesity.

Conclusions: Both sarcopenia and sarcopenic obesity are associated with an increased long-term risk of impaired cognitive function in older adults.

Keywords

Sarcopenia; obesity; cognition

The population of older adults with obesity in the United States continues to grow, with recent estimates exceeding 40%.[1] The impact of obesity in this population is compounded by sarcopenia, age-related changes in muscle mass and strength, that naturally occurs with aging.[2] The confluence of these 2 epidemics, sarcopenic obesity, has been proposed to be a harbinger of incident disability,[3] reduced quality of life,[4] and mortality.[5] The prevalence of this entity varies 18-fold, in part due to the inability to solidify an accurate definition in both clinical and research settings.[6]

The pathophysiology of sarcopenic obesity remains unclear, but it is likely due to a multitude of underlying changes in body composition, sex-specific hormonal and inflammatory changes, and myocellular mechanisms that stimulate infiltration of fat into muscle.[7,8] Common mechanisms believed to accelerate cognitive changes that lead to dementia also include insulin resistance, oxidative damage,[9,10] central obesity, and cardiometabolic factors.[11–15] There has been mixed epidemiological evidence to suggest that obesity may be protective in the development of cognitive impairment[16] but the interaction between muscle and bone has not been controlled for in analyses, prompting prevention efforts that focus predominantly on aerobic physical activity rather than dietary interventions.[17]

The purpose of this study was to investigate whether sarcopenia or sarcopenic obesity, defined using the consensus definitions of the Foundation of the National Institutes of Health (FNIH),[18] to a sample of cognitively intact persons, are associated with greater long-term risk of impaired cognitive function over the course of the 8-year study. Although measures of sarcopenia have been shown to have differing relationships with dementia,[19–21] it is unclear whether the interplay with adiposity impacts this relationship. This approach permits for the determination of whether a higher risk of impaired cognitive function is driven more heavily by weight status or sarcopenic status. Gaining an understanding of these 3 separate classifications (sarcopenia, obesity, sarcopenic obesity) is important in the future design of health promotion interventions to mitigate the development of long-term impaired cognitive function. In addition, as it is unknown how different definitions of sarcopenic obesity impact incident impaired cognitive function, we sought to conduct a sensitivity analysis to ascertain this effect.

Methods

Study Design and Participants

The National Health and Aging Trends Study (NHATS) is a nationally representative cohort of Medicare beneficiaries age 65 and older in the United States. Its aim is to provide information and an understanding of trends in late-life functioning. This cohort study over-samples ethnic and diverse populations gathering self-report interview data in addition to objective, standardized physical function data. Trained staff assessed cognitive and physical function measures in participants' homes. For this analysis, we evaluated baseline participants in Round 1 with annual follow-up through Round 8 (n = 8245). Participants were excluded if they lacked data for grip strength (n = 1177), lacked information on height or weight preventing the calculation of body mass index (BMI) (n = 147), or had baseline impaired cognitive function (n = 1099) as described later in this article. This yielded a final sample of 5822 participants who were included in the initial analysis. The Committee for the Protection of Human Subjects at Dartmouth College exempted this study from review, as the data were downloaded and analyzed in a de-identified manner.

Outcome Variable: Impaired Cognitive Function

Cognitive function was assessed in the NHATS sample as previously described (https://www.nhats.org/scripts/TechnicalDementiaClass.htm). For this study, we created a composite outcome variable of impaired cognitive function (yes/no) based on the Alzheimer's Disease 8 (AD-8) score and 3 domains: memory, orientation, and executive function. The AD-8 is a brief interview designed to help discriminate between signs of normal aging and mild dementia. It consists of 8 items that assess memory, temporal orientation, and judgment.[22,23] This instrument in NHATS was used unless the informant was not a relative and had known the person for less than 1 year. A score of ≥2 classified a participant as having impaired cognitive function. AD-8 items were not asked if AD-8 was coded in a prior round. Each individual domain was assigned a binary score (impaired/not impaired). As participants did not have full cognitive data within and across each round, if an AD-8 score was not present, individual domain scores were summed, defined as an impairment in any domain that characterized the presence/absence of impaired cognitive function. Verbal learning and memory were assessed with a 10-word recall task using a computer-assisted personal interviewing system. The participant read a list of 10 words and was asked to recall as many of the words as possible immediately, and then again after a 5-minute delay. Orientation was evaluated by asking participants the date, month, year, and day of the week and naming the first and last name of the president/vice-president. Executive function was assessed using the clock-drawing test,[24] which measures planning, numerical knowledge, concept of time, gross motor functioning, concentration, and comprehension. Participants were given a blank sheet of paper and a pen and given 2 minutes to draw a clock face, place the numbers, and place the hands at 11:10.

Primary Predictor Variables

Obesity was defined in 2 ways: using a cutoff of BMI 30 kg/m^2[25] or a waist circumference of ≥88 cm for women or ≥102 cm for men.[26] Current self-reported height and weight were assessed using a questionnaire. Waist circumference was measured using a flexible tape

measure on the participant's abdomen aligned with his or her navel in a snug, but not tight manner, holding their breath at the end of exhalation, recording to the nearest one-quarter inch.

