

Abdominal Adiposity and Mortality in Chinese Women

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Background: Increased abdominal adiposity has been linked to an increase in mortality in populations where many are overweight or obese; it is unclear whether the same is true in relatively lean populations.

Methods: We examined the association between waist-hip ratio and mortality in the Shanghai Women's Health Study, a population-based, prospective cohort study of Chinese women aged 40 to 70 years enrolled from December 28, 1996, through May 23, 2000, 95% of whom had a body mass index (calculated as weight in kilograms divided by height in meters squared) of less than 30.0. Included in this analysis were 72 773 nonsmoking women who had anthropometrics taken by trained interviewers at enrollment and who were followed up through December 31, 2004. Deaths were ascertained by biennial home visits and linkage with the vital statistics registry.

Results: During a mean follow-up of 5.7 years, 1456 deaths occurred. The waist-hip ratio was positively and

significantly associated with deaths from all causes, cardiovascular disease, and diabetes ($P < .01$ for trend). A less significant positive association was found for death from cancer. After adjustment for body mass index and other potential confounders, the relative risks of total mortality were 1 (reference group), 1.28 (95% confidence interval [CI], 1.04-1.58), 1.40 (95% CI, 1.14-1.72), 1.54 (95% CI, 1.26-1.88), and 1.95 (95% CI, 1.60-2.38) across the lowest to the highest waist-hip ratio quintiles. The positive association appeared to be more evident in women with a lower body mass index. The relative risks of total mortality comparing the extreme waist-hip ratio quintiles were 2.36 (95% CI, 1.71-3.27), 1.60 (95% CI, 1.10-2.34), and 1.46 (95% CI, 0.97-2.20) for women with a body mass index of less than 22.3, 22.3 to 25.1, and 25.2 or greater, respectively.

Conclusion: Abdominal adiposity independently predicts mortality risk, particularly for nonobese women.

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OBESITY HAS BEEN CLEARLY shown to increase mortality. However, it remains controversial whether the relationship between body weight and mortality can be best described as linear, J-shaped, or U-shaped.¹⁻⁶ Many methodologic limitations may have contributed to the conflicting results and led to an underestimate of the impact of obesity on mortality.^{5,7,8} Among them is the reduced validity of body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) as a measure of adiposity in the elderly, who tend to lose lean body mass and have a shift of body fat from peripheral to central sites with a concomitant increase in waist-hip ratio (WHR) (waist circumference divided by hip circumference) at the same level of BMI.⁷⁻⁹ Growing evidence suggests that the adverse effects of obesity are closely re-

lated to the distribution of body fat and that central (intra-abdominal) obesity is particularly detrimental.¹⁰ Measures of central adiposity, such as WHR, have been shown to be a better marker than BMI in predicting certain obesity-related health risks.^{6,11} A recent large-scale case-control

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study involving 27 098 participants from 52 countries reported that WHR, in comparison with BMI and other anthropometric measures, best predicted the risk of myocardial infarction across all age and ethnic groups.¹¹ Waist-hip ratio was also found in a few cohort studies to be a significant predictor of mortality^{1,6,12,13} and potentially better than BMI at predicting mortality risk.^{6,12,13} Investigations of body fat distribution and mortality to date have

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been conducted mainly in Western populations, where overweight and obesity are prevalent, and most have used self-reported waist and hip circumferences. The accuracy of these self-reported measurements has been shown to decrease with increasing body size, and the measurement errors are further compounded in their ratio.¹⁴ Studies in relatively lean populations and, more importantly, with directly measured anthropometric variables are needed to further quantify the effect of body fat distribution on mortality.

We evaluated the association between WHR and the risk of death from all causes and from specific causes in a large cohort of middle-aged and older Chinese women with an average BMI of 24.0 at the time of enrollment in the Shanghai Women's Health Study (SWHS).

