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Survival in Older Men May Benefit From Being Slightly Overweight and Centrally Obese—A 5-Year Follow-up Study in 4,000 Older Adults Using DXA

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Background. Whether overweight in old age is hazardous remains controversial. Body mass index (BMI) overestimates adiposity and fails to measure central adiposity. We used dual-energy x-ray absorptiometry (DXA) to measure adiposity and hypothesized that overall adiposity, distribution of adiposity, and muscle mass might individually affect survival.

Methods. We recruited 2000 men and 2000 women aged 65 years or older. Baseline BMI, waist–hip ratio (WHR), body fat index (BFI = total body fat/height square), relative truncal fat (RTF = trunk fat/total body fat), and body muscle mass index (BMMI = total body muscle mass/height square) were measured. Mortality was ascertained by death registry after 63.3 (median) months.

Results. Two hundred and forty-two men and 78 women died. In men, mortality hazard ratio (HR) decreased consistently by 0.85 ($p < .005$), 0.86 ($p < .005$), and 0.86 ($p < .005$) per every quintile increase in BMI, BFI, and BMMI, respectively. A J-shaped relationship was observed in central adiposity (RTF and WHR) quintiles; the minimum values were at the 3rd WHR quintile (0.92–0.94) and 4th RTF quintile (mean WHR, 0.94). When RTF was tested with BFI, both high and low central adiposity were unfavorable while general adiposity became marginally insignificant ($p = 0.062$). When BFI and BMMI were tested together, increasing adiposity rather than muscle mass favored survival (BFI quintile, HR 0.97, p .015; BMMI quintile, HR 1.00, p .997).

Conclusions. Older men were resistive to hazards of overweight and adiposity; and mild-grade overweight, obesity, and even central obesity might be protective. This may bear significant implication on the recommended cutoff values for BMI and WHR in the older population.

***Key Words*:** mortality—adiposity—BMI—muscles.

NUMEROUS reports have observed a J- or U-shaped relationship between body mass index (BMI) and mortality in the general population (1–4). On the one hand, there exists robust evidence that underweight is deleterious in older adults (5–10), whereas on the other, this relationship remains controversial at the high end of BMI range. In contrary to the young or middle-aged population, a high BMI in old age may either be beneficial (7,9,10–12), insignificant (5,13), unfavorable for survival up to the age of 75 years (14), or only became hazardous at very high BMI values (15). Even in studies that described the deleterious effect of being overweight, the associated excess risk was attenuated in old age when compared with the younger population (4,16). Therefore, it is possible that high BMI is either genuinely protective or that older people are more resistive to the harmful effect of adiposity.

Owing to the progressive age-related decline in stature (17), BMI has been known to overestimate adiposity in older persons (18–20). Furthermore, being a composite index of both fat and muscle mass, BMI is not measuring adiposity alone. In addition, there exists a strong positive association between BMI and muscle mass in older persons (21,22). Therefore, it is possible that a high BMI in older age represents an optimal combination of adiposity and muscle rather than obesity. Participants with high BMI might in actual fact be simply nonobese and nonsarcopenic and therefore survive most favorably. Furthermore, it has been increasingly recognized that central obesity, which BMI fails to measure, may be more detrimental than general obesity (9,12,23–27). All these perspectives may account for the spurious relationship between high BMI and mortality.

To overcome the limitation of BMI serving as an adiposity parameter in old age, past studies have attempted to use methods such as bioelectrical impedance (28), total body potassium measurements (29), or skinfold thickness (30) to estimate fat mass and fat-free mass, aiming to separate the effect of muscle and adiposity on mortality. Using these methods, both high fat- and low fat–free mass have been found to be independent predictors of mortality (28–30).

Dual-energy x-ray absorptiometry (DXA) offers more precise in vivo measurements of fat and muscle mass as well as the regional distribution of fat, which may account for discrepant interpretations surrounding BMI and mortality. We therefore used DXA to examine the relationship between mortality and body composition and adiposity distribution and hypothesized that overall adiposity, the distribution of adiposity, and muscle mass might individually affect survival in old age.

