



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

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6 **the prediction of cardiovascular disease risk in women: a cross-**
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9 **sectional study**
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44 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.

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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.

For peer review only

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.

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3 We aim to assess the associations between general and central obesity anthropometric measures with
4 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart
5 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk
6 calculated from the Framingham risk score model for 10-year CVD incidence or death,[17] SCORE
7 risk chart for high-risk regions for 10-year CVD death,[18] general CVD and simplified general CVD
8 risk score model for 10-year CVD incidence and death[19] would also be assessed. To aid comparison
9 between obesity indices, which are measured in different units, the incremental shift in CVD risk with
10 one standard deviation increment in each anthropometric measurement above the mean would be
11 assessed. Finally, we would determine which indices of obesity are most sensitive and specific for
12 identifying females at increased 10-year CVD risk.
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25 **METHODS**

26 **Study cohort and measurements**

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28 We selected 4487 women aged 20-69 years with no history of heart disease, diabetes or stroke from
29 the population representative sample of 4727 women from the National Heart Foundation (NHF)
30 Risk Factor Prevalence Study.[20] Information on demographic characteristics and conventional CVD
31 risk variables recorded in this prevalence study include: anthropometric measures, smoking status,
32 systolic and diastolic blood pressure, and lipid levels. Physical measurements of height (to the nearest
33 centimetre), weight (to the nearest 10th of a kilogram), and waist and hip circumference were
34 collected according to standardised methodologies[21 22] using two observers. The mean of two
35 measurements was taken at each site to the nearest centimetre.
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48 **Variables in risk score models**

49 The Framingham 10-year predicted risk for CVD incidence or death[17] was calculated using these
50 variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level,
51 high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status. Fewer variables
52 were used in the calculation of the 10-year predicted CVD death with the SCORE risk chart for high-
53 risk regions (Denmark, Finland and Norway),[18 23] these included: age, sex, smoking status, mean
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3 total cholesterol level, mean HDL cholesterol level and mean SBP. Similar risk variables (age, SBP,
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5 current antihypertensive treatment, smoking status and diabetes status) were used in both the general
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7 CVD and simplified general CVD risk score model.[19] In the simplified general CVD risk score
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9 model, however, the total cholesterol level and HDL cholesterol level were replaced by BMI in the
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11 calculation of the 10-year risk for CVD incidence and death.
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13 14 15 **Statistical analysis**

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17 The data on the representative sample of 4487 Australian females was described using mean \pm
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19 standard deviation for continuous variables, while counts and percentages were used for categorical
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21 variables. Non-parametric Spearman's rank correlation was used to assess the associations between
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23 anthropometric measurements of obesity with CVD risk factors, and with the calculated 10-year
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25 predicted risks, due to the skewness in the distribution of some variables. Anthropometric
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27 measurements were also converted to z-scores (original value subtracted by the mean and result
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29 divided by the standard deviation) to represent the number of standard deviations above and below the
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31 mean for each subject. Logistic regression was used to assess the effects of each standardised
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33 anthropometric measurement of being above the recommended treatment thresholds for various risk
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35 score models as a result of a one standard deviation increment above the mean for each
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37 anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals represented
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39 the likelihood of being above the recommended treatment thresholds for the specific risk score models
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41 (20% for the Framingham risk score model for 10-year CVD incidence or death; 10% for SCORE risk
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43 chart for high-risk regions for 10-year CVD death; 10% and 20% for the general CVD and simplified
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45 general CVD risk score models for 10-year CVD incidence and death). The predictive ability of these
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47 anthropometric measures to identify individuals above and below the treatment thresholds was
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49 assessed using sensitivity, specificity and area under the receiver operating characteristic (ROC)
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51 curve. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses
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53 were performed with IBM SPSS Statistics Version 20.
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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

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3 anthropometric measurement above the mean on the likelihood of being above the recommended
4 thresholds or being indicated for treatment. All anthropometric measures of central obesity (WC,
5 WHR and WSR) generally recorded higher odds-ratios than general measures of obesity and they
6 increased the likelihood of individuals being above the respective treatment thresholds.
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12 Anthropometric measurements of central obesity (WC, WHR and WSR) recorded higher area under
13 the ROC curves, higher sensitivity and specificity, than BMI in identifying females above and below
14 the 20% treatment threshold for the Framingham model for 10-year CVD incidence (Figure 1a) and
15 general CVD model for 10-year CVD incidence and death (Figure 1b). Although BMI is included in
16 the simplified general CVD model, high area under the ROC curve (> 0.76) were reported for both
17 WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements
18 as compared to general obesity measurement in predicting the increased risk of CVD.
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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
<i>Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[24 25]</i>					
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)
<i>Framingham 10-year predicted risk for CVD death (threshold = 20%)[24 25]</i>					
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)
<i>SCORE-HIGH 10-year predicted risk for CVD death (threshold = 10%)[18]</i>					
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)
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[26]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[26]</i>					
#	2.16 ^{***} (1.99 - 2.34)	1.66 ^{***} (1.54 - 1.80)	2.16 ^{***} (1.98 - 2.35)	#	#
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[19 27]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[19 27]</i>					
#	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.

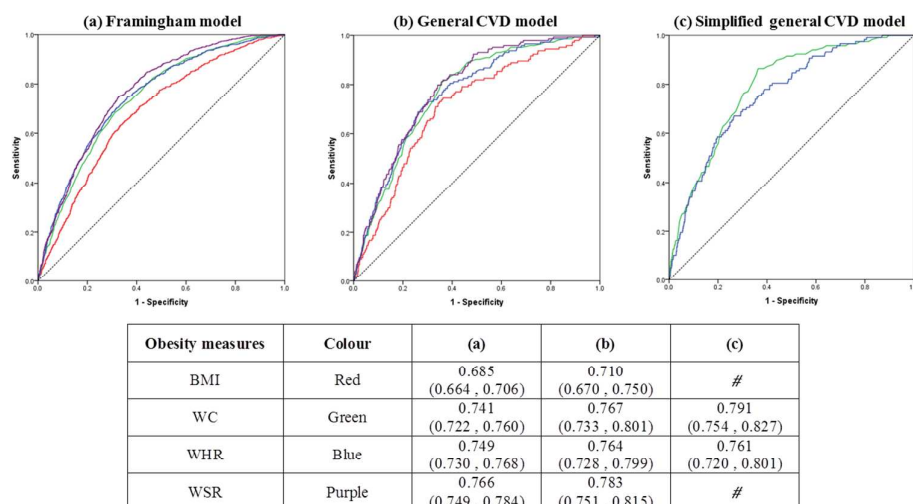


Figure 1 ROC curves to compare the predictive ability of obesity measures for being above the 20% cut-off of three CVD models: (a) Framingham risk score model for 10-year CVD incidence; (b) General cardiovascular disease risk score model for 10-year CVD incidence and death; (c) Simplified general cardiovascular 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

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3 between obesity and CVD risk. BMI is also a flawed measure as it does not correctly identify
4 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does
5 not account for the effect of age and ethnicity on body fat distribution.[44-48] An increase in muscle
6 or fat-free mass would, however, be reflected in the central obesity measures.
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12 Among central obesity measures, we found their performance to be comparable in our study. It
13 remains unclear which measurement should be incorporated into CVD risk score models. To date,
14 BMI is included in the simplified general CVD risk score model as an alternative to total and HDL
15 cholesterol level considering its ease of measurement and calculation,[19] and in the QRISK score
16 model.[49] A collaborative analysis of 58 prospective studies, however, reported that both measures
17 of general and central obesity did not improve CVD risk assessment when information is available on
18 SBP, diabetes and lipids.[50] Overweight and obesity is nevertheless important in CVD prevention,
19 with one out of three of fatal and one out of seven of non-fatal CVD cases attributable to it.[32]
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31 Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and
32 its association with CVD risk.[35] Some studies recommended the use of WC in clinical assessment
33 and research studies.[51-52] In a systematic review and meta-analysis study of Caucasians without
34 CVD, WC was most highly correlated with all CVD risk factors, compared to BMI, WHR, WSR and
35 body fat percentage, in women.[51] In other studies, WC was also more closely associated with CVD
36 risk factors than other measures of central obesity and BMI in women.[53-56] The advantages of WC
37 are, it is easy to measure and interpret, and it is less prone to measurement and calculation error.[52]
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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

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

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40 WSR is the least commonly used measure of central obesity. In a systematic review and meta-analysis
41 study, WSR reported the weakest correlations with CVD risk factors, compared to BMI and other
42 measures of central obesity,[51] which is contrary to our study findings. In contrast, WSR was most
43 highly correlated with CHD risk predicted using the Framingham model[17] in women from England,
44 compared to BMI, WC and WHR in another study.[65] WSR, however, reported lower correlations
45 than WC and BMI following adjustments for age.[65] The advantage of WSR include, the same cut
46 point could be applied across a wide range of populations. A cut-off value of 0.5 indicates increased
47 risk for men and women, people of different ethnic groups and this value may also be used in both
48 children and adults, unlike WC which requires different cut-offs.[66 67] More research is required to
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3 assess the association between WSR and CVD risk in women, in comparison with WC, WHR and
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5 BMI.
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9 Our study has limitations. This study is cross-sectional, however, it is a representative sample of the
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11 Australian female population. There is only one set of baseline measurements recorded for some risk
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13 variables but important variables including anthropometric measures of obesity are measured twice.
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15 Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against
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17 the treatment thresholds of the various models, and are not prospective CVD events.
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20 21 **CONCLUSIONS**

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23 The significant and independent effect of obesity measures on CVD risk substantiates its inclusion
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25 into risk score models. Central obesity is more strongly associated with CVD risk than general
26
27 obesity. The deposition of adipose tissue is associated with systemic inflammation which has a direct
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29 effect on CVD risk. Therefore, increments in central obesity have a more detrimental effect on CVD
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31 risk compared to increments in general obesity.
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35 When used alone, BMI is inadequate for identifying individuals at increased risk of CVD as it does
36
37 not differentiate between fat and fat-free mass. On the other hand, anthropometric measurements of
38
39 central obesity have higher sensitivity and specificity. These measures are also more sensitive to
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41 lifestyle modifications. An increase in muscle mass through diet and training would lead to changes in
42
43 measures such as WC and WSR but little change might be indicated with BMI.[68] It would be more
44
45 useful to measure a patient's central obesity during clinical assessment to evaluate the effect of
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47 lifestyle changes in relation to CVD risk compared to BMI. Central obesity measures are also
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49 significant and independent predictors of CVD risk, accounting for additional risk above BMI. These
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51 measurements should be incorporated into CVD risk assessment, particularly when assessing the risk
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53 in women and the elderly.[52 69-72]
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3 Future prospective studies are required to elucidate which anthropometric measurements of central
4 obesity are better indicators or predictors of CVD risk.[68] Studies measuring body fat distribution
5 using computerised tomography or magnetic resonance imaging are desirable to better understand the
6 association between body fat distribution and mortality, but costly.[73]
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13 In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of
14 waist circumference, should be considered for incorporation into the clinical assessment of CVD risk.
15 Treatment of well-established CVD risk factors coupled with reducing overweight and obesity
16 through lifestyle modifications would be an advisable goal in the primary prevention of CVD.[4] It is
17 equally important to maintain a healthy weight and to prevent central or abdominal obesity
18 concurrently.
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13 **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.