METHODS

The SWHS is a population-based, prospective cohort study of adult Chinese women. The study was approved by the relevant institutional review boards for human research in China and the United States. The design and methods of the SWHS have been described in detail elsewhere.¹⁵ Briefly, from December 28, 1996, through May 23, 2000, the study recruited 74 942 women aged 40 to 70 years from selected urban communities of Shanghai, with a participation rate of 92.7%. Recruitment activities, including a detailed baseline survey and anthropometric measurements, were carried out at participants' homes by trained interviewers. Structured questionnaires were used during the survey to obtain information on demographics, diet and lifestyle habits, menstrual and reproductive history, hormone use, and medical history. The validity and reproducibility of the food frequency and physical activity questionnaires used in the SWHS have been demonstrated previously.^{16,17}

ANTHROPOMETRY

Participants were asked to wear light indoor clothing when they were measured for weight, height, and circumferences of the waist and hips by trained interviewers. The measurement was conducted uniformly according to a standard protocol. Waist circumference was measured at 2.5 cm above the umbilicus and hip circumference at the level of maximum width of the buttocks with the subject in a standing position. Circumferences and heights were measured to the nearest 0.1 cm. Weight was measured to the nearest 0.1 kg using a digital weight scale that was calibrated every 6 months. All measurements were taken twice. A tolerance limit of 1 kg was set for weight measurement and 1 cm for height and circumference measurements. A third measurement was taken if the difference of the first 2 measurements was greater than the tolerance limit. Using the average of the 2 closest measurements, WHR and BMI were then calculated for the analysis.

END POINT ASCERTAINMENT

Participants were followed up by means of biennial in-person contact and record linkage to the Shanghai Cancer Registry and the Shanghai Vital Statistics Registry. For the present analysis, we used outcome data through December 31, 2004. As of this date, follow-up for the vital status of participants was more than 99% complete. The underlying cause of death was determined on the basis of death certificates and coded according to the codes of the *International Classification of Diseases, Ninth Revision (ICD-9)*. The primary end point for the present analysis

was death from all causes that occurred after the baseline survey, with follow-up through 2004. In addition, we examined deaths from cancer (*ICD-9* codes 140-208), cardiovascular disease (CVD) (*ICD-9* codes 390-459), stroke (*ICD-9* codes 430-438), diabetes mellitus (*ICD-9* code 250), and all other causes.

STATISTICAL ANALYSIS

Of the 74 942 SWHS participants, only 2113 (2.8%) had ever smoked cigarettes, and they were excluded from the present analysis to avoid confounding by cigarette use. We also excluded those women who were pregnant ($n=10$), were lost to follow-up ($n=10$), or had missing data for anthropometric measurements ($n=39$). After these exclusions (not mutually exclusive), 72 773 women remained for the analysis. Study participants were classified into 5 categories according to quintiles of WHR, with the lowest quintile serving as the reference category. Cox proportional hazards models were used, with age as the time scale to estimate relative risks (RRs) of death associated with WHR and their 95% confidence intervals (CIs) and to adjust for potential confounders.¹⁸ Entry time was defined as age at enrollment, and exit time was defined as age at death or censoring. Covariates included birth calendar year (7 categories); education level (4 categories); occupation (3 categories); family income (4 categories); menopausal status (premenopausal or postmenopausal); use of hormone therapy (yes or no); amount of regular exercise (hours per week, 4 categories); alcohol consumption (yes or no; using finer categories did not change the estimates because few women ever drank); intake of saturated fat, vegetables, and fruits (continuous); and BMI (linear and quadratic terms; both low and high BMI values were associated with increased mortality). Tests for linear trend in risk across WHR categories were performed by using the median value for each WHR category and modeling them as continuous variables. We also conducted analyses stratified by BMI level, age, menopausal status, and amount of exercise to further examine the independent effect of WHR and to evaluate possible effect modification. In addition, we used restricted cubic spline regression, a flexible statistical technique, to evaluate the association between WHR and mortality.¹⁹ Four knots were used for the analysis of total mortality and placed at the 5th, 35th, 65th, and 95th percentiles of the WHR distribution among the cohort. As relatively fewer deaths from specific causes occurred, 3 knots (placed at the 5th, 50th, and 95th percentiles) were used for the analysis of cause-specific mortality. To make the graph more stable and meaningful, those with a WHR below the 1st percentile or above the 99th percentile were deleted from the data set used to fit the spline model. Finally, we examined waist circumference and the ratio of waist circumference to height (waist-height ratio) and calculated c statistics to compare their predictive values. Statistical analyses were performed using SAS statistical software (version 9.1; SAS Institute Inc, Cary, NC). All statistical tests were based on 2-sided probability.

