



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

Journal:	<i>BMJ Open</i>
Manuscript ID:	bmjopen-2013-004138
Article Type:	Research
Date Submitted by the Author:	27-Sep-2013
Complete List of Authors:	Goh, Louise; Curtin University, School of Public Health Dhaliwal, Satvinder; Curtin University, School of Public Health Welborn, Timothy; Sir Charles Gairdner Hospital, Lee, Andy; Curtin University, School of Public Health Della, Phillip; Curtin University, School of Nursing and Midwifery
<b>Primary Subject Heading</b>:	Public health
Secondary Subject Heading:	Epidemiology
Keywords:	PUBLIC HEALTH, EPIDEMIOLOGY, CARDIOLOGY

SCHOLARONE™  
Manuscripts

1  
2  
3 **Anthropometric measurements of general and central obesity and**  
4  
5  
6 **the prediction of cardiovascular disease risk in women: a cross-**  
7  
8  
9 **sectional study**  
10

14 Correspondence to: Professor Satvinder S Dhaliwal

15 School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
16 U1987, Perth, WA 6845, Australia

17  
18  
19 Tel: +618 9266 2949

20  
21  
22 Fax: +618 9266 2958

23  
24 E-mail: [s.dhaliwal@curtin.edu.au](mailto:s.dhaliwal@curtin.edu.au)  
25  
26  
27

28 Louise GH Goh,<sup>1</sup> Satvinder S Dhaliwal,<sup>1</sup> Timothy A Welborn,<sup>2</sup> Andy H Lee,<sup>1</sup> Phillip R Della<sup>3</sup>  
29  
30  
31

32 <sup>1</sup>School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
33 U1987, Perth, WA 6845, Australia  
34  
35

36 <sup>2</sup>Sir Charles Gairdner Hospital, Hospital Avenue, Nedlands, Perth, WA 6009, Australia  
37

38 <sup>3</sup>School of Nursing and Midwifery, Curtin Health Innovation Research Institute, Curtin University,  
39 GPO Box U1987, Perth, WA 6845, Australia  
40  
41  
42

43  
44 Anthropometric obesity measures and CVD risk  
45  
46  
47

48  
49 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female  
50  
51

52  
53 Word count: 3963  
54  
55  
56  
57  
58  
59  
60

**ABSTRACT**

**Objectives:** It is important to ascertain which anthropometric measurements of obesity, general or central, are better predictors of CVD risk in women. Ten-year CVD risk was calculated from the Framingham risk score model, SCORE risk chart for high-risk regions, general CVD and simplified general CVD risk score model. Increase in CVD risk associated with one standard deviation increment in each anthropometric measurement above the mean was calculated, and the diagnostic utility of obesity measures in identifying subjects with increased likelihood of being above the treatment threshold was assessed.

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

**Setting:** Population-based survey in Australia.

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

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

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

**Conclusions:** Central obesity measures should be included in the clinical assessment of CVD risk in place or in addition to BMI. It is equally important to maintain a healthy weight and to prevent central obesity concurrently.

For peer review only

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

## 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<sup>1.5</sup>, and subtracting 18 from the result). BMI or WC is most commonly used to measure body fatness.[9]

It is, however, unclear which anthropometric measurements are better correlated with CVD risk factors and CVD risk in women, considering adiposity is highly heterogeneous with age, sex and ethnic differences in body fat distribution.[8] Previous studies reported that BMI identified individuals at increased risk of CVD as effectively as WC.[10 11] In another study, BMI was a better predictor of CVD than WC.[12] Conversely, some studies reported that WC was a better indicator of CVD risk than BMI and WHR, in ethnically diverse groups.[13 14] Another study, however, reported that WC and WHR but not BMI were independent predictors of CVD risk, accounting for conventional risk factors in the Framingham risk score model.[16] More research is needed to ascertain which measures are better correlated with CVD risk factors and subsequent CVD risk in women.

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

## **METHODS**

### **Study cohort and measurements**

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

### **Variables in risk score models**

The Framingham 10-year predicted risk for CVD incidence or death[17] was calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status. Fewer variables were used in the calculation of the 10-year predicted CVD death with the SCORE risk chart for high-risk regions (Denmark, Finland and Norway),[18 23] these included: age, sex, smoking status, mean

1  
2  
3 total cholesterol level, mean HDL cholesterol level and mean SBP. Similar risk variables (age, SBP,  
4 current antihypertensive treatment, smoking status and diabetes status) were used in both the general  
5 CVD and simplified general CVD risk score model.[19] In the simplified general CVD risk score  
6 model, however, the total cholesterol level and HDL cholesterol level were replaced by BMI in the  
7 calculation of the 10-year risk for CVD incidence and death.  
8  
9

