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Body Mass Index, Dementia, and Mortality in the Elderly

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Abstract

Objectives—To explore the association between body mass index and mortality in the elderly taking the diagnosis of dementia into account.

Design—Cohort study.

Setting—cohort study of aging in Medicare recipients in New York City.

Participants—1,452 elderly individuals 65 years and older of both genders.

Measurements—We used proportional hazards regression for longitudinal multivariate analyses relating body mass index (BMI) and weight change to all-cause mortality.

Results—There were 479 deaths during 9,974 person-years of follow-up. There were 210 cases of prevalent dementia at baseline, and 209 cases of incident dementia during follow-up. Among 1,372 persons with BMI information, the lowest quartile of BMI was associated with a higher mortality risk compared to the second quartile (HR = 1.5; 95% CI: 1.1,2.0) after adjustment for age, gender, education, ethnic group, smoking, cancer, and dementia. When persons with dementia were excluded, both the lowest (HR = 1.9; 95% CI =1.3,2.6) and highest (HR = 1.6; 95% CI : 1.1,2.3) quartiles of BMI were related to higher mortality. Weight loss was related to a higher mortality risk (HR = 1.5; 95% CI: 1.2,1.9) but this association was attenuated when persons with short follow-up or persons with dementia were excluded.

Conclusion—The presence of dementia does not explain the association between low BMI and higher mortality in the elderly. However, dementia may explain the association between weight loss and higher mortality.

Keywords

mortality; dementia; body mass index; weight change

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Dr. Luchsinger had the main responsibility in the conception of the analysis, interpretation of the results, and drafting of the manuscript. Dr. Tang supervised the statistical analyses and writing of the manuscript. Dr. Mayeux was responsible for the collection of the data, definition of the outcomes, supervision of the project, and supervised the writing of the manuscript. None of the authors have conflicts of interest to report.

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INTRODUCTION

The relation of anthropometric measures to mortality in the elderly may be confounded by pre-morbid conditions such as cancer [2], heart disease [3] and habits such as smoking[4] that cause weight loss, but these conditions do not seem to account for the association between low BMI and mortality [5, 6], implying that low BMI directly causes mortality through unknown mechanisms, or that there is confounding by other conditions. Dementia has been identified as a condition associated with weight loss [7, 8], low BMI [9], and increased mortality [10], but most studies relating BMI and mortality do not control for the presence of dementia. Our objective was to explore the associations of BMI and weight change to mortality in a prospective study of elderly persons in Northern New York City taking into account the presence of dementia.

METHODS

Participants and Setting

Participants were enrolled in a cohort study by a random sampling of Medicare recipients 65 years or older residing in northern Manhattan (Washington Heights, Hamilton Heights, Inwood)[11]. Each participant underwent an in-person interview of general health and function at the time of study entry followed by a standard assessment, including medical history, physical and neurological examination as well as a neuropsychological battery. Baseline data were collected from 1992 through 1994. Follow-up data were collected at sequential intervals of approximately 18 months. Some participants did not complete follow-up at all intervals due to refusal to participate further, relocation or death. The institutional review board of Columbia-Presbyterian Medical Center approved this study.

Anthropometric measurements were conducted beginning in the first follow up interval of the cohort for all participants. Of the 2,126 individuals who underwent clinical assessment at baseline, 1,452 had anthropometric measurements at the first follow-up interval. For analyses with BMI, 80 of the available 1,452 persons were excluded due to missing height or improbable measurements, leaving a final analytic sample for BMI analyses of 1,372 participants and 1,113 persons had a second weight measurement.

Body Mass Index and Waist Circumference

BMI was calculated as weight in kilograms divided by height in meters-squared (kg/m^2) at the first follow-up examination. BMI was categorized as quartiles for the main analyses. Since the association between BMI and adverse outcomes has been reported to be U or J shaped[5] we used the second quartile as the reference for all analyses. We conducted secondary analyses categorizing BMI into four categories according to the National Heart Lung and Blood Institute (NHLBI) guidelines: underweight ($< 18.5\ kg/m^2$), normal ($18.5 - 24.9\ kg/m^2$), overweight ($25.0 - 29.9\ kg/m^2$), and obese ($\geq 30.0\ kg/m^2$)[12]. However, this classification has been reported to be of poor predictive value in the elderly[13], and we did not use it for the main analyses.