Grip strength was tested using a digital hand dynamometer placed on a stable horizontal surface with the participant in the sitting position. After the interviewer demonstrated the activity and proper arm position, the participant squeezed as hard as possible and the value was recorded to the nearest tenth of a kilogram. The maximum of 2 trials conducted with the same dominant hand was used. Two separate cutpoints using the criteria for sarcopenia were used: (1) grip strength <35.5 kg in men and <20 kg in women; grip strength divided by BMI <1.05 in men and <0.79 in women.[27] Although there is no consensus for categorizing sarcopenic obesity,[7] it was defined in 3 manners: low grip strength with BMI-defined obesity; low grip strength with waist circumference–defined obesity; low grip strength to BMI ratio.

Covariates

Age was considered a restricted variable; hence, age categories were presented and used in the analysis. Race information was collected in Round 1 only (White, Black, Other, Hispanic). Smoking status was defined as current, past, or never smoked using the question "ever smoked cigarettes regularly at least 1 cigarette a day." Education status was classified by schooling type ranging from no schooling to a master's professional or doctoral level. Categories were classified as less than high school, high school to some college, college, and more than college. A physical activity proxy was defined using the answer to the question "in the last month, did you (respondent) ever go walking for exercise?" Information regarding health conditions was asked using a self-reported questionnaire to participants as to whether a doctor had ever told them if they had any of several conditions. Previous research demonstrates good concordance between self-reported and medical record extracted health conditions,[28,29] variables that were based on a validated algorithm from the Medicare Chronic Condition Data Warehouse.[30,31]

Statistical Analysis

All data were aggregated according to NHATS guidelines (http://www.nhats.org). Demographic and baseline characteristics were evaluated using descriptive statistics. An unpaired *t*-test and χ^2 test, or their nonparametric equivalents, assessed differences between categories (sarcopenia, obesity, sarcopenic obesity, neither). Participants with impaired cognitive function classified as described previously were censored at baseline. Our primary composite outcome was the development of incident impaired cognitive function (defined previously). Secondary outcomes consisted of each specific subcategory of impaired cognitive function (AD-8 score, memory, orientation, executive function). Primary predictor was cohort category of sarcopenia, sarcopenic obesity, obesity only, and neither obesity nor sarcopenia (referent group). Proportional hazard models evaluated time-to-event with the outcome of impaired cognitive function: in these models, impaired cognitive function was binarized according to NHATS-established guidelines.[32,33] Unadjusted and adjusted models were evaluated and fulfilled the proportional hazard assumption using the Schoenfeld residual test. Model 2 adjusted for age group, sex, race, smoking status, education status,

and physical activity measure. Model 3 additionally adjusted for comorbid conditions (heart disease, hypertension, arthritis, diabetes, lung disease, stroke, cancer). Study data were analyzed using R version 3.5.2 (www.R-project.org). Data wrangling was conducted using *tidyverse* (v. 1.2.1) and linear mixed effects modeling with package *lme4* (v. 1.1.20) and *ImerTest* (v. 3.1.0). A *P* value of <.05 was considered statistically significant.

Results

Baseline characteristics are presented in Table 1. Of the 5822 participants at baseline, 750 (12.9%) were classified as having sarcopenic obesity and 2292 (39.4%) with sarcopenia, 841 (14.4%) with obesity alone, and 1939 (33.3%) with neither condition. Participants were predominantly female (55.7%) and white (71.5%). Grip strength was lower in the sarcopenia alone (21.5 ± 7.6 kg) or sarcopenic obesity (20.7 ± 7.4) groups than in the other 2 groups. Supplementary Table 1 outlines the differences in those included versus excluded in the study. Table 2 outlines the proportion fulfilling criteria for impaired cognitive function in each year group, and by cognitive domain/test. Rates at 8 years were highest in the sarcopenia and sarcopenic obesity groups (28.3 vs. 22.6%). Supplementary Table 2 lists the number of missing counts in each of the measures across years, categories, and cognitive measures.

Table 3 outlines the unadjusted and adjusted Cox-proportional hazard models for incident impaired cognitive function for the 3 different definitions of sarcopenic obesity and Figure 1a–c represent the proportional hazard modeling. Adjusting for comorbidities did not markedly alter the hazard ratios. By using either BMI or waist circumference as a measure for obesity, both sarcopenia and sarcopenic obesity were significantly associated with greater incident impaired cognitive function. Estimates were higher using waist circumference than BMI for obesity. These values persisted for the subdomains of executive function, memory, and orientation (Supplementary Figures 1 and 2). Obesity alone did not increase the risk of incident impaired cognitive function. In the interaction model, although sarcopenia was significant in the overall model, the term between sarcopenia and obesity was nonsignificant. Using the FNIH definition[18] (grip strength divided by BMI), we observed a significant impairment among the primary and secondary measures of cognition. Using a linear mixed modeling and clustering longitudinal observations by patients, we observed only sarcopenia having significance using BMI for the obesity variable (Table 4). Sarcopenia and sarcopenic obesity were observed to be significantly associated with reduced scores in immediate and delayed recall, orientation, and executive function using the waist circumference definition or FNIH definitions (Supplementary Tables 3 and 4).

