Published in final edited form as:

Ann Epidemiol. 2009 October ; 19(10): 724–731. doi:10.1016/j.annepidem.2009.05.003.

Waist-Hip-Ratio as a Predictor of All-Cause Mortality in High-Functioning Older Adults

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Abstract

Purpose—The relationship between obesity and mortality in older adults is debated, with concern that body mass index (BMI) may be an imperfect measure of obesity in this age group. We assessed the relationship between three measures of obesity and all-cause mortality in a group of healthy older adults.

Methods—We analyzed data from the MacArthur Successful Aging Study, a longitudinal study of highfunctioning men and women, aged 70–79 years at baseline. We examined 12-year, all-cause mortality risk by BMI, waist circumference (WC) and waist-to-hip circumference ratio (WHR). Proportional hazards regression was used to adjust for gender, race, baseline age, and smoking status. We tested for obesity interactions with gender, race, and smoking status, and conducted stratified analyses based on the results of interaction testing.

Results—There was no association between all-cause mortality and BMI or WC in either unadjusted or adjusted analyses. In contrast, all-cause mortality increased with WHR. There was an interaction with sex, so that there was a graded relationship between WHR and mortality in women (relative hazard, 1.28 per 0.1 increase in WHR, 95% confidence interval 1.05, 1.55) and a threshold relationship in men (relative hazard 1.75 for WHR>1.0 compared to WHR≤1.0, 95% confidence interval 1.06, 2.91).

Conclusion—WHR rather than BMI, appears to be the more appropriate yardstick for risk stratification of high-functioning older adults.

Keywords

MeSH Heading Keywords: Waist-Hip Ratio; Mortality; Obesity; Geriatric Assessment; Health Status Indicators

INTRODUCTION

Obesity, which contributes to multiple morbidities, has been increasing in the United States for sometime (1, 2) and has been associated with approximately 300,000 excess deaths annually (3). Data from longitudinal cohort studies, suggest that the incidence of obesity continues to increase with age, even after age 60 (4). In 1999–2000, 33% of American men

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and 39% of American women 65 to 74 (5) years old met criteria for obesity(6). In older adults, obesity is thought to contribute to insulin resistance and type 2 diabetes mellitus (7), hypertension, dyslipidemia (8), and ultimately to coronary artery disease (9); however the magnitude of the impact of obesity on the health of older adults, is unclear.

It has been suggested that the positive association between high body mass index (BMI) and mortality, seen in younger populations (10) wanes in those more than 65 years old, and that death rates in obese older adults may even be lower than in older adults who don't meet criteria for obesity (11, 12). As a result, there have been calls to raise the BMI threshold for obesity in older adults above the threshold used in young and middle-aged adults (13). There is however, a more fundamental question of whether or not BMI is the appropriate measure of obesity in older adults. Changes in body size and composition that commonly occur with aging may limit the usefulness of BMI in assessing the extent of adiposity in older adults: 1) Measured height decreases with aging in many individuals because of changes in standing posture (14), which induces an artifactual increase in BMI of 1.5 kg/m² on average in men and 2.5 kg/m² in women, despite minimal change in body weight (15). 2) Muscle loss is common with aging (16), which leads to reduction in BMI even if the muscle is replaced by fat. 3) Unintentional weight loss is common in many older individuals, as a result of diseases such as cancer, or as part of the syndrome of frailty, and both are associated increased mortality risk (17), which confounds the BMI-mortality relationship.

Since abdominal adipose tissue may be the real culprit in the health risks associated with obesity, measures of absolute and relative waist size such as waist circumference (WC) and waist-hip ratio (WHR) may be the more relevant clinical measures of the extent of adiposity (18). Central fat increases the risk of developing both metabolic syndrome and diabetes mellitus(19–21) and contributes to cardiovascular risk by increasing peripheral vascular resistance (22) and secreting pro-inflammatory cytokines (23, 24). In fact, measures of central adiposity predict cardiovascular risk better than measures of generalized obesity such as BMI(25–30) and the association between central fat and glucose disorders is also independent of measures of generalized obesity (31) (32, 33). Further, WC has been associated with increase in all cause mortality (34, 35) as has WHR (36–38) suggesting a broader mortality impact of central obesity.