RESULTS

The mean age of the study population was 52 years at enrollment, and the mean WHR was 0.81. The prevalence of overweight or obesity ($\text{BMI} \geq 25.0$) was 35.2%, and the prevalence of obesity ($\text{BMI} \geq 30.0$) was 5.0%. Very few women ever drank alcoholic beverages or used hormonal therapy. **Table 1** presents the age-adjusted baseline characteristics of study participants according to quintiles of WHR. Compared with those with a lower WHR, women with a higher WHR tended to be older, less edu-

Table 1. Age-Adjusted Baseline Characteristics of the Study Population According to Quintiles of WHR*

Characteristic	All Subjects (n = 72 773)	Quintile of WHR				
		<0.77 (n = 14 558)	0.77-0.78 (n = 14 103)	0.79-0.81 (n = 14 981)	0.82-0.84 (n = 14 553)	≥0.85 (n = 14 578)
Age, mean, y	52.0	48.8	49.9	51.5	53.1	56.3
Postmenopausal	48.9	48.5	48.7	48.9	49.0	49.7
Hormone therapy	2.1	2.5	2.5	2.2	1.9	1.9
Education level						
≤Elementary school	20.6	16.1	17.6	19.3	21.3	25.3
Middle school	37.3	34.8	36.5	37.4	39.7	40.9
High school	28.3	31.3	30.1	29.1	26.5	24.1
≥College	13.9	17.8	15.8	14.2	12.5	9.7
Occupation						
Professionals, technicians, administrators	29.2	34.2	31.7	30.1	27.2	23.5
Clerical and service workers	20.7	19.4	20.0	20.9	21.0	22.3
Manufacturing and agricultural workers	50.1	46.4	48.3	49.0	51.7	54.2
Annual family income, yuan						
<10 000	15.8	14.4	14.5	15.5	16.3	18.2
10 000 to 19 999	38.3	37.7	37.8	38.0	39.0	39.7
20 000 to 29 999	28.3	29.5	29.1	28.3	27.7	26.6
≥30 000	17.7	18.4	18.6	18.1	17.0	15.5
Regular exercise, mean, h/wk	2.0	2.1	2.1	2.0	2.0	1.9
Ever drank alcohol	1.9	1.7	1.8	2.0	2.1	1.9
Daily intake, mean, g						
Saturated fat	9.4	9.7	9.6	9.4	9.3	9.1
Vegetables	299.0	297.7	298.8	299.0	300.0	299.2
Fruits	272.7	272.5	273.2	273.9	273.7	270.4
Mean BMI	24.0	21.9	23.1	24.1	24.9	26.1

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); WHR, waist-hip ratio (waist circumference divided by hip circumference).

*Adjusted for age in 5-year categories; all tests for trend were significant ($P < .05$) except for vegetable and fruit intakes. Unless otherwise indicated, data are expressed as percentage of subjects.

cated, less likely to have a professional or administrative position, and more likely to have low family income. In addition, they exercised a little less and consumed less saturated fat. Vegetable or fruit intake did not appear to differ by WHR. Waist-hip ratio was moderately correlated with BMI (Pearson correlation, $r = 0.46$), whereas the correlation between waist circumference and BMI was high ($r = 0.84$).