Discussion

To our knowledge, this is the first study that evaluates the incidence of impaired cognitive function using recently validated grip strength definitions of sarcopenia and sarcopenic obesity in a sample of older adults in the United States. Both sarcopenia and sarcopenic obesity lead to a future risk of impaired cognitive function that is unlikely due to the interaction between fat and muscle strength. Previous studies have focused on the specific relationship between handgrip strength or muscle mass and cognitive function.[19,34,35]

Although these studies lack the specificity that our analysis provides, our findings suggest that sarcopenia may be the major predictor of impaired long-term cognitive function.

Few studies evaluate the impact of sarcopenia and sarcopenic obesity on cognitive function. Of those, there is reliance on nonstandard different definitions of both obesity and sarcopenia that have demonstrated conflicting and weaker estimates.[19,21,36–40] For example, Jeong et al[41] used the Korean Longitudinal Study of Aging to ascertain the association of low handgrip strength in those with a BMI ≥25 kg/m^2 and dementia. In their study, and in line with our results, those with sarcopenic obesity had larger drops in cognition (as measured by the mini-mental status examination) as compared with those without obesity or normal strength. Our study expands on these previous reports and provides additional evidence that sarcopenia with and without obesity is related to cognitive impairment. These aforementioned studies had numerous limitations, including a reliance of bioelectrical impedance to assess muscle mass, their cross-sectional study design, small sample sizes, and use of 1 measure to assess cognition. Additional longitudinal studies focusing on established definitions of sarcopenia and obesity are needed to confirm the causality with incident cognitive impairment; this prevents the methodological challenges that have plagued this field on long-term physical function and disability.[7]

A priori, we expected that there would be a synergistic effect between sarcopenia and obesity based on the existing underlying molecular and pathophysiological mechanisms.[7] Our findings were confirmed, but not in the direction we had expected; obesity appeared to diminish the estimates having a protective effect on long-term incident impaired cognitive function in individuals with coexistent sarcopenia. Whether this is due to the differences between subcutaneous or visceral adiposity, or due to an impact on muscle strength is unclear. Muscle, fat, and cognition all share common underlying physiological pathways.[10,42] Although purely speculative, obesity may selectively alter a biological target that diminishes this relationship.

We used commonly used definitions of sarcopenia that are increasingly related to incident disability as compared with muscle mass.[43] A major debate[7] and an existing study limitation of this study is how to best classify persons with sarcopenia and obesity. The recent FNIH consortium has challenged whether muscle mass should be used for the definition of sarcopenia, suggesting that muscle mass (assessed using dual-energy x-ray absorptiometry), should not be used in defining sarcopenia. Others argue that sarcopenia should be a distinct entity from dynapenia (low muscle strength).[44] Previous studies have defined sarcopenia obesity using a ratio of appendicular skeletal mass normalized for total body fat, the European Working Group on Sarcopenia in Older Persons[45] defined sarcopenia, and overall skeletal muscle mass.[8,46,47] These all have been related to impaired walking speed and physical limitations, reduced functional impairment post-hospitalization, and a higher degree of medical comorbidity, respectively. There is a new consensus statement conducted by the European Association for the Study of Obesity and the European Society for Clinical Nutrition and Metabolism that aims to obtain consensus on this issue (personal communication, L. Donini, MD, 2020).

Although the sensitivity of BMI is poor in ascertaining adiposity in older adults, it continues to be used as a common population-based measure in clinical settings.[48] Our use of waist circumference in lieu of BMI suggested similar estimates, despite the differential ability to ascertain subcutaneous versus visceral adiposity. We deliberately conducted a sensitivity analysis to ascertain whether incident impaired cognitive function was definition-dependent. Although percent body fat should be evaluated as a component in defining obesity, the inability to perform accurate assessment using computed tomography, magnetic resonance imaging, or dual-energy x-ray absorptiometry in clinical practice that is cost-effective or is covered by third-party payers continues to be problematic. As such, our results were consistent using all 3 definitions. In fact, the estimates of overall cognitive impairment (and its secondary outcomes) using grip strength with waist circumference approximated those of grip strength alone, and were higher than those using grip strength/BMI. The implications of these findings are not trivial. Previous studies have demonstrated poor concordance with different definitions.[49–51] Although researchers search for the "ideal" definition to ascertain risk of an adverse outcome, for long-term impaired cognitive impairment, it may be inconsequential, irrespective of the manner in which one identifies sarcopenia or sarcopenic obesity, such participants are at higher risk.

Our large sample size with specific cognitive metrics enhances the validity of our results. NHATS has previously validated the analysis by grouping cognitive variables that permit the creation of composite and domain-specific measures.[32,33] This approach permits evaluation not only of a composite outcome, but also 3 distinct cognitive domains (executive function, orientation, and memory). Although exploratory, this suggests a robust effect where the decline in cognitive impairment may be global/general rather than domain-specific. Future research may want to evaluate domain-specific impairments in relation to muscle strength measures, particularly in epidemiological work evaluating associations between cognition and obesity.