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

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3 **Anthropometric measurements of general and central obesity and**
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6 **the prediction of cardiovascular disease risk in women: a cross-**
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9 **sectional study**
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44 Anthropometric obesity measures and CVD risk
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48 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female
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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 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.

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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.

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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

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3 risk factors in the Framingham risk score model.[17] More research is thus needed to ascertain which
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5 measures are better correlated with CVD risk factors and subsequent CVD risk in women.
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9 We aim to assess the associations between general and central obesity anthropometric measures with
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11 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart
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13 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk
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15 calculated from the Framingham risk score model for 10-year CVD incidence or death,[18] SCORE
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17 risk chart for high-risk regions for 10-year CVD death,[19] general CVD and simplified general CVD
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19 risk score models for 10-year CVD incidence and death[20] would also be assessed. To aid
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21 comparison between obesity indices, which are measured in different units, the incremental shift in
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23 CVD risk with one standard deviation increment in each anthropometric measurement above the mean
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25 would be assessed. Finally, we determined which indices of obesity are most sensitive and specific for
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27 identifying females at increased 10-year CVD risk.
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30 31 **METHODS**

32 33 **Study cohort and measurements**

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35 We selected 4487 women aged 20-69 years with no history of heart disease, diabetes or stroke from
36
37 the population representative sample of 4727 women from the National Heart Foundation (NHF)
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39 Risk Factor Prevalence Study.[21] Participants taking medications to lower their CVD risk factors
40
41 were also excluded. The participants of the NHF study consisted of residents on the federal electoral
42
43 rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart,
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45 Darwin and Canberra in a systematic probability sampling by sex and 5-year age groups. Information
46
47 on demographic characteristics was collected using a self-administered questionnaire and
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49 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,
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51 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of
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53 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip
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55 circumference were collected according to standardised methodologies[22 23] using two observers.
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57 The waist circumference was measured from the front at the narrowest point between the rib cage and
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3 iliac crest after full expiration while the hip circumference was measured from the side at the maximal
4 extension of buttocks by one observer using a metal tape. A second observer recorded another set of
5 measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of
6 two measurements was taken at each site to the nearest centimetre.
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10 11 12 13 **Risk score models**

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15 The Framingham 10-year predicted risk for CVD incidence or death was developed using data from
16 the American Framingham Heart Study.[18] Participants aged 30-74 years who were free of CVD and
17 cancer were included in the model development. The 10-year risk for CVD incidence or death was
18 calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total
19 cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status.
20 The SCORE risk chart was developed by pooling 12 cohort studies to predict the 10-year CVD death
21 risk in Europe. The cohorts consisted of participants aged 19-80 years with no previous history of
22 heart attack.[19] It was derived from a much larger dataset than the Framingham, general CVD and
23 simplified general CVD risk score models. Fewer variables were used in the calculation of the 10-year
24 predicted CVD death risk with the SCORE risk chart for high-risk regions (Denmark, Finland and
25 Norway),[19 24] these included: age, sex, smoking status, mean total cholesterol level, mean HDL
26 cholesterol level and mean SBP. The general CVD risk score model was also developed using data
27 from the American Framingham Heart Study but using a larger cohort than the Framingham
28 model.[20] Individuals without CVD were used in the development of the general CVD risk score
29 model.[20] The simplified general CVD risk score model was developed similarly as the general CVD
30 risk score model. It is, however, a simpler CVD risk prediction model which is calculated using non-
31 laboratory predictors. Risk variables (age, SBP, current antihypertensive treatment, smoking status
32 and diabetes status) were used in both of the models.[20] The only difference is, BMI is included in
33 the simplified general CVD risk score model instead of total and HDL cholesterol which is used in the
34 general CVD risk score model.
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58 **Statistical analysis**

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3 The data on the representative sample of 4487 Australian females was described using mean \pm
4 standard deviation for continuous variables, while counts and percentages were used for categorical
5 variables. Non-parametric Spearman's rank correlation was used to assess the associations between
6 anthropometric measurements of obesity with CVD risk factors, and with the calculated 10-year
7 predicted risks, due to the skewness in the distribution of some variables. Anthropometric
8 measurements were also converted to z-scores (original value subtracted by the mean and result
9 divided by the standard deviation) to represent the number of standard deviations above and below the
10 mean for each subject. Logistic regression was used to assess the effects of each standardised
11 anthropometric measurement of being above the recommended treatment thresholds for various risk
12 score models as a result of a one standard deviation increment above the mean for each
13 anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals represented
14 the likelihood of being above the recommended treatment thresholds for the specific risk score models
15 (20% for the Framingham risk score model for 10-year CVD incidence or death; 10% for SCORE risk
16 chart for high-risk regions for 10-year CVD death; 10% and 20% for the general CVD and simplified
17 general CVD risk score models for 10-year CVD incidence and death). The predictive ability of these
18 anthropometric measures to identify individuals above and below the treatment thresholds was
19 assessed using sensitivity, specificity and area under the receiver operating characteristic (ROC)
20 curve. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses
21 were performed with IBM SPSS Statistics Version 21.
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43 RESULTS

44 The sample of 4487 women aged 20-69 years from the NHF Risk Factor Prevalence Study is a
45 representative sample of the Australian female population, free of heart disease, diabetes and stroke.
46 The characteristics of the sample are summarised in Table 1. In addition to the conventional risk
47 factors for CVD, all anthropometric measurements of general and central obesity were presented.
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56 The 10-year CVD risk of each participant in the sample was calculated using four risk score models.
57 The frequency distribution of calculated risks is presented in Table 2. Except for the Framingham
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3 model for CVD incidence, all other models predicted risks of less than 10% for at least 85% of the
4 sample. The Framingham model for CVD incidence, general CVD model for CVD incidence and
5 death, and simplified general CVD model for CVD incidence and death, predicted risk values across
6 the entire range from 0% to greater than 40%.
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13 Anthropometric measurements of obesity were positively correlated with age, SBP, total cholesterol
14 and total cholesterol to HDL cholesterol ratio (all Spearman's $r \geq 0.195$, $p < 0.001$), with HC
15 recording the lowest correlations. These obesity measures were negatively correlated with HDL
16 cholesterol (all Spearman's $r \leq -0.160$, $p < 0.001$). Measures of central obesity that included a
17 measure of waist circumference (WC, WHR and WSR) generally recorded better correlations
18 compared to measures of general obesity (BMI and BAI).
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27 The associations between anthropometric measurements of obesity and the 10-year predicted risks
28 calculated using the four models are presented in Table 3. All Spearman's rank correlations were
29 statistically significant ($p < 0.0005$). All anthropometric measures of central obesity (WC, WHR and
30 WSR) generally had consistently higher correlations with the predicted risks calculated using the four
31 CVD risk score models, as compared to measures of general obesity
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40 Recommended treatment thresholds for the four CVD risk models were identified from a review of
41 the literature. Table 4 presents the effects of a one standard deviation increment in each
42 anthropometric measurement above the mean on the likelihood of being above the recommended
43 thresholds or being indicated for treatment. All anthropometric measures of central obesity (WC,
44 WHR and WSR) generally recorded higher odds-ratios than general measures of obesity and they
45 increased the likelihood of individuals being above the respective treatment thresholds.
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54 Anthropometric measurements of central obesity (WC, WHR and WSR) also recorded higher area
55 under the ROC curves, higher sensitivity and specificity, than BMI in identifying females above and
56 below the 20% treatment threshold for the Framingham model for 10-year CVD incidence (Figure 1a)
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3 and general CVD model for 10-year CVD incidence and death (Figure 1b). Although BMI is included
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5 in the simplified general CVD model, high area under the ROC curve (> 0.76) are reported for both
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7 WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements
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9 as compared to general obesity measurement in predicting the increased risk of CVD.
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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
<i>Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[25 26]</i>					
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)
<i>Framingham 10-year predicted risk for CVD death (threshold = 20%)[25 26]</i>					
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)
<i>SCORE-HIGH 10-year predicted risk for CVD death (threshold = 10%)[19]</i>					
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)
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
#	2.16*** (1.99 - 2.34)	1.66*** (1.54 - 1.80)	2.16*** (1.98 - 2.35)	#	#
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
#	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.

