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

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Anthropometric obesity measures and CVD risk

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ABSTRACT

Objectives: It is important to ascertain which anthropometric measurements of obesity, general or central, are better predictors of CVD risk in women. Ten-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 model. Increase in CVD risk associated with one standard deviation increment in each anthropometric measurement above the mean was calculated, and the diagnostic utility of obesity measures in identifying subjects 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, one standard deviation above the mean, and obesity measures that had the greatest diagnostic utility in identifying subjects 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 to body mass index (BMI). These central obesity measures also had higher sensitivity and specificity in identifying females above and below the 20% treatment threshold than BMI. Central obesity measures also recorded better correlations with CVD risk compared to 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 should be included in the clinical assessment of CVD risk in place or in addition to BMI. It is equally important to maintain a healthy weight and to prevent central obesity concurrently.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study provided evidence that anthropometric measures of central obesity are better predictors of CVD risk compared to general obesity measures in women.
- Central obesity measures add prognostic information on CVD risk in women above 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 CVD events was not used.

INTRODUCTION

The prevalence of obesity has reached epidemic or pandemic proportions. 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 ischemic 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 Study[7] have all shown that overweight and obesity are associated with increased 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] Both general and central obesity are associated with CVD risk.[5 9-14] 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 index[15] (BAI; HC divided by height$^{1.5}$, and subtracting 18 from the result). BMI or WC is most commonly used to measure body fatness.[9]

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 reported that BMI identified individuals at increased risk of CVD as effectively as WC.[10 11] In another study, BMI was a better predictor of CVD than WC.[12] Conversely, some studies reported that WC was a better indicator of CVD risk than BMI and WHR, in ethnically diverse groups.[13 14] Another study, however, reported that WC and WHR but not BMI were independent predictors of CVD risk, accounting for conventional risk factors in the Framingham risk score model.[16] More research is 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 females 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,[17] SCORE risk chart for high-risk regions for 10-year CVD death,[18] general CVD and simplified general CVD risk score model for 10-year CVD incidence and death[19] would also be assessed. To aid comparison between obesity indices, which are measured in different units, the incremental shift in CVD risk with one standard deviation increment in each anthropometric measurement above the mean would be assessed. Finally, we would determine which indices of obesity are most sensitive and specific for identifying females 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.[20] Information on demographic characteristics 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 hip circumference were collected according to standardised methodologies[21 22] using two observers. The mean of two measurements was taken at each site to the nearest centimetre.

Variables in risk score models

The Framingham 10-year predicted risk for CVD incidence or death[17] 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. Fewer variables were used in the calculation of the 10-year predicted CVD death with the SCORE risk chart for high-risk regions (Denmark, Finland and Norway),[18 23] these included: age, sex, smoking status, mean

total cholesterol level, mean HDL cholesterol level and mean SBP. Similar risk variables (age, SBP, current antihypertensive treatment, smoking status and diabetes status) were used in both the general CVD and simplified general CVD risk score model.[19] In the simplified general CVD risk score model, however, the total cholesterol level and HDL cholesterol level were replaced by BMI in the calculation of the 10-year risk for CVD incidence and death.

Statistical analysis

The data on the representative sample of 4487 Australian females was described using mean ± standard deviation 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 standard deviation) to represent the number of standard deviations above and below the mean for each subject. 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 a one standard deviation increment above the mean for each anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals 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 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 Version 20.

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 were 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 subject in the sample was calculated using four risk score models. The frequency distribution of calculated risks was 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 waist circumference (WC, WHR and WSR) generally recorded better correlations compared to 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 were 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.

Recommended treatment thresholds for the four CVD risk models were identified from a review of the literature. Table 4 presented the effects of a one standard deviation 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 odds-ratios 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) recorded higher area under the ROC curves, higher sensitivity and specificity, than BMI in identifying females 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) were reported for both WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements as compared to general obesity measurement in predicting the increased risk of CVD.

Table 1 Characteristics of a representative Australian sample of 4487 females (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%)
Smoking status – n (%)	
Non-smoker	2652 (59.1%)
Previous smoker	880 (19.6%)
Current smoker	955 (21.3%)
SBP (mmHg)	122.1 ± 18.4
DBP (mmHg)	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

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

Table 2 Frequency distribution of 10-year predicted CVD incidence and mortality using various risk prediction models, in incremental risk categories of 10%. Counts and percentages of females were presented.