Methods

Four thousand community-dwelling men and women aged 65 years or older were invited to attend a health check carried out in the School of Public Health of The Chinese University of Hong Kong between August 2001 and December 2003 by placing recruitment notices in community centers for the elderly participants and housing estates. This project was primarily examining the bone mineral density of older Chinese adults. Talks were also given at these centers explaining the purpose, procedures, and investigations to be carried out. Written informed consents were obtained. Only ethnical Chinese participants were recruited. We excluded those who (a) were unable to walk without assistance of another person; (b) had had a bilateral hip replacement because that would have affected the bone mineral density measurement; (c) were not competent to give informed consent; and (d) had medical conditions, in the judgment of the study physicians, which made it unlikely that they would survive the duration of the study (3 years). The sample was stratified so that approximately 33% were in each of the age groups: 65–69, 70–74, and 75 years and older. The study was approved by the Clinical Research Ethics Committee of the Chinese University of Hong Kong.

Stature and body weight were measured to calculate BMI (body weight in kilograms divided by the square of stature in meters). The participants were asked to stand upright without shoes and look straight ahead, and their standing heights were measured by the Holtain Harpenden stadiometer (Holtain Ltd, Crosswell, UK). Body weight was measured, with the participants wearing a light gown, by the Physician Balance Beam Scale (Health-O-Meter, Inc., Alsip, IL). Waist circumferences (the narrowest circumference around the trunk between the rib cage and the pelvis) and hip circumferences (the maximum circumference around the buttock posteriorly and the pubis symphysis anteriorly) were measured with a flexible measuring tape.

We measured body composition: total body muscle mass, total body fat mass, and truncal fat mass by DXA using a Hologic QDR 2000 densitometer (Hologic, Waltham, WA; Hologic Delphi, software version 11.2). In measuring the trunk fat, a line of delineation was drawn between the head of the humerus and the glenoid fossa of the scapula to separate the upper limb from the trunk, and the leg consisted of the parts of the body between the inferior border of the ischial tuberosity to the most distal tip of the toes. The maximum coefficient of variation for fat mass and muscle mass is 1.47% and 0.84%, respectively. Calibration with a Hologic body composition step phantom was performed at least three times per week.

Mortality status was ascertained annually by registration search in the Death Registry of the Hong Kong Government on the March 31 of each year. The last search was undertaken on March 31, 2008.

Statistical Methods

All body composition measurements were corrected to body built in the same way as the calculation of BMI. General adiposity or body fat index (BFI) was expressed by total body fat mass in kilograms divided by the square of stature in meters. Likewise, body muscle mass index (BMMI) was represented by the total body muscle mass in kilograms divided by the square of stature in meters. We used relative truncal fat (RTF) (31,32) to indicate central adiposity, which was the ratio between truncal fat mass to that of total body fat mass.

Men and women were analyzed separately. Mortality rate was expressed in number of deaths per 1000 person-years and plotted against BMI, BFI, RFT, and waist–hip ratio (WHR) quintiles to demonstrate the shape of the mortality curve.

The relationship between mortality and body composition was tested by Cox-regression analysis. Three multivariate analyses were undertaken. Model 1 included age, BFI, and RTF. Model 2 included age, BFI, and BMMI. Model 3 included age, RTF, and WHR. All body composition variables (in quintiles) that related to mortality in a J-shaped pattern were taken as categorical and as ordinal if either an increasing or a decreasing trend was observed. All tests were two-sided, and p values less than .05 were considered statistically significant. Statistical analysis was conducted using SPSS version 15.0.

Results

A total of 2000 men and 2000 women were assessed at baseline. The mean age of the cohort was 72.5 ± 5.2 (*SD*) years (in men, 72.3 ± 5.0 years; in women, 72.5 ± 5.3 years); 242 (12.1%) men and 78 (3.9%) women died after a median follow-up period of 63.3 months.