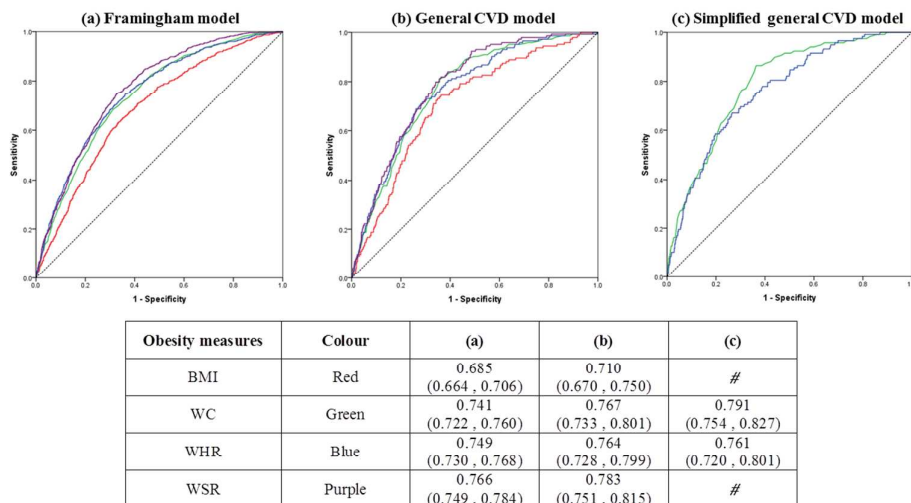


Figure 1 ROC curves to compare the predictive ability of obesity measures for being above the 20% cut-off of three CVD models: (a) Framingham risk score model for 10-year CVD incidence; (b) General cardiovascular disease risk score model for 10-year CVD incidence and death; (c) Simplified general cardiovascular 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

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3 inflammation which directly contributes to CVD risk.[35] Hence, measures that account for the
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5 accumulation of excess abdominal fat would report stronger associations and are desirable for
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7 assessing adiposity. They would also be more accurate at indicating CVD risk and should be
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9 incorporated into CVD assessment.[36-39] The addition of central obesity measures to BMI has also
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11 been shown to improve the accuracy of stratifying participants into lower and higher risk categories
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13 for mortality[40] and provides incremental value in predicting CVD above and beyond that provided
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15 by general obesity measures.[41-45] BMI is a flawed measure as it does not correctly identify
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17 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does
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19 not account for the effect of age and ethnicity on body fat distribution.[46-50] An increase in muscle
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21 or fat-free mass would, however, be reflected in the central obesity measures.
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25 Among central obesity measures, we found their performance to be comparable in our study. It
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27 remains unclear which measurement should be incorporated into CVD risk score models. A
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29 collaborative analysis of 58 prospective studies, however, reported that both measures of general and
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31 central obesity did not improve CVD risk assessment when information is available on SBP, diabetes
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33 and lipids.[51] Overweight and obesity is nevertheless important in CVD prevention, with one out of
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35 three of fatal and one out of seven of non-fatal CVD cases attributable to it.[34]
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39 Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and
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41 its association with CVD risk.[37] Some studies recommended the use of WC in clinical assessment
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43 and research studies.[52-53] In a systematic review and meta-analysis study of Caucasians without
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45 CVD, WC was most highly correlated with all CVD risk factors, compared to BMI, WHR, WSR and
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47 body fat percentage, in women.[52] In other studies, WC was also more closely associated with CVD
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49 risk factors than other measures of central obesity and BMI in women.[54-57] The advantages of WC
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51 are, it is easy to measure and interpret, and it is less prone to measurement and calculation error.[53]
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53 Appropriate sex, age and ethnic-specific WC cut points would need to be established.[44] It would
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55 also be difficult to use WC in today's multicultural societies due to requirements for different cut
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57 points.[50]
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5 The use of WHR is also supported as it is less strongly associated with BMI than WC and is thus a
6 more specific surrogate for fat distribution.[40] A longitudinal population study on 1462 women from
7 Sweden reported stronger relations between WHR and CVD endpoints, compared to BMI, WC and
8 HC.[58] These relations were mostly independent of age, BMI and either SBP, cholesterol level or
9 smoking habit.[58] In a meta-regression analysis of prospective studies, WHR was also more strongly
10 associated with CVD compared to WC, although the difference was not significant.[37] Another study
11 reported that WHR was associated with CVD mortality but not WC in elderly women from the United
12 Kingdom.[59] Elevated WHR was also independently associated with a higher CVD risk in the
13 Nurses' Health Study and in the Swedish Women's Lifestyle and Health Cohort Study.[45 60]
14 Women with a WHR of ≥ 0.88 were 3.25 times more at risk of CHD compared to women with a
15 WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted
16 odds-ratios were also reported with WHR and CHD and CVD mortality, compared to WC and BMI,
17 in an Australian population without heart disease, diabetes or stroke.[61] Similar results were
18 presented in other studies. WHR reported the highest age standardised hazard ratios in relation to
19 CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include,
20 it has low measurement error, high precision and no bias over a wide range of ethnic groups.[64]
21 WHR, however, may not be suitable for assessing central obesity in the elderly[65] due to laxity of
22 abdominal muscles which would undermine the predictive value of abdominal circumferences.[55] It
23 is also more difficult to measure than WC.[37] Despite its limitations, WHR has been recommended
24 for incorporation into CVD risk assessment.[37]

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48 WSR is the least commonly used measure of central obesity. In a systematic review and meta-analysis
49 study, WSR reported the weakest correlations with CVD risk factors, compared to BMI and other
50 measures of central obesity,[52] which is contrary to our study findings. In contrast, WSR was most
51 highly correlated with CHD risk predicted using the Framingham model[18] in women from England,
52 compared to BMI, WC and WHR in another study.[66] WSR, however, reported lower correlations
53 than WC and BMI following adjustments for age.[66] The advantage of WSR include, the same cut
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3 point could be applied across a wide range of populations. A cut-off value of 0.5 indicates increased
4 risk for men and women, people of different ethnic groups and this value may also be used in both
5 children and adults, unlike WC which requires different cut-offs.[67 68] More research is required to
6 assess the association between WSR and CVD risk in women, in comparison with WC, WHR and
7 BMI.
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15 Our study has limitations. This study is cross-sectional, however, it is a representative sample of the
16 Australian female population. There is only one set of baseline measurements recorded for some risk
17 variables but important variables including anthropometric measures of obesity are measured twice.
18 Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against
19 the treatment thresholds of the various models, and are not prospective CVD events.
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26 27 **CONCLUSIONS**

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29 Central obesity is more strongly associated with CVD risk than general obesity. The deposition of
30 adipose tissue is associated with systemic inflammation which has a direct effect on CVD risk.
31 Therefore, increments in central obesity have a more detrimental effect on CVD risk compared to
32 increments in general obesity.
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40 When used alone, BMI is inadequate for identifying individuals at increased risk of CVD as it does
41 not differentiate between fat and fat-free mass. On the other hand, anthropometric measurements of
42 central obesity have higher sensitivity and specificity. These measures are also more sensitive to
43 lifestyle modifications. An increase in muscle mass through diet and training would lead to changes in
44 measures such as WC and WSR but little change might be indicated with BMI.[69] It would be more
45 useful to measure a patient's central obesity during clinical assessment to evaluate the effect of
46 lifestyle changes in relation to CVD risk compared to BMI. Central obesity measures are also
47 significant and independent predictors of CVD risk, accounting for additional risk above BMI. These
48 measurements should be incorporated into CVD risk assessment, particularly when assessing the risk
49 in women and the elderly.[53 70-73]
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5 Future prospective studies are required to elucidate which anthropometric measurements of central
6 obesity are better indicators or predictors of CVD risk.[69] Studies measuring body fat distribution
7 using computerised tomography or magnetic resonance imaging are desirable to better understand the
8 association between body fat distribution and mortality, but costly.[74]
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15 In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of
16 waist circumference, should be considered for incorporation into the clinical assessment of CVD risk.
17 Treatment of well-established CVD risk factors coupled with reducing overweight and obesity
18 through lifestyle modifications would be an advisable goal in the primary prevention of CVD.[4] It is
19 equally important to maintain a healthy weight and to prevent central or abdominal obesity
20 concurrently.
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4
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6
7 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin
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9 University. This study was carried out in accordance with the Declaration of Helsinki.
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13 **Data sharing statement** No additional data are available.
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7 **Anthropometric measurements of general and central obesity and**
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9 **the prediction of cardiovascular disease risk in women: a cross-**
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11 **sectional study**
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42 Anthropometric obesity measures and CVD risk
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46 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female
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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 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.

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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.

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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] ~~It has also been suggested that another study,~~ BMI ~~was~~ a better predictor of CVD than WC.[13] Conversely, some studies reported that WC ~~was~~ 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](#)

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7 independent predictors of CVD risk but not BMI, accounting for conventional risk factors in the
8 Framingham risk score model.[17] More research is thus needed to ascertain which measures are
9 better correlated with CVD risk factors and subsequent CVD risk in women.
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13 We aim to assess the associations between general and central obesity anthropometric measures with
14 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart
15 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk
16 calculated from the Framingham risk score model for 10-year CVD incidence or death,[18] SCORE
17 risk chart for high-risk regions for 10-year CVD death,[19] general CVD and simplified general CVD
18 risk score models s for 10-year CVD incidence and death[20] would also be assessed. To aid
19 comparison between obesity indices, which are measured in different units, the incremental shift in
20 CVD risk with one standard deviation increment in each anthropometric measurement above the mean
21 would be assessed. Finally, we ~~would-determined~~ which indices of obesity are most sensitive and
22 specific for identifying females at increased 10-year CVD risk.
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33 METHODS

34 Study cohort and measurements

35 We selected 4487 women aged 20-69 years with no history of heart disease, diabetes or stroke from
36 the population representative sample of 4727 women from the National Heart Foundation (NHF)
37 Risk Factor Prevalence Study.[21] Participants taking medications to lower their CVD risk factors
38 were also excluded. The participants of the NHF study consisted of residents on the federal electoral
39 rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart,
40 Darwin and Canberra in a systematic probability sampling by sex and 5-year age groups. Information
41 on demographic characteristics was collected using a self-administered questionnaire and
42 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,
43 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of
44 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip
45 circumference were collected according to standardised methodologies[22 23] using two observers.
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7 The waist circumference was measured from the front at the narrowest point between the rib cage and
8 iliac crest after full expiration while the hip circumference was measured from the side at the maximal
9 extension of buttocks by one observer using a metal tape. A second observer recorded another set of
10 measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of
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14 two measurements was taken at each site to the nearest centimetre.

