

BMJ Open Anthropometric measurements of general and central obesity and the prediction of cardiovascular disease risk in women: a cross-sectional study

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ABSTRACT

Objectives: It is important to ascertain which anthropometric measurements of obesity, general or central, are better predictors of cardiovascular disease (CVD) risk in women. 10-year CVD risk was calculated from the Framingham risk score model, SCORE risk chart for high-risk regions, general CVD and simplified general CVD risk score models. Increase in CVD risk associated with 1 SD increment in each anthropometric measurement above the mean was calculated, and the diagnostic utility of obesity measures in identifying participants with increased likelihood of being above the treatment threshold was assessed.

Design: Cross-sectional data from the National Heart Foundation Risk Factor Prevalence Study.

Setting: Population-based survey in Australia.

Participants: 4487 women aged 20–69 years without heart disease, diabetes or stroke.

Outcome measures: Anthropometric obesity measures that demonstrated the greatest increase in CVD risk as a result of incremental change, 1 SD above the mean, and obesity measures that had the greatest diagnostic utility in identifying participants above the respective treatment thresholds of various risk score models.

Results: Waist circumference (WC), waist-to-hip ratio (WHR) and waist-to-stature ratio had larger effects on increased CVD risk compared with body mass index (BMI). These central obesity measures also had higher sensitivity and specificity in identifying women above and below the 20% treatment threshold than BMI. Central obesity measures also recorded better correlations with CVD risk compared with general obesity measures. WC and WHR were found to be significant and independent predictors of CVD risk, as indicated by the high area under the receiver operating characteristic curves (>0.76), after controlling for BMI in the simplified general CVD risk score model.

Conclusions: Central obesity measures are better predictors of CVD risk compared with general obesity measures in women. It is equally important to maintain a healthy weight and to prevent central obesity concurrently.

Strengths and limitations of this study

- This study provided evidence that anthropometric measures of central obesity are better predictors of cardiovascular disease (CVD) risk compared with general obesity measures in women.
- Central obesity measures add prognostic information on CVD risk in women above the measures of general obesity and should be considered for incorporation into the clinical assessment of CVD risk.
- Although this study is cross-sectional, it is a representative sample of the Australian female population.
- Only one set of baseline measurements is recorded for some risk variables but some important variables are measured twice.
- The predicted 10-year CVD risks are calculated using risk score models to stratify individuals against the treatment thresholds for various risk score models. Prospective data of CVD events were not used.

INTRODUCTION

In 2008, more than 200 million men and approximately 300 million women were obese.¹ Overweight and obesity is one of the leading risk factors for mortality, estimated to account for 23% of the ischaemic heart disease burden.¹ It results in the deterioration of the entire cardiovascular risk profile.^{2–3} Large prospective studies such as the Framingham Heart Study,⁴ the Nurses' Health Study^{5–6} and the Buffalo Health Study⁷ have all shown that overweight and obesity are associated with increased cardiovascular disease (CVD) risk. Excess adipose tissue contributes to the cardiovascular and other risks associated with being overweight or obese.⁸

The American Heart Association released a Scientific Statement emphasising the importance of assessing adiposity.⁸ New guidelines have also been released by the American College of Cardiology, American Heart Association Task Force on Practice Guidelines and The Obesity Society for the management of overweight and obesity in adults to prevent CVD.⁹ General and central obesity are associated with CVD risk.^{5 10–15} Currently used general and central obesity anthropometric measures for assessing adiposity-related risk include: body mass index (BMI; weight in kilograms divided by square of height in meters), waist circumference (WC), hip circumference (HC), waist-to-hip ratio (WHR; ratio of WC to HC), waist-to-stature ratio (WSR; ratio of WC to height) and body adiposity index¹⁶ (BAI; HC divided by height^{1.5}, and subtracting 18 from the result). BMI or WC is most commonly used to measure body fatness.¹⁰

It is, however, unclear which anthropometric measurements are better correlated with CVD risk factors and CVD risk in women, considering adiposity is highly heterogeneous with age, sex and ethnic differences in body fat distribution.⁸ Previous studies have reported that BMI identified individuals at increased risk of CVD as effectively as WC.^{11 12} It has also been suggested that BMI is a better predictor of CVD than WC.¹³ Conversely, some studies reported that WC is a better indicator of CVD risk than BMI and WHR, in ethnically diverse groups.^{14 15} WC and WHR have also been identified as independent predictors of CVD risk but not BMI, accounting for conventional risk factors in the Framingham risk score model.¹⁷ More research is thus needed to ascertain which measures are better correlated with CVD risk factors and subsequent CVD risk in women.