In men, mortality decreased progressively across BMI (except the 2nd BMI quintile), BFI, and BMMI quintiles until the 5th at which the beneficial effect was slightly attenuated (Table 1). The hazard ratio (HR) decreased consistently by 0.85 ($p < .005$), 0.86 ($p < .005$), and 0.86

Table 1. Crude Mortality and Comparison Between Individual Quintile to the Quintile with the Lowest Mortality by Cox-Regression

	BMI	BFI	BMMI	RTF	WHR
	Deaths per 1,000 person-years, HR (95% CI)				
Men					
1st quintile	34.65, 1.96 (1.33–2.90)†	29.52, 1.76 (1.17–2.64)†	35.01, 2.23 (1.49–3.34)‡	31.42, 2.51 (1.62–3.91)‡	25.84, 1.43 (0.95–2.14)
2nd quintile	19.49, 1.09 (0.71–1.69)	25.52, 1.51 (0.99–2.28)	20.69, 1.30 (0.83–2.02)	21.67, 1.72 (1.07–2.74)*	18.40, 1.01 (0.65–1.57)
3rd quintile	21.46, 1.21 (0.79–1.85)	22.12, 1.31 (0.85–2.01)	18.90, 1.18 (0.75–1.85)	23.60, 1.88 (1.18–2.98)†	18.15, 1.00
4th quintile	17.87, 1.00	16.95, 1.00	15.93, 1.00	12.67, 1.00	21.44, 1.18 (0.77–1.81)
5th quintile	18.46, 1.04 (0.67–1.61)	17.48, 1.03 (0.65–1.63)	21.42, 1.34 (0.86–2.09)	22.45, 1.76 (1.11–2.81)*	27.70, 1.53 (1.02–2.29)*
Women					
1st quintile	8.67, 1.55 (0.72–3.30)	8.69, 1.32 (0.64–2.72)	8.07, 1.24 (0.60–2.58)	9.82, 1.76 (0.84–3.71)	7.98, 1.98 (0.85–4.63)
2nd quintile	5.62, 1.00	6.6, 1.00	6.67, 1.03 (0.48–2.21)	5.59, 1.00	4.01, 1.00
3rd quintile	8.67, 1.54 (0.72–3.29)	8.22, 1.26 (0.60–2.61)	6.56, 1.00	10.22, 1.83 (0.88–3.83)	6.25, 1.57 (0.64–3.83)
4th quintile	8.11, 1.44 (0.67–3.10)	7.07, 1.07 (0.50–2.28)	10.21, 1.56 (0.78–3.14)	7.63, 1.37 (0.63–2.98)	10.64, 2.66 (1.18–6.01)*
5th quintile	8.67, 1.54 (0.72–3.29)	9.18, 1.39 (0.68–2.84)	8.23, 1.25 (0.60–2.60)	6.53, 1.17 (0.53–2.62)	10.95, 2.76 (1.22–6.23)*

Notes: In men, BMI quintiles: 1st, <21.01; 2nd, 21.02–22.74; 3rd, 22.75–24.16; 4th, 24.17–25.91; 5th, >25.91. In men, WHR quintiles: 1st, <0.87; 2nd, 0.88–0.91; 3rd, 0.92–0.94; 4th, 0.95–0.97; 5th, >0.98. In women, BMI quintiles: 1st, <21.08; 2nd, 21.09–22.95; 3rd, 22.96–24.58; 4th, 24.59–26.66; 5th, >26.67. In women, WHR quintiles: 1st, <0.85; 2nd, 0.86–0.89; 3rd, 0.90–0.94; 4th, 0.95–0.99; 5th, >1.00. BFI = body fat index; BMI = body mass index; BMMI = body muscle mass index; CI = confidence interval; HR = hazard ratio; RTF = relative truncal fat; WHR = waist–hip ratio.

*$p < .05$, †$p < .01$, ‡$p < .001$.

($p < .005$) per every quintile increase in BMI, BFI, and BMMI, respectively. However, a possible J-shaped relationship was observed across RTF and WHR quintiles, both measures of central adiposity. The minimums of the mortality curve were at the 3rd and 4th quintiles of WHR and RTF, respectively (Figure 1 and Table 1).