	Risk categories				
	0-9%	**10-19%**	**20-29%**	**30-39%**	**≥40%**
Framingham 10-year predicted risk for CVD incidence[17]	2936 (67.0%)	764 (17.4%)	417 (9.5%)	179 (4.1%)	89 (2.0%)
Framingham 10-year predicted risk for CVD death[17]	4354 (99.3%)	29 (0.7%)	2 (0%)	0 (0%)	0 (0%)
SCORE-HIGH 10-year predicted risk for CVD death[18]	4318 (98.5%)	53 (1.2%)	9 (0.2%)	4 (0.1%)	1 (0%)
GCVD 10-year predicted risk for CVD incidence and death[19]	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[19]	3809 (85.7%)	519 (11.7%)	90 (2.0%)	19 (0.4%)	9 (0.2%)

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

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[17]	0.380	0.450	0.301	0.409	0.485	0.378
Framingham 10-year predicted risk for CVD death[17]	0.394	0.452	0.307	0.404	0.483	0.377
SCORE-HIGH 10-year predicted risk for CVD death[18]	0.309	0.381	0.253	0.348	0.419	0.338
GCVD 10-year predicted risk for CVD incidence and death[19]	0.385	0.452	0.307	0.405	0.487	0.383
SGCVD 10-year predicted risk for CVD incidence and death[19]	#	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.

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

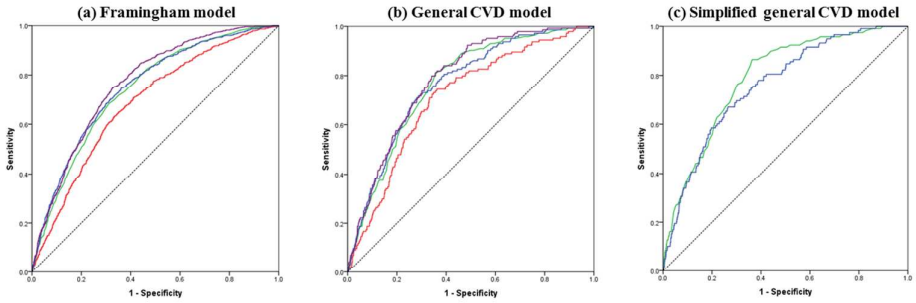
Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity

BMI	WC	HC	WHR	WSR	BAI
Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[24 25]					
1.71*** (1.59 - 1.85)	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%)[24 25]					
1.68 (0.98 - 2.87)	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%)[18]					
1.53*** (1.29 - 1.82)	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%)[26]					
1.72*** (1.59 - 1.86)	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%)[26]					
#	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%)[19 27]					
1.64*** (1.44 - 1.86)	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%)[19 27]					
#	2.26*** (1.96 - 2.60)	1.72*** (1.50 - 1.99)	2.11*** (1.82 - 2.45)	#	#

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

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

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



Obesity measures	Colour	(a)	(b)	(c)
BMI	Red	0.685 (0.664 , 0.706)	0.710 (0.670 , 0.750)	#
WC	Green	0.741 (0.722 , 0.760)	0.767 (0.733 , 0.801)	0.791 (0.754 , 0.827)
WHR	Blue	0.749 (0.730 , 0.768)	0.764 (0.728 , 0.799)	0.761 (0.720 , 0.801)
WSR	Purple	0.766 (0.749 , 0.784)	0.783 (0.751 , 0.815)	#

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.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

DISCUSSION

Overall, 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 model, compared to general measures of obesity. Central obesity measures also recorded higher odds-ratios and increased the likelihood of being above the recommended treatment threshold of the respective models with one standard deviation increase above the mean. Consistent with our study findings, previous studies also reported stronger associations between central obesity measures and CVD risk. Higher standardised odds-ratios adjusted for BMI were reported for WC and CVD, compared to BMI, in women from the International Day for the Evaluation of Abdominal Obesity (IDEA) study.[28 29] An increase in WC was associated with being 4.25 times more likely of stroke and transient ischemic attacks.[30] Central obesity measures which incorporated the measure of waist circumference 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. Conversely, some studies reported that the association between BMI and CVD was similar to measures of central obesity.[31 32]

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.[33] 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.[34-37] 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 mortality[38] and provides incremental value in predicting CVD above and beyond that provided by general obesity measures.[39-43] BMI alone is thus insufficient to account for the association

between obesity and CVD risk. BMI is also 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.[44-48] 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. To date, BMI is included in the simplified general CVD risk score model as an alternative to total and HDL cholesterol level considering its ease of measurement and calculation,[19] and in the QRISK score model.[49] A collaborative analysis of 58 prospective studies, however, reported that both measures of general and central obesity did not improve CVD risk assessment when information is available on SBP, diabetes and lipids.[50] Overweight and obesity is nevertheless important in CVD prevention, with one out of three of fatal and one out of seven of non-fatal CVD cases attributable to it.[32]

Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and its association with CVD risk.[35] Some studies recommended the use of WC in clinical assessment and research studies.[51 52] In a systematic review and meta-analysis study of Caucasians without CVD, WC was most highly correlated with all CVD risk factors, compared to BMI, WHR, WSR and body fat percentage, in women.[51] In other studies, WC was also more closely associated with CVD risk factors than other measures of central obesity and BMI in women.[53-56] The advantages of WC are, it is easy to measure and interpret, and it is less prone to measurement and calculation error.[52] Appropriate sex, age and ethnic-specific WC cut points would need to be established.[42] It would also be difficult to use WC in today's multicultural societies due to requirements for different cut points.[48]

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.[38] A longitudinal population study on 1462 women from Sweden reported stronger relations between WHR and CVD endpoints, compared to BMI, WC and

HC.[57] These relations were mostly independent of age, BMI and either SBP, cholesterol level or smoking habit.[57] In a meta-regression analysis of prospective studies, WHR was also more strongly associated with CVD compared to WC, although the difference was not significant.[35] Another study reported that WHR was associated with CVD mortality but not WC in elderly women from the United Kingdom.[58] 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.[43 59] Women with a WHR of ≥ 0.88 were 3.25 times more at risk of CHD compared to women with a WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[43] Higher age and sex adjusted odds-ratios were also reported with WHR and CHD and CVD mortality, compared to WC and BMI, in an Australian population without heart disease, diabetes or stroke.[60] Similar results were presented in other studies. WHR reported the highest age standardised hazard ratios in relation to CVD mortality, followed by WSR, WC and BMI in women.[61 62] The advantages of WHR include, it has low measurement error, high precision and no bias over a wide range of ethnic groups.[63] WHR, however, may not be suitable for assessing central obesity in the elderly[64] due to laxity of abdominal muscles which would undermine the predictive value of abdominal circumferences.[54] It is also more difficult to measure than WC.[35] Despite its limitations, WHR has been recommended for incorporation into CVD risk assessment.[35]

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 to BMI and other measures of central obesity,[51] which is contrary to our study findings. In contrast, WSR was most highly correlated with CHD risk predicted using the Framingham model[17] in women from England, compared to BMI, WC and WHR in another study.[65] WSR, however, reported lower correlations than WC and BMI following adjustments for age.[65] The advantage of WSR include, 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, people of different ethnic groups and this value may also be used in both children and adults, unlike WC which requires different cut-offs.[66 67] 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

The significant and independent effect of obesity measures on CVD risk substantiates its inclusion into risk score models. 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 to 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.[68] 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 to 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.[52 69-72]

Future prospective studies are required to elucidate which anthropometric measurements of central obesity are better indicators or predictors of CVD risk.[68] Studies measuring body fat distribution using computerised tomography or magnetic resonance imaging are desirable to better understand the association between body fat distribution and mortality, but costly.[73]

In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of waist circumference, 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 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.

Competing interests None.

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Ethics approval We have ethics approval for the use of the National Heart Foundation data from the Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin University. This study was carried out in accordance with the Declaration of Helsinki.

Data sharing statement No additional data are available.

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STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cross-sectional studies*

Section/Topic	Item #	Recommendation	Reported on page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	5
Objectives	3	State specific objectives, including any prespecified hypotheses	6
Methods			
Study design	4	Present key elements of study design early in the paper	6
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	6
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	6
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	6
Bias	9	Describe any efforts to address potential sources of bias	*6*
Study size	10	Explain how the study size was arrived at	N.A.
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	7
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	7
		(*b*) Describe any methods used to examine subgroups and interactions	N.A.
		(*c*) Explain how missing data were addressed	N.A.
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	N.A.
		(*e*) Describe any sensitivity analyses	N.A.
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	8
		(b) Give reasons for non-participation at each stage	N.A.
		(c) Consider use of a flow diagram	N.A.
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	8,10
		(b) Indicate number of participants with missing data for each variable of interest	N.A.
Outcome data	15*	Report numbers of outcome events or summary measures	8-14
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	8-14
		(*b*) Report category boundaries when continuous variables were categorized	8,10,11
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N.A.
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	9,14
Discussion			
Key results	18	Summarise key results with reference to study objectives	15
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	18
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	18
Generalisability	21	Discuss the generalisability (external validity) of the study results	18
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	N.A.

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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

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Anthropometric obesity measures and CVD risk

Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female

ABSTRACT

Objectives: It is important to ascertain which anthropometric measurements of obesity, general or central, are better predictors of CVD risk in women. Ten-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 one standard deviation 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, one standard deviation above the mean, and obesity measures that had the greatest diagnostic utility in identifying subjects 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 to body mass index (BMI). These central obesity measures also had higher sensitivity and specificity in identifying females above and below the 20% treatment threshold than BMI. Central obesity measures also recorded better correlations with CVD risk compared to 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 to general obesity measures in women. It is equally important to maintain a healthy weight and to prevent central obesity concurrently.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study provided evidence that anthropometric measures of central obesity are better predictors of CVD risk compared to general obesity measures in women.
- Central obesity measures add prognostic information on CVD risk in women above 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 CVD events was not used.