We aim to assess the associations between general and central obesity anthropometric measures with CVD risk factors, using a representative sample of 4487 women aged 20–69 years without heart disease, diabetes or stroke. The associations between these indices of obesity with predicted risk calculated from the Framingham risk score model for 10-year CVD incidence or death,¹⁸ SCORE risk chart for high-risk regions for 10-year CVD death,¹⁹ general CVD and simplified general CVD risk score models for 10-year CVD incidence and death²⁰ were examined. To aid comparison between obesity indices, which are measured in different units, the incremental shift in CVD risk with 1 SD increment in each anthropometric measurement above the mean would be assessed. Finally, we determined which indices of obesity are most sensitive and specific for identifying women at increased 10-year CVD risk.

METHODS

Study cohort and measurements

We selected 4487 women aged 20–69 years with no history of heart disease, diabetes or stroke from the population representative sample of 4727 women from

the National Heart Foundation (NHF) Risk Factor Prevalence Study.²¹ Participants taking medications to lower their CVD risk factors were also excluded. The participants of the NHF study consisted of residents on the federal electoral rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart, Darwin and Canberra in a systematic probability sampling by sex and 5-year age groups. Information on demographic characteristics was collected using a self-administered questionnaire, and conventional CVD risk variables recorded in this prevalence study include anthropometric measures, smoking status, systolic and diastolic blood pressure and lipid levels. Physical measurements of height (to the nearest centimetre), weight (to the nearest 10th of a kilogram) and waist and HC were collected according to standardised methodologies^{22 23} using two observers. The WC was measured from the front at the narrowest point between the rib cage and iliac crest after full expiration while the HC was measured from the side at the maximal extension of buttocks by one observer using a metal tape. A second observer recorded another set of measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of two measurements was taken at each site to the nearest centimetre. Participants were classified as non-smokers, previous smokers or current smokers.²¹ Mercury sphygmomanometers were used to record blood pressure levels on the right arm of seated participants 5 min apart.²¹ Two readings were taken and the average was used in the analysis. Fasting blood samples were also collected in EDTA tubes and dispatched to the central laboratory at the Division of Clinical Chemistry, Institute of Medical and Veterinary Science, Adelaide each week for lipid levels to be assayed.²¹

Risk score models

The Framingham 10-year predicted risk for CVD incidence or death was developed using data from the American Framingham Heart Study.¹⁸ Participants aged 30–74 years who were free of CVD and cancer were included in the model development. The 10-year risk for CVD incidence or death was calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status. The SCORE risk chart was developed by pooling 12 cohort studies to predict the 10-year CVD death risk in Europe. The cohorts consisted of participants aged 19–80 years with no previous history of heart attack.¹⁹ The SCORE model was derived from a much larger dataset than the Framingham, general CVD and simplified general CVD risk score models. Fewer variables were used in the calculation of the 10-year predicted CVD death risk with the SCORE risk chart for high-risk regions (Denmark, Finland and Norway),^{19 24} these included: age, sex, smoking status, mean total cholesterol level, mean HDL cholesterol level and mean

Table 1 Characteristics of a representative Australian sample of 4487 women (aged 20–69 years) free of heart disease, diabetes and stroke

Variables	Summary statistics
Age (years), n (%)	
20–29	840 (18.7)
30–39	1116 (24.9)
40–49	1139 (25.4)
50–59	743 (16.6)
≥60	649 (14.4)
Ethnicity	
Australia	3329 (76.5%)
UK and Ireland	416 (9.5%)
Northern Europe	180 (4.1%)
Southern Europe	234 (5.4%)
Asia	195 (4.5%)
Smoking status, n (%)	
Non-smoker	2652 (59.1)
Previous smoker	880 (19.6)
Current smoker	955 (21.3)
SBP (mm Hg)	122.1±18.4
DBP (mm Hg)	75.7±10.8
Total cholesterol (mmol/L)	5.5±1.2
HDL cholesterol (mmol/L)	1.5±0.4
Ratio of total cholesterol to HDL cholesterol	3.9±1.3
BMI (kg/m ²)	24.8±4.7
WC (cm)	76.2±11.1
HC (cm)	100.1±10.0
WHR	0.76±0.06
WSR	0.47±0.07
BAI (%)	30.6±5.4