WC is the excess fat in the abdomen out of proportion to total body fat and is used as an predictor of risk factors and morbidity [14]. WC was measured at the level of the iliac crest while the subject was at minimal inspiration to the closest 0.1cm.

Weight change

We had a second weight measurement in 1,113 persons taken approximately 18 months after the first one. We calculated the yearly change in weight by subtracting the second weight measurement from the first one and dividing the difference by the interval in years between the two measurements. We classified weight change as weight loss (> 1 kg per year), stable

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weight (1 kg loss to 1 kg gain per year), and weight gain (> 1 kg per year). We also explored other category cutoffs for weight change (e.g. 10th and 90th percentile, tertiles, quartiles).

Clinical diagnoses

History of diabetes mellitus, hypertension, heart disease, cancer and smoking were ascertained by self report. Heart disease included a history of atrial fibrillation and other arrhythmias, myocardial infarction, congestive heart failure or angina pectoris. Smoking was classified into current and past smoking. In our cohort, only current smoking was associated with BMI and past smoking was not included in the analyses.

Plasma Lipids

Fasting plasma total cholesterol and triglyceride levels were determined at the first follow-up using standard enzymatic techniques. High-density lipoprotein (HDL) cholesterol levels were determined after precipitation of apolipoprotein B containing lipoproteins with phosphotungstic acid [15]. Low-density lipoprotein (LDL) cholesterol was recalculated using the formula of Friedewald et al. [16].

Diagnosis of Dementia

Diagnosis of dementia and assignment of specific cause was made by consensus of 2 neurologists, 1 psychiatrist, and 2 neuropsychologists based on baseline and follow-up information. The diagnosis of dementia was based on DSM-IV criteria [17] and required evidence of cognitive deficit on the neuropsychological test battery as well as evidence of impairment in social or occupational function (Clinical Dementia Rating of 1 or more) [18]. Diagnosis of AD was based on the NINCDS-ADRDA criteria [19]. A diagnosis of *probable* AD was made when the dementia could not be explained by any other disorder. A diagnosis of *possible* AD was made when the most likely cause of dementia was AD, but there were other disorders that could contribute to the dementia such as stroke and Parkinson's disease. A diagnosis of vascular dementia was made when the dementia started within 3 months of the stroke and in whom the local effects of stroke were thought to be the primary mechanism for dementia. Brain imaging was available in 85% of cases of stroke; in the remainder, World Health Organization (WHO) criteria were used to define stroke [20].

All-cause mortality

We obtained vital status from the National Death Index until December 31st 2003. Thus, individuals who did not have reported deaths were censored at that date.

Statistical Methods

Bivariate analyses compared variables among the BMI quartiles. If there were statistically significant global differences, pairwise comparisons were made between underweight, overweight and obese persons to those with normal BMI. Continuous variables were compared using analysis of variance, and categorical variables were compared using chi-squared test[21]. Cox proportional hazards regression models[22] were used in multivariate analyses exploring the association of BMI, WC, and weight change to all cause mortality[22]. We examined the exposures as continuous variables, and as described above. The time-to-event variable was time from measurement of BMI to death; individuals who did not die were censored at the last time of death ascertainment (12/31/2003). The multivariate models were constructed adding all relevant covariates, one at a time, and we present 3 models in the tables: one adjusted for age and gender, one also adjusted for education, ethnic group, cancer, current smoking, and dementia, and a third model adding diabetes, hypertension, LDL, and heart disease. The first two models adjust for potential confounders, while the third model includes variables that are probably in the causal pathway between BMI and mortality. Thus, we mostly describe the

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results for the second model in the text, and anticipated that any attenuation in the hazard ratios (HR) of the exposure of interest in the third model was indicative of mediation and not of confounding. For analyses with weight change as the exposure we included baseline BMI in all the models, and calculated the time to event from the time of the second weight measurement.

The predictive value of anthropometric measures may change by gender [23] and age [24, 25] and we conducted secondary analyses stratifying by gender and age. Current smoking, cancer, and dementia are also associated with lower BMI[5, 9]; thus we conducted secondary analyses relating BMI and WC to mortality excluding persons with cancer, current smoking, and dementia. Lower BMI may also be associated to unidentified co-morbid conditions, and subjects with early mortality can account for associations between low BMI and higher mortality [26]. Thus, we conducted secondary analyses excluding individuals with less than 2 years of follow-up after the measurement of BMI. All analyses were conducted using SAS 9.1 for Windows (Cary, NC).