15 16 17 **Variables in risk score models**

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19 The Framingham 10-year predicted risk for CVD incidence or death[18] was developed using data
20 from the American Framingham Heart Study.[18] Participants aged 30-74 years who were free of
21 CVD and cancer were included in the model development. The 10-year risk for CVD incidence or
22 death was calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood
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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 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

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7 | ~~cholesterol level and HDL cholesterol level were replaced by BMI in the calculation of the 10-year~~
8 | ~~risk for CVD incidence and death.~~
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10 11 **Statistical analysis**

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13 The data on the representative sample of 4487 Australian females was described using mean \pm
14 standard deviation for continuous variables, while counts and percentages were used for categorical
15 variables. Non-parametric Spearman's rank correlation was used to assess the associations between
16 anthropometric measurements of obesity with CVD risk factors, and with the calculated 10-year
17 predicted risks, due to the skewness in the distribution of some variables. Anthropometric
18 measurements were also converted to z-scores (original value subtracted by the mean and result
19 divided by the standard deviation) to represent the number of standard deviations above and below the
20 mean for each subject. Logistic regression was used to assess the effects of each standardised
21 anthropometric measurement of being above the recommended treatment thresholds for various risk
22 score models as a result of a one standard deviation increment above the mean for each
23 anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals represented
24 the likelihood of being above the recommended treatment thresholds for the specific risk score models
25 (20% for the Framingham risk score model for 10-year CVD incidence or death; 10% for SCORE risk
26 chart for high-risk regions for 10-year CVD death; 10% and 20% for the general CVD and simplified
27 general CVD risk score models for 10-year CVD incidence and death). The predictive ability of these
28 anthropometric measures to identify individuals above and below the treatment thresholds was
29 assessed using sensitivity, specificity and area under the receiver operating characteristic (ROC)
30 curve. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses
31 were performed with IBM SPSS Statistics Version 21⁰.
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48 49 **RESULTS**

50 The sample of 4487 women aged 20-69 years from the NHF Risk Factor Prevalence Study is a
51 representative sample of the Australian female population, free of heart disease, diabetes and stroke.
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7 The characteristics of the sample ~~were-are~~ summarised in Table 1. In addition to the conventional risk
8 factors for CVD, all anthropometric measurements of general and central obesity were presented.
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11 The 10-year CVD risk of each ~~subject-participant~~ in the sample was calculated using four risk score
12 models. The frequency distribution of calculated risks ~~was-is~~ presented in Table 2. Except for the
13 Framingham model for CVD incidence, all other models predicted risks of less than 10% for at least
14 85% of the sample. The Framingham model for CVD incidence, general CVD model for CVD
15 incidence and death, and simplified general CVD model for CVD incidence and death, predicted risk
16 values across the entire range from 0% to greater than 40%.
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24 Anthropometric measurements of obesity were positively correlated with age, SBP, total cholesterol
25 and total cholesterol to HDL cholesterol ratio (all Spearman's $r \geq 0.195$, $p < 0.001$), with HC
26 recording the lowest correlations. These obesity measures were negatively correlated with HDL
27 cholesterol (all Spearman's $r \leq -0.160$, $p < 0.001$). Measures of central obesity that included a
28 measure of waist circumference (WC, WHR and WSR) generally recorded better correlations
29 compared to measures of general obesity (BMI and BAI).
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37 The associations between anthropometric measurements of obesity and the 10-year predicted risks
38 calculated using the four models ~~arewere~~ presented in Table 3. All Spearman's rank correlations were
39 statistically significant ($p < 0.0005$). All anthropometric measures of central obesity (WC, WHR and
40 WSR) generally had consistently higher correlations with the predicted risks calculated using the four
41 CVD risk score models, as compared to measures of general obesity-
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47 Recommended treatment thresholds for the four CVD risk models were identified from a review of
48 the literature. Table 4 ~~presented-presents~~ the effects of a one standard deviation increment in each
49 anthropometric measurement above the mean on the likelihood of being above the recommended
50 thresholds or being indicated for treatment. All anthropometric measures of central obesity (WC,
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7 WHR and WSR) generally recorded higher odds-ratios than general measures of obesity and they
8 increased the likelihood of individuals being above the respective treatment thresholds.
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11 Anthropometric measurements of central obesity (WC, WHR and WSR) also recorded higher area
12 under the ROC curves, higher sensitivity and specificity, than BMI in identifying females above and
13 below the 20% treatment threshold for the Framingham model for 10-year CVD incidence (Figure 1a)
14 and general CVD model for 10-year CVD incidence and death (Figure 1b). Although BMI is included
15 in the simplified general CVD model, high area under the ROC curve (> 0.76) ~~are~~ were reported for
16 both WC and WHR (Figure 1c), indicating the independent contribution of central obesity
17 measurements as compared to general obesity measurement in predicting the increased risk of CVD.
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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

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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
<i>Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[25 26]</i>					
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)
<i>Framingham 10-year predicted risk for CVD death (threshold = 20%)[25 26]</i>					
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)
<i>SCORE-HIGH 10-year predicted risk for CVD death (threshold = 10%)[19]</i>					
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)
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
#	2.16*** (1.99 - 2.34)	1.66*** (1.54 - 1.80)	2.16*** (1.98 - 2.35)	#	#
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
#	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.

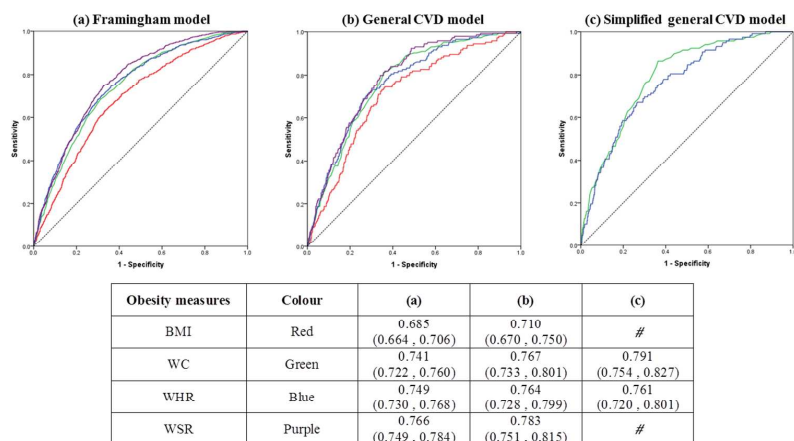


Figure 1 ROC curves to compare the predictive ability of obesity measures for being above the 20% cut-off of three CVD models: (a) Framingham risk score model for 10-year CVD incidence; (b) General cardiovascular disease risk score model for 10-year CVD incidence and death; (c) Simplified general cardiovascular 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~~

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7 ~~for by the general obesity measure.~~ Conversely, some studies reported that the association between
8 BMI and CVD was similar to measures of central obesity.[33 34]
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11 There are several possible explanations for our study findings that measures of central obesity are
12 better predictors of CVD risk than BMI. Greater central obesity is associated with systemic
13 inflammation which directly contributes to CVD risk.[35] Hence, measures that account for the
14 accumulation of excess abdominal fat would report stronger associations and are desirable for
15 assessing adiposity. They would also be more accurate at indicating CVD risk and should be
16 incorporated into CVD assessment.[36-39] The addition of central obesity measures to BMI has also
17 been shown to improve the accuracy of stratifying participants into lower and higher risk categories
18 for mortality[40] and provides incremental value in predicting CVD above and beyond that provided
19 by general obesity measures.[41-45] ~~BMI alone is thus insufficient to account for the association~~
20 ~~between obesity and CVD risk.~~ BMI is ~~also~~ a flawed measure as it does not correctly identify
21 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does
22 not account for the effect of age and ethnicity on body fat distribution.[46-50] An increase in muscle
23 or fat-free mass would, however, be reflected in the central obesity measures.
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37 Among central obesity measures, we found their performance to be comparable in our study. It
38 remains unclear which measurement should be incorporated into CVD risk score models. ~~To date,~~
39 ~~BMI is included in the simplified general CVD risk score model as an alternative to total and HDL~~
40 ~~cholesterol level considering its ease of measurement and calculation,[20] and in the QRISK score~~
41 ~~model.[29]~~ A collaborative analysis of 58 prospective studies, however, reported that both measures
42 of general and central obesity did not improve CVD risk assessment when information is available on
43 SBP, diabetes and lipids.[51] Overweight and obesity is nevertheless important in CVD prevention,
44 with one out of three of fatal and one out of seven of non-fatal CVD cases attributable to it.[34]
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52 Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and
53 its association with CVD risk.[37] Some studies recommended the use of WC in clinical assessment
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7 and research studies.[52 53] In a systematic review and meta-analysis study of Caucasians without
8 CVD, WC was most highly correlated with all CVD risk factors, compared to BMI, WHR, WSR and
9 body fat percentage, in women.[52] In other studies, WC was also more closely associated with CVD
10 risk factors than other measures of central obesity and BMI in women.[54-57] The advantages of WC
11 are, it is easy to measure and interpret, and it is less prone to measurement and calculation error.[53]
12 Appropriate sex, age and ethnic-specific WC cut points would need to be established.[44] It would
13 also be difficult to use WC in today's multicultural societies due to requirements for different cut
14 points.[50]