INTRODUCTION

The prevalence of obesity has reached epidemic proportions. 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 ischemic 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 Study[7] have all shown that overweight and obesity are associated with increased 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] Both 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 index[16] (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 females 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 death[20] would also be assessed. To aid comparison between obesity indices, which are measured in different units, the incremental shift in CVD risk with one standard deviation 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 females 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 hip circumference were collected according to standardised methodologies[22 23] using two observers. The waist circumference was measured from the front at the narrowest point between the rib cage and

iliac crest after full expiration while the hip circumference 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.

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] It 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 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 of the models.[20] The only difference is, 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 females was described using mean ± standard deviation 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 standard deviation) to represent the number of standard deviations above and below the mean for each subject. 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 a one standard deviation increment above the mean for each anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals 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 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 Version 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 waist circumference (WC, WHR and WSR) generally recorded better correlations compared to 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 to 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 a one standard deviation 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 odds-ratios 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 females 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 both WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements as compared to general obesity measurement in predicting the increased risk of CVD.

Table 1 Characteristics of a representative Australian sample of 4487 females (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%)
United Kingdom 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 (mmHg)	122.1 ± 18.4
DBP (mmHg)	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

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

Table 2 Frequency distribution of 10-year predicted CVD incidence and mortality using various risk prediction models, in incremental risk categories of 10%. Counts and percentages of females were presented.

	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%)

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

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.

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

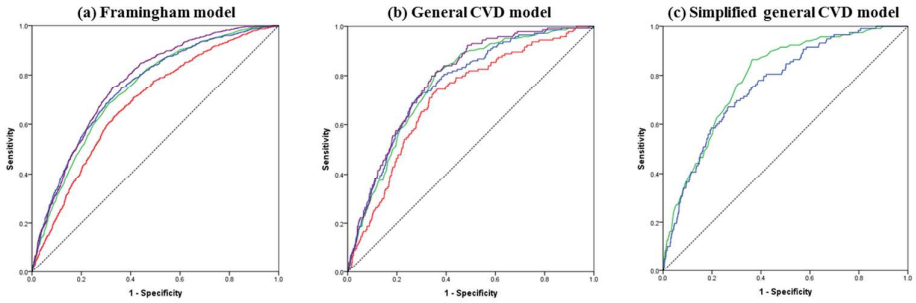
Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity

BMI	WC	HC	WHR	WSR	BAI
Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[25 26]					
1.71*** (1.59 - 1.85)	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]					
1.68 (0.98 - 2.87)	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]					
1.53*** (1.29 - 1.82)	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]					
1.72*** (1.59 - 1.86)	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]					
1.64*** (1.44 - 1.86)	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)	#	#

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

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

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



Obesity measures	Colour	(a)	(b)	(c)
BMI	Red	0.685 (0.664 , 0.706)	0.710 (0.670 , 0.750)	#
WC	Green	0.741 (0.722 , 0.760)	0.767 (0.733 , 0.801)	0.791 (0.754 , 0.827)
WHR	Blue	0.749 (0.730 , 0.768)	0.764 (0.728 , 0.799)	0.761 (0.720 , 0.801)
WSR	Purple	0.766 (0.749 , 0.784)	0.783 (0.751 , 0.815)	#

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.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

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 model, compared to general measures of obesity. Central obesity measures also recorded higher odds-ratios and increased the likelihood of being above the recommended treatment threshold of the respective models with one standard deviation increase above the mean. Central obesity measures which incorporated the measure of waist circumference 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 odds-ratios adjusted for BMI were reported for WC and CVD, compared to 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 ischemic 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 mortality[40] 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 both 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 is nevertheless important in CVD prevention, with one out of three of fatal and one out of seven of 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 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 to 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 to 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 to 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 United Kingdom.[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 to women with a WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted odds-ratios were also reported with WHR and CHD and CVD mortality, compared to 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 hazard ratios in relation to CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include, 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 elderly[65] 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 to 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 model[18] in women from England, compared to 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 include, 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, people of different ethnic groups and this value may also be used in both 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 to 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 to 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 computerised tomography or magnetic resonance imaging are desirable to better understand the association between body fat distribution and mortality, but costly.[74]

In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of waist circumference, 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 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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Anthropometric measurements of general and central obesity and the prediction of cardiovascular disease risk in women: a cross-sectional study

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Anthropometric obesity measures and CVD risk

Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female

ABSTRACT

Objectives: It is important to ascertain which anthropometric measurements of obesity, general or central, are better predictors of CVD risk in women. Ten-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 one standard deviation increment in each anthropometric measurement above the mean was calculated, and the diagnostic utility of obesity measures in identifying participants~~subjects~~ 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, one standard deviation above the mean, and obesity measures that had the greatest diagnostic utility in identifying subjects 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 to body mass index (BMI). These central obesity measures also had higher sensitivity and specificity in identifying females above and below the 20% treatment threshold than BMI. Central obesity measures also recorded better correlations with CVD risk compared to 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 to general obesity measures in women. ~~Central obesity measures should be included in the clinical assessment of CVD risk in place or in addition to BMI.~~ It is equally important to maintain a healthy weight and to prevent central obesity concurrently.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study provided evidence that anthropometric measures of central obesity are better predictors of CVD risk compared to general obesity measures in women.
- Central obesity measures add prognostic information on CVD risk in women above 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 CVD events was not used.

INTRODUCTION

The prevalence of obesity has reached epidemic ~~or pandemic~~ proportions. 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 ischemic 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 Study[7] have all shown that overweight and obesity are associated with increased 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] Both 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 index[16] (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] ~~In~~ It has also been suggested that ~~another study,~~ BMI i~~wa~~s a better predictor of CVD than WC.[13] Conversely, some studies reported that WC i~~wa~~s a better indicator of CVD risk than BMI and WHR, in ethnically diverse groups.[14 15] ~~Another study, however, reported that~~ WC and WHR ~~but not BMI were~~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 females 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 death[20] would also be assessed. To aid comparison between obesity indices, which are measured in different units, the incremental shift in CVD risk with one standard deviation increment in each anthropometric measurement above the mean would be assessed. Finally, we ~~would~~ determined which indices of obesity are most sensitive and specific for identifying females 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 hip circumference were collected according to standardised methodologies[22 23] using two observers.

The waist circumference was measured from the front at the narrowest point between the rib cage and iliac crest after full expiration while the hip circumference 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.

~~Variables in r~~Risk score models

The Framingham 10-year predicted risk for CVD incidence or death[18] 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] It was derived from a much larger dataset than the Framingham, ~~and~~ 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 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. ~~Similar r~~Risk variables (age, SBP, current antihypertensive treatment, smoking status and diabetes status) were used in both of the ~~the general CVD and simplified general CVD risk score~~ models.[20]~~-~~ The only difference is, 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. ~~In the simplified general CVD risk score model, however, the total~~

~~cholesterol level and HDL cholesterol level were replaced by BMI in the calculation of the 10-year risk for CVD incidence and death.~~

Statistical analysis

The data on the representative sample of 4487 Australian females was described using mean ± standard deviation 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 standard deviation) to represent the number of standard deviations above and below the mean for each subject. 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 a one standard deviation increment above the mean for each anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals 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 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 Version 21~~0~~.

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 ~~were~~ 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 ~~subject~~ participant in the sample was calculated using four risk score models. The frequency distribution of calculated risks ~~was~~ 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 waist circumference (WC, WHR and WSR) generally recorded better correlations compared to 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~~were~~ 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 to measures of general obesity~~.~~

Recommended treatment thresholds for the four CVD risk models were identified from a review of the literature. Table 4 ~~presented~~ presents the effects of a one standard deviation 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 odds-ratios 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 females 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~~were~~ reported for both WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements as compared to general obesity measurement in predicting the increased risk of CVD.

Table 1 Characteristics of a representative Australian sample of 4487 females (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%)
United Kingdom 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 (mmHg)	122.1 ± 18.4
DBP (mmHg)	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

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

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Table 2 Frequency distribution of 10-year predicted CVD incidence and mortality using various risk prediction models, in incremental risk categories of 10%. Counts and percentages of females were presented.

	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%)

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

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.

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

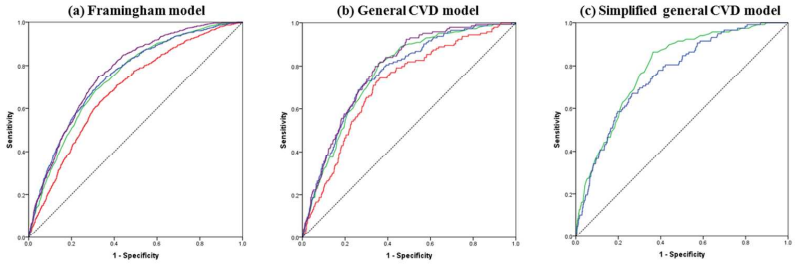
Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity

BMI	WC	HC	WHR	WSR	BAI
Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[25 26]					
1.71*** (1.59 - 1.85)	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]					
1.68 (0.98 - 2.87)	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]					
1.53*** (1.29 - 1.82)	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]					
1.72*** (1.59 - 1.86)	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]					
1.64*** (1.44 - 1.86)	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)	#	#

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

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

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



Obesity measures	Colour	(a)	(b)	(c)
BMI	Red	0.685 (0.664 , 0.706)	0.710 (0.670 , 0.750)	#
WC	Green	0.741 (0.722 , 0.760)	0.767 (0.733 , 0.801)	0.791 (0.754 , 0.827)
WHR	Blue	0.749 (0.730 , 0.768)	0.764 (0.728 , 0.799)	0.761 (0.720 , 0.801)
WSR	Purple	0.766 (0.749 , 0.784)	0.783 (0.751 , 0.815)	#

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.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

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, ~~Overall,~~ 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 model, compared to general measures of obesity. Central obesity measures also recorded higher odds-ratios and increased the likelihood of being above the recommended treatment threshold of the respective models with one standard deviation increase above the mean. Central obesity measures which incorporated the measure of waist circumference 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 odds-ratios adjusted for BMI were reported for WC and CVD, compared to 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 ischemic attacks.[32] ~~Central obesity measures which incorporated the measure of waist circumference 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. 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 mortality[40] and provides incremental value in predicting CVD above and beyond that provided by general obesity measures.[41-45] BMI alone is thus insufficient to account for the association between obesity and CVD risk. BMI is also 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. To date, BMI is included in the simplified general CVD risk score model as an alternative to total and HDL cholesterol level considering its ease of measurement and calculation,[20] and in the QRISK score model.[29] A collaborative analysis of 58 prospective studies, however, reported that both 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 is nevertheless important in CVD prevention, with one out of three of fatal and one out of seven of 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 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 to 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 to 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 to 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 United Kingdom.[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 to women with a WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted odds-ratios were also reported with WHR and CHD and CVD mortality, compared to 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 hazard ratios in relation to CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include, 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 elderly[65] 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 to 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 model[18] in women from England, compared to 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 include, 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, people of different ethnic groups and this value may also be used in both 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

~~The significant and independent effect of obesity measures on CVD risk substantiates its inclusion into risk score models.~~ 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 to 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 to 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 computerised tomography or magnetic resonance imaging are desirable to better understand the association between body fat distribution and mortality, but costly.[74]

In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of waist circumference, 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 data and revising the manuscript critically for important intellectual content. SSD conceived the study, performed the

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STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cross-sectional studies*

Section/Topic	Item #	Recommendation	Reported on page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	5
Objectives	3	State specific objectives, including any prespecified hypotheses	6
Methods			
Study design	4	Present key elements of study design early in the paper	6
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	6
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	6
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	6
Bias	9	Describe any efforts to address potential sources of bias	*6*
Study size	10	Explain how the study size was arrived at	N.A.
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	8
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	8
		(*b*) Describe any methods used to examine subgroups and interactions	N.A.
		(*c*) Explain how missing data were addressed	N.A.
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	N.A.
		(*e*) Describe any sensitivity analyses	N.A.
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	8
		(b) Give reasons for non-participation at each stage	N.A.
		(c) Consider use of a flow diagram	N.A.
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	8,11
		(b) Indicate number of participants with missing data for each variable of interest	N.A.
Outcome data	15*	Report numbers of outcome events or summary measures	8-15
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	8-15
		(*b*) Report category boundaries when continuous variables were categorized	8,11,12
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N.A.
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	9-10,15
Discussion			
Key results	18	Summarise key results with reference to study objectives	16
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	19
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	19
Generalisability	21	Discuss the generalisability (external validity) of the study results	19
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	N.A.

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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

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

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Anthropometric obesity measures and CVD risk

Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female

ABSTRACT

Objectives: It is important to ascertain which anthropometric measurements of obesity, general or central, are better predictors of CVD risk in women. Ten-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 one standard deviation 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, one standard deviation above the mean, and obesity measures that had the greatest diagnostic utility in identifying subjects 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 to body mass index (BMI). These central obesity measures also had higher sensitivity and specificity in identifying females above and below the 20% treatment threshold than BMI. Central obesity measures also recorded better correlations with CVD risk compared to 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 to general obesity measures in women. It is equally important to maintain a healthy weight and to prevent central obesity concurrently.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study provided evidence that anthropometric measures of central obesity are better predictors of CVD risk compared to general obesity measures in women.
- Central obesity measures add prognostic information on CVD risk in women above 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 CVD events was 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 ischemic 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 Study[7] have all shown that overweight and obesity are associated with increased 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] Both 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 index[16] (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 females 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 death[20] were examined. To aid comparison between obesity indices, which are measured in different units, the incremental shift in CVD risk with one standard deviation 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 females 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 hip circumference were collected according to standardised methodologies[22 23] using two observers. The waist circumference was measured from the front at the narrowest point between the rib cage and