When central adiposity (RTF) was tested with general adiposity (BFI) with adjustment for age, BFI became

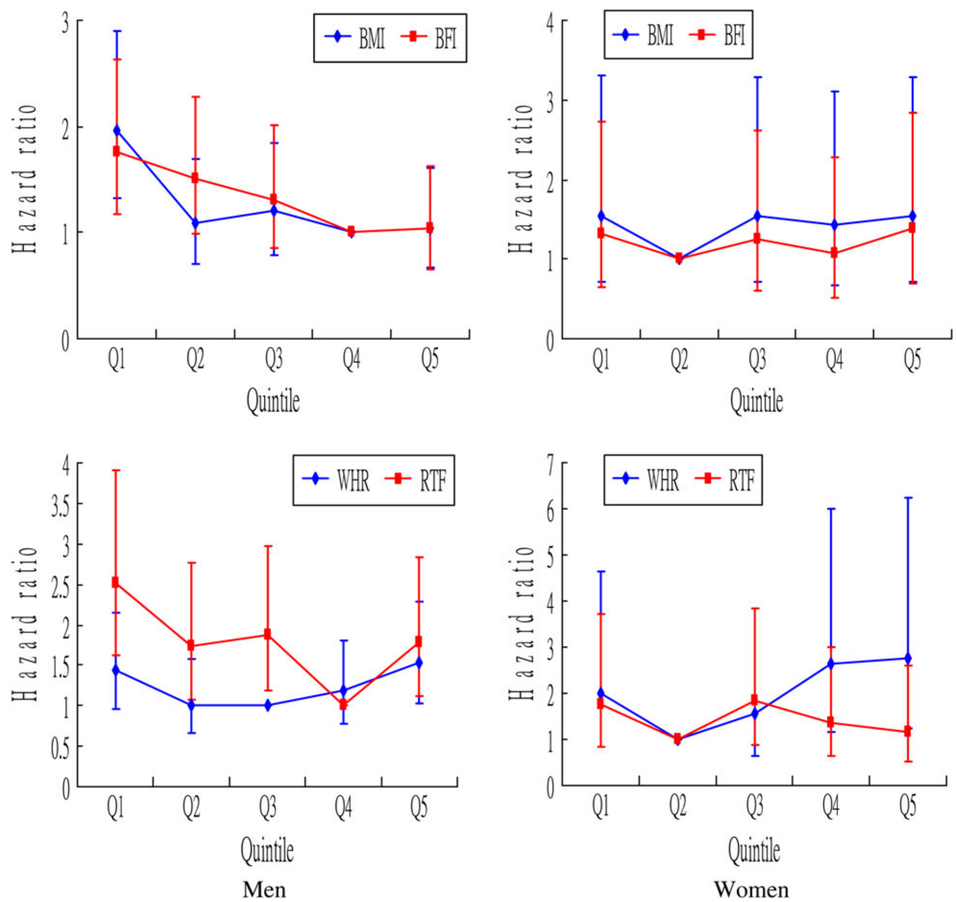

Figure 1. Relationship between body composition and mortality. *Notes:* BFI = body fat index; BMI = body mass index; RTF = relative truncal fat; WHR = waist–hip ratio.

marginally insignificant ($p = 0.06$) while RTF maintained a J-shaped relationship. Comparing to the 4th quintile, which was the minimum, mortality was higher in the 1st, 3rd, and 5th RTF quintiles (Model 1, Table 2). When total body fat mass, represented by BFI and total body muscle mass, by BMMI, were tested together with adjustment for age, increasing adiposity rather than muscles favored survival (Model 2, Table 2). RTF maintained a J-shaped mortality relationship when placed into the same model with another measure of central obesity, the WHR (Model 3, Table 2).

In women, no corresponding relationship was observed except for WHR, in which a J-shaped curve was observed. The mortality in the highest two quintiles was more than 2.5 times higher than that of the 2nd quintile (the lowest mortality quintile; Table 1 and Figure 1). The relationship between BMI and other adiposity measurements was tabulated in Table 3.

Discussion

In older men, mortality was related to body composition, whereas in women, no corresponding relationship could be revealed except in WHR. The insufficient number of mortality events in women (242 deaths in men vs 78 deaths in women) may account for the absence of relationship. That remains to be elucidated by future studies with longer follow-up period.

Our observation suggests that overweight is favorable for survival in older men. The 4th BMI quintile (24.16–25.91), which had the lowest mortality, actually corresponded to the overweight to obesity category according to the World Health Organization Asia-Pacific cutoff (overweight BMI > 23 and obesity BMI > 25) (33). The beneficial effect of high BMI for survival was only slightly attenuated at the highest quintile (5th vs 4th quintile: 18.4 vs 17.8 per 1,000 person-years and statistically insignificant, *p* .866). On the other side, the mortality in the lowest weight quintile (BMI < 21.01) was 1.96 times that of the 4th BMI quintile (Table 1). Our results are consistent with several previous reports in other populations, which concluded that underweight was hazardous and overweight advantageous (7,9–12).