RESULTS

The mean age of the sample was 78.3 ± 6.4 years, 69.9% were women. 32.5% were African American, 46.8% were Hispanic, and 20.7% were White. The mean number of years of education was 8.2 ± 4.6 years. The mean BMI was 26.5 ± 5.2 kg/m^2. The mean LDL was 118.9 ± 35.5 mg/dl; 57.4% reported hypertension, 19.9% diabetes, 28.3% heart disease, 4.9% cancer, and 10.1% current smoking. There were 479 deaths during 9,974 years of follow-up. There were 210 cases of prevalent dementia at baseline, and 209 cases of incident dementia during follow-up. The BMI was 26.8 kg/m^2 in persons without dementia, 25.9 kg/m^2 in persons with incident dementia, and 25.8 kg/m^2 in person with prevalent dementia (p=0.005). Prevalent dementia (HR=2.1; 95% CI 1.7,2.5) but not incident dementia (HR=0.9;0.7,1.2) was related to higher mortality after adjustment for age and gender. Compared to persons in the second quartile of BMI (Table 1), persons in the first quartile were older, had a lower prevalence of diabetes, hypertension, and cancer, a higher prevalence of current smoking and dementia, and a shorter time of follow-up. Persons in the third quartile of BMI had a higher proportion of women, and a higher prevalence of hypertension and heart disease. Persons in the fourth quartile of BMI were younger, had a higher proportion of women and African Americans, a lower proportion of Whites, and a higher prevalence of hypertension and heart disease.

Relation of body mass index (BMI) to mortality

BMI as a continuous variable was not related to mortality (HR = 0.9, 95% CI: 0.9,1.0) after adjustment for age, gender, education, ethnic group, cancer, current smoking, and dementia. In analyses relating BMI quartiles to mortality risk (Table 2), the first BMI quartile was associated with higher mortality compared to the second quartile after adjustment for age, gender, education, ethnic group, cancer, current smoking, and dementia (HR=1.5;95% CI: 1.1,2.0). The results did not change in 1,071 persons without current smoking and cancer (HR for the first quartile = 1.5; 95% CI: 1.1,1.9) or when prevalent and incident dementia were introduced as different variables in the model or when prevalent and incident dementia were introduced as different variables in the model (HR= 1.4; 95% CI: 1.4; 1.1,1.8). When persons with dementia were excluded, the HR relating the first quartile of BMI to mortality increased to 1.9 (95% CI= 1.3,2.6), and there was an increased risk of mortality for the highest quartile of BMI (HR=1.6; 95% CI= 1.1,2.3) after adjustment for age, gender, education, ethnic group, current smoking, and cancer; the third quartile of BMI was not associated to mortality (HR= 1.3,0.9,1.9). Among the 419 persons with dementia there was no association of BMI quartile to mortality; the HR for the first quartile was 1.1 (95% CI:0.8,1.6), 1.1 for the third quartile (95 % CI: 0.7,1.7), and 0.7 for the 4th quartile (95% CI: 0.5,1.1). These associations did not

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change after exclusion of persons with cancer, current smoking, and less than 2 years of follow-up. The association between the first quartile of BMI and mortality for the whole sample was modestly attenuated after 94 persons with less than 2 years of follow-up were excluded (HR=1.3; 95% CI: 1.0,1.8). In analyses stratified by median age (77 years), the first quartile of BMI was associated with a higher mortality risk for older and younger persons, but the association was stronger for younger persons. In persons aged 65 to 77 years the third and fourth quartiles of BMI were also associated with an increased risk of mortality. These associations were stronger for persons under 77 years of age without dementia: the HR of mortality was 2.2 (95% CI: 1.2,4.1) for the lowest BMI quartile compared to the second,1.8 (95% CI: 1.0,3.2) for the third quartile, and 2.2 (95% CI: 1.2,3.8) for the fourth quartile. For persons over 77 years of age without dementia, the lowest BMI was associated with a higher mortality risk (HR = 1.7; 95% CI: 1.1,2.5) but not the third or fourth quartiles. There was no effect modification by gender.

We conducted secondary analyses relating BMI classified by NHLBI guidelines to mortality, and found that underweight status was not associated with higher mortality (HR= 1.2;95% CI: 0.8,1.8) compared to normal. Overweight (HR= 0.8;95% CI: 0.7,0.9), and obese (HR= 0.7;95% CI: 0.5,0.9) status were related to a lower risk of mortality. These associations were not modified by excluding persons with current smoking, cancer, or less than 2 years of follow-up; however, when persons with dementia were excluded from the analyses, the associations of overweight (HR=0.9,0.7,1.2) and obesity (HR=0.8, 0.6,1.2) were attenuated. These analyses classifying BMI by NHLBI guidelines were limited by the low number of persons who were underweight (n=55), and a larger number of persons who were classified as normal (n= 515) and overweight (n=507) compared to the categorization of BMI by quartiles.