During a mean follow-up of 5.7 years, 1456 deaths were documented, including 732 from cancer (17.8% lung cancer, 12.4% colorectal cancer, 11.8% stomach cancer, 10.3% liver cancer, 8.5% breast cancer, and 39.3% other cancers), 357 from CVD (57.4% stroke, 20.2% coronary heart disease, and 22.4% other circulatory diseases), 99 from diabetes mellitus, and 268 from other causes. In age-adjusted analyses, BMI showed U-shaped associations with total and CVD mortality and no association with cancer mortality (data not shown). Compared with the third BMI quintile (22.9-24.5), the RRs of total mortality for the first (<21.1) and fifth (≥26.7) BMI quintiles were 1.46 (95% CI, 1.23-1.73) and 1.20 (95% CI, 1.02-1.41), respectively. The corresponding RRs for CVD mortality were 2.22 (95% CI, 1.51-3.25) and 2.06 (95% CI, 1.44-2.93), respectively. Further adjustment for sociodemographic and lifestyle factors did not markedly alter the results.

Table 2 summarizes the RRs and 95% CIs of total and cause-specific mortality according to WHR quintiles. Waist-hip ratio was positively and significantly as-

sociated with risk of death from all causes as well as from CVD, stroke, and diabetes in a dose-response fashion ($P < .01$ for trend). A less significant positive association was found for death from cancer. After adjustment for BMI and other potential confounders, women in the highest WHR quintile compared with those in the lowest quintile had an RR of 1.95 (95% CI, 1.60-2.38) for death from all causes, 2.74 (95% CI, 1.73-4.32) for death from CVD, 2.62 (95% CI, 1.40-4.89) for death from stroke, and 1.31 (95% CI, 1.00-1.71) for death from cancer. Additional adjustment for history of hypertension and diabetes (potential biological mediators) attenuated the RR for total mortality to 1.79 (95% CI, 1.47-2.19), CVD-related mortality to 2.41 (95% CI, 1.52-3.81), and stroke-related mortality to 2.23 (95% CI, 1.19-4.18), but did not change the RR estimate for cancer-related mortality.

The **Figure** shows the shape of the dose-response relation between all-cause and cause-specific mortality and WHR on a continuous basis. Overall, as WHR increased, so did the adjusted RRs of mortality. When WHR was analyzed as a continuous variable, for each 0.05 (1-SD) increase in WHR, the risk for total mortality increased by 18% (RR, 1.18; 95% CI, 1.12-1.25).

In further stratified analyses (**Table 3**), the positive association between WHR and total mortality persisted across all strata defined by BMI level, menopausal status, age, or amount of regular exercise. The magnitude of the association appeared to be greater in women with

Table 2. Relative Risks (RRs) of Deaths From All Causes and From Specific Causes According to Quintiles of WHR