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22 The use of WHR is also supported as it is less strongly associated with BMI than WC and is thus a
23 more specific surrogate for fat distribution.[40] A longitudinal population study on 1462 women from
24 Sweden reported stronger relations between WHR and CVD endpoints, compared to BMI, WC and
25 HC.[58] These relations were mostly independent of age, BMI and either SBP, cholesterol level or
26 smoking habit.[58] In a meta-regression analysis of prospective studies, WHR was also more strongly
27 associated with CVD compared to WC, although the difference was not significant.[37] Another study
28 reported that WHR was associated with CVD mortality but not WC in elderly women from the United
29 Kingdom.[59] Elevated WHR was also independently associated with a higher CVD risk in the
30 Nurses' Health Study and in the Swedish Women's Lifestyle and Health Cohort Study.[45 60]
31 Women with a WHR of ≥ 0.88 were 3.25 times more at risk of CHD compared to women with a
32 WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted
33 odds-ratios were also reported with WHR and CHD and CVD mortality, compared to WC and BMI,
34 in an Australian population without heart disease, diabetes or stroke.[61] Similar results were
35 presented in other studies. WHR reported the highest age standardised hazard ratios in relation to
36 CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include,
37 it has low measurement error, high precision and no bias over a wide range of ethnic groups.[64]
38 WHR, however, may not be suitable for assessing central obesity in the elderly[65] due to laxity of
39 abdominal muscles which would undermine the predictive value of abdominal circumferences.[55] It
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7 is also more difficult to measure than WC.[37] Despite its limitations, WHR has been recommended
8 for incorporation into CVD risk assessment.[37]
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11 WSR is the least commonly used measure of central obesity. In a systematic review and meta-analysis
12 study, WSR reported the weakest correlations with CVD risk factors, compared to BMI and other
13 measures of central obesity,[52] which is contrary to our study findings. In contrast, WSR was most
14 highly correlated with CHD risk predicted using the Framingham model[18] in women from England,
15 compared to BMI, WC and WHR in another study.[66] WSR, however, reported lower correlations
16 than WC and BMI following adjustments for age.[66] The advantage of WSR include, the same cut
17 point could be applied across a wide range of populations. A cut-off value of 0.5 indicates increased
18 risk for men and women, people of different ethnic groups and this value may also be used in both
19 children and adults, unlike WC which requires different cut-offs.[67 68] More research is required to
20 assess the association between WSR and CVD risk in women, in comparison with WC, WHR and
21 BMI.
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33 Our study has limitations. This study is cross-sectional, however, it is a representative sample of the
34 Australian female population. There is only one set of baseline measurements recorded for some risk
35 variables but important variables including anthropometric measures of obesity are measured twice.
36 Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against
37 the treatment thresholds of the various models, and are not prospective CVD events.
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43 CONCLUSIONS

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45 ~~The significant and independent effect of obesity measures on CVD risk substantiates its inclusion~~
46 ~~into risk score models.~~ Central obesity is more strongly associated with CVD risk than general
47 obesity. The deposition of adipose tissue is associated with systemic inflammation which has a direct
48 effect on CVD risk. Therefore, increments in central obesity have a more detrimental effect on CVD
49 risk compared to increments in general obesity.
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7 When used alone, BMI is inadequate for identifying individuals at increased risk of CVD as it does
8 not differentiate between fat and fat-free mass. On the other hand, anthropometric measurements of
9 central obesity have higher sensitivity and specificity. These measures are also more sensitive to
10 lifestyle modifications. An increase in muscle mass through diet and training would lead to changes in
11 measures such as WC and WSR but little change might be indicated with BMI.[69] It would be more
12 useful to measure a patient's central obesity during clinical assessment to evaluate the effect of
13 lifestyle changes in relation to CVD risk compared to BMI. Central obesity measures are also
14 significant and independent predictors of CVD risk, accounting for additional risk above BMI. These
15 measurements should be incorporated into CVD risk assessment, particularly when assessing the risk
16 in women and the elderly.[53 70-73]

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26 Future prospective studies are required to elucidate which anthropometric measurements of central
27 obesity are better indicators or predictors of CVD risk.[69] Studies measuring body fat distribution
28 using computerised tomography or magnetic resonance imaging are desirable to better understand the
29 association between body fat distribution and mortality, but costly.[74]

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35 In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of
36 waist circumference, should be considered for incorporation into the clinical assessment of CVD risk.
37 Treatment of well-established CVD risk factors coupled with reducing overweight and obesity
38 through lifestyle modifications would be an advisable goal in the primary prevention of CVD.[4] It is
39 equally important to maintain a healthy weight and to prevent central or abdominal obesity
40 concurrently.
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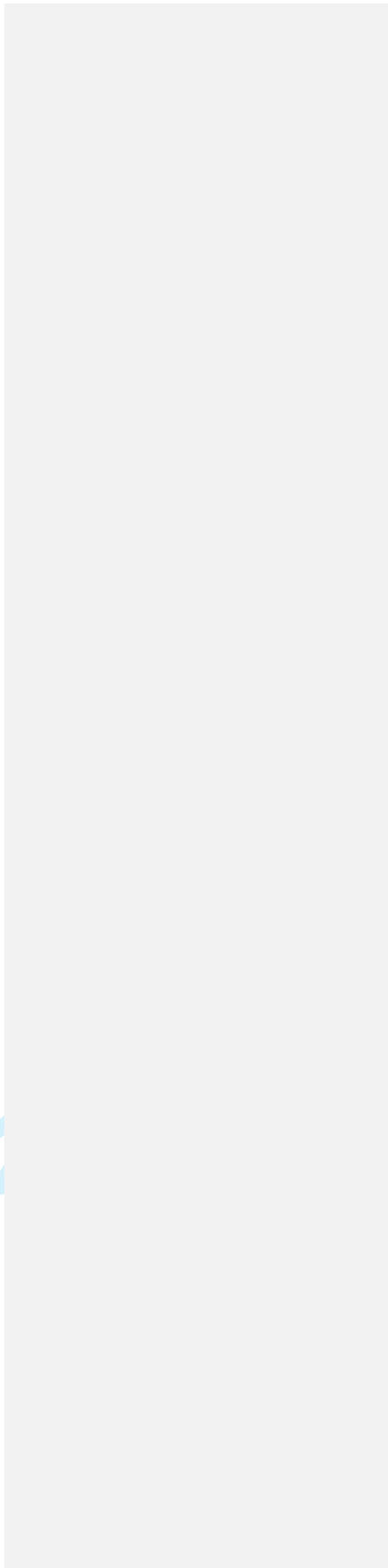
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analysis and data interpretation and revised the manuscript critically for important intellectual content.
TAW participated in the study design, acquired the data and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

Competing interests None.

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7 **Ethics approval** We have ethics approval for the use of the National Heart Foundation data from the
8 Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth
9 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin
10 University. This study was carried out in accordance with the Declaration of Helsinki.
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15 **Data sharing statement** No additional data are available.
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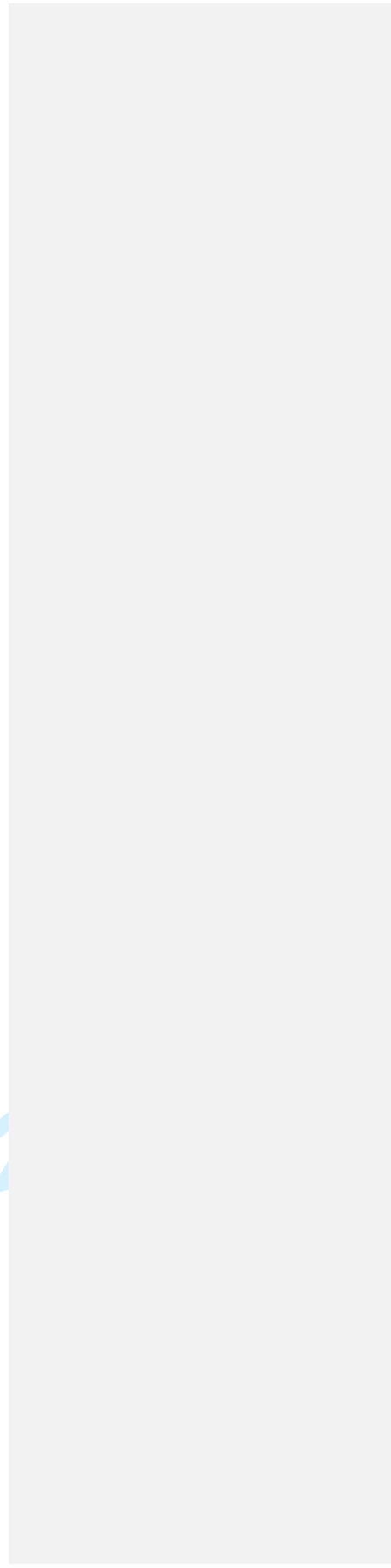
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STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cross-sectional studies*

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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9 **sectional study**
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44 Anthropometric obesity measures and CVD risk

48 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.

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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.

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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

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3 risk factors in the Framingham risk score model.[17] More research is thus needed to ascertain which
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5 measures are better correlated with CVD risk factors and subsequent CVD risk in women.
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9 We aim to assess the associations between general and central obesity anthropometric measures with
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11 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart
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13 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk
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15 calculated from the Framingham risk score model for 10-year CVD incidence or death,[18] SCORE
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17 risk chart for high-risk regions for 10-year CVD death,[19] general CVD and simplified general CVD
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19 risk score models for 10-year CVD incidence and death[20] were examined. To aid comparison
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21 between obesity indices, which are measured in different units, the incremental shift in CVD risk with
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23 one standard deviation increment in each anthropometric measurement above the mean would be
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25 assessed. Finally, we determined which indices of obesity are most sensitive and specific for
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27 identifying females at increased 10-year CVD risk.
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30 31 **METHODS**

32 33 **Study cohort and measurements**

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35 We selected 4487 women aged 20-69 years with no history of heart disease, diabetes or stroke from
36
37 the population representative sample of 4727 women from the National Heart Foundation (NHF)
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39 Risk Factor Prevalence Study.[21] Participants taking medications to lower their CVD risk factors
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41 were also excluded. The participants of the NHF study consisted of residents on the federal electoral
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43 rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart,
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45 Darwin and Canberra in a systematic probability sampling by sex and 5-year age groups. Information
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47 on demographic characteristics was collected using a self-administered questionnaire and
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49 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,
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51 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of
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53 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip
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55 circumference were collected according to standardised methodologies[22 23] using two observers.
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57 The waist circumference was measured from the front at the narrowest point between the rib cage and
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3 iliac crest after full expiration while the hip circumference was measured from the side at the maximal
4 extension of buttocks by one observer using a metal tape. A second observer recorded another set of
5 measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of
6 two measurements was taken at each site to the nearest centimetre. Participants were classified as
7 non-smokers, previous smokers or current smokers.[21] Mercury sphygmomanometers were used to
8 record blood pressure levels on the right arm of seated participants five minutes apart.[21] Two
9 readings were taken and the average was used in the analysis. Fasting blood samples were also
10 collected in EDTA tubes and despatched to the central laboratory at the Division of Clinical
11 Chemistry, Institute of Medical and Veterinary Science, Adelaide each week for lipid levels to be
12 assayed.[21]
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24 25 **Risk score models**