### 14 **Statistical analysis**

15  
16  
17 The data on the representative sample of 4487 Australian females was described using mean  $\pm$   
18 standard deviation for continuous variables, while counts and percentages were used for categorical  
19 variables. Non-parametric Spearman's rank correlation was used to assess the associations between  
20 anthropometric measurements of obesity with CVD risk factors, and with the calculated 10-year  
21 predicted risks, due to the skewness in the distribution of some variables. Anthropometric  
22 measurements were also converted to z-scores (original value subtracted by the mean and result  
23 divided by the standard deviation) to represent the number of standard deviations above and below the  
24 mean for each subject. Logistic regression was used to assess the effects of each standardised  
25 anthropometric measurement of being above the recommended treatment thresholds for various risk  
26 score models as a result of a one standard deviation increment above the mean for each  
27 anthropometric measure of obesity. Odds-ratios and associated 95% confidence intervals represented  
28 the likelihood of being above the recommended treatment thresholds for the specific risk score models  
29 (20% for the Framingham risk score model for 10-year CVD incidence or death; 10% for SCORE risk  
30 chart for high-risk regions for 10-year CVD death; 10% and 20% for the general CVD and simplified  
31 general CVD risk score models for 10-year CVD incidence and death). The predictive ability of these  
32 anthropometric measures to identify individuals above and below the treatment thresholds was  
33 assessed using sensitivity, specificity and area under the receiver operating characteristic (ROC)  
34 curve. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses  
35 were performed with IBM SPSS Statistics Version 20.  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60



## 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

1  
2  
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.  
7  
8  
9

10  
11  
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.  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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 <sup>2</sup> )	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.

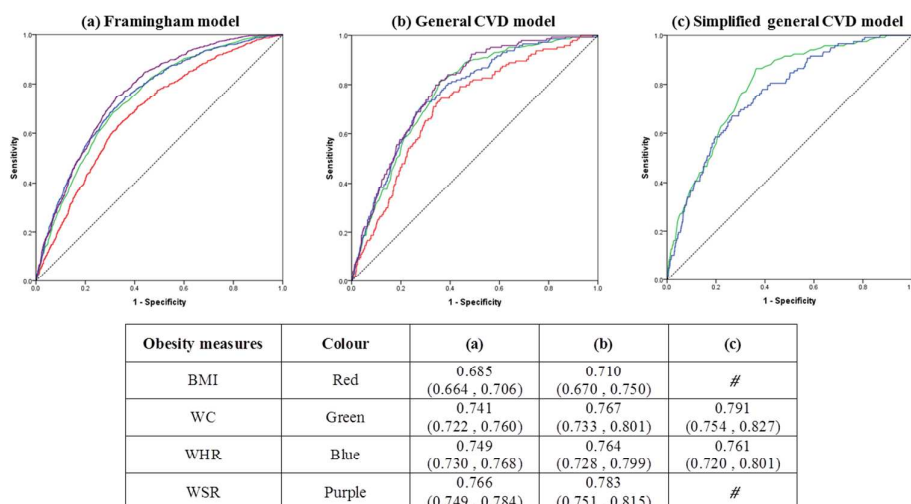
**Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity**

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



1  
2  
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.  
7  
8  
9

10  
11  
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]  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29

30  
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]  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

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

1  
2  
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  $\geq 0.88$  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]

20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60  
WSR is the least commonly used measure of central obesity. In a systematic review and meta-analysis  
study, WSR reported the weakest correlations with CVD risk factors, compared to BMI and other  
measures of central obesity,[51] which is contrary to our study findings. In contrast, WSR was most  
highly correlated with CHD risk predicted using the Framingham model[17] in women from England,  
compared to BMI, WC and WHR in another study.[65] WSR, however, reported lower correlations  
than WC and BMI following adjustments for age.[65] The advantage of WSR include, the same cut  
point could be applied across a wide range of populations. A cut-off value of 0.5 indicates increased  
risk for men and women, people of different ethnic groups and this value may also be used in both  
children and adults, unlike WC which requires different cut-offs.[66 67] More research is required to

1  
2  
3 assess the association between WSR and CVD risk in women, in comparison with WC, WHR and  
4  
5 BMI.  
6  
7

8  
9 Our study has limitations. This study is cross-sectional, however, it is a representative sample of the  
10  
11 Australian female population. There is only one set of baseline measurements recorded for some risk  
12  
13 variables but important variables including anthropometric measures of obesity are measured twice.  
14  
15 Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against  
16  
17 the treatment thresholds of the various models, and are not prospective CVD events.  
18  
19

## 20 21 **CONCLUSIONS**

22  
23 The significant and independent effect of obesity measures on CVD risk substantiates its inclusion  
24  
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  
28  
29 effect on CVD risk. Therefore, increments in central obesity have a more detrimental effect on CVD  
30  
31 risk compared to increments in general obesity.  
32  
33

34  
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  
40  
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  
46  
47 lifestyle changes in relation to CVD risk compared to BMI. Central obesity measures are also  
48  
49 significant and independent predictors of CVD risk, accounting for additional risk above BMI. These  
50  
51 measurements should be incorporated into CVD risk assessment, particularly when assessing the risk  
52  
53 in women and the elderly.[52 69-72]  
54  
55  
56  
57  
58  
59  
60