Our cohort, although of a fair size, did not include too many very obese participants: the highest BMI quintile was 25.91 and higher. It is uncertain whether the inclusion of more obese participants might alter the shape of the mortality curve. Nevertheless, sufficient number of our participants already belonged to the obesity range according to Asia-Pacific cutoff for obesity; hence, this mortality pattern could be considered representative of the Chinese population (34).

The DXA measurements in the present study offered direct assessment of body fat mass and enabled scrutiny of the relationship between mortality and total body adiposity. The analysis of BFI, however, resulted in a pattern similar to that of BMI, with the higher adiposity being protective rather than hazardous. In fact, the reduction in HR for every quintile increment was nearly identical among BMI (HR = 0.85, $p < .005$), BFI (HR = 0.86, $p < .005$), and

Table 2. Multivariate Analysis of General Adiposity, Central Adiposity, and Body Muscle Mass in Men

	Unit	HR (95% CI)	*p* value
Model 1: general and central adiposity			
BFI	Quintile	0.90 (0.81–1.00)	.062
RTF			
1st quintile	—	1.87 (1.15–3.01)	.010*
2nd quintile	—	1.45 (0.90–2.33)	.120
3rd quintile	—	1.71 (1.07–2.72)	.022*
4th quintile	—	1.00	—
5th quintile	—	1.84 (1.15–2.93)	.010*
Model 2: fat and muscle mass			
BFI	Quintile	0.87 (0.79–0.97)	.015*
BMMI	Quintile	1.00 (0.89–1.11)	.977
Model 3: central adiposity—RTF and WHR			
RTF			
1st quintile	—	2.51 (1.57–4.01)	.0001*
2nd quintile	—	1.65 (1.03–2.66)	.036*
3rd quintile	—	1.77 (1.11–2.82)	.014*
4th quintile	—	1.00	—
5th quintile	—	1.66 (1.04–2.66)	.029*
WHR			
1st quintile	—	0.97 (0.63–1.49)	.896
2nd quintile	—	0.89 (0.57–1.39)	.632
3rd quintile	—	1.00	—
4th quintile	—	1.18 (0.77–1.80)	.444
5th quintile	—	1.44 (0.96–2.16)	.078

Notes: Multivariate analysis was undertaken using Cox-regression model. BFI and BMMI were taken as ordinal; RTF and WHR were taken as categorical. Model 1 covariates: age, BFI, RTF. Model 2 covariates: age, BFI, BMMI. Model 3 covariates: age, RTF, WHR. BFI = body fat index; BMMI = body muscle mass index; CI = confidence interval; HR = hazard ratio; RTF = relative truncal fat; WHR = waist–hip ratio.

*$p < .05$.

Table 3. Relationship Between BMI and Other Body Composition Measurements

	BFI	BMMI	RTF	WHR
	Mean (*SD*)			
Men				
BMI				
1st quintile	3.59 (1.06)	14.60 (1.05)	0.502 (0.060)	0.87 (0.06)
2nd quintile	5.02 (0.79)	15.81 (0.76)	0.555 (0.052)	0.91 (0.06)
3rd quintile	5.78 (0.82)	16.54 (0.79)	0.570 (0.042)	0.93 (0.05)
4th quintile	6.50 (0.86)	17.38 (0.82)	0.581 (0.041)	0.94 (0.05)
5th quintile	7.84 (1.19)	18.73 (1.21)	0.584 (0.039)	0.96 (0.06)
p Value for trend	<.0001	<.0001	<.0001	<.0001
Women				
BMI				
1st quintile	5.51 (1.22)	13.17 (0.92)	0.491 (0.066)	0.88 (0.08)
2nd quintile	7.35 (0.79)	14.12 (0.80)	0.519 (0.051)	0.91 (0.07)
3rd quintile	8.27 (0.88)	14.74 (0.90)	0.526 (0.050)	0.93 (0.08)
4th quintile	9.40 (0.86)	15.47 (0.88)	0.528 (0.049)	0.94 (0.08)
5th quintile	11.38 (1.51)	16.72 (1.34)	0.522 (0.043)	0.95 (0.08)
p Value for trend	<.0001	<.0001	<.0001	<.0001

Notes: BFI = body fat index; BMI = body mass index; BMMI = body muscle mass index; RTF = relative truncal fat; WHR = waist–hip ratio.