iliac crest after full expiration while the hip circumference 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 five minutes apart.[21] Two readings were taken and the average was used in the analysis. Fasting blood samples were also collected in EDTA tubes and despatched 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 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 of the models.[20] The only difference is, 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 females were described using mean ± standard deviation 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 standard deviation) to represent the number of standard deviations above and below the mean for each subject. 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 a one standard deviation increment above the mean for each anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals 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 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 Version 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 waist circumference (WC, WHR and WSR) generally recorded better correlations compared to 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 to 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 a one standard deviation 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 odds-ratios 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 females 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 both WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements as compared to general obesity measurement in predicting the increased risk of CVD.

Table 1 Characteristics of a representative Australian sample of 4487 females (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%)
United Kingdom 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 (mmHg)	122.1 ± 18.4
DBP (mmHg)	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

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

Table 2 Frequency distribution of 10-year predicted CVD incidence and mortality using various risk prediction models, in incremental risk categories of 10%. Counts and percentages of females were presented.

	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%)

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

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.

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

Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation 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]					
1.71*** (1.59 - 1.85)	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]					
1.68 (0.98 - 2.87)	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]					
1.53*** (1.29 - 1.82)	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]					
1.72*** (1.59 - 1.86)	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]					
1.64*** (1.44 - 1.86)	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)	#	#

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

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

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

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 model, compared to general measures of obesity. Central obesity measures also recorded higher odds-ratios and increased the likelihood of being above the recommended treatment threshold of the respective models with one standard deviation increase above the mean. Central obesity measures which incorporated the measure of waist circumference 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 odds-ratios adjusted for BMI were reported for WC and CVD, compared to 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 ischemic 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 mortality[40] 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 both 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 is nevertheless important in CVD prevention, with one out of three of fatal and one out of seven of 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 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 to 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 to 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 to 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 United Kingdom.[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 to women with a WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted odds-ratios were also reported with WHR and CHD and CVD mortality, compared to 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 hazard ratios in relation to CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include, 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 elderly[65] 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 to 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 model[18] in women from England, compared to 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 include, 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, people of different ethnic groups and this value may also be used in both 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 to 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 to 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 computerised tomography or magnetic resonance imaging are desirable to better understand the association between body fat distribution and mortality, but costly.[74]

In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of waist circumference, 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.

Figure legend

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.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

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Contributors LGHG was involved in drafting the manuscript, interpretation of 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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Anthropometric measurements of general and central obesity and the prediction of cardiovascular disease risk in women: a cross-sectional study

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Anthropometric obesity measures and CVD risk

Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female

ABSTRACT

Objectives: It is important to ascertain which anthropometric measurements of obesity, general or central, are better predictors of CVD risk in women. Ten-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 one standard deviation 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, one standard deviation above the mean, and obesity measures that had the greatest diagnostic utility in identifying subjects 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 to body mass index (BMI). These central obesity measures also had higher sensitivity and specificity in identifying females above and below the 20% treatment threshold than BMI. Central obesity measures also recorded better correlations with CVD risk compared to 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 to general obesity measures in women. It is equally important to maintain a healthy weight and to prevent central obesity concurrently.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study provided evidence that anthropometric measures of central obesity are better predictors of CVD risk compared to general obesity measures in women.
- Central obesity measures add prognostic information on CVD risk in women above 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 CVD events was not used.

INTRODUCTION

~~The prevalence of obesity has reached epidemic proportions.~~ 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 ischemic 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 Study[7] have all shown that overweight and obesity are associated with increased 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] Both 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 index[16] (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 females 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 death[20] ~~would also be assessed~~were examined. To aid comparison between obesity indices, which are measured in different units, the incremental shift in CVD risk with one standard deviation 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 females 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 hip circumference were collected according to standardised methodologies[22 23] using two observers. The waist circumference was measured from the front at the narrowest point between the rib cage and

iliac crest after full expiration while the hip circumference 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 five minutes apart.[21] Two readings were taken and the average was used in the analysis. Fasting blood samples were also collected in EDTA tubes and despatched 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] ~~It~~ 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 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 of the models.[20] The only difference is, 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 females ~~was~~ were described using mean ± standard deviation 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 standard deviation) to represent the number of standard deviations above and below the mean for each subject. 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 a one standard deviation increment above the mean for each anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals 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 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 Version 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 waist circumference (WC, WHR and WSR) generally recorded better correlations compared to 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 to 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 a one standard deviation 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 odds-ratios 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 females 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 both WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements as compared to general obesity measurement in predicting the increased risk of CVD.