1  
2  
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]  
7  
8  
9

10  
11  
12  
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.  
19  
20  
21  
22  
23  
24

25  
26  
27 **Acknowledgements** Curtin University provided educational support to LGHG through the Curtin  
28 International Postgraduate Research Scholarship.  
29  
30  
31

32  
33 **Contributors** LGHG was involved in drafting the manuscript, interpretation of data and revising the  
34 manuscript critically for important intellectual content. SSD conceived the study, performed the  
35 analysis and data interpretation and revised the manuscript critically for important intellectual content.  
36 TAW participated in the study design, acquired the data and revised the manuscript critically for  
37 important intellectual content. All authors read and approved the final manuscript.  
38  
39  
40  
41  
42  
43

44  
45  
46 **Competing interests** None.  
47

48  
49 **Funding** None.  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
3 **Ethics approval** We have ethics approval for the use of the National Heart Foundation data from the  
4 Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth  
5 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin  
6 University. This study was carried out in accordance with the Declaration of Helsinki.  
7  
8  
9  
10

11  
12  
13 **Data sharing statement** No additional data are available.  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

## REFERENCES

1. World Health Organization. Obesity and overweight. Secondary Obesity and overweight 2012. <http://www.who.int/mediacentre/factsheets/fs311/en/>.
2. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. *Circulation* 2002;**106**(25):3143-421
3. Kannel WB. Metabolic risk factors for coronary heart disease in women: Perspective from the Framingham Study. *Am. Heart J.* 1987;**114**(2):413-19 doi: [http://dx.doi.org/10.1016/0002-8703\(87\)90511-4](http://dx.doi.org/10.1016/0002-8703(87)90511-4)[published Online First: Epub Date].
4. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation* 1983;**67**:968-77
5. Manson JE, Colditz GA, Stampfer MJ, et al. A Prospective Study of Obesity and Risk of Coronary Heart Disease in Women. *N. Engl. J. Med.* 1990;**322**(13):882-89 doi: doi:10.1056/NEJM199003293221303[published Online First: Epub Date].
6. Manson JE, Willett WC, Stampfer MJ, et al. Body Weight and Mortality among Women. *N. Engl. J. Med.* 1995;**333**(11):677-85 doi: doi:10.1056/NEJM199509143331101[published Online First: Epub Date].
7. Dorn JM, Schisterman EF, Winkelstein W, Trevisan M. Body Mass Index and Mortality in a General Population Sample Women of Men and Women: The Buffalo Health Study. *American Journal of Epidemiology* 1997;**146**(11):919-31
8. Cornier M-A, Després J-P, Davis N, et al. Assessing Adiposity: A Scientific Statement From the American Heart Association. *Circulation* 2011;**124**(18):1996-2019 doi: 10.1161/CIR.0b013e318233bc6a[published Online First: Epub Date].
9. Park YS, Kim J-S. Obesity Phenotype and Coronary Heart Disease Risk as Estimated by the Framingham Risk Score. *Journal of Korean Medical Science* 2012;**27**(3):243-49

- 1  
2  
3 10. Satoh H, Kishi R, Tsutsui H. Body Mass Index can Similarly Predict the Presence of Multiple  
4 Cardiovascular Risk Factors in Middle-aged Japanese Subjects as Waist Circumference.  
5 Internal Medicine 2010;**49**(11):977-82  
6  
7  
8  
9 11. Ryan MC, Fenster Farin HM, Abbasi F, Reaven GM. Comparison of Waist Circumference Versus  
10 Body Mass Index in Diagnosing Metabolic Syndrome and Identifying Apparently Healthy  
11 Subjects at Increased Risk of Cardiovascular Disease. The American Journal of Cardiology  
12 2008;**102**(1):40-46 doi: <http://dx.doi.org/10.1016/j.amjcard.2008.02.096>[published  
13 Online  
14 First: Epub Date]].  
15  
16  
17  
18 12. Ying X, Song Z, Zhao C, Jiang Y. Body mass index, waist circumference, and cardiometabolic  
19 risk factors in young and middle-aged Chinese women. J. Zhejiang Univ. Sci. B  
20 2010;**11**(9):639-46 doi: 10.1631/jzus.B1000105[published Online First: Epub Date]].  
21  
22  
23  
24 13. Zhu S, Heymsfield SB, Toyoshima H, Wang Z, Pietrobelli A, Heshka S. Race-ethnicity-specific  
25 waist circumference cutoffs for identifying cardiovascular disease risk factors. The American  
26 Journal of Clinical Nutrition 2005;**81**(2):409-15  
27  
28  
29  
30 14. Huang K-C, Lee M-S, Lee S-D, et al. Obesity in the Elderly and Its Relationship with  
31 Cardiovascular Risk Factors in Taiwan. Obes. Res. 2005;**13**(1):170-78 doi:  
32 10.1038/oby.2005.22[published Online First: Epub Date]].  
33  
34  
35  
36 15. Bergman RN, Stefanovski D, Buchanan TA, et al. A Better Index of Body Adiposity. Obesity  
37 (