BMI, body mass index; BAI, body adiposity index; DBP, diastolic blood pressure; HC, hip circumference; HDL cholesterol, high-density lipoprotein cholesterol; SBP, systolic blood pressure; WC, waist circumference; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

SBP. The general CVD risk score model was also developed using data from the American Framingham Heart Study but using a larger cohort than the Framingham

model.²⁰ Individuals without CVD were used in the development of the general CVD risk score model.²⁰ The simplified general CVD risk score model was developed similarly as the general CVD risk score model. It is, however, a simpler CVD risk prediction model which is calculated using non-laboratory predictors. Risk variables (age, SBP, current antihypertensive treatment, smoking status and diabetes status) were used in both models.²⁰ The only difference is that, BMI is included in the simplified general CVD risk score model instead of total and HDL cholesterol which is used in the general CVD risk score model.

Statistical analysis

The data on the representative sample of 4487 Australian women were described using mean±SD for continuous variables, while counts and percentages were used for categorical variables. Non-parametric Spearman's rank correlation was used to assess the associations between anthropometric measurements of obesity with CVD risk factors, and with the calculated 10-year predicted risks, due to the skewness in the distribution of some variables. Anthropometric measurements were also converted to z-scores (original value subtracted by the mean and result divided by the SD) to represent the number of SDs above and below the mean for each participant. Logistic regression was used to assess the effects of each standardised anthropometric measurement of being above the recommended treatment thresholds for various risk score models as a result of 1 SD increment above the mean for each anthropometric measure of obesity. ORs and associated 95% CIs represented the likelihood of being above the recommended treatment thresholds for the specific risk score models (20% for the Framingham risk score model for 10-year CVD incidence or death; 10% for SCORE risk chart for high-risk regions for 10-year CVD death; 10% and 20% for the general CVD and simplified general CVD risk score models for 10-year CVD incidence and death). The predictive ability of these anthropometric measures to identify individuals above and below the

Table 2 Frequency distribution of 10-year predicted CVD incidence and mortality using various risk prediction models, in incremental risk categories of 10%

	Risk categories				
	0–9%	10–19%	20–29%	30–39%	≥40%
Framingham 10-year predicted risk for CVD incidence ¹⁸	2936 (67.0%)	764 (17.4%)	417 (9.5%)	179 (4.1%)	89 (2.0%)
Framingham 10-year predicted risk for CVD death ¹⁸	4354 (99.3%)	29 (0.7%)	2 (0%)	0 (0%)	0 (0%)
SCORE-HIGH 10-year predicted risk for CVD death ¹⁹	4318 (98.5%)	53 (1.2%)	9 (0.2%)	4 (0.1%)	1 (0%)
GCVD 10-year predicted risk for CVD incidence and death ²⁰	3738 (85.2%)	503 (11.5%)	109 (2.5%)	21 (0.5%)	14 (0.3%)
SGCVD 10-year predicted risk for CVD incidence and death ²⁰	3809 (85.7%)	519 (11.7%)	90 (2.0%)	19 (0.4%)	9 (0.2%)

Counts and percentages of women were presented.

CVD, cardiovascular disease; GCVD, general cardiovascular disease risk score model; SCORE-HIGH, SCORE risk chart for high-risk regions; SGCVD, simplified general cardiovascular disease risk score model.

treatment thresholds was assessed using sensitivity, specificity and area under the receiver operating characteristic (ROC) curve. *p* Values of less than 0.05 were considered to be statistically significant. All statistical analyses were performed with IBM SPSS Statistics V.21.

RESULTS

The sample of 4487 women aged 20–69 years from the NHF Risk Factor Prevalence Study is a representative sample of the Australian female population, free of heart disease, diabetes and stroke. The characteristics of the sample are summarised in [table 1](#). In addition to the conventional risk factors for CVD, all anthropometric measurements of general and central obesity were presented.

