Association of changes in waist circumference with cardiovascular disease and all-cause mortality among the elderly Chinese population: a retrospective cohort study

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ABSTRACT

BACKGROUND To examine the association of baseline waist circumference (WC) and changes in WC with cardiovascular disease (CVD) and all-cause mortality among elderly people.

METHODS A total of 30,041 eligible participants were included from a retrospective cohort in China. The same questionnaire, anthropometric and laboratory measurements were performed at baseline (2010) and the first follow-up (2013). The percent change in WC between baseline and the first follow-up was calculated to evaluate three years change of WC. We collected the occurrence of CVD and all-cause death from the first follow-up to December 31, 2018. Restricted cubic splines and Cox proportional-hazards regression models were used to evaluate the relationship between baseline WC/ changes in WC and mortality.

RESULTS The dose-response relationships between baseline WC and CVD mortality were U- or J-shaped. In low WC group, compared with stable group, the fully adjusted hazard ratio (aHR) for CVD mortality was 1.60 (95% CI: 1.24–2.06) in WC gain group among men. In normal WC group, the CVD mortality risk increased with WC gain (men: aHR = 1.86, 95% CI: 1.36–2.56; women: aHR = 1.83, 95% CI: 1.29–2.58). In moderate-high WC group, the CVD mortality risk increased with WC gain (men: aHR = 1.76, 95% CI: 1.08–2.88; women: aHR = 1.46, 95% CI: 1.04–2.05) and risk decreased with WC loss (men: aHR = 0.54, 95% CI: 0.30–0.98; women: aHR = 0.59, 95% CI: 0.37–0.96).

CONCLUSIONS For the elderly population, WC gain may increase CVD mortality risk regardless of baseline WC, whereas WC reduction could decrease the risk only in the moderate-high WC group.

Obesity is a major risk factor for many chronic diseases, such as high blood pressure, diabetes and dyslipidemia. Further, more serious obesity could cause cardiovascular disease (CVD) and death.[1–3] However, recent evidence shows that there is a more obvious obesity paradox in elderly people; namely, overweight and obesity are more beneficial to CVD disease.[4] The relationship between obesity and CVD in elderly people is controversial. Most of the studies indicating the existence of an obesity paradox only have used body mass index (BMI) as an indicator of obesity.[5] Obesity is commonly measured by BMI and waist circumference (WC).[6, 7] WC increases at a faster rate than BMI; as a result, the obesity-related health burden may be underestimated by using BMI alone and may contribute to the obesity paradox.[8, 9] Abdominal obesity defined by WC may be a better indicator than overall obesity defined by BMI with respect to predicting CVD and all-cause mortality.[10, 11]

However, most studies are often based on WC at a certain point, ignoring the changes in WC during follow-up.[12] Previous studies on the effect of WC change in European and American populations were limited and conflicting.[13–17] Some studies of Chinese populations only explored the association of changes in WC and chronic disease, without considering the time of outcome variable.[18–20] Most studies based on cross-sectional study design can-
not determine whether WC changes had taken place before CVD and all-cause mortality, and the purpose of cause and effect inference could not be achieved. To our knowledge, only a few cohort studies assessed the relationship between changes in BMI and the risk of CVD death in Chinese populations. However, the risk of CVD mortality with changes in WC has not been studied in the Chinese population. Thus, the purpose of this study was to explore the risk of CVD mortality and all-cause mortality with baseline WC and WC change and to identify dose-response relationships among baseline WC and WC change and CVD mortality and all-cause mortality in an elderly Chinese population.

METHODS

Study Population

This retrospective cohort study from an annual health check-up project was carried out in 2010 in Xinzhen City, Henan Province, Central China. Health check-up costs were paid by the Chinese government. Elderly people aged 60 and above in the city were given an annual free health check-up, including questionnaire interviews, physical examinations and laboratory tests, and an electronic health check-up database for the elderly population was formed, which can be integrated with the hospital information system and the cause of death registration system. We used health check-up data from January 2010 to December 2010 as baseline WC in the present study, and data from January 2013 to December 2013 were used as the first follow-up WC. The changes in WC from baseline to first follow-up were calculated. The health check-up cohort was followed until December 2018. From the end of the first follow-up to December 31, 2018, we collected the occurrence of outcome events from the hospital information system and the Register of Causes of Death that could provide direct access to all deaths, dates of death, and causes of death. The exclusion criteria in this study were as follows: (1) age < 60 years; (2) CVD or CVD medical history at baseline; (3) missing WC values at baseline or WC < 50 cm or > 130 cm; (4) missing WC values at follow-up; and (5) death before the first follow-up. A total of 30,041 participants were included in this cohort study. This study was approved by the Ethics Committee of Zhengzhou University in China, and all participants gave written informed consent.