Notably, our estimates were unaffected by inclusion of salient sociodemographic variables and medical comorbidities. Length of chronic illnesses may be a factor; however, we lack information on duration of health conditions before study onset. Other important covariates that may impact our estimates include the lack of objectively measured physical activity, lack of nutritional measures, and preexisting life-course factors that may influence different trajectories. NHATS does not objective measure height or weight as other epidemiological studies (eg, Health and Retirement Survey). Although this is a limitation of this current analysis, a comparison study noted that the BMI of NHATS and Health and Retirement Survey demonstrated similar BMIs.[52] We recognize that measures of physical activity were limited to the question on walking; ideally, a life-course history of aerobic or resistance exercises would be integrated into our modeling or in a sample of objectively measured physical activity. The exclusion of participants without key data (eg, grip strength, BMI) was low (15.9%). However, the excluded cohort was older, less educated and active, weaker (based on grip strength), and had fewer comorbidities, suggesting that these participants may not affect our estimates.

Conclusions and Implications

This analysis provides empirical evidence for the need to identify participants with both sarcopenia and sarcopenic obesity in clinical practice. Multicomponent interventional studies targeting elements of both sarcopenia and obesity are critically needed to determine and alter the trajectories of developing dementia in these groups. The implications are significant, as reducing the risks of functional decline may reduce institutionalization. Importantly, future research should evaluate the effect of cognitive decline and resource use in this population, as these may drive up health care costs. Although treatments for dementia are mainly supportive and nonpharmacological, our findings suggest the importance of targeting this population to implement such interventions early on in the disease trajectory. In addition, evaluating sarcopenia from a life-course standpoint may be even more important than its identification/evaluation, to mitigate its development through physical activity and dietary interventions.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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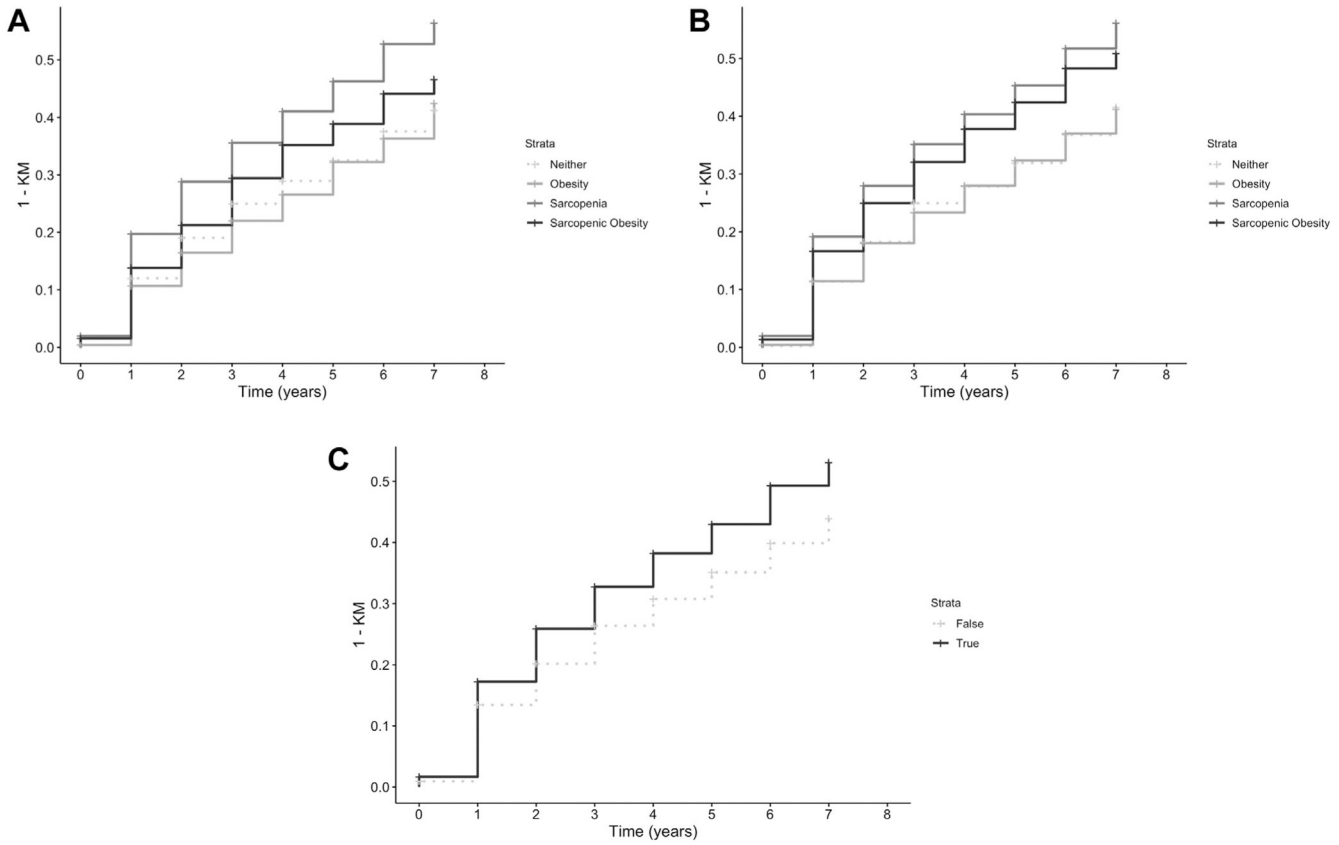

Fig. 1.