In several recent studies, examining the usefulness of WC and WHR as predictors of mortality risk in mixed groups of patients between 20 and 75 years, it has been noted that WHR has a monotonic relationship with mortality(34, 39–41), whereas BMI has had a J shaped relation(41) or little relation at all(40) with mortality risk. However, studies in older adults have shown varying findings by gender (42, 43) and ethnicity (44). A recent, United Kingdom study in a predominantly Caucasian population demonstrated the better prediction ability of WHR over BMI for both total and cardiovascular mortality over median follow up of 5.9 years, in adults, 75 years of age or older. Compared with the lowest quintile of BMI, there was no increase in mortality associated with being in the top two quintiles of BMI, but in non-smokers, mortality trended upwards from the lowest to the highest quintile of WHR (45). It is not clear, however, whether the failure of BMI to predict mortality in this study was a result of recent weight loss associated with frailty.

Therefore, the primary objective of our study was to compare BMI, WHR and WC as predictors of mortality over a longer period (12 years) in a cohort of older Americans selected to be high-functioning (i.e. who are not frail and less likely to have had recent weight loss that might confound the obesity-mortality association).

METHODS

Study Sample

We analyzed data from the MacArthur Successful Aging Study, a longitudinal study of relatively high functioning men and women aged 70–79 years at baseline (46). More than 4000 non-institutionalized, 70–79 year-old men and women from three communities (Durham, NC, East Boston, MA, and New Haven, Conn.) were screened on the basis of 4 criteria of physical functioning and 2 criteria of cognitive functioning, to identify those in the top tertile of functioning. The screening criteria were: 1) no self-reported disability on the Katz Activities of Daily Living scale (47), 2) no more than one disability on the Rosow-Breslau (48) and Nagi (49) self-reported scales of disability in physical function, 3) ability to hold a semi-tandem balance for at least 10 seconds, 4) ability to stand up from a seated position 5 times within 20 seconds, 5) score of 6 or better on the 9-item Short Portable Mental Status Questionnaire (50) and 6) ability to recall 3 or more of 6 elements from a short story after delay. Of the 1313 individuals who met all 6 criteria, 91% (N=1189) agreed to participate and provided informed consent. Informed consent was obtained from all subjects after the respective human subjects institutional review boards approved the protocols.

Measurements

Baseline data (including self-reported demographics, chronic conditions, and health behaviors) were collected, starting in 1988. Follow up data were collected after a mean interval of 28 months, starting in 1991, and once again, after a mean interval of 57 months from the first follow up, starting in 1995. Age (years), gender, ethnicity (white versus black), current smoking status, pack-years of smoking exposure, height, and body weight, were obtained by self-report. BMI was computed as weight (in kilograms) divided by the square of the height (in meters squared). Using procedures outlined in the 1988 Anthropometric standardization reference manual (51), WC and hip circumference were measured, and WHR was computed as the ratio of WC to hip circumference.

Deaths were identified through contact with next of kin at the time of the follow-ups (1991 and 1995), ongoing local monitoring of obituary notices, and a National Death Index search for deaths up to the year 2000. Date of death information from the NDI was available for all but three of the deceased participants, whose deaths were confirmed by next of kin contact.

Analysis

Initial, unadjusted analysis examined 12-year, all-cause mortality risk by 7–8 equal-length categories of the 3 candidate predictors: BMI, WC and WHR; categories were chosen after examination of predictor distributions, to ensure that at least 5% of the sample was in each category. Proportional hazards regression was then used to adjust for gender, race, age at baseline, and smoking. If the adjusted association between candidate predictor and mortality was found to be monotonic, we replaced the categorical predictor by its continuous version, for further analysis.

Sensitivity Analyses—One postulated reason for the poor predictive ability of obesity measures in older adults is confounding by recent weight loss in those who are close to death. Thus we conducted sensitivity analyses, in which we excluded individuals who died in the early part of the study (first 2.5 years and first 7 years), and examined mortality in the later years.