Causes of Deaths	Quintile of WHR					P Value for Trend
	<0.77 (n = 14 558)	0.77-0.78 (n = 14 103)	0.79-0.81 (n = 14 981)	0.82-0.84 (n = 14 553)	≥0.85 (n = 14 578)	
All causes						
No. of deaths	160	201	258	316	521	
Crude RR (95% CI)*	1 [Reference]	1.15 (0.93-1.41)	1.18 (0.97-1.44)	1.29 (1.06-1.56)	1.61 (1.35-1.93)	<.001
Multivariate RR (95% CI)†	1 [Reference]	1.28 (1.04-1.58)	1.40 (1.14-1.72)	1.54 (1.26-1.88)	1.95 (1.60-2.38)	<.001
Cancer						
No. of deaths	101	115	134	162	220	
Crude RR (95% CI)*	1 [Reference]	1.05 (0.81-1.37)	1.00 (0.77-1.30)	1.09 (0.85-1.40)	1.17 (0.92-1.49)	.15
Multivariate RR (95% CI)†	1 [Reference]	1.12 (0.85-1.47)	1.10 (0.84-1.44)	1.21 (0.93-1.58)	1.31 (1.00-1.71)	.04
Noncancer causes						
No. of deaths	59	86	124	154	301	
Crude RR (95% CI)*	1 [Reference]	1.31 (0.94-1.83)	1.50 (1.10-2.04)	1.62 (1.20-2.19)	2.32 (1.75-3.09)	<.001
Multivariate RR (95% CI)†	1 [Reference]	1.54 (1.10-2.16)	1.91 (1.39-2.63)	2.10 (1.53-2.88)	3.04 (2.24-4.14)	<.001
CVD						
No. of deaths	26	40	59	87	145	
Crude RR (95% CI)*	1 [Reference]	1.37 (0.84-2.25)	1.59 (1.00-2.52)	2.02 (1.30-3.14)	2.45 (1.60-3.74)	<.001
Multivariate RR (95% CI)†	1 [Reference]	1.52 (0.92-2.50)	1.84 (1.15-2.96)	2.31 (1.46-3.66)	2.74 (1.73-4.32)	<.001
Stroke						
No. of deaths	13	18	33	50	91	
Crude RR (95% CI)*	1 [Reference]	1.23 (0.60-2.51)	1.76 (0.93-3.36)	2.32 (1.26-4.29)	3.09 (1.71-5.56)	<.001
Multivariate RR (95% CI)†	1 [Reference]	1.21 (0.59-2.48)	1.69 (0.87-3.26)	2.11 (1.12-3.99)	2.62 (1.40-4.89)	<.001
Diabetes mellitus						
No. of deaths	7	7	13	25	47	
Crude RR (95% CI)*	1 [Reference]	0.86 (0.30-2.46)	1.21 (0.48-3.04)	1.93 (0.83-4.47)	2.45 (1.10-5.48)	<.001
Multivariate RR (95% CI)†	1 [Reference]	1.06 (0.37-3.06)	1.62 (0.62-4.21)	2.60 (1.07-6.34)	3.23 (1.35-7.75)	<.001
Other causes						
No. of deaths	26	39	52	42	109	
Crude RR (95% CI)*	1 [Reference]	1.38 (0.84-2.27)	1.50 (0.94-2.41)	1.08 (0.66-1.77)	2.15 (1.39-3.34)	<.001
Multivariate RR (95% CI)†	1 [Reference]	1.72 (1.04-2.84)	2.09 (1.28-3.40)	1.58 (0.94-2.65)	3.35 (2.08-5.42)	<.001

Abbreviations: CI, confidence interval; CVD, cardiovascular disease; WHR, waist-hip ratio (waist circumference divided by hip circumference).

*The crude RR is estimated from Cox model using age as the time scale (model 1).

†Model 1 is further stratified on birth year and adjusted for education; occupation; annual family income; menopausal status; hormone therapy; amount of regular exercise; alcohol consumption; intakes of saturated fat, vegetables, and fruits; body mass index; and the square of body mass index (model 2).

lower BMIs. The multivariate RRs of total mortality comparing the extreme WHR quintiles were 2.36 (95% CI, 1.71-3.27), 1.60 (95% CI, 1.10-2.34), and 1.46 (95% CI, 0.97-2.20) for women with BMIs of less than 22.3, 22.3 to 25.1, and 25.2 or greater, respectively. Results of tests for multiplicative interaction, however, were not significant. There was no indication of effect modification by menopausal status, age, or amount of regular exercise.

We also examined waist and hip circumferences individually in relation to mortality (data not shown). When an adjustment was not made for BMI in multivariate analyses, only mortality from stroke was related to greater waist circumference, with an RR of 2.00 (95% CI, 1.08-3.71) for the highest vs the lowest quintile. After adjustment for BMI, however, positive associations emerged for total mortality (RR, 1.95; 95% CI, 1.46-2.60) and death from CVD (RR, 1.68; 95% CI, 0.94-3.01), diabetes (RR, 6.37; 95% CI, 2.00-20.33), cancer (RR, 1.61; 95% CI, 1.07-2.42), and other causes (RR, 2.22; 95% CI, 1.15-4.27) ($P < .05$ for trend). Waist circumference appeared to be more predictive for women 50 years or younger than for women older than 50 years, with an RR for total mortality of 4.16 (95% CI, 2.09-8.31) for younger women vs 1.62 (95% CI, 1.17-2.23) for older women ($P = .02$ for in-

teraction). Overall, no independent association was found for hip circumference.