(A–C) Incident overall cognitive impairment over an 8-year period. Kaplan-Meier curves represent the incidence of overall impaired cognitive function in the cohort of individuals with sarcopenia, sarcopenic obesity, obesity, and neither obesity nor sarcopenia. Sarcopenia was defined as a grip strength <35.5 kg in men or <20 kg in women; obesity was defined as BMI ≥ 30 kg/m^2; sarcopenic obesity was defined as participants fulfilling definitions of both sarcopenia and obesity; neither was defined as participants fulfilling neither sarcopenia or obesity. (A) Definition using BMI, (B) definition using waist circumference, and (C) grip strength divided by BMI definition of men <1.05, women <0.79.

Table 1

Baseline Characteristics of Cohort (n = 5822)

Characteristics	Overall	Sarcopenia	Obesity	Sarcopenic Obesity	Neither	*P* value
	n = 5822	n = 2292	n = 841	n = 750	n = 1939	
Age category, y						<.001
65–70	1170 (20.1)	194 (8.5)	297 (35.3)	117 (15.6)	562 (29.0)	
70–75	1314 (22.6)	344 (15.0)	272 (32.3)	180 (24.0)	518 (26.7)	
75–80	1191 (20.5)	438 (19.1)	166 (19.7)	178 (23.7)	409 (21.1)	
80–85	1125 (19.3)	586 (25.6)	82 (9.8)	159 (21.2)	298 (15.4)	
85+	1022 (17.6)	730 (31.8)	24 (2.9)	116 (15.5)	152 (7.8)	
Female sex	3244 (55.7)	1122 (49.0)	517 (61.5)	432 (57.6)	1173 (60.5)	<.001
Race						<.001
White	4165 (71.4)	1698 (74.1)	540 (64.2)	488 (65.1)	1439 (74.2)	
Black	1192 (20.5)	378 (16.5)	266 (31.6)	172 (22.9)	376 (19.4)	
Hispanic	302 (5.2)	139 (6.1)	19 (2.3)	72 (9.6)	72 (3.7)	
Other/Don’t know	163 (2.8)	77 (3.4)	16 (1.9)	18 (2.4)	52 (2.7)	
Smoking status						<.001
Current	476 (8.2)	173 (7.5)	54 (6.4)	33 (4.4)	216 (11.1)	
Former	2527 (43.4)	1002 (43.7)	384 (45.7)	346 (46.1)	795 (41.0)	
Never	2818 (48.4)	1117 (48.7)	402 (47.8)	371 (49.5)	928 (47.9)	
Not reported	1 (0.0)	0 (0.0)	1 (0.1)	0 (0.0)	0 (0.0)	
Education level						<.001
< High school	1416 (24.3)	645 (28.1)	185 (22.0)	232 (30.9)	354 (18.3)	
High school to some college	2810 (48.3)	1048 (45.7)	438 (52.1)	364 (48.5)	960 (49.5)	
College	966 (16.6)	374 (16.3)	140 (16.6)	94 (12.5)	358 (18.5)	
Post college	626 (10.8)	223 (9.7)	78 (9.3)	60 (8.0)	265 (13.7)	
Not reported	4 (0.1)	2 (0.1)	0 (0.0)	0 (0.0)	2 (0.1)	
Physical activity						
Ever walk	3584 (61.6)	1361 (59.4)	488 (58.0)	374 (49.9)	1361 (70.2)	<.001
Objective measures						
Grip strength, kg	26.4 ± 10.5	21.5 ± 7.6	32.6 ± 10.5	20.7 ± 7.4	31.9 ± 10.2	<.001

Characteristics	Overall	Sarcopenia	Obesity	Sarcopenic Obesity	Neither	*P* value
	n = 5822	n = 2292	n = 841	n = 750	n = 1939	
BMI, kg/m^2	27.5 ± 5.5	24.6 ± 3.1	34.9 ± 4.5	34.2 ± 4.2	25.2 ± 2.9	<.001
WC, cm	99.8 ± 16.5	94.9 ± 14.3	113.7 ± 14.9	114.8 ± 14.5	93.9 ± 12.8	<.001
GS:BMI ratio	1.0 ± 0.4	0.9 ± 0.3	1.0 ± 0.3	0.6 ± 0.2	1.3 ± 0.4	<.001
Comorbidities						
Heart disease	1451 (24.9)	664 (29.0)	193 (22.9)	237 (31.6)	357 (18.4)	<.001
Hypertension	3844 (66.0)	1484 (64.7)	651 (77.4)	573 (76.4)	1136 (58.6)	<.001
Arthritis	3000 (51.5)	1216 (53.1)	482 (57.3)	498 (66.4)	804 (41.5)	<.001
Diabetes	1404 (24.1)	499 (21.8)	281 (33.4)	322 (42.9)	302 (15.6)	<.001
Lung disease	835 (14.3)	338 (14.7)	145 (17.2)	134 (17.9)	218 (11.2)	<.001
Stroke	575 (9.9)	304 (13.3)	56 (6.7)	90 (12.0)	125 (6.4)	<.001
Cancer	1503 (25.8)	646 (28.2)	187 (22.2)	189 (25.2)	481 (25.0)	.012

GS, grip strength; WC, waist circumference.