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27 The Framingham 10-year predicted risk for CVD incidence or death was developed using data from
28 the American Framingham Heart Study.[18] Participants aged 30-74 years who were free of CVD and
29 cancer were included in the model development. The 10-year risk for CVD incidence or death was
30 calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total
31 cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status.
32 The SCORE risk chart was developed by pooling 12 cohort studies to predict the 10-year CVD death
33 risk in Europe. The cohorts consisted of participants aged 19-80 years with no previous history of
34 heart attack.[19] The SCORE model was derived from a much larger dataset than the Framingham,
35 general CVD and simplified general CVD risk score models. Fewer variables were used in the
36 calculation of the 10-year predicted CVD death risk with the SCORE risk chart for high-risk regions
37 (Denmark, Finland and Norway),[19 24] these included: age, sex, smoking status, mean total
38 cholesterol level, mean HDL cholesterol level and mean SBP. The general CVD risk score model was
39 also developed using data from the American Framingham Heart Study but using a larger cohort than
40 the Framingham model.[20] Individuals without CVD were used in the development of the general
41 CVD risk score model.[20] The simplified general CVD risk score model was developed similarly as
42 the general CVD risk score model. It is, however, a simpler CVD risk prediction model which is
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3 calculated using non-laboratory predictors. Risk variables (age, SBP, current antihypertensive
4 treatment, smoking status and diabetes status) were used in both of the models.[20] The only
5 difference is, BMI is included in the simplified general CVD risk score model instead of total and
6 HDL cholesterol which is used in the general CVD risk score model.
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10 11 12 13 **Statistical analysis**

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15 The data on the representative sample of 4487 Australian females were described using mean \pm
16 standard deviation for continuous variables, while counts and percentages were used for categorical
17 variables. Non-parametric Spearman's rank correlation was used to assess the associations between
18 anthropometric measurements of obesity with CVD risk factors, and with the calculated 10-year
19 predicted risks, due to the skewness in the distribution of some variables. Anthropometric
20 measurements were also converted to z-scores (original value subtracted by the mean and result
21 divided by the standard deviation) to represent the number of standard deviations above and below the
22 mean for each subject. Logistic regression was used to assess the effects of each standardised
23 anthropometric measurement of being above the recommended treatment thresholds for various risk
24 score models as a result of a one standard deviation increment above the mean for each
25 anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals represented
26 the likelihood of being above the recommended treatment thresholds for the specific risk score models
27 (20% for the Framingham risk score model for 10-year CVD incidence or death; 10% for SCORE risk
28 chart for high-risk regions for 10-year CVD death; 10% and 20% for the general CVD and simplified
29 general CVD risk score models for 10-year CVD incidence and death). The predictive ability of these
30 anthropometric measures to identify individuals above and below the treatment thresholds was
31 assessed using sensitivity, specificity and area under the receiver operating characteristic (ROC)
32 curve. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses
33 were performed with IBM SPSS Statistics Version 21.
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53 54 55 56 **RESULTS**

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3 The sample of 4487 women aged 20-69 years from the NHF Risk Factor Prevalence Study is a
4 representative sample of the Australian female population, free of heart disease, diabetes and stroke.
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6 The characteristics of the sample are summarised in Table 1. In addition to the conventional risk
7 factors for CVD, all anthropometric measurements of general and central obesity were presented.
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13 The 10-year CVD risk of each participant in the sample was calculated using four risk score models.
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15 The frequency distribution of calculated risks is presented in Table 2. Except for the Framingham
16 model for CVD incidence, all other models predicted risks of less than 10% for at least 85% of the
17 sample. The Framingham model for CVD incidence, general CVD model for CVD incidence and
18 death, and simplified general CVD model for CVD incidence and death, predicted risk values across
19 the entire range from 0% to greater than 40%.
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27 Anthropometric measurements of obesity were positively correlated with age, SBP, total cholesterol
28 and total cholesterol to HDL cholesterol ratio (all Spearman's $r \geq 0.195$, $p < 0.001$), with HC
29 recording the lowest correlations. These obesity measures were negatively correlated with HDL
30 cholesterol (all Spearman's $r \leq -0.160$, $p < 0.001$). Measures of central obesity that included a
31 measure of waist circumference (WC, WHR and WSR) generally recorded better correlations
32 compared to measures of general obesity (BMI and BAI).
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42 The associations between anthropometric measurements of obesity and the 10-year predicted risks
43 calculated using the four models are presented in Table 3. All Spearman's rank correlations were
44 statistically significant ($p < 0.0005$). All anthropometric measures of central obesity (WC, WHR and
45 WSR) generally had consistently higher correlations with the predicted risks calculated using the four
46 CVD risk score models, as compared to measures of general obesity
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54 Recommended treatment thresholds for the four CVD risk models were identified from a review of
55 the literature. Table 4 presents the effects of a one standard deviation increment in each
56 anthropometric measurement above the mean on the likelihood of being above the recommended
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3 thresholds or being indicated for treatment. All anthropometric measures of central obesity (WC,
4 WHR and WSR) generally recorded higher odds-ratios than general measures of obesity and they
5 increased the likelihood of individuals being above the respective treatment thresholds.
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11 Anthropometric measurements of central obesity (WC, WHR and WSR) also recorded higher area
12 under the ROC curves, higher sensitivity and specificity, than BMI in identifying females above and
13 below the 20% treatment threshold for the Framingham model for 10-year CVD incidence (Figure 1a)
14 and general CVD model for 10-year CVD incidence and death (Figure 1b). Although BMI is included
15 in the simplified general CVD model, high area under the ROC curve (> 0.76) are reported for both
16 WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements
17 as compared to general obesity measurement in predicting the increased risk of CVD.
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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
<i>Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[25 26]</i>					
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)
<i>Framingham 10-year predicted risk for CVD death (threshold = 20%)[25 26]</i>					
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)
<i>SCORE-HIGH 10-year predicted risk for CVD death (threshold = 10%)[19]</i>					
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)
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
#	2.16*** (1.99 - 2.34)	1.66*** (1.54 - 1.80)	2.16*** (1.98 - 2.35)	#	#
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
#	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

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3 inflammation which directly contributes to CVD risk.[35] Hence, measures that account for the
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5 accumulation of excess abdominal fat would report stronger associations and are desirable for
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7 assessing adiposity. They would also be more accurate at indicating CVD risk and should be
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9 incorporated into CVD assessment.[36-39] The addition of central obesity measures to BMI has also
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11 been shown to improve the accuracy of stratifying participants into lower and higher risk categories
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13 for mortality[40] and provides incremental value in predicting CVD above and beyond that provided
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15 by general obesity measures.[41-45] BMI is a flawed measure as it does not correctly identify
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17 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does
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19 not account for the effect of age and ethnicity on body fat distribution.[46-50] An increase in muscle
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21 or fat-free mass would, however, be reflected in the central obesity measures.

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

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39 Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and
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41 its association with CVD risk.[37] Some studies recommended the use of WC in clinical assessment
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43 and research studies.[52 53] In a systematic review and meta-analysis study of Caucasians without
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45 CVD, WC was most highly correlated with all CVD risk factors, compared to BMI, WHR, WSR and
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47 body fat percentage, in women.[52] In other studies, WC was also more closely associated with CVD
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49 risk factors than other measures of central obesity and BMI in women.[54-57] The advantages of WC
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51 are, it is easy to measure and interpret, and it is less prone to measurement and calculation error.[53]
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53 Appropriate sex, age and ethnic-specific WC cut points would need to be established.[44] It would
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55 also be difficult to use WC in today's multicultural societies due to requirements for different cut
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57 points.[50]

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5 The use of WHR is also supported as it is less strongly associated with BMI than WC and is thus a
6 more specific surrogate for fat distribution.[40] A longitudinal population study on 1462 women from
7 Sweden reported stronger relations between WHR and CVD endpoints, compared to BMI, WC and
8 HC.[58] These relations were mostly independent of age, BMI and either SBP, cholesterol level or
9 smoking habit.[58] In a meta-regression analysis of prospective studies, WHR was also more strongly
10 associated with CVD compared to WC, although the difference was not significant.[37] Another study
11 reported that WHR was associated with CVD mortality but not WC in elderly women from the United
12 Kingdom.[59] Elevated WHR was also independently associated with a higher CVD risk in the
13 Nurses' Health Study and in the Swedish Women's Lifestyle and Health Cohort Study.[45 60]
14 Women with a WHR of ≥ 0.88 were 3.25 times more at risk of CHD compared to women with a
15 WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted
16 odds-ratios were also reported with WHR and CHD and CVD mortality, compared to WC and BMI,
17 in an Australian population without heart disease, diabetes or stroke.[61] Similar results were
18 presented in other studies. WHR reported the highest age standardised hazard ratios in relation to
19 CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include,
20 it has low measurement error, high precision and no bias over a wide range of ethnic groups.[64]
21 WHR, however, may not be suitable for assessing central obesity in the elderly[65] due to laxity of
22 abdominal muscles which would undermine the predictive value of abdominal circumferences.[55] It
23 is also more difficult to measure than WC.[37] Despite its limitations, WHR has been recommended
24 for incorporation into CVD risk assessment.[37]

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48 WSR is the least commonly used measure of central obesity. In a systematic review and meta-analysis
49 study, WSR reported the weakest correlations with CVD risk factors, compared to BMI and other
50 measures of central obesity,[52] which is contrary to our study findings. In contrast, WSR was most
51 highly correlated with CHD risk predicted using the Framingham model[18] in women from England,
52 compared to BMI, WC and WHR in another study.[66] WSR, however, reported lower correlations
53 than WC and BMI following adjustments for age.[66] The advantage of WSR include, the same cut
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3 point could be applied across a wide range of populations. A cut-off value of 0.5 indicates increased
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5 risk for men and women, people of different ethnic groups and this value may also be used in both
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7 children and adults, unlike WC which requires different cut-offs.[67 68] More research is required to
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9 assess the association between WSR and CVD risk in women, in comparison with WC, WHR and
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11 BMI.