In addition, we assessed RRs of death associated with waist-height ratio, another measure of central obesity. As with waist circumference, positive associations emerged only when the multivariate analyses adjusted for BMI (RR of total mortality comparing the extreme quintiles of waist-height ratio, 2.18; 95% CI, 1.62-2.93). The *c* statistics were 0.773, 0.771, and 0.772 for fully adjusted models of total mortality that included WHR, waist circumference, and waist-height ratio, respectively.

COMMENT

In this large cohort study of Chinese women, a positive monotonic dose-response relationship was observed between WHR and the risk of death. This positive association was independent of BMI and sociodemographic and lifestyle factors. Several features distinguish this study from previous studies. To our knowledge, this is the first cohort study that has evaluated mortality risk associated with body fat distribution in Asian women. Nearly two thirds of the study population had a BMI of less than 25.0, providing a unique opportunity to evalu-

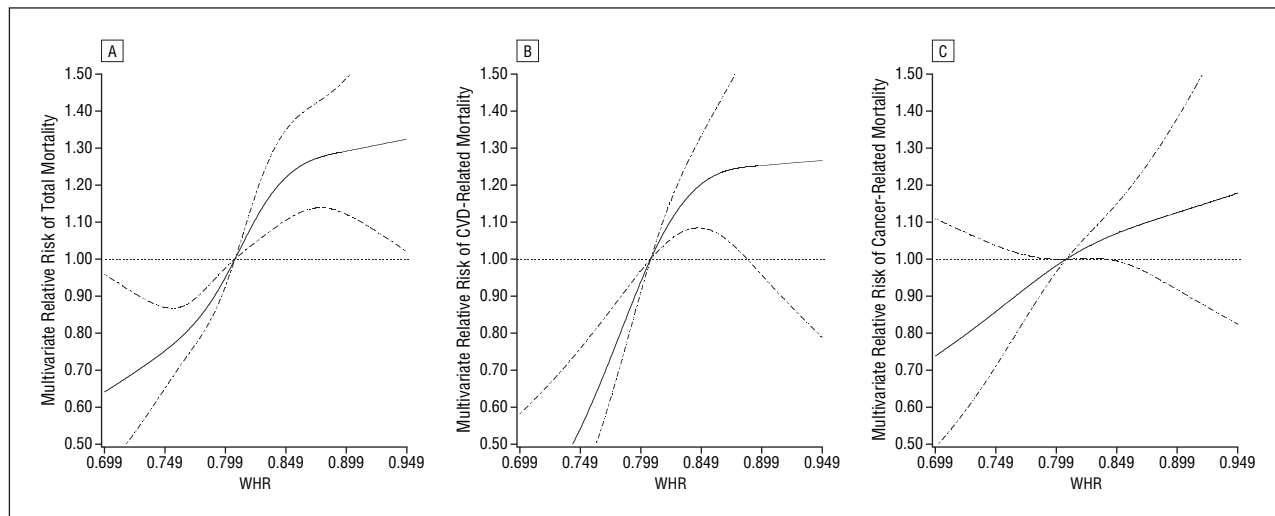


Figure 3. Multivariate relative risks of total mortality (A) and mortality from cardiovascular disease (CVD) (B) and cancer (C) according to waist-hip ratio (WHR). Multivariate relative risks were estimated from a restricted cubic spline Cox regression model. Point estimates are indicated by a solid line and 95% confidence intervals by dashed lines. The median value was treated as the reference point.