BMMI (HR = 0.86, $p < .005$), which may suggest that overweight is advantageous for survival through increasing adiposity and muscle mass.

Nevertheless, the apparent U-shaped relationship between BMI and mortality has been explained by the opposing effect of fat and muscle mass, with increasing obesity being harmful but increasing muscle mass beneficial for survival (15,28–30). Following this hypothesis, we have examined the independent effect of fat and muscle mass on mortality by placing them in the same multivariate model (Model 2, Table 2). Although a higher muscle mass, as expressed by the BMMI favored survival (Table 1), it became insignificant after adjusting for BFI, which represented body fatness (Model 2, Table 2). Yet high body fat (BFI) persisted to favor survival after adjustment for muscle mass (BMMI), which was in contrary to previous works (28–30), which reported that increasing adiposity was hazardous. Our data, however, suggested that increasing adiposity, independent of muscle mass, might be beneficial in older adults, at least in the range of adiposity that we examined. We therefore have to postulate that adiposity, and not muscle mass, favors survival in older men and that we did not observe any opposing effect of muscle loss and fat gain on mortality, as both favored survival in the same direction (Table 1).

Reaffirming the unfavorable effect of central obesity, we have also observed that the mortality in the highest WHR quintile (WHR > 0.98) was 1.58 times that of the middle quintile (WHR, 0.92–0.94), which had the lowest mortality. Such WHR values, nevertheless, were already considered central obesity. Again, similar to general adiposity and overweight, older men were likely to be more resistive to the hazard of central obesity or might even benefit from mild-grade central obesity.

The other measure of central obesity, RTF, on the other hand, demonstrated a very robust J-shaped relationship with mortality (Table 1). The central accumulation of fat appeared again to be advantageous for survival until it reached the 4th quintile, after which it became disadvantageous. The lowest mortality RTF quintile corresponded to a mean WHR of 0.94. Such WHR again is considered central obesity, reinforcing the notion that older men may benefit from mild-grade central obesity.

Based on the favorable effect of general adiposity on mortality that we had found, we postulated that the total amount of fat (general adiposity) and the distribution of fat (central adiposity) might have opposing effects on mortality, with the former being favorable and the latter unfavorable. On examining the relative importance of truncal versus general adiposity in relation to mortality, we found that the protective effect of general adiposity (BFI) ceased and the J-shaped relationship between truncal adiposity and mortality remained robust (Model 1, Table 2). This analysis suggested that it was the distribution of adiposity rather than total adiposity that determined mortality and that survival would be affected adversely by having either too little or too much central fat. It is uncertain how this observation contributes to the U-shaped relationship between BMI and mortality.

We have used RTF and WHR separately to represent central obesity. RTF, being a more sophisticated measurement, was able to discriminate mortality difference among all quintiles, whereas WHR, the other parameter of central obesity, could only demonstrate excess risk in the highest quintile in men (Table 1 and Model 3, Table 2). Albeit this finding should not underscore the validity of WHR, which is a simpler and more readily available measurement.

Our study has several limitations. The participants were healthy community dwellers who were free from any disability, and the relationship between body composition and mortality may differ in the disabled or frail (13). Our results, therefore, cannot be generalized to the disabled or institutionalized populations. DXA-measured adiposity, although is reliable, may not relate to mortality as closely as computerized tomography-measured or DXA-derived visceral adiposity. The adiposity mortality curve could be more accurately described if the latter two modalities were used. Lastly, the effect of central adiposity may vary across different ethnic groups. Our participants were all ethnic Chinese, and as there may be ethnic differences in fat distribution, our observations may not be extrapolated to the Western population.

Conclusions

Older men were resistive to the hazard of overweight and adiposity, and mild-grade overweight, obesity, and even central obesity might be protective. This may bear significant implication on the recommended cutoff values for BMI and WHR in the older population. In addition, favorable survival was related more to increasing adiposity than muscle mass. No corresponding relationship, except in WHR, was observed in older women.

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