Relation of weight change to mortality

We also related weight change to the risk of mortality (Table 3). Only persons with more than 1 kg of yearly weight loss had an increased mortality risk (HR =1.5; 95% CI: 1.2,1.9) after adjustment for age, gender, baseline BMI, education, ethnic group, cancer, current smoking, and dementia. The association attenuated and became non-significant when persons with less than two years of follow-up were excluded (HR=1.2; 95% CI= 0.9,1.6). It was also attenuated when persons with dementia were excluded (HR = 1.3;0.9,1.9), but did not change when current smokers and persons with cancer were excluded (HR=1.5; 95% CI: 1.1,1.9). The association of weight loss to mortality was not modified by age or gender.

DISCUSSION

We found that in individuals over the age of 65 years that low BMI is related to higher mortality risk even after taking into account conditions associated with weight loss, such as cancer, current smoking, and dementia. However, we found that dementia, either prevalent or developed during follow-up, was not an appreciable confounder of the association between low BMI and mortality in the expected direction. When persons with dementia were excluded from the analyses, the association between low BMI and mortality became stronger, and the highest quartile of BMI was associated with higher mortality. We confirmed that age is an important modifier in the association of BMI to mortality in the elderly.

The association between low BMI and mortality in the elderly does not seem to be explained by traditional confounders, such as cancer, and smoking[5, 6, 27-29]. This may indicate that known pre-morbid conditions (e.g. cancer, smoking, heart disease) are not important confounders in the association between low BMI and mortality, or that there is residual or unmeasured confounding by other conditions[6]. We sought to explore whether dementia, which is associated with weight loss [30], lower BMI [9], and higher mortality [1, 31-35], could explain the association between low BMI and mortality. Dementia may cause weight loss and lower BMI due to

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feeding difficulties[7] and other catabolic changes that are not totally understood. The main contribution of our study to the literature of the association of anthropometric measures to mortality in the elderly is the addition of dementia as a covariate and potential confounder. We also found that higher BMI was related to higher mortality once persons with dementia were excluded from the analyses. Persons with dementia tend to be older (80.7 vs. 76.5 year; $p < 0.001$), and higher BMI is related to higher mortality in younger persons[25], and this may explain our results. Our stratified analyses by age were in general consistent with the published literature, and showed that high BMI tended to predict increases in mortality in younger elderly. However, low BMI was a predictor of increased mortality for the whole sample, even in younger persons.

The mechanisms for the association of lower BMI to increased mortality remain unclear. The relationship between low BMI and higher mortality may be explained by lower fat free mass in thin individuals rather than overall leanness [28]. Low BMI and weight loss be a proxy for malnutrition, which is common in sick elderly persons [36]. Low BMI and low body cell mass are predictors of higher mortality in nursing home residents [37], and in hospitalized elderly after discharge [38]. Lower BMI may also be a proxy for weight loss, which is related to higher mortality [39]. Voluntary weight loss by dieting may be related to higher mortality as well as involuntary weight loss [39], raising the possibility that there are specific mechanisms related to leanness that increase mortality. For example, some have postulated that decreased estrogen synthesis in lean individuals may account for this association [40]. The results of our study help to establish that the presence of dementia does not explain the association of low BMI to increased mortality, and support further investigation to elucidate the mechanisms that explain this association. However, our results indicate that dementia could partially explain the relation between weight loss and increased mortality.

There are several potential explanations for our finding of no association of higher BMI and mortality in the oldest old, and WC and mortality for the whole sample. Aging is related to a decrease in body weight mainly due to lean body mass loss[41], and consequently, BMI does not capture adiposity in the elderly, particularly the oldest old. Another important explanation is bias related to the study of an elderly cohort.

The main limitations of our study are the lack of data on cause-specific mortality and of weight history prior to inclusion in the cohort. We had BMI measurements of BMI for a time point before the onset of dementia for persons with incident dementia, but not at the time of dementia diagnosis. Thus we could properly adjust for dementia as a confounder only for those with prevalent dementia. All anthropometric measures have limitations in the measurement of adiposity in the elderly and direct measurement of body composition may be needed to capture this construct [5].