ate the influence of body fat distribution on mortality among lean or normal-weight individuals. None of the participants included in the analyses had ever smoked cigarettes and, thus, confounding by smoking was not a concern for this study. All anthropometric variables were measured directly rather than self-reported, which eliminated the possibility of any bias from self-reports. Our finding of a positive relationship between WHR and mortality regardless of BMI underscores the importance of incorporating fat distribution measurement in assessing obesity-related health risk and supports the use of WHR as a measure of choice to improve risk assessment. Given the possibility that a healthy body weight, currently defined as a BMI of 18.5 to 24.9, might provide some false assurance to those who are apparently lean but have a high degree of abdominal adiposity, it is important to raise awareness among those subjects and their health care providers of the deleterious effects of abdominal obesity.

Our results are consistent with those from studies of white women that support an independent contribution of body fat distribution to mortality and the usefulness of WHR as a predictor of mortality.^{1,6,12} In the Nurses' Health Study^{1,20} and the Iowa Women's Health Study,^{6,21} a positive association of WHR with mortality, independent of BMI, was consistently found during short- and longer-term follow-ups. Waist-hip ratio showed a stronger association with mortality from CVD than from cancer in the Iowa Women's Health Study, as it did in our study. Coronary heart disease was the cause that contributed most to the association with CVD-related mortality in the Iowa study, whereas stroke was the predominant cause in our study. Likewise, in a Swedish cohort of middle-aged and older women, WHR was directly related to the risk of all-cause mortality after adjustment for overall body fat.¹² No association, however, was observed between WHR and mortality in a small Dutch cohort of elderly women.²² The WHR also appears to predict mortality risk in men, but the evidence has been limited and inconclusive.^{12,13,22}

Waist circumference has been proposed in recent years to replace WHR as an indicator of abdominal adiposity, because it is simpler to measure and interpret.²³ However, waist circumference is highly correlated not only with visceral fat but also with BMI, and it reflects both general and central adiposity, whereas WHR correlates less strongly with BMI and thus may provide more specific information about regional fat distribution.⁶ In our study and in the Iowa Women's Health Study,^{6,21} WHR appeared to be a more robust predictor of mortality than waist circumference was. The WHR was also found to be a better predictor than waist circumference of coronary heart disease in our previous study of the same cohort²⁴ and in the Nurses' Health Study.²⁵ The analysis with *c* statistics in the present study, however, failed to demonstrate that one measure of abdominal adiposity is superior to another.

It is believed that abdominal obesity directly underlies insulin resistance and systemic inflammation, which, in turn, leads to the metabolic syndrome, CVD, and certain cancers.^{9,10} Expanded abdominal fat deposits provide a major source of increased production of C-reactive protein, tumor necrosis factor α , interleukin 6, plasminogen activator inhibitor 1, angiotensinogen, and vascular endothelial growth factor, all of which contribute importantly to the development and progression of many obesity-related diseases.¹⁰

Limitations of our study need to be considered when interpreting the results. The maximum follow-up time of 7.5 years in our study is relatively short. Although the short interval between anthropometric measurement and event reduces the influence of fluctuations in body weight and fat distribution over time on the results, it raises concern about the effect of baseline illness. We therefore conducted sensitivity analyses excluding deaths that occurred within the first 2 or 3 years of follow-up and excluding subjects with a history of CVD or cancer at baseline and found no material change in the risk estimates for total mortality. Despite having carefully adjusted for a range of potential confounding variables, including so-

Table 3. Multivariate Relative Risks (RRs) of Total Mortality According to WHR, Stratified by BMI, Menopausal Status, Age, or Exercise*