Data Collection

The demographic data and clinical information of the subjects were collected at the time when they underwent the health check-up. Demographic data included age, sex (male/female), daily alcohol consumption (yes/no), current smoking status (yes/no), living alone (yes/no), physical activity (yes/no), history of diabetes mellitus, and history of hypertension. The definition of current smoking status is that subjects used to smoke 100 cigarettes or above in their lifetime, and were currently still smoking regularly. Regular physical activity was defined as 30 min moderate intensity exercise more than three times a week.

The clinical records included anthropometric measurements (such as height, weight, blood pressure, WC) and laboratory data. The measurements of height and weight were performed with the participants in light clothing without shoes. BMI was calculated as the ratio of weight (kg) to height (m$^2$) squared. Blood pressure of the subjects was measured twice by using an electronic sphygmomanometer (Omron HEM-7125, Kyoto, Japan), and the mean value of the two measurements was recorded, with participants resting for 5 min. WC was measured at the midpoint of the distance between the lowest costal ridge and the upper border of the iliac crest. According to WHO classifications, WC was categorized as < 80.0, 80.0 to 87.9, 88.0 to 93.9, ≥ 94 cm for Asian men, and < 72, 72.0 to 79.9, 80.0 to 87.9, ≥ 88 cm for Asian women, corresponding to low, normal, moderate-high, and high WC groups, respectively. Moderate-high and high WC groups were defined as abdominal obesity. We used baseline WC as a subgroup to explore the association of changes in WC with mortality risk in each subgroup. The relative change in WC was calculated according to the following formula: Percent change in WC = (WC$_{\text{follow-up}}$ - WC$_{\text{baseline}}$) / WC$_{\text{baseline}}$ × 100%. The percent change in WC was classified as follows: ≤ −5%, −5% to 5%, and > 5%, corresponding to loss, stable, and gain, respectively. A WC change of −5% to 5% was considered the reference group.
This retrospective cohort was obtained from annual health check-ups; therefore, all laboratory tests were performed by qualified technicians from health check-up hospitals. The blood samples of subjects were collected after fasting for 8 h. All plasma and serum samples were frozen at −20°C until laboratory testing was performed. Fasting plasma glucose (FPG) was measured using an oxidase enzymatic method. The concentrations of triglycerides (TG) and high-density lipoprotein cholesterol (HDL-C) were assessed enzymatically using an automatic biochemical detector (DIRUI CS380, Changchun, China) and commercial reagents.

Outcome Definition

The outcome events of interest were CVD mortality and all-cause mortality. CVD mortality consisted of coronary heart disease (CHD) and stroke mortality. Outcome events are defined by the International Classification of Diseases, Tenth Revision (ICD-10). Cases of CHD and stroke were identified by the use of ICD-10 codes I20–I25 and I60–I69, respectively. The date and cause of death were identified from the data of the annual health check-up project with a digital link to hospital information system and the Register of Causes of Death.

Statistical Analysis

Continuous data are described as the mean ± SD or median (interquartile range), and categorical data are presented as frequencies (percentage). Kruskal-Wallis test or $\chi^2$ test was used to test differences in baseline variables among groups. We used Cox proportional hazards regression to examine whether baseline WC and 3-year change in WC were associated with total mortality and CVD mortality. Before applying Cox proportional hazard models, we tested the proportional hazard assumptions. If all variables and the entire model met the proportional risk assumption, Cox proportional hazard regression models were used. To assess the association between outcome events and the main research variables accurately, we employed a series of incremental Cox proportional hazards models: model 1—adjusted for age, living alone, smoking status, alcohol consumption, physical activity, history of hypertension and diabetes mellitus; model 2—additional adjustment for BMI, SBP, DBP, RHR, FPG, TC, TG, HDL-C.