The association between low BMI and higher mortality in the elderly is not explained by dementia, and more research is needed to understand this phenomenon.

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Table 1

Comparison of relevant clinical characteristics among body mass index quartiles

Characteristics	1st quartile (< 23.0g/m^2)	2nd quartile (23.0 −26.1 kg/m^2)	3rd quartile (26.2 −29.4 kg/m^2)	4th quartile (> 29.4 kg/m^2)
Sample Size	342	344	343	343
Age (yrs ± S.D.)	79.7±6.4†	78.1±6.4	77.3±5.9	77.1±5.6*
Women (%)	228 (66.7)	207 (60.2)	241 (70.3)†	279 (81.3)‡
African American (%)	110 (32.2)	92 (26.7)	92 (26.8)	143 (41.7)‡
Hispanic (%)	148 (43.3)	170 (49.4)	178 (51.9)	147 (42.9)
White (%)	84 (24.6)	82 (23.8)	73 (21.3)	53 (15.5)†
Education (yrs ± S.D.)	8.1±4.6	8.4±4.8	8.1±4.5	8.1±4.7
LDL (mg/dl ± S.D.)	116±37.0	118±34.1	120.3±36.2	122.9±34.3
Hypertension (%)	170 (49.7)*	178 (51.7)	213 (62.1)†	233 (67.9)‡
Diabetes (%)	46 (13.5)*	63 (18.3)	78 (22.7)	78 (22.7)
Heart Disease (%)	82 (23.9)	81 (23.6)	106 (30.9)*	109 (31.8)*
Cancer (%)	7 (2.1)*	19 (5.5)	26 (7.6)	20 (5.8)
Dementia (%)	125 (37.1)*	105 (30.7)	96 (28.4)	93 (27.3)
Current smoker (%)	52 (15.2)*	31 (9.0)	29 (8.5)	26 (7.6)
Follow-up (yrs ± S.D.)	6.5±2.8‡	7.3±2.2	7.1±2.4	7.2±2.3

* $p < 0.05$

† $p < 0.01$

‡ $p < 0.001$

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Table 2

Hazard ratios (HR) and 95% confidence intervals relating body mass index (BMI) quartiles to all-cause mortality. Model 1 is adjusted for age and gender. Model 2 is adjusted also for education, ethnic group, cancer, current smoking, and dementia. Model 3 is also adjusted for diabetes, hypertension, low density lipoprotein (LDL), and heart disease. Washington Heights Inwood Columbia Aging Project 1992 − 2003.

BMI quartiles	At Risk	Deaths (rate per 100)	Model 1 HR (95% CI)	Model 2 HR (95% CI)	Model 3 HR (95% CI)
1 (< 23.0 kg/m^2)	342	152 (6.8)	1.5 (1.1,1.9)	1.5 (1.1,2.9)	1.6 (1.2,2.0)
2 (23.0−26.1 kg/m^2)	344	107 (4.3)	1.0	1.0	1.0
3 (26.2 − 29.4 kg/m^2)	343	111 (4.5)	1.2 (0.9,1.5)	1.2 (0.9,1.6)	1.1 (0.8,1.4)
4 (> 29.4 kg/m^2)	343	109 (4.4)	1.2 (0.9,1.6)	1.2 (0.9,1.5)	1.1 (0.8,1.4)

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Table 3

Hazard ratios (HR) and 95% confidence intervals relating weight change to all-cause mortality. Model 1 is adjusted for age, gender, and baseline body mass index (BMI). Model 2 is adjusted also for education, ethnic group, cancer, current smoking, and dementia. Model 3 is also adjusted for diabetes, hypertension, low density lipoprotein (LDL), heart disease. Washington Heights Inwood Columbia Aging Project 1992 − 2003.

Weight change	At Risk	Deaths (rate per 100)	Model 1 HR (95% CI)	Model 2 HR (95% CI)	Model 3 HR (95% CI)
Loss (> 1 kg/year)	441	167 (6.6)	1.6 (1.3,2.1)	1.5 (1.2,1.9)	1.5 (1.2,1.9)
Stable (≤ 1 kg loss/year or gain/year)	388	102 (4.2)	1.0	1.0	1.0
Gain (> 1 kg/year)	284	88 (4.9)	1.2 (0.9,1.6)	1.1 (0.8,1.5)	1.1 (0.8,1.5)