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15 Our study has limitations. This study is cross-sectional, however, it is a representative sample of the
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17 Australian female population. There is only one set of baseline measurements recorded for some risk
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19 variables but important variables including anthropometric measures of obesity are measured twice.
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21 Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against
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23 the treatment thresholds of the various models, and are not prospective CVD events.
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26 27 **CONCLUSIONS**

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29 Central obesity is more strongly associated with CVD risk than general obesity. The deposition of
30
31 adipose tissue is associated with systemic inflammation which has a direct effect on CVD risk.
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33 Therefore, increments in central obesity have a more detrimental effect on CVD risk compared to
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35 increments in general obesity.
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39 When used alone, BMI is inadequate for identifying individuals at increased risk of CVD as it does
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41 not differentiate between fat and fat-free mass. On the other hand, anthropometric measurements of
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43 central obesity have higher sensitivity and specificity. These measures are also more sensitive to
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45 lifestyle modifications. An increase in muscle mass through diet and training would lead to changes in
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47 measures such as WC and WSR but little change might be indicated with BMI.[69] It would be more
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49 useful to measure a patient's central obesity during clinical assessment to evaluate the effect of
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51 lifestyle changes in relation to CVD risk compared to BMI. Central obesity measures are also
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53 significant and independent predictors of CVD risk, accounting for additional risk above BMI. These
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55 measurements should be incorporated into CVD risk assessment, particularly when assessing the risk
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57 in women and the elderly.[53 70-73]
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3 Future prospective studies are required to elucidate which anthropometric measurements of central
4 obesity are better indicators or predictors of CVD risk.[69] Studies measuring body fat distribution
5 using computerised tomography or magnetic resonance imaging are desirable to better understand the
6 association between body fat distribution and mortality, but costly.[74]
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13 In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of
14 waist circumference, should be considered for incorporation into the clinical assessment of CVD risk.
15 Treatment of well-established CVD risk factors coupled with reducing overweight and obesity
16 through lifestyle modifications would be an advisable goal in the primary prevention of CVD.[4] It is
17 equally important to maintain a healthy weight and to prevent central or abdominal obesity
18 concurrently.
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27 **Figure legend**

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29 **Figure 1 ROC curves to compare the predictive ability of obesity measures for being above the**
30 **20% cut-off of three CVD models: (a) Framingham risk score model for 10-year CVD**
31 **incidence; (b) General cardiovascular disease risk score model for 10-year CVD incidence and**
32 **death; (c) Simplified general cardiovascular disease risk score model for 10-year CVD incidence**
33 **and death**
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36 # Area under the ROC curve is not calculated for this obesity measure as it contains height which is
37 also used in the calculation of the simplified general CVD model.
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39 Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WSR,
40 waist-to-stature ratio.
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3 analysis and data interpretation and revised the manuscript critically for important intellectual content.
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5 TAW participated in the study design, acquired the data and revised the manuscript critically for
6
7 important intellectual content. All authors read and approved the final manuscript.
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3 **Ethics approval** We have ethics approval for the use of the National Heart Foundation data from the
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5 Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth
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7 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin
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9 University. This study was carried out in accordance with the Declaration of Helsinki.
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13 **Data sharing statement** No additional data are available.
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3 **Anthropometric measurements of general and central obesity and**
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6 **the prediction of cardiovascular disease risk in women: a cross-**
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9 **sectional study**
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44 Anthropometric obesity measures and CVD risk
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48 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female
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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 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.

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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.

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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

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3 risk factors in the Framingham risk score model.[17] More research is thus needed to ascertain which
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5 measures are better correlated with CVD risk factors and subsequent CVD risk in women.
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9 We aim to assess the associations between general and central obesity anthropometric measures with
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11 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart
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13 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk
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15 calculated from the Framingham risk score model for 10-year CVD incidence or death,[18] SCORE
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17 risk chart for high-risk regions for 10-year CVD death,[19] general CVD and simplified general CVD
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19 risk score models for 10-year CVD incidence and death[20] ~~would also be assessed~~were examined.

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21 To aid comparison between obesity indices, which are measured in different units, the incremental
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23 shift in CVD risk with one standard deviation increment in each anthropometric measurement above
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25 the mean would be assessed. Finally, we determined which indices of obesity are most sensitive and
26
27 specific for identifying females at increased 10-year CVD risk.
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30 31 **METHODS**

32 33 **Study cohort and measurements**

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35 We selected 4487 women aged 20-69 years with no history of heart disease, diabetes or stroke from
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37 the population representative sample of 4727 women from the National Heart Foundation (NHF)
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39 Risk Factor Prevalence Study.[21] Participants taking medications to lower their CVD risk factors
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41 were also excluded. The participants of the NHF study consisted of residents on the federal electoral
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43 rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart,
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45 Darwin and Canberra in a systematic probability sampling by sex and 5-year age groups. Information
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47 on demographic characteristics was collected using a self-administered questionnaire and
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49 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,
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51 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of
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53 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip
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55 circumference were collected according to standardised methodologies[22 23] using two observers.
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57 The waist circumference was measured from the front at the narrowest point between the rib cage and
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3 iliac crest after full expiration while the hip circumference was measured from the side at the maximal
4 extension of buttocks by one observer using a metal tape. A second observer recorded another set of
5 measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of
6 two measurements was taken at each site to the nearest centimetre. Participants were classified as
7 non-smokers, previous smokers or current smokers.[21] Mercury sphygmomanometers were used to
8 record blood pressure levels on the right arm of seated participants five minutes apart.[21] Two
9 readings were taken and the average was used in the analysis. Fasting blood samples were also
10 collected in EDTA tubes and despatched to the central laboratory at the Division of Clinical
11 Chemistry, Institute of Medical and Veterinary Science, Adelaide each week for lipid levels to be
12 assayed.[21]

23 24 25 **Risk score models**

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27 The Framingham 10-year predicted risk for CVD incidence or death was developed using data from
28 the American Framingham Heart Study.[18] Participants aged 30-74 years who were free of CVD and
29 cancer were included in the model development. The 10-year risk for CVD incidence or death was
30 calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total
31 cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status.
32 The SCORE risk chart was developed by pooling 12 cohort studies to predict the 10-year CVD death
33 risk in Europe. The cohorts consisted of participants aged 19-80 years with no previous history of
34 heart attack.[19] ~~†~~The SCORE model was derived from a much larger dataset than the Framingham,
35 general CVD and simplified general CVD risk score models. Fewer variables were used in the
36 calculation of the 10-year predicted CVD death risk with the SCORE risk chart for high-risk regions
37 (Denmark, Finland and Norway),[19 24] these included: age, sex, smoking status, mean total
38 cholesterol level, mean HDL cholesterol level and mean SBP. The general CVD risk score model was
39 also developed using data from the American Framingham Heart Study but using a larger cohort than
40 the Framingham model.[20] Individuals without CVD were used in the development of the general
41 CVD risk score model.[20] The simplified general CVD risk score model was developed similarly as
42 the general CVD risk score model. It is, however, a simpler CVD risk prediction model which is
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3 calculated using non-laboratory predictors. Risk variables (age, SBP, current antihypertensive
4 treatment, smoking status and diabetes status) were used in both of the models.[20] The only
5 difference is, BMI is included in the simplified general CVD risk score model instead of total and
6 HDL cholesterol which is used in the general CVD risk score model.
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10 11 12 13 **Statistical analysis**

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15 The data on the representative sample of 4487 Australian females ~~was~~were described using mean \pm
16 standard deviation for continuous variables, while counts and percentages were used for categorical
17 variables. Non-parametric Spearman's rank correlation was used to assess the associations between
18 anthropometric measurements of obesity with CVD risk factors, and with the calculated 10-year
19 predicted risks, due to the skewness in the distribution of some variables. Anthropometric
20 measurements were also converted to z-scores (original value subtracted by the mean and result
21 divided by the standard deviation) to represent the number of standard deviations above and below the
22 mean for each subject. Logistic regression was used to assess the effects of each standardised
23 anthropometric measurement of being above the recommended treatment thresholds for various risk
24 score models as a result of a one standard deviation increment above the mean for each
25 anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals represented
26 the likelihood of being above the recommended treatment thresholds for the specific risk score models
27 (20% for the Framingham risk score model for 10-year CVD incidence or death; 10% for SCORE risk
28 chart for high-risk regions for 10-year CVD death; 10% and 20% for the general CVD and simplified
29 general CVD risk score models for 10-year CVD incidence and death). The predictive ability of these
30 anthropometric measures to identify individuals above and below the treatment thresholds was
31 assessed using sensitivity, specificity and area under the receiver operating characteristic (ROC)
32 curve. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses
33 were performed with IBM SPSS Statistics Version 21.
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54 55 56 **RESULTS**

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3 The sample of 4487 women aged 20-69 years from the NHF Risk Factor Prevalence Study is a
4 representative sample of the Australian female population, free of heart disease, diabetes and stroke.
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6 The characteristics of the sample are summarised in Table 1. In addition to the conventional risk
7 factors for CVD, all anthropometric measurements of general and central obesity were presented.
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13 The 10-year CVD risk of each participant in the sample was calculated using four risk score models.
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15 The frequency distribution of calculated risks is presented in Table 2. Except for the Framingham
16 model for CVD incidence, all other models predicted risks of less than 10% for at least 85% of the
17 sample. The Framingham model for CVD incidence, general CVD model for CVD incidence and
18 death, and simplified general CVD model for CVD incidence and death, predicted risk values across
19 the entire range from 0% to greater than 40%.
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27 Anthropometric measurements of obesity were positively correlated with age, SBP, total cholesterol
28 and total cholesterol to HDL cholesterol ratio (all Spearman's $r \geq 0.195$, $p < 0.001$), with HC
29 recording the lowest correlations. These obesity measures were negatively correlated with HDL
30 cholesterol (all Spearman's $r \leq -0.160$, $p < 0.001$). Measures of central obesity that included a
31 measure of waist circumference (WC, WHR and WSR) generally recorded better correlations
32 compared to measures of general obesity (BMI and BAI).
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42 The associations between anthropometric measurements of obesity and the 10-year predicted risks
43 calculated using the four models are presented in Table 3. All Spearman's rank correlations were
44 statistically significant ($p < 0.0005$). All anthropometric measures of central obesity (WC, WHR and
45 WSR) generally had consistently higher correlations with the predicted risks calculated using the four
46 CVD risk score models, as compared to measures of general obesity
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54 Recommended treatment thresholds for the four CVD risk models were identified from a review of
55 the literature. Table 4 presents the effects of a one standard deviation increment in each
56 anthropometric measurement above the mean on the likelihood of being above the recommended
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3 thresholds or being indicated for treatment. All anthropometric measures of central obesity (WC,
4 WHR and WSR) generally recorded higher odds-ratios than general measures of obesity and they
5 increased the likelihood of individuals being above the respective treatment thresholds.
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11 Anthropometric measurements of central obesity (WC, WHR and WSR) also recorded higher area
12 under the ROC curves, higher sensitivity and specificity, than BMI in identifying females above and
13 below the 20% treatment threshold for the Framingham model for 10-year CVD incidence (Figure 1a)
14 and general CVD model for 10-year CVD incidence and death (Figure 1b). Although BMI is included
15 in the simplified general CVD model, high area under the ROC curve (> 0.76) are reported for both
16 WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements
17 as compared to general obesity measurement in predicting the increased risk of CVD.
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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
<i>Framingham 10-year predicted risk for CVD incidence (threshold = 20%)[25 26]</i>					
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)
<i>Framingham 10-year predicted risk for CVD death (threshold = 20%)[25 26]</i>					
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)
<i>SCORE-HIGH 10-year predicted risk for CVD death (threshold = 10%)[19]</i>					
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)
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 10%)[27]</i>					
#	2.16*** (1.99 - 2.34)	1.66*** (1.54 - 1.80)	2.16*** (1.98 - 2.35)	#	#
<i>GCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
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)
<i>SGCVD 10-year predicted risk for CVD incidence and death (threshold = 20%)[20 28]</i>					
#	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.