The 10-year CVD risk of each participant in the sample was calculated using four risk score models. The frequency distribution of calculated risks is presented in [table 2](#). Except for the Framingham model for CVD incidence, all other models predicted risks of less than 10% for at least 85% of the sample. The Framingham model for CVD incidence, general CVD model for CVD incidence and death and simplified general CVD model for CVD incidence and death predicted risk values across the entire range from 0% to greater than 40%.

Anthropometric measurements of obesity were positively correlated with age, SBP, total cholesterol and total cholesterol to HDL cholesterol ratio (all Spearman's $r \geq 0.195$, $p < 0.001$), with HC recording the lowest correlations. These obesity measures were negatively correlated with HDL cholesterol (all Spearman's $r \leq -0.160$, $p < 0.001$). Measures of central obesity that included a measure of WC (WHR and WSR) generally recorded better correlations compared with measures of general obesity (BMI and BAI).

The associations between anthropometric measurements of obesity and the 10-year predicted risks calculated using the four models are presented in [table 3](#). All Spearman's rank correlations were statistically significant ($p < 0.0005$). All anthropometric measures of central obesity (WC, WHR and WSR) generally had consistently

higher correlations with the predicted risks calculated using the four CVD risk score models, as compared with measures of general obesity.

Recommended treatment thresholds for the four CVD risk models were identified from a review of the literature. [Table 4](#) presents the effects of 1 SD increment in each anthropometric measurement above the mean on the likelihood of being above the recommended thresholds or being indicated for treatment. All anthropometric measures of central obesity (WC, WHR and WSR) generally recorded higher ORs than general measures of obesity and they increased the likelihood of individuals being above the respective treatment thresholds.

Anthropometric measurements of central obesity (WC, WHR and WSR) also recorded higher area under the ROC curves, higher sensitivity and specificity, than BMI in identifying women above and below the 20% treatment threshold for the Framingham model for 10-year CVD incidence ([figure 1A](#)) and general CVD model for 10-year CVD incidence and death ([figure 1B](#)). Although BMI is included in the simplified general CVD model, high area under the ROC curve (>0.76) are reported for WC and WHR ([figure 1C](#)), indicating the independent contribution of central obesity measurements as compared with general obesity measurement in predicting the increased risk of CVD.

DISCUSSION

Measures of obesity are generally not included in the prediction of CVD risk. BMI is the only measure of obesity currently included in CVD risk score models such as the simplified general CVD risk score model, as an alternative to total and HDL cholesterol level for ease of measurement and calculation,²⁰ and in the QRISK score model.²⁹

In our study, anthropometric measurements of central obesity (WC, WHR and WSR) were more strongly associated with conventional CVD risk factors and the 10-year predicted risk calculated using the Framingham risk score model, SCORE risk chart for high-risk regions, general CVD and simplified general CVD risk score

Table 3 Non-parametric correlations between anthropometric measurements of general and central obesity and 10-year predicted risk of CVD incidence and mortality in 4487 women

	BMI	WC	HC	WHR	WSR	BAI
Framingham 10-year predicted risk for CVD incidence ¹⁸	0.380	0.450	0.301	0.409	0.485	0.378
Framingham 10-year predicted risk for CVD death ¹⁸	0.394	0.452	0.307	0.404	0.483	0.377
SCORE-HIGH 10-year predicted risk for CVD death ¹⁹	0.309	0.381	0.253	0.348	0.419	0.338
GCVD 10-year predicted risk for CVD incidence and death ²⁰	0.385	0.452	0.307	0.405	0.487	0.383
SGCVD 10-year predicted risk for CVD incidence and death ²⁰	*	0.446	0.320	0.384	*	*

All Spearman's rank correlations significant at the $p < 0.0005$ level.

*Correlation is not calculated for this obesity measure as it contains variables that are also used in the calculation of the simplified general CVD model.