Interaction testing and Stratified Analyses—The distributions of waist size and WHR are substantially different in men and women, and the relationship of these

anthropometric measures to total mortality may differ by gender. We tested for gender differences in the obesity-mortality association by adding gender interaction terms to the model. We also tested for interactions with current smoking, because others have noted that the relationship between obesity and mortality is modified by smoking status (45). Based on the results of the interaction testing, we examined the obesity-mortality relationship stratified by the effect-modifying variable.

RESULTS

Study Sample

The average age of participants was 74 years, and the study sample was 81% white. BMI was similar in men and women (mean 26 in both), but both WC and WHR were higher in men than in women (Table 1). There were 71 deaths from all causes by the time of the first follow up (1991), another 202 deaths by the time of the second follow up (1995), and an additional 219 deaths by final NDI follow up in 2000.

Unadjusted Mortality Associations

Examination of the 12-year unadjusted risks for all-cause mortality as a function of baseline values of the three candidate measures of obesity revealed that mortality risk increased with measures of central obesity (WC and WHR) but was essentially constant over the range of BMI (Figure 1). The increase in mortality risk was most dramatic over the range of WHR compared to WC.

Adjusted Mortality Associations

After adjusting for age, sex, race, and smoking (current status and total pack-years) in a proportional hazards model, only WHR showed a monotonically increasing association with mortality rate; the other candidate predictors had neither monotonic nor U-shaped associations with mortality (Fig. 2, panels a – c).

Sensitivity Analyses

To test if the poor predictive performance of a weight-based measure, such as BMI, might be the result of confounding by recent weight loss in frail older adults approaching death, we excluded those who died before the first follow up in 1991 (approximately 2.5 years after the baseline assessments of obesity measures). Again, only WHR showed a near-monotonic relationship with mortality rate after 1991, and the other 2 measures had neither monotonic nor U-shaped associations (Fig 2). Only WHR showed near-monotonic associations with mortality after the second follow up in 1995 (7 years after baseline obesity assessments); the other measures showed no consistent pattern (Fig 2). Analyses with continuous versions of the obesity measures as predictors also confirmed a linear mortality trend with WHR for deaths since baseline, deaths after 1991, and deaths after 1995; there was no linear trend with the other 2 measures (See Table 2).

Interactions and Stratified Analyses

Gender modified the relationship between continuous WHR and mortality (p values 0.04, 0.07, and 0.6 for mortality after baseline, after 1991, and after 1995 respectively), and current smoking did not modify the WHR-mortality association ($p > 0.6$ for smoking interactions). In gender-stratified analyses, there was a strong relationship between continuous WHR and mortality in women, but not in men (See Table 3). To allow for the possibility that the WHR relationship to mortality in men may not be graded, we examined the adjusted mortality rate as a function of WHR categories in men, and found that only the highest category (WHR > 1.0) was associated with increased mortality in men (HR for

mortality after 1995: 1.75, 95% CI 1.06, 2.91). We also examined the relationship of the other 2 obesity measures (as both categorical and continuous predictors) with mortality separately in men and women, and neither monotonic nor U-shaped relationships were seen for any of the measures in either gender (data not shown).

DISCUSSION

Abdominal girth relative to body size, as indexed by WHR, is positively associated with all cause mortality in high functioning older men and women, however neither BMI, the accepted measure of generalized obesity, nor WC, an absolute measure of abdominal girth, has significant associations with all-cause mortality in high-functioning older adults of either gender. These findings are consistent with previous studies (42–44) that have found BMI to be a poor predictor of mortality in older adults, and with studies (52) that have noted WHR to have a more consistent positive relationship with mortality, even in older ages.

The poor predictive ability of BMI in previous studies in the elderly may have been a result of confounding by recent weight loss and frailty. Our study in high functioning older adults, was designed to minimize such confounding, and suggests that markers of generalized obesity (such as BMI) should not be used to assess obesity related health risks in older adults (even if they are not frail), and that markers of central obesity might be more relevant in all older adults.