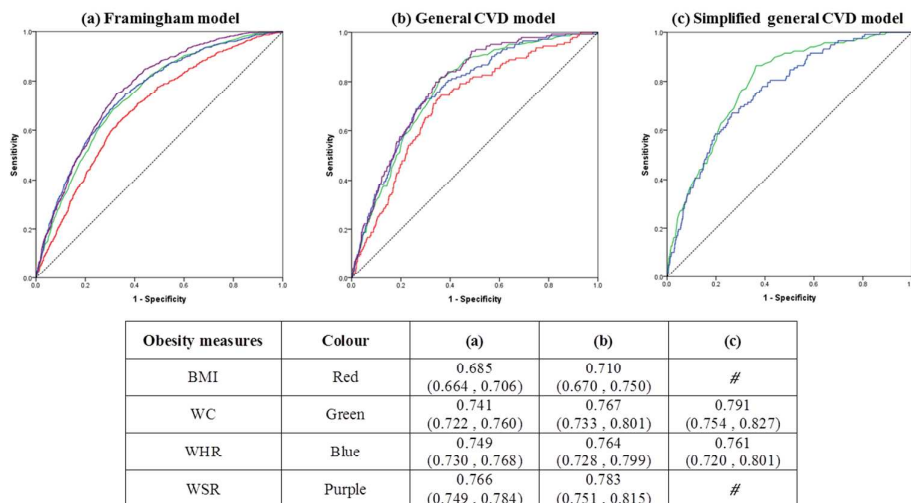


Figure 1 ROC curves to compare the predictive ability of obesity measures for being above the 20% cut-off of three CVD models: (a) Framingham risk score model for 10-year CVD incidence; (b) General cardiovascular disease risk score model for 10-year CVD incidence and death; (c) Simplified general cardiovascular 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

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3 inflammation which directly contributes to CVD risk.[35] Hence, measures that account for the
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5 accumulation of excess abdominal fat would report stronger associations and are desirable for
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7 assessing adiposity. They would also be more accurate at indicating CVD risk and should be
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9 incorporated into CVD assessment.[36-39] The addition of central obesity measures to BMI has also
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11 been shown to improve the accuracy of stratifying participants into lower and higher risk categories
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13 for mortality[40] and provides incremental value in predicting CVD above and beyond that provided
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15 by general obesity measures.[41-45] BMI is a flawed measure as it does not correctly identify
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17 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does
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19 not account for the effect of age and ethnicity on body fat distribution.[46-50] An increase in muscle
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21 or fat-free mass would, however, be reflected in the central obesity measures.
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25 Among central obesity measures, we found their performance to be comparable in our study. It
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27 remains unclear which measurement should be incorporated into CVD risk score models. A
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29 collaborative analysis of 58 prospective studies, however, reported that both measures of general and
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31 central obesity did not improve CVD risk assessment when information is available on SBP, diabetes
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33 and lipids.[51] Overweight and obesity is nevertheless important in CVD prevention, with one out of
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35 three of fatal and one out of seven of non-fatal CVD cases attributable to it.[34]
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39 Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and
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41 its association with CVD risk.[37] Some studies recommended the use of WC in clinical assessment
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43 and research studies.[52 53] In a systematic review and meta-analysis study of Caucasians without
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45 CVD, WC was most highly correlated with all CVD risk factors, compared to BMI, WHR, WSR and
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47 body fat percentage, in women.[52] In other studies, WC was also more closely associated with CVD
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49 risk factors than other measures of central obesity and BMI in women.[54-57] The advantages of WC
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51 are, it is easy to measure and interpret, and it is less prone to measurement and calculation error.[53]
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53 Appropriate sex, age and ethnic-specific WC cut points would need to be established.[44] It would
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55 also be difficult to use WC in today's multicultural societies due to requirements for different cut
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57 points.[50]
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5 The use of WHR is also supported as it is less strongly associated with BMI than WC and is thus a
6 more specific surrogate for fat distribution.[40] A longitudinal population study on 1462 women from
7 Sweden reported stronger relations between WHR and CVD endpoints, compared to BMI, WC and
8 HC.[58] These relations were mostly independent of age, BMI and either SBP, cholesterol level or
9 smoking habit.[58] In a meta-regression analysis of prospective studies, WHR was also more strongly
10 associated with CVD compared to WC, although the difference was not significant.[37] Another study
11 reported that WHR was associated with CVD mortality but not WC in elderly women from the United
12 Kingdom.[59] Elevated WHR was also independently associated with a higher CVD risk in the
13 Nurses' Health Study and in the Swedish Women's Lifestyle and Health Cohort Study.[45 60]
14 Women with a WHR of ≥ 0.88 were 3.25 times more at risk of CHD compared to women with a
15 WHR of < 0.72 after adjusting for BMI and other CVD risk factors.[45] Higher age and sex adjusted
16 odds-ratios were also reported with WHR and CHD and CVD mortality, compared to WC and BMI,
17 in an Australian population without heart disease, diabetes or stroke.[61] Similar results were
18 presented in other studies. WHR reported the highest age standardised hazard ratios in relation to
19 CVD mortality, followed by WSR, WC and BMI in women.[62 63] The advantages of WHR include,
20 it has low measurement error, high precision and no bias over a wide range of ethnic groups.[64]
21 WHR, however, may not be suitable for assessing central obesity in the elderly[65] due to laxity of
22 abdominal muscles which would undermine the predictive value of abdominal circumferences.[55] It
23 is also more difficult to measure than WC.[37] Despite its limitations, WHR has been recommended
24 for incorporation into CVD risk assessment.[37]

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48 WSR is the least commonly used measure of central obesity. In a systematic review and meta-analysis
49 study, WSR reported the weakest correlations with CVD risk factors, compared to BMI and other
50 measures of central obesity,[52] which is contrary to our study findings. In contrast, WSR was most
51 highly correlated with CHD risk predicted using the Framingham model[18] in women from England,
52 compared to BMI, WC and WHR in another study.[66] WSR, however, reported lower correlations
53 than WC and BMI following adjustments for age.[66] The advantage of WSR include, the same cut
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3 point could be applied across a wide range of populations. A cut-off value of 0.5 indicates increased
4 risk for men and women, people of different ethnic groups and this value may also be used in both
5 children and adults, unlike WC which requires different cut-offs.[67 68] More research is required to
6 assess the association between WSR and CVD risk in women, in comparison with WC, WHR and
7 BMI.
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15 Our study has limitations. This study is cross-sectional, however, it is a representative sample of the
16 Australian female population. There is only one set of baseline measurements recorded for some risk
17 variables but important variables including anthropometric measures of obesity are measured twice.
18 Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against
19 the treatment thresholds of the various models, and are not prospective CVD events.
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26 27 **CONCLUSIONS**

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29 Central obesity is more strongly associated with CVD risk than general obesity. The deposition of
30 adipose tissue is associated with systemic inflammation which has a direct effect on CVD risk.
31 Therefore, increments in central obesity have a more detrimental effect on CVD risk compared to
32 increments in general obesity.
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39 When used alone, BMI is inadequate for identifying individuals at increased risk of CVD as it does
40 not differentiate between fat and fat-free mass. On the other hand, anthropometric measurements of
41 central obesity have higher sensitivity and specificity. These measures are also more sensitive to
42 lifestyle modifications. An increase in muscle mass through diet and training would lead to changes in
43 measures such as WC and WSR but little change might be indicated with BMI.[69] It would be more
44 useful to measure a patient's central obesity during clinical assessment to evaluate the effect of
45 lifestyle changes in relation to CVD risk compared to BMI. Central obesity measures are also
46 significant and independent predictors of CVD risk, accounting for additional risk above BMI. These
47 measurements should be incorporated into CVD risk assessment, particularly when assessing the risk
48 in women and the elderly.[53 70-73]
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3 Future prospective studies are required to elucidate which anthropometric measurements of central
4 obesity are better indicators or predictors of CVD risk.[69] Studies measuring body fat distribution
5 using computerised tomography or magnetic resonance imaging are desirable to better understand the
6 association between body fat distribution and mortality, but costly.[74]
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13 In conclusion, WC, WHR and WSR, or measures of central obesity that include a measurement of
14 waist circumference, should be considered for incorporation into the clinical assessment of CVD risk.
15 Treatment of well-established CVD risk factors coupled with reducing overweight and obesity
16 through lifestyle modifications would be an advisable goal in the primary prevention of CVD.[4] It is
17 equally important to maintain a healthy weight and to prevent central or abdominal obesity
18 concurrently.
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37 important intellectual content. All authors read and approved the final manuscript.
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4
5 Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth
6
7 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin
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9 University. This study was carried out in accordance with the Declaration of Helsinki.
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13 **Data sharing statement** No additional data are available.
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For peer review only

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			

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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.