All values represented are counts (%) or mean ± SD. An analysis of variance for all continuous variables, and χ^2 test used for all category *P* values.

Physical activity is defined as an affirmative response to “In the last month, did you ever go walking for exercise?” Sarcopenia was defined as a grip strength <35.5 kg in men or <20 kg in women; obesity was defined as BMI ≥30 kg/m^2; sarcopenic obesity was defined as participants fulfilling definitions of both sarcopenia and obesity; neither was defined as participants fulfilling neither sarcopenia or obesity.

Table 2

Rates of Impaired Cognition by Year of Survey

	Year 2	Year 3	Year 4	Year 5	Year 6	Year 7	Year 8
Overall impaired cognitive function							
Totals	887/4778 (18.6)	722/3905 (18.5)	662/3278 (20.2)	578/2975 (19.4)	530/2665 (19.9)	522/2363 (22.1)	452/2132 (21.2)
Sarcopenia	514/1822 (28.2)	407/1425 (28.6)	354/1160 (30.5)	294/998 (29.5)	247/856 (28.9)	242/734 (33.0)	178/630 (28.3)
Obesity	69/698 (9.9)	54/585 (9.2)	57/510 (11.2)	52/490 (10.6)	63/461 (13.7)	62/420 (14.8)	73/397 (18.4)
Sarcopenic obesity	117/623 (18.8)	95/520 (18.3)	97/432 (22.5)	84/395 (21.3)	79/355 (22.3)	75/302 (24.8)	61/270 (22.6)
Neither	187/1635 (11.4)	166/1375 (12.1)	154/1176 (13.1)	148/1092 (13.6)	141/993 (14.2)	143/907 (15.8)	140/835 (16.8)
Memory domain							
Totals	527/4706 (11.2)	428/3836 (11.2)	345/3188 (10.8)	318/2907 (10.9)	306/2605 (11.7)	274/2308 (11.9)	237/2084 (11.4)
Sarcopenia	320/1769 (18.1)	243/1375 (17.7)	190/1099 (17.3)	162/953 (17.0)	149/822 (18.1)	122/701 (17.4)	89/604 (14.7)
Obesity	37/697 (5.3)	22/583 (3.8)	20/505 (4.0)	24/486 (4.9)	28/457 (6.1)	26/416 (6.2)	33/389 (8.5)
Sarcopenic Obesity	70/615 (11.4)	58/512 (11.3)	56/419 (13.4)	44/383 (11.5)	42/343 (12.2)	42/294 (14.3)	37/265 (14.0)
Neither	100/1625 (6.2)	105/1366 (7.7)	79/1165 (6.8)	88/1085 (8.1)	87/983 (8.9)	84/897 (9.4)	78/826 (9.4)
Orientation							
Totals	309/4706 (6.6)	266/3836 (6.9)	237/3188 (7.4)	224/2915 (7.7)	196/2605 (7.5)	214/2308 (9.3)	190/2084 (9.1)
Sarcopenia	199/1769 (11.2)	161/1375 (11.7)	142/1099 (12.9)	113/960 (11.8)	93/822 (11.3)	101/701 (14.4)	76/604 (12.6)
Obesity	20/697 (2.9)	16/583 (2.7)	15/505 (3.0)	18/486 (3.7)	23/457 (5.0)	26/416 (6.2)	21/389 (5.4)
Sarcopenic obesity	37/615 (6.0)	33/512 (6.4)	27/419 (6.4)	30/384 (7.8)	24/343 (7.0)	26/294 (8.8)	28/265 (10.6)
Neither	53/1625 (3.3)	56/1366 (4.1)	53/1165 (4.5)	63/1085 (5.8)	56/983 (5.7)	61/897 (6.8)	65/826 (7.9)
Executive function (clock test)							
Totals	279/4706 (5.9)	209/3836 (5.4)	193/3188 (6.1)	145/2915 (5.0)	120/2605 (4.6)	116/2308 (5.0)	107/2084 (5.1)
Sarcopenia	151/1769 (8.5)	122/1375 (8.9)	104/1099 (9.5)	79/960 (8.2)	67/822 (8.2)	62/701 (8.8)	45/604 (7.5)
Obesity	26/697 (3.7)	18/583 (3.1)	19/505 (3.8)	13/486 (2.7)	15/457 (3.3)	11/416 (2.6)	16/389 (4.1)
Sarcopenic obesity	40/615 (6.5)	30/512 (5.9)	26/419 (6.2)	26/384 (6.8)	15/343 (4.4)	19/294 (6.5)	14/265 (5.3)
Neither	62/1625 (3.8)	39/1366 (2.9)	44/1165 (3.8)	27/1085 (2.5)	23/983 (2.3)	24/897 (2.7)	32/826 (3.9)

All values represented are counts (%).