A non-linear test was used to examine whether there was a non-linear correlation between the study variables and the outcome events. Likelihood ratio test was used to verify whether the association of the overall model was significant. Spline regression was used to further illustrate the non-linear association of baseline WC and changes in WC change and the hazard ratio (HR) for mortality. A restricted cubic spline regression model was set to four knots, which were automatically placed at fifth, 35th, 65th, and 95th percentiles.

Restricted cubic splines were created by using Stata 14 (Stata Corp, College Station, TX). Cox proportional hazards models, the cumulative survival curves and other data were analyzed with R version 4.0.0 (R Foundation for Statistical Computing, Vienna, Austria). Statistical significance was determined at $P < 0.05$ based on two-sided probability.

RESULTS

A total of 30,041 survey respondents (age: 68.69 ± 6.87 years; 58.75% male) were eligible for inclusion in the present study and were followed for 115,468.9 person-years. Individuals were observed for a median of 4.39 years of follow-up (interquartile range: 3.33–4.60). Table 1 shows the participant characteristics at baseline according to the four categories of WC.

Baseline Characteristics of the Study Participants

The baseline characteristics of the study population of different genders according to baseline WC category are shown in Table 1. Living alone, smoking status, alcohol consumption, physical activity, history of hypertension and diabetes mellitus, BMI, SBP, DBP, TC, TG, and HDL-C were associated with WC categories. For both sexes, compared with subjects with low WC, those with higher WC were more likely to be current smokers, and have a history of hypertension and diabetes mellitus than those with a low WC. Similarly, WC was positively associated with BMI, systolic and diastolic blood pressure, and more favourable blood biochemistry profiles. For men, participants in the higher WC group were more likely to be daily drinkers (Table 1).
Table 1  Baseline characteristics of the study population by baseline waist circumference.