Table 1 Characteristics of a representative Australian sample of 4487 females (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%)
United Kingdom 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 (mmHg)	122.1 ± 18.4
DBP (mmHg)	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

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

Table 2 Frequency distribution of 10-year predicted CVD incidence and mortality using various risk prediction models, in incremental risk categories of 10%. Counts and percentages of females were presented.

	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%)

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

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.

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

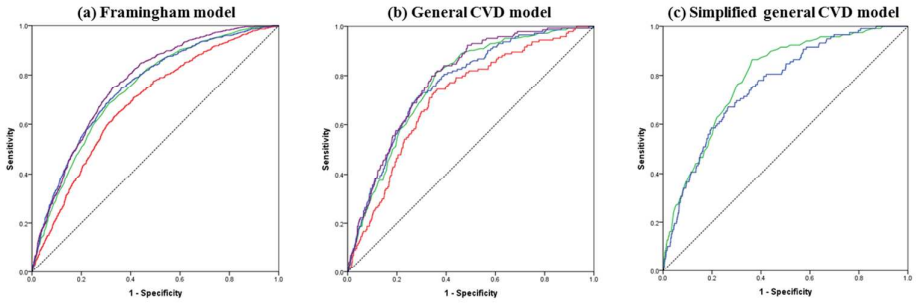
Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity

BMI	WC	HC	WHR	WSR	BAI
Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[25 26]					
1.71*** (1.59 - 1.85)	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]					
1.68 (0.98 - 2.87)	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]					
1.53*** (1.29 - 1.82)	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]					
1.72*** (1.59 - 1.86)	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]					
1.64*** (1.44 - 1.86)	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)	#	#

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

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

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



Obesity measures	Colour	(a)	(b)	(c)
BMI	Red	0.685 (0.664 , 0.706)	0.710 (0.670 , 0.750)	#
WC	Green	0.741 (0.722 , 0.760)	0.767 (0.733 , 0.801)	0.791 (0.754 , 0.827)
WHR	Blue	0.749 (0.730 , 0.768)	0.764 (0.728 , 0.799)	0.761 (0.720 , 0.801)
WSR	Purple	0.766 (0.749 , 0.784)	0.783 (0.751 , 0.815)	#

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.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WSR, waist-to-stature ratio.

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 model, compared to general measures of obesity. Central obesity measures also recorded higher odds-ratios and increased the likelihood of being above the recommended treatment threshold of the respective models with one standard deviation increase above the mean. Central obesity measures which incorporated the measure of waist circumference 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 odds-ratios adjusted for BMI were reported for WC and CVD, compared to 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 ischemic 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 mortality[40] 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 both 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 is nevertheless important in CVD prevention, with one out of three of fatal and one out of seven of 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 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 to 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 to 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 to 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 United Kingdom.[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 to women with a WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted odds-ratios were also reported with WHR and CHD and CVD mortality, compared to 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 hazard ratios in relation to CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include, 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 elderly[65] 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 to 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 model[18] in women from England, compared to 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 include, 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, people of different ethnic groups and this value may also be used in both 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 to 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 to 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 computerised tomography or magnetic resonance imaging are desirable to better understand the association between body fat distribution and mortality, but costly.[74]

In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of waist circumference, 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 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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Ethics approval We have ethics approval for the use of the National Heart Foundation data from the Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin University. This study was carried out in accordance with the Declaration of Helsinki.

Data sharing statement No additional data are available.

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STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cross-sectional studies*

Section/Topic	Item #	Recommendation	Reported on page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	5
Objectives	3	State specific objectives, including any prespecified hypotheses	6
Methods			
Study design	4	Present key elements of study design early in the paper	6
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants	6
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	6
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	6
Bias	9	Describe any efforts to address potential sources of bias	*6*
Study size	10	Explain how the study size was arrived at	N.A.
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	8
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	8
		(*b*) Describe any methods used to examine subgroups and interactions	N.A.
		(*c*) Explain how missing data were addressed	N.A.
		(*d*) If applicable, describe analytical methods taking account of sampling strategy	N.A.
		(*e*) Describe any sensitivity analyses	N.A.
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	8
		(b) Give reasons for non-participation at each stage	N.A.
		(c) Consider use of a flow diagram	N.A.
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	8,11
		(b) Indicate number of participants with missing data for each variable of interest	N.A.
Outcome data	15*	Report numbers of outcome events or summary measures	8-15
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	8-15
		(*b*) Report category boundaries when continuous variables were categorized	8,11,12
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	N.A.
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	9-10,15
Discussion			
Key results	18	Summarise key results with reference to study objectives	16
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	19
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	19-20
Generalisability	21	Discuss the generalisability (external validity) of the study results	19-20
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	N.A.

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.