Sarcopenia was defined as a grip strength <35.5 kg in men or < 20 kg in women; obesity was defined as BMI ≥30 kg/m^2; sarcopenic obesity was defined as participants fulfilling definitions of both sarcopenia and obesity; neither was defined as participants fulfilling neither sarcopenia or obesity. Overall impaired cognitive function consists of an abnormal score on either the AD8 scale or on any of the 3 domains (memory, orientation, executive function). Each individual domain binarized based on NHATS cutoffs (0 = not impaired, 1 = impaired). If AD8 score not present, individual domain scores

summed – impaired cognitive function defined as impairment in any domain (summed score 1, 2, or 3). Executive function consists of clock-drawing test; Memory domain (immediate word recall, delayed word recall). Orientation consists of date recall and president/vice president naming

Table 3

Cox-Proportional Hazard Models of Incident Impaired Cognitive Function

Definition	Model 1			Model 2			Model 3		
	Sarcopenia	Obesity	Sarcopenic Obesity	Sarcopenia	Obesity	Sarcopenic Obesity	Sarcopenia	Obesity	Sarcopenic Obesity
Events*	**n = 991**	**n = 203**	**n = 277**	**n = 991**	**n = 203**	**n = 277**	**n = 983**	**n = 202**	**n = 274**
GS/BMI									
Overall impaired cognitive function	2.49 (2.23–2.78)	0.98 (0.83–1.16)	1.76 (1.52–2.05)	1.64 (1.46–1.84)	0.99 (0.84–1.17)	1.27 (1.09–1.48)	1.6 (1.42–1.8)	0.98 (0.82–1.16)	1.2 (1.03–1.4)
Executive function	2.29 (1.96–2.67)	0.97 (0.76–1.22)	1.86 (1.51–2.28)	1.66 (1.41–1.96)	0.97 (0.76–1.23)	1.47 (1.19–1.81)	1.63 (1.38–1.92)	0.97 (0.76–1.23)	1.41 (1.14–1.74)
Memory	2.71 (2.39–3.08)	0.91 (0.74–1.11)	1.82 (1.53–2.16)	1.7 (1.49–1.94)	0.9 (0.74–1.1)	1.26 (1.06–1.49)	1.69 (1.47–1.93)	0.9 (0.74–1.11)	1.23 (1.03–1.47)
Orientation	2.72 (2.33–3.17)	0.87 (0.67–1.11)	1.78 (1.44–2.2)	1.65 (1.4–1.95)	0.91 (0.71–1.18)	1.2 (0.96–1.48)	1.65 (1.39–1.94)	0.94 (0.73–1.22)	1.18 (0.95–1.47)
Events*	**n = 492**	**n = 432**	**n = 732**	**n = 492**	**n = 432**	**n = 732**	**n = 487**	**n = 431**	**n = 726**
GS/Waist circumference									
Overall impaired cognitive function	2.52 (2.15–2.94)	1.05 (0.9–1.23)	2.23 (1.92–2.59)	1.6 (1.36–1.89)	0.99 (0.84–1.17)	1.45 (1.24–1.69)	1.59 (1.35–1.87)	0.99 (0.84–1.17)	1.39 (1.19–1.63)
Executive function	2.13 (1.71–2.66)	0.97 (0.78–1.22)	2.07 (1.68–2.54)	1.52 (1.21–1.91)	0.98 (0.78–1.24)	1.58 (1.27–1.95)	1.5 (1.2–1.89)	0.98 (0.78–1.24)	1.53 (1.23–1.9)
Memory	2.91 (2.42–3.5)	1.12 (0.93–1.36)	2.56 (2.15–3.06)	1.78 (1.47–2.15)	1.07 (0.89–1.3)	1.6 (1.34–1.92)	1.79 (1.48–2.16)	1.09 (0.9–1.32)	1.59 (1.33–1.92)
Orientation	2.84 (2.26–3.56)	1.09 (0.86–1.38)	2.55 (2.05–3.17)	1.67 (1.32–2.11)	1.0 (0.79–1.28)	1.46 (1.16–1.82)	1.67 (1.32–2.12)	1.02 (0.8–1.3)	1.46 (1.16–1.83)
Events*			**n = 1044**			**n = 1044**			**n = 1035**
GS:BMI ratio									
Overall impaired cognitive function			1.72 (1.57–1.88)			1.34 (1.22–1.47)			1.31 (1.19–1.45)
Executive function			1.58 (1.4–1.79)			1.37 (1.2–1.57)			1.35 (1.18–1.55)
Memory			1.72 (1.55–1.9)			1.3 (1.17–1.45)			1.3 (1.17–1.46)
Orientation			1.8 (1.59–2.04)			1.25 (1.1–1.43)			1.26 (1.1–1.45)

GS, grip strength; SDOC, Sarcopenia Definitions Outcome Consortium.

All values represented are hazard ratios (95%) confidence intervals.

Overall impaired cognitive function consists of an abnormal score on either the AD8 scale or on any of the 3 domains (memory, orientation, executive function).

Sarcopenia was defined as a grip strength <35.5 kg in men or <20 kg in women; obesity was defined as a BMI ≥30 kg/m^2; sarcopenic obesity was defined as participants fulfilling definitions of both sarcopenia and obesity; neither was defined as participants fulfilling neither sarcopenia or obesity. Cutpoints for WC: men < 102 cm; females < 88 cm; Cutpoints for GS: BMI: men < 1.05; females < 0.79

Model 1: Unadjusted; Model 2: Age category, sex, smoking, status, education; Model 3: Model 2 + Comorbidities (heart disease, hypertension, diabetes, lung disease, stroke, cancer, ever walk).