There are several possible explanations for our finding. First, generalized obesity is thought to provide a nutritional reserve against acute illnesses that are more common in older ages (53, 54), and provide protection against fall-related injuries (that can lead to increased mortality (55)) by both increasing soft tissue padding around vulnerable bone, and increasing bone mass as a result of fatty tissue synthesis of estrogen and greater weight bearing bone formation (56). Secondly, replacement of muscle mass by lighter adipose tissue, commonly seen in aging, makes weight-based measures less reliable as indicators of obesity in older adults. Finally, as mentioned previously, there are age related changes in measured height (57, 58), which produce artifactual changes in BMI (59).

Waist girth is an easily assessed measure of central obesity, and it is positively associated with risk of coronary artery disease and cardiovascular disease mortality (42, 60, 61). However, there is tremendous variation in normal body size in the population, and waist size normalized by body size might be a better marker of obesity than waist size by itself. Further, WHR is more than a simple measure of truncal obesity (62, 63) - it reflects the relative abundance of visceral fat over peripheral fat and muscle (specifically, gluteal muscle). This may be particularly pertinent in older adults, as muscle loss and alterations in regional fat distribution are common with aging. Thus our finding that WHR is a better predictor of all-cause mortality, than WC, in high-functioning older adults (with a graded relationship in women over the entire range of WHR and a threshold effect at WHR>1.0 in men) suggests the greater mortality import of relative abdominal adiposity (relative to body size, peripheral adiposity, and muscle mass) compared to absolute abdominal adiposity. This finding is consistent with studies that have found decreased health risks in individuals with more peripheral muscle and greater hip circumference, adjusted for body size and/or BMI (64–67).

Tice et al. (68) have also noted a monotonic relationship between WHR (but not BMI) and mortality in women. The stronger, graded relationship in women may be the result of greater fat mass increase with WHR in older women compared to older men (69). WHR associations with mortality were stronger when early deaths were excluded, which is consistent with the hypothesized metabolic dysregulation pathway, which takes time to

impact mortality. Competing causes for near-term mortality, such as concurrent illnesses, undiagnosed pathology, and frailty, dilute the near-term impact of obesity on mortality. The higher prevalence of these conditions in men compared to women, also contributes to the observed sex difference in WHR-mortality associations..

Our study had some important limitations. Body height and weight were obtained by self report, which may have led to underestimation of BMI, primarily because older adults tend to report their peak heights at younger ages (70) and overweight and obese individuals tend to round downwards in reporting their weights (70). However, such underestimation of BMI would only have strengthened a BMI-mortality gradient, had it been present. No such gradient was seen in this study. Secondly, our study was based on single measurements of WHR, WC and BMI at one time point. This limits our ability to draw inferences re: the effect of changing body size on mortality.

Strengths of our study include the fact that at baseline, our cohort represented high functioning adults, thus minimizing confounding by frailty. To further reduce any residual confounding by recent weight-loss caused by a terminal condition, we excluded deaths in the first two and half years of follow-up (and first 7 years in a second sensitivity analysis), and noted minimal effects of these exclusions on the nature of the WHR relationship to mortality.

In conclusion, WHR, rather than BMI or WC, appears to be the more appropriate yardstick for obesity-related risk stratification of high-functioning older adults, and possibly all older adults. However, given our use of self reported weight and height data, these findings need to be confirmed in other cohorts of older adults. Further research into the mechanisms underlying the increased health risks associated with high WHR is also needed, specifically to delineate the role of intra-abdominal visceral fat (measured by magnetic resonance imaging, for instance) in older adults' health risks.

Selected Abbreviations and Acronyms

BMI	body mass index
WC	waist circumference
WHR	waist-to-hip circumference ratio
NDI	National Death Index

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Figure 1. 12-year All-Cause Mortality Risk as Function of Obesity Indices

BMI: Body mass index, WC: Waist circumference, WHR: Waist-to-hip circumference ratio

P values for trend: BMI 0.17, WC 0.01, WHR <0.0001

Fig 2.