BMI, body mass index; BAI, body adiposity index; CVD, cardiovascular disease; GCVD, general cardiovascular disease risk score model; HC, hip circumference; WC, waist circumference; SCORE-HIGH, SCORE risk chart for high-risk regions; SGCVD, simplified general cardiovascular disease risk score model; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

Table 4 OR and associated 95% CIs of being above the recommended treatment thresholds for various risk score models as a result of a 1 SD increment above the mean for each anthropometric measure of obesity

BMI	WC	HC	WHR	WSR	BAI
Framingham 10-year predicted risk for CVD incidence (threshold=20%) ^{25, 26}	2.12*** (1.95–2.29)	1.55*** (1.44–1.68)	2.27*** (2.08–2.47)	2.35*** (2.17–2.56)	1.92*** (1.77–2.09)
Framingham 10-year predicted risk for CVD death (threshold=20%) ^{25, 26}	3.13* (1.30–7.54)	1.60* (1.04–2.46)	2.52* (1.09–5.83)	3.33* (1.32–8.39)	1.58* (1.05–2.36)
SCORE-HIGH 10-year predicted risk for CVD death (threshold=10%) ¹⁹	1.91*** (1.59–2.29)	1.40*** (1.18–1.66)	2.01*** (1.66–2.42)	2.04*** (1.70–2.46)	1.58*** (1.31–1.90)
GCVD 10-year predicted risk for CVD incidence and death (threshold=10%) ²⁷	2.11*** (1.95–2.29)	1.57*** (1.45–1.70)	2.23*** (2.04–2.43)	2.34*** (2.15–2.55)	1.94*** (1.79–2.11)
SGCVD 10-year predicted risk for CVD incidence and death (threshold=10%) ²⁷	2.16*** (1.99–2.34)	1.66*** (1.54–1.80)	2.16*** (1.98–2.35)	†	†
GCVD 10-year predicted risk for CVD incidence and death (threshold=20%) ^{20, 28}	2.03*** (1.77–2.31)	1.52*** (1.33–1.74)	2.08*** (1.81–2.39)	2.15*** (1.88–2.45)	1.72*** (1.49–1.97)
SGCVD 10-year predicted risk for CVD incidence and death (threshold=20%) ^{20, 28}	2.26*** (1.96–2.60)	1.72*** (1.50–1.99)	2.11*** (1.82–2.45)	†	†

*p<0.05, **p<0.01, ***p<0.001.

†OR is not calculated for this obesity measure as it contains variables that are also used in the calculation of the simplified general CVD model.

BMI, body mass index; BAI, body adiposity index; CVD, cardiovascular disease; GCVD, general cardiovascular disease risk score model; HC, hip circumference; WC, waist circumference; SCORE-HIGH, SCORE risk chart for high-risk regions; SGCVD, simplified general cardiovascular disease risk score model; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

model, compared with general measures of obesity. Central obesity measures also recorded higher ORs and increased the likelihood of being above the recommended treatment threshold of the respective models with 1 SD increase above the mean. Central obesity measures which incorporated the measure of WC also exhibited higher sensitivity and specificity than BMI. Although BMI is included in the calculation of the simplified general CVD model, high area under the ROC curves were reported for WC and WHR, thus confirming that anthropometric measures of central obesity independently and significantly predicts CVD risk that is not accounted for by the general obesity measure. Hence, BMI alone is insufficient to account for the association between obesity and CVD risk.

Consistent with our study findings, previous studies also reported stronger associations between central obesity measures and CVD risk. Higher standardised ORs adjusted for BMI were reported for WC and CVD, compared with BMI, in women from the International Day for the Evaluation of Abdominal Obesity (IDEA) study.^{30, 31} An increase in WC was associated with being 4.25 times more likely of stroke and transient ischaemic attacks.³² Conversely, some studies reported that the association between BMI and CVD was similar to measures of central obesity.^{33, 34}

There are several possible explanations for our study findings that measures of central obesity are better predictors of CVD risk than BMI. Greater central obesity is associated with systemic inflammation which directly contributes to CVD risk.³⁵ Hence, measures that account for the accumulation of excess abdominal fat would report stronger associations and are desirable for assessing adiposity. They would also be more accurate at indicating CVD risk and should be incorporated into CVD assessment.^{36–39} The addition of central obesity measures to BMI has also been shown to improve the accuracy of stratifying participants into lower and higher risk categories for mortality⁴⁰ and provides incremental value in predicting CVD above and beyond that provided by general obesity measures.^{41–45} BMI is a flawed measure as it does not correctly identify individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does not account for the effect of age and ethnicity on body fat distribution.^{46–50} An increase in muscle or fat-free mass would, however, be reflected in the central obesity measures.