| Baseline characteristics | Male (n = 17,648) | Female (n = 12,923) |
|--------------------------|------------------|---------------------|
|                          | Low | Normal | Moderate | High | P      | Low | Normal | Moderate | High | P      |
| Age, yrs                 | 66.98 ± 5.95 | 66.91 ± 5.67 | 66.69 ± 5.49 | 66.88 ± 6.00 | 0.188 | 73.09 ± 8.11 | 71.16 ± 7.47 | 70.46 ± 7.00 | 70.14 ± 6.95 | < 0.001 |
| Living alone             | 833 (12.49%) | 864 (12.93%) | 275 (10.56%) | 352 (8.96%) | < 0.001 | 1,114 (38.84%) | 1,154 (31.27%) | 1,234 (32.01%) | 564 (28.50%) | < 0.001 |
| Smoking status           | 1,289 (19.33%) | 1,386 (20.75%) | 646 (24.82%) | 434 (25.57%) | < 0.001 | 78 (2.72%) | 75 (1.44%) | 67 (1.74%) | 39 (1.97%) | 0.002 |
| Alcohol consumption      | 628 (9.42%) | 903 (13.32%) | 444 (17.06%) | 303 (17.86%) | < 0.001 | 57 (1.99%) | 75 (2.03%) | 64 (1.66%) | 40 (2.02%) | 0.614 |
| Physical activity        | 1,002 (15.18%) | 1,331 (19.92%) | 518 (19.90%) | 322 (18.97%) | < 0.001 | 637 (22.21%) | 848 (22.97%) | 971 (25.19%) | 455 (22.99%) | 0.022 |
| History of hypertension  | 1,510 (22.65%) | 2,014 (30.15%) | 957 (36.77%) | 751 (44.25%) | < 0.001 | 979 (34.14%) | 1,246 (33.76%) | 1,624 (42.13%) | 1,064 (53.76%) | < 0.001 |
| History of diabetes mellitus | 518 (7.77%) | 731 (10.94%) | 393 (15.10%) | 307 (18.09%) | < 0.001 | 297 (10.36%) | 493 (13.36%) | 705 (18.29%) | 448 (22.64%) | < 0.001 |
| BMI, kg/m²               | 22.68 ± 2.04 | 23.89 ± 2.32 | 25.06 ± 2.49 | 26.97 ± 3.42 | < 0.001 | 22.29 ± 2.73 | 23.26 ± 2.63 | 24.46 ± 3.01 | 26.56 ± 3.70 | < 0.001 |
| SBP, mmHg               | 125.61 ± 14.35 | 127.99 ± 16.17 | 130.42 ± 16.48 | 134.07 ± 18.98 | < 0.001 | 129.02 ± 19.42 | 129.75 ± 18.57 | 132.81 ± 19.83 | 138.41 ± 21.96 | < 0.001 |
| DBP, mmHg               | 77.3 ± 8.37 | 76.69 ± 8.66 | 79.66 ± 8.94 | 80.60 ± 9.52 | < 0.001 | 77.34 ± 9.70 | 77.91 ± 9.15 | 78.68 ± 9.48 | 79.87 ± 10.17 | < 0.001 |
| RHR, beats/min          | 75 (72-78) | 75 (71-78) | 75 (70-78) | 75 (70-78) | 0.019 | 75 (70-78) | 75 (72-78) | 75 (70-79) | 75 (70-80) | 0.001 |
| FPG, mmol/L             | 5.39 (5.26-5.45) | 5.43 (5.25-5.55) | 5.48 (5.30-5.61) | 5.58 (5.51-5.74) | < 0.001 | 5.31 (4.90-5.50) | 5.40 (5.15-5.57) | 5.45 (5.10-5.66) | 5.56 (5.13-5.81) | < 0.001 |
| TC, mmol/L              | 4.72 (4.64-4.78) | 4.74 (4.61-4.83) | 4.77 (4.66-4.87) | 4.84 (4.71-4.97) | < 0.001 | 4.72 (4.55-4.98) | 4.75 (4.64-4.96) | 4.81 (4.66-5.19) | 4.92 (4.71-5.34) | < 0.001 |
| TG, mmol/L              | 1.34 (1.23-1.40) | 1.38 (1.21-1.49) | 1.44 (1.28-1.57) | 1.55 (1.30-1.70) | < 0.001 | 1.30 (1.12-1.48) | 1.35 (1.20-1.50) | 1.42 (1.21-1.61) | 1.51 (1.25-1.77) | < 0.001 |
| HDL-C, mmol/L           | 1.57 (1.56-1.59) | 1.57 (1.55-1.58) | 1.57 (1.54-1.58) | 1.56 (1.53-1.58) | < 0.001 | 1.57 (1.54-1.59) | 1.57 (1.54-1.59) | 1.56 (1.53-1.58) | 1.55 (1.51-1.57) | < 0.001 |

Continuous data were described as mean ± SD or median (interquartile range), and categorical data were presented as frequencies (percentage). BMI: body mass index; DBP: diastolic blood pressure; FPG: fasting plasma glucose; HDL-C: high-density lipoprotein cholesterol; RHR: resting heart rate; SBP: systolic blood pressure; TC: total cholesterol; TG: triglyceride.

Association Between Baseline WC and Mortality Risk

During a median of 4.39 years of follow-up, 685 men and 647 women died from CVD, with mortality rates of 10.30 and 13.22 per 1,000 person-year, respectively. For men with low, normal, moderate-high, and high baseline WC, the CVD mortality was 10.92, 8.79, 10.71, and 13.11 per 1,000 person-years, respectively. For women with low, normal, moderate-high, and high baseline WC, the CVD mortality was 15.89, 10.74, 13.03, and 14.33 per 1,000 person-years, respectively.

All variables and the entire model met the proportional risk assumption, and Cox proportional hazard models were used. Table 2 lists the hazard ratios for CVD and all-cause death, grouped according to the sex-specific category of baseline WC. Cox proportional-hazards models revealed a significantly increased risk of CVD mortality for men with low and high versus normal WC after adjusting for some potential confounders (Table 2). The aHR was 1.42 (95% CI: 1.18–1.70) for the low group and 1.45 (95% CI: 1.11–1.89) for the high group. Risks of CVD mortality were increased for women with low (aHR = 1.29; 95% CI: 1.04–1.61), moderate-high (aHR = 1.25; 95% CI: 1.01–1.55), and high (aHR = 1.32; 95% CI: 1.02–1.71) versus normal WC. Similar effects on all-cause mortality were also observed across different WC categories. The HR (95% CI) for all-cause mortality for men with high WC was 1.20 (1.03–1.40). The HRs (95% CIs) for all-cause mortality for women with low, moderate-high, and high were 1.19 (1.05–1.35), 1.20 (1.06–1.36), and 1.26 (1.08–1.48) versus normal WC, respectively.