Each individual domain binarized based on NHATS cutoffs (0 = not impaired, 1 = impaired). If AD8 score not present, individual domain scores summed – overall impaired cognitive function defined as impairment in any domain (summed score 1, 2, or 3). Executive function consists of clock-drawing test; memory domain (immediate word recall, delayed word recall). Orientation consists of date recall and president/vice president naming.

* The number of events will differ depending on missingness of data.

Table 4

Linear Mixed Effects Modeling Using Grip Strength/Body Mass Index for Impaired Cognitive Function

	Model 1		Model 2		Model 3		Interaction Model	
	β ± SE	*P* value	β ± SE	*P* value	β ± SE	*P* value	β ± SE	*P* value
Overall impaired cognitive function								
Sarcopenia	0.330 ± 0.019	<.001	0.164 ± 0.019	<.001	0.157 ± 0.019	<.001	0.157 ± 0.019	<.001
Obesity	−0.032 ± 0.025	.201	−0.021 ± 0.023	.381	−0.015 ± 0.024	.25	−0.015 ± 0.024	.521
Sarcopenic obesity	0.142 ± 0.026	<.001	0.034 ± 0.025	.162	0.028 ± 0.025	.19	−0.114 ± 0.033	.001
Self-rated memory								
Sarcopenia	0.160 ± 0.025	<.001	0.061 ± 0.026	.020	0.030 ± 0.027	.266	0.030 ± 0.027	.266
Obesity	0.072 ± 0.033	.030	0.041 ± 0.033	.206	0.009 ± 0.033	.784	0.009 ± 0.033	.784
Sarcopenic obesity	0.202 ± 0.035	<.001	0.088 ± 0.035	.011	0.031 ± 0.036	.387	−0.008 ± 0.047	.865
Memory domain								
Immediate recall								
Sarcopenia	−0.997 ± 0.045	<.001	−0.393 ± 0.042	<.001	−0.376 ± 0.042	<.001	−0.376 ± 0.042	<.001
Obesity	0.030 ± 0.060	.614	0.015 ± 0.052	.779	0.026 ± 0.052	.625	0.026 ± 0.052	.625
Sarcopenic obesity	−0.658 ± 0.063	<.001	−0.243 ± 0.055	<.001	−0.207 ± 0.056	<.001	0.144 ± 0.073	.050
Delayed recall								
Sarcopenia	−1.089 ± 0.053	<.001	−0.414 ± 0.050	<.001	−0.401 ± 0.050	<.001	−0.401 ± 0.050	<.001
Obesity	0.084 ± 0.071	.234	0.079 ± 0.062	.198	0.084 ± 0.062	.180	0.084 ± 0.062	.180
Sarcopenic obesity	−0.719 ± 0.074	<.001	−0.258 ± 0.065	<.001	−0.229 ± 0.066	.001	0.088 ± 0.087	.314
Orientation								
Sarcopenia	−0.875 ± 0.049	<.001	−0.374 ± 0.047	<.001	−0.355 ± 0.047	<.001	−0.355 ± 0.047	<.001
Obesity	−0.013 ± 0.065	.840	−0.030 ± 0.059	.606	−0.032 ± 0.059	.589	−0.032 ± 0.059	.589
Sarcopenic obesity	−0.561 ± 0.068	<.001	−0.183 ± 0.062	.003	−0.157 ± 0.063	.013	0.231 ± 0.083	.005
Executive function								
Sarcopenia	−0.538 ± 0.029	<.001	−0.276 ± 0.027	<.001	−0.265 ± 0.027	<.001	−0.265 ± 0.027	<.001
Obesity	−0.006 ± 0.038	.879	0.012 ± 0.034	.729	0.011 ± 0.034	.750	0.011 ± 0.034	.750
Sarcopenic obesity	−0.347 ± 0.040	<.001	−0.143 ± 0.036	<.001	−0.127 ± 0.037	.001	0.127 ± 0.048	.008

All values represented as β-coefficient ± SE. Note: Interaction model includes Demographics and Comorbidities (heart disease, hypertension, diabetes, lung disease, stroke, cancer, ever walk).

Overall impaired cognitive function consists of an abnormal score on either the AD8 scale or on any of the 3 domains (memory, orientation, executive function).

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Sarcopenia was defined as a grip strength <35.5 kg in men or <20 kg in women; obesity was defined as BMI ≥ 30 kg/m^2; sarcopenic obesity was defined as participants fulfilling definitions of both sarcopenia and obesity; neither was defined as participants fulfilling neither sarcopenia or obesity. Model 1: Unadjusted; Model 2: Age category, sex, smoking, status, education; Model 3: Model 2 + Comorbidities (heart disease, hypertension, diabetes, lung disease, stroke, cancer, ever walk).

Each individual domain binarized based on NHATS cutoffs (0 = not impaired, 1 = impaired). If AD8 score not present, individual domain scores summed – impaired cognitive function defined as impairment in any domain (summed score 1, 2, or 3). Executive function consists of clock-drawing test; memory domain (immediate word recall, delayed word recall). Orientation consists of date recall and president/vice president naming.

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