Among central obesity measures, we found their performance to be comparable in our study. It remains unclear which measurement should be incorporated into CVD risk score models. A collaborative analysis of 58 prospective studies, however, reported that measures of general and central obesity did not improve CVD risk assessment when information is available on SBP, diabetes and lipids.⁵¹ Overweight and obesity are, nevertheless, important in CVD prevention, with one of three fatal and one of seven non-fatal CVD cases attributable to it.³⁴

Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and its association

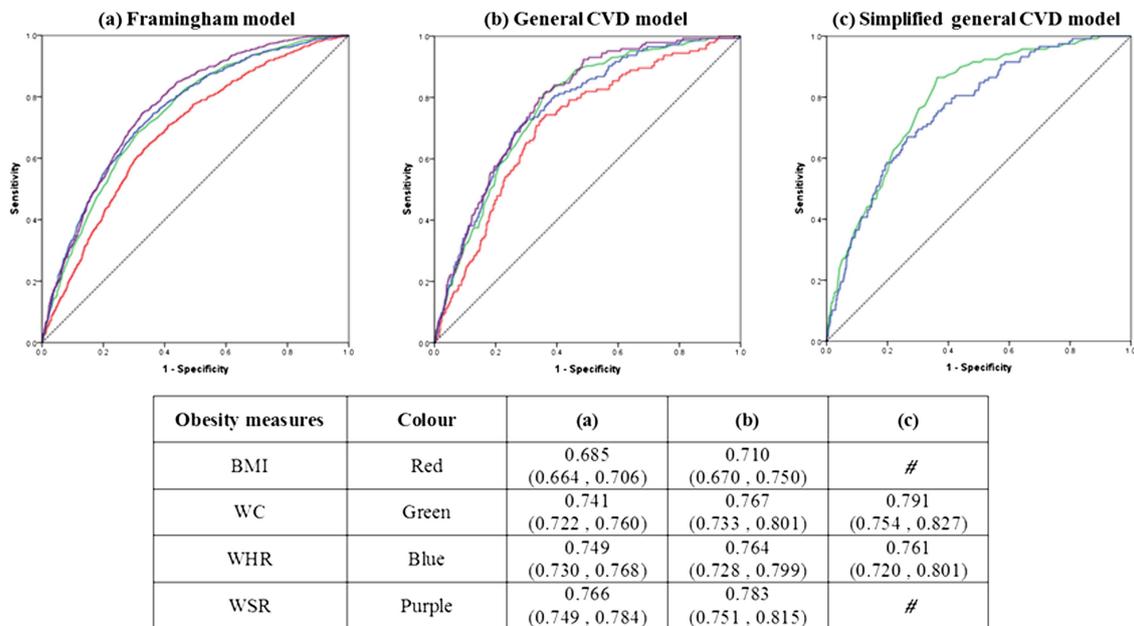


Figure 1 ROC curves to compare the predictive ability of obesity measures for being above the 20% cut-off of three CVD models: (A) Framingham risk score model for 10-year CVD incidence; (B) general cardiovascular disease risk score model for 10-year CVD incidence and death; (C) simplified general cardiovascular disease risk score model for 10-year CVD incidence and death. #Area under the ROC curve is not calculated for this obesity measure as it contains height which is also used in the calculation of the simplified general CVD model. BMI, body mass index; CVD, cardiovascular disease; ROC, receiver operating characteristic; WC, waist circumference; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

with CVD risk.³⁷ Some studies recommended the use of WC in clinical assessment and research studies.^{52–53} In a systematic review and meta-analysis study of Caucasians without CVD, WC was most highly correlated with all CVD risk factors, compared with BMI, WHR, WSR and body fat percentage, in women.⁵² In other studies, WC was also more closely associated with CVD risk factors than other measures of central obesity and BMI in women.^{54–57} The advantages of WC are: it is easy to measure and interpret and it is less prone to measurement and calculation error.⁵³ Appropriate sex, age and ethnic-specific WC cut-points would need to be established.⁴⁴ It would also be difficult to use WC in today's multicultural societies due to requirements for different cut-points.⁵⁰