Restricted cubic spline analyses were used to examine the association between baseline WC and CVD mortality (Figure 1). For men, the association between baseline WC on a continuous scale and CVD mortality was a pronounced U-shape: both low and high concentrations were associated with high CVD mortality. For women, a slight J-shape was apparent, although the 95% CI at the lower range of WC was not significant. The concentrations of WC associated with the lowest CVD mortality were 93 cm for men and 86 cm for women.

Association Between Changes in WC and Mortality Risk

We examined the association of relative values of changes in WC and CVD mortality risk stratified by gender and baseline WC (Table 3). In the low WC group, compared with the stable group, the fully
Table 2  Adjusted Cox proportional-hazards regression models for the association of baseline WC categories with CVD mortality and all-cause mortality in an elderly Chinese population.

| Baseline WC | Person-years | CVD mortality | All-cause mortality |
|-------------|--------------|---------------|---------------------|
|             | Events | Mortality | Model1 | Model2 | Events | Mortality | Model1 | Model2 |
| Male        |        |           |       |        |        |           |       |        |
| Low         | 25,186.60 | 275 | 10.92 | 1.37 (1.14–1.64) | 1.42 (1.18–1.70) | 847 | 33.63 | 1.11 (1.01–1.23) | 1.08 (0.98–1.2) |
| Normal      | 25,035.56 | 220 | 8.79  | 1.00  | 1.00  | 774 | 30.92 | 1.00  | 1.00  |
| Moderate-high | 9,896.18 | 106 | 10.71 | 1.16 (0.92–1.47) | 1.18 (0.93–1.49) | 284 | 28.70 | 0.87 (0.76–1.01) | 0.89 (0.78–1.03) |
| High        | 6,409.67  | 84  | 13.11 | 1.44 (1.12–1.85) | 1.45 (1.11–1.89) | 222 | 34.64 | 1.13 (0.97–1.31) | 1.20 (1.03–1.4) |
| Female      |          |      |      |       |       |      |       |       |       |
| Low         | 11,391.76 | 181 | 15.89 | 1.24 (1.01–1.55) | 1.29 (1.04–1.61) | 547 | 48.02 | 1.20 (1.06–1.36) | 1.19 (1.05–1.35) |
| Normal      | 14,613.06 | 157 | 10.74 | 1.00  | 1.00  | 486 | 33.26 | 1.00  | 1.00  |
| Moderate-high | 15,190.36 | 198 | 13.03 | 1.25 (1.01–1.54) | 1.25 (1.01–1.55) | 548 | 36.08 | 1.16 (1.03–1.32) | 1.20 (1.06–1.36) |
| High        | 7,745.72  | 111 | 14.33 | 1.31 (1.02–1.68) | 1.32 (1.02–1.71) | 274 | 35.37 | 1.14 (0.98–1.33) | 1.26 (1.08–1.48) |

*Model1: adjusted for age, living alone, smoking status, alcohol consumption, physical activity, history of hypertension and diabetes mellitus. †Model2: Model1 + further adjusted for body mass index, systolic blood pressure, diastolic blood pressure, resting heart rate, fasting plasma glucose, total cholesterol, triglyceride, and high-density lipoprotein cholesterol. CVD: cardiovascular disease; WC: waist circumference.

Figure 1  HRs for morbidity according to baseline WC in the elderly population. (A): Dose-response association between baseline WC (cm) and risk of CVD mortality for man; (B): dose-response association between baseline WC (cm) and risk of all-cause mortality for woman; (C): dose-response association between baseline WC (cm) and risk of CVD morbidity for man; and (D): dose-response association between baseline WC (cm) and risk of all-cause morbidity for woman. WC: waist circumference; CVD: cardiovascular disease.

aHR for CVD mortality was 1.60 (95% CI: 1.24–2.06) in the WC gain group for men. In the normal WC group, the CVD mortality risk significantly increased with WC gain for both sexes (men: aHR 1.86; 95% CI: 1.36–2.56; women: aHR 1.83; 95% CI: 1.29–2.58). In the moderate-high WC group, the multivariate-adjusted risk for CVD mortality increased with WC gain (men: aHR = 1.76, 95% CI: 1.08–2.88; women: aHR = 1.46, 95% CI: 1.04–2.05). In the moderate-high WC group, WC loss was associated with decrease of CVD mortality risk (men: aHR = 0.54, 95% CI: 0.30–0.98; women: aHR = 0.59, 95% CI: 0.37–0.96).