Fig 2a. Adjusted Mortality Hazard as Function of 1988 Body Mass Index
Adjusted for age, sex, race, and smoking

P values for trend: 0.3 (mortality after 1988), 0.5 (after 1991), 0.8 (after 1995)

Fig 2b Adjusted Mortality Hazard as Function of 1988 Waist Size

Adjusted for age, sex, race, and smoking

P values for trend: 0.6 (mortality after 1988), 0.7 (after 1991), 0.8 (after 1995)

Fig 2c Adjusted Mortality Hazard as Function of 1988 Waist-Hip Ratio

Adjusted for age, sex, race, and smoking

P values for trend: 0.06 (mortality after 1988), 0.15 (after 1991), 0.07 (after 1995)

Table 1

Descriptive Statistics of the Study Sample Mean (standard deviation) or Number (%)

	All (N=1189)	Women (N=659)	Men (N=530)
Age (years)	74 (2.7)	74 (2.7)	74 (2.7)
Race: White	960 (81%)	522 (80%)	438 (83%)
Current smokers	184 (15%)	96 (15%)	88 (17%)
Packyears of smoking	22 (34)	14 (24)	33 (40)
Body mass index (kg/m²)	26 (4.2)	26 (4.7)	26 (3.6)
Waist circumference (inches)	36.3 (4.8)	34.6 (4.8)	38.5 (3.9)
Waist circumference (cms)	92.3 (12.2)	88.0 (12.1)	97.7 (9.9)
Waist-hip ratio	0.88 (0.08)	0.84 (0.07)	0.94 (0.06)
Waist-hip ratio: ≤ 0.75	64 (5%)	63 (10%)	1 (.2%)
(0.75, 0.80)	128 (11%)	124 (19%)	4 (1%)
(0.80, 0.85)	205 (17%)	187 (29%)	18 (3%)
(0.85, 0.90)	239 (20%)	141 (21%)	98 (19%)
(0.90, 0.95)	310 (26%)	94 (14%)	216 (41%)
(0.95, 1.00)	177 (15%)	38 (8%)	139 (26%)
>1.00	62 (5%)	9 (1%)	53 (10%)
All deaths by 2000	492 (41%)	208 (32%)	284 (54%)
Deaths after 1991 follow-up	421 (35%)	187 (28%)	234 (44%)
Deaths after 1995 follow-up	219 (18%)	103 (16%)	116 (22%)

Table 2Adjusted mortality hazard ratios ^a for 1988 obesity measures as continuous predictors

	Mortality since baseline (1988)	Mortality since 1991	Mortality since 1995
Body Mass Index (kg/m ²)	0.99 (0.97 to 1.01)	0.99 (0.97 to 1.02)	1.00 (0.96 to 1.03)
Waist Circumference (cms)	1.00 (0.99 to 1.01)	1.00 (0.99 to 1.01)	1.00 (0.99 to 1.02)
Waist-Hip Ratio (per 0.1)	1.11 (0.97 to 1.28)	1.08 (0.93 to 1.26)	1.24 (1.00 to 1.53) *

^aHazard ratios reported as *point estimate* (95% confidence interval), and are adjusted for age, gender, race, current smoking status, and total pack-years of smoking.

*
 $p < 0.05$

Table 3Adjusted Mortality Hazard Ratios ^a for Waist-Hip Ratio after Stratification by Gender

	Mortality since baseline [1988]	Mortality since 1991	Mortality since 1995
Women ^b	<i>1.28</i> (1.05 to 1.55) *	<i>1.22</i> (1.00 to 1.50) *	<i>1.30</i> (0.98 to 1.72)
Men	<i>0.95</i> (0.77 to 1.12]	<i>0.91</i> (0.72 to 1.16)	<i>1.15</i> (0.82 to 1.60)

^a Hazard ratios reported as *point estimate* (95% confidence interval) per 0.1 increase in WHR, and are adjusted for age, race, current smoking status, and total pack-years of smoking.

^b p values for test of gender interaction: *0.04* for mortality since baseline, *0.07* for mortality since 1991, and *0.6* for mortality since 1995.

* p < 0.05.