The use of WHR is also supported as it is less strongly associated with BMI than WC, and is thus a more specific surrogate for fat distribution.⁴⁰ A longitudinal population study on 1462 women from Sweden reported stronger relations between WHR and CVD endpoints, compared with BMI, WC and HC.⁵⁸ These relations were mostly independent of age, BMI and either SBP, cholesterol level or smoking habit.⁵⁸ In a meta-regression analysis of prospective studies, WHR was also more strongly associated with CVD compared with WC, although the difference was not significant.³⁷ Another study reported that WHR was associated with CVD mortality but not WC in elderly women from the UK.⁵⁹ Elevated WHR was also independently associated with a higher CVD risk in the Nurses' Health Study and in the Swedish Women's Lifestyle and Health Cohort Study.^{45–60} Women with a WHR of ≥ 0.88 were 3.25 times

more at risk of CHD compared with women with a WHR of < 0.72 after adjusting for BMI and other CVD risk factors.⁴⁵ Higher age-adjusted and sex-adjusted ORs were also reported with WHR and CHD and CVD mortality, compared with WC and BMI, in an Australian population without heart disease, diabetes or stroke.⁶¹ Similar results were presented in other studies. WHR reported the highest age standardised HRs in relation to CVD mortality, followed by WSR, WC and BMI in women.^{62–63} The advantages of WHR are: it has low measurement error, high precision and no bias over a wide range of ethnic groups.⁶⁴ WHR, however, may not be suitable for assessing central obesity in the elderly⁶⁵ due to laxity of abdominal muscles which would undermine the predictive value of abdominal circumferences.⁵⁵ It is also more difficult to measure than WC.³⁷ Despite its limitations, WHR has been recommended for incorporation into CVD risk assessment.³⁷

WSR is the least commonly used measure of central obesity. In a systematic review and meta-analysis study, WSR reported the weakest correlations with CVD risk factors, compared with BMI and other measures of central obesity,⁵² which is contrary to our study findings. In contrast, WSR was most highly correlated with CHD risk predicted using the Framingham model¹⁸ in women from England, compared with BMI, WC and WHR in another study.⁶⁶ WSR, however, reported lower correlations than WC and BMI following adjustments for age.⁶⁶ The advantage of WSR is that the same cut-point could be applied across a wide range of populations. A cut-off value of 0.5 indicates increased risk for men and women and people of different ethnic groups, and this

value may also be used in children and adults, unlike WC which requires different cut-offs.^{67 68} More research is required to assess the association between WSR and CVD risk in women, in comparison with WC, WHR and BMI.

Our study has limitations. This study is cross sectional; however, it is a representative sample of the Australian female population. There is only one set of baseline measurements recorded for some risk variables but important variables including anthropometric measures of obesity are measured twice. Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against the treatment thresholds of the various models, and are not prospective CVD events.

CONCLUSIONS

Central obesity is more strongly associated with CVD risk than general obesity. The deposition of adipose tissue is associated with systemic inflammation which has a direct effect on CVD risk. Therefore, increments in central obesity have a more detrimental effect on CVD risk compared with increments in general obesity.

When used alone, BMI is inadequate for identifying individuals at increased risk of CVD as it does not differentiate between fat and fat-free mass. On the other hand, anthropometric measurements of central obesity have higher sensitivity and specificity. These measures are also more sensitive to lifestyle modifications. An increase in muscle mass through diet and training would lead to changes in measures such as WC and WSR but little change might be indicated with BMI.⁶⁹ It would be more useful to measure a patient's central obesity during clinical assessment to evaluate the effect of lifestyle changes in relation to CVD risk compared with BMI. Central obesity measures are also significant and independent predictors of CVD risk, accounting for additional risk above BMI. These measurements should be incorporated into CVD risk assessment, particularly when assessing the risk in women and the elderly.^{53 70-73}

Future prospective studies are required to elucidate which anthropometric measurements of central obesity are better indicators or predictors of CVD risk.⁶⁹ Studies measuring body fat distribution using CT or MRI are desirable to better understand the association between body fat distribution and mortality, but are costly.⁷⁴

In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of WC, should be considered for incorporation into the clinical assessment of CVD risk. Treatment of well-established CVD risk factors coupled with reducing overweight and obesity through lifestyle modifications would be an advisable goal in the primary prevention of CVD.⁴ It is equally important to maintain a healthy weight and to prevent central or abdominal obesity concurrently.

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Contributors LGHG was involved in drafting the manuscript, interpretation of the data and revising the manuscript critically for important intellectual content. SSD conceived the study, performed the analysis and data

interpretation and revised the manuscript critically for important intellectual content. TAW participated in the study design, acquired the data and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

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