We found a significant nonlinear relationship...
between the percent changes in WC and CVD mortality for non-abdominal obesity, which remained after adjustment for all covariates. For abdominal obesity, there was a linear association between changes in WC and mortality (Table S1). The overall tests of association (Likelihood ratio tests) indicated a significant association between changes in WC and outcomes (Table S2). The spline regression analyses showed that for non-abdominal obesity, the lowest risk was found around a zero change in WC, indicating a U-shaped association between change in WC and CVD mortality in both sexes (Figure 2A). The shape of all-cause mortality was similar to that of CVD mortality, but the risk of CVD mortality attributed to changes in WC was higher than the risk of all-cause mortality overall (Figure 2B). For abdominal obesity, the risk of CVD and all-cause death increased with WC gain (Figure 2C and Figure 2D).

### DISCUSSION

Our study revealed that both baseline WC and changes in WC were associated with CVD mortality and all-cause mortality among elderly people in China. Not considering changes in WC, those in the low and high WC groups had a higher risk of CVD mortality than those in the normal WC group. Con-
Considering the changes in WC, the dose-response relationship between changes in WC and mortality was U-shaped for those with non-abdominal obesity at baseline. For those with abdominal obesity at baseline, significantly higher risks of CVD mortality were found with increased changes in WC, especially for those in the moderate-high WC group. The overall dose-response relationship between baseline WC and CVD mortality was U- or J-shaped, which was consistent with the results of a Japanese community study. Although some studies have reported that a greater WC was associated with increased CVD mortality, most of these studies were conducted in American or European populations. In addition, these studies may not have collected information on populations with a low WC. The convincing reason for this discrepancy may be that the distribution of WC and the main cause of death vary in different populations. According to a report, more than 30% of US adults are estimated to be obese and the leading cause of death in America is CVD. Because obesity is a long-term independent risk factor for CVD mortality, a monotonic rise dose-response relationship between WC and CVD mortality is more likely to be observed. Our study found that lower WC was associated with a higher risk of all-cause and CVD deaths. On the one hand, a possible reason is that underweight-related morbidities, such as cancer, diabetes, respiratory disease, and kidney disease, are the major causes of death in Asia. On the other hand, it might be explained by the association of a low WC with malnutrition and sarcopenia. In addition, elderly people with a low WC may have low-grade inflammation and may be more frail. These mechanisms may lead to vulnerability to external hazards, which further leads to death. The CARDIA cohort of 5,115 participants found that a high WC was associated with a higher blood pressure, triglyceride and total cholesterol levels, higher diabetes rates, and lower HDL cholesterol. Studies have found that increased WC can lead to an increased risk of CVD death through disorders of metabolic factors such as impaired glucose tolerance, dyslipidemia and hypertension. However, the association between a high WC and CVD mortality remained after adjusting for these factors. Possible mechanisms are cardiomyocyte hypertrophy, myocardial fibrosis, sustained expression and secretion of proinflammatory adipocytes, disordered breathing during sleep, and changes in cardiac structure and function.

Figure 2 HRs for morbidity according to percent changes in WC in the elderly population. Dose-response association between percent changes in WC (%) and risk of CVD mortality for (A) those with non-abdominal obesity at baseline and (C) those with abdominal obesity at baseline. Dose-response association between percent changes in WC and risk of all-cause mortality for (B) those with non-abdominal obesity at baseline and (D) those with abdominal obesity at baseline. CVD: cardiovascular disease; WC: waist circumference.
The above studies only evaluated the association of abdominal obesity and CVD mortality. However, WC changed dynamically during the follow-up period. Research conclusions about the health consequences of long-term dynamic changes in WC are different and controversial. A study of 1,805 Iranian men showed that there was no association between changes in WC and mortality.[41] A study involving 2,584 men and 14,041 women in Denmark found that changes in WC were positively associated with mortality in healthy middle-aged individuals.[14] Two studies of a rural population in China found that a WC gain was associated with increased hypertension risk and dyslipidemia risk.[19, 20] These studies were merely based on the association of changes in WC and ending events. However, changes in different baseline WC levels may have different meanings or effects on outcomes. It would be inaccurate to explore the role of changes in WC in the entire population without distinguishing baseline WC levels. Therefore, for the first time, by establishing four baseline WC categories, we investigated the impact of changes in WC in each baseline WC group on CVD mortality during a follow-up period.

This study confirmed that WC loss in elderly women with a normal WC may increase the risk of CVD and all-cause mortality, and WC loss in elderly men is associated with an increased risk of all-cause mortality. This may be due to nutritional deficiencies, diabetes, cancer and other malignant diseases resulting in reduced body fat.[11, 24] The population in the moderate-high WC group is the most worthy of attention. WC loss within the normal range in this group could decrease the risk of CVD mortality, which may be caused by the positive effects of lifestyle changes, such as a healthy diet and regular physical activity. The mechanism by which WC gain increases mortality could be explained by higher visceral adipose tissue. Visceral adipose tissue is more pathogenic than subcutaneous tissue,[42, 43] because it can secrete some mediators that may develop into cardiac metabolic diseases.[44] In addition, WC gain is associated with inflammation,[45, 46] insulin resistance,[47, 48] T2DM,[49, 50] and CHD,[51, 52] all of which may progress to CVD death. People with high WC have no significant changes in the risk of CVD death regardless of whether there is a decrease or an increase in WC, which is consistent with the conclusion of a study of Nordic women study.[13] Obesity is a serious risk factor that has had an impact on outcome. This finding was supported in this article, in which a high baseline WC was associated with a high risk of CVD mortality. The change in WC during follow-up was not sufficient to affect the HR of a baseline high WC. We also found that after adjusting for BMI, a strong correlation between WC and mortality remained. Abdominal fat accumulation has proven to be an influencing factor for many adverse health outcomes including diabetes, metabolic syndrome, CVD and all-cause mortality, independent of BMI.[53–55] Our study shows that regular monitoring of WC and keeping WC within a normal range can help prevent CVD deaths and all-cause deaths. Unhealthy diet, lack of exercise and smoking are the major lifestyle risk factors associated with abdominal obesity.[56] Controlling WC within the normal range through lifestyle changes can prevent CVD events and reduce the mortality rate.

Our study has several strengths, including the large sample size and the longitudinal design. Additionally, baseline WC, follow-up WC, weight and height were objectively measured and not self-reported. Abdominal obesity was defined according to the WHO recommendations for Chinese people, which could improve the accuracy of the analyses in this study. In contrast to other studies, this study additionally investigated the relationship between dynamic changes in WC in four baseline WC groups and CVD mortality. This could provide more evidence of the association between adiposity and mortality. Inevitably, this study has some limitations. First, the study participants were selected from one city in the middle of China and the body composition is quite different in Asians than in the United States and Europe; thus, our proposed cutoff values for the indices may not be applicable to non-Asian populations. Our findings require confirmation in other ethnic groups. Secondly, because information on smoking and drinking was self-reported, measurement errors were inevitable. Finally, although we adjusted three models for a variety of covariates, there is still a possibility of residual confounding. Muscle mass and strength, psychological factors (depression or
stress), medical care and economic level, all have an important role in evaluating mortality. However, the related information was not available in this study.

In conclusion, our study found that among elderly people, those with a low WC and high WC have a higher risk of CVD death and all-cause death than those with a normal WC. In the moderate-high baseline WC group, there was an increased risk of CVD death with WC gain and a decreased CVD death risk with WC loss. In the normal baseline WC group, the CVD mortality risk significantly increased with WC gain. This underscores the importance of preventing abdominal fat deposition in elderly people. Therefore, a healthy lifestyle should be implemented earlier to prevent abdominal adiposity and maintain a healthy waist size to reduce the occurrence of CVD death.

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CONFLICTS OF INTEREST

None.

ETHICAL APPROVAL

This study was approved by the Ethics Committee of Zhengzhou University in China, and all participants gave written informed consent.

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