Variable	Quintile of WHR					P Value for Trend
	<0.77	0.77-0.78	0.79-0.81	0.82-0.84	≥0.85	
BMI tertile 1 (<22.3)						
No. of subjects	9081	6244	4396	2953	1559	
No. of deaths	99	90	94	79	70	
Multivariate RR (95% CI)	1 [Reference]	1.42 (1.06-1.90)	1.89 (1.41-2.53)	1.97 (1.44-2.68)	2.36 (1.71-3.27)	<.001
BMI tertile 2 (22.3-25.1)						
No. of subjects	3917	5037	5797	5332	4151	
No. of deaths	36	54	90	98	136	
Multivariate RR (95% CI)	1 [Reference]	1.00 (0.66-1.53)	1.26 (0.85-1.86)	1.24 (0.84-1.82)	1.60 (1.10-2.34)	.002
BMI tertile 3 (≥25.2)						
No. of subjects	1560	2822	4788	6268	8868	
No. of deaths	25	57	74	139	315	
Multivariate RR (95% CI)	1 [Reference]	1.25 (0.78-2.00)	0.89 (0.56-1.39)	1.16 (0.75-1.77)	1.46 (0.97-2.20)	.001
Premenopausal						
No. of subjects	9555	8520	7908	6605	4626	
No. of deaths	40	48	52	40	45	
Multivariate RR (95% CI)	1 [Reference]	1.35 (0.89-2.07)	1.53 (1.00-2.36)	1.32 (0.82-2.10)	1.81 (1.11-2.93)	.04
Postmenopausal						
No. of subjects	5003	5583	7073	7948	9952	
No. of deaths	120	153	206	276	476	
Multivariate RR (95% CI)	1 [Reference]	1.26 (0.99-1.60)	1.35 (1.07-1.71)	1.55 (1.24-1.95)	1.94 (1.55-2.42)	<.001
Age ≤50 y						
No. of subjects	9932	8824	8150	6699	4690	
No. of deaths	48	52	49	43	52	
Multivariate RR (95% CI)	1 [Reference]	1.28 (0.86-1.91)	1.32 (0.87-2.00)	1.35 (0.87-2.10)	2.09 (1.33-3.28)	.003
Age >50 y						
No. of subjects	4626	5279	6831	7854	9888	
No. of deaths	112	149	209	273	469	
Multivariate RR (95% CI)	1 [Reference]	1.28 (1.00-1.64)	1.42 (1.12-1.80)	1.57 (1.24-1.98)	1.93 (1.54-2.42)	<.001
Physically active						
No. of subjects	4766	4640	5208	5288	5884	
No. of deaths	64	82	119	146	231	
Multivariate RR (95% CI)	1 [Reference]	1.33 (0.96-1.85)	1.55 (1.13-2.12)	1.70 (1.24-2.32)	2.01 (1.48-2.74)	<.001
Physically inactive						
No. of subjects	9792	9463	9773	9265	8694	
No. of deaths	96	119	139	170	290	
Multivariate RR (95% CI)	1 [Reference]	1.25 (0.96-1.65)	1.31 (1.00-1.71)	1.44 (1.10-1.88)	1.93 (1.48-2.50)	<.001

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); CI, confidence interval; WHR, waist-hip ratio (waist circumference divided by hip circumference).

*The RRs were stratified on birth year and adjusted for education; occupation; annual family income; menopausal status; hormone therapy; amount of regular exercise; alcohol consumption; intakes of saturated fat, vegetables, and fruits; BMI; and the square of BMI. None of the results of tests for interaction were significant.

ciodemographic factors, physical activity, and other lifestyle and dietary factors, we could not completely rule out the possibility of residual confounding due to unmeasured or inaccurately measured covariates. For example, the questionnaire-based physical activity assessment used in our study, although validated, is crude and prone to misclassification; consequently, residual confounding from physical activity or fitness status cannot be dismissed. On the other hand, our study focused on WHR, a more specific measure of abdominal fat distribution, which may be less subject than BMI to the influence of these confounders.

Nevertheless, our study of relatively lean Chinese women, taken with studies of white women, suggests that increased abdominal adiposity contributes independently to an increase in mortality; WHR predicts mortality risk and can be used as a measure of choice to enhance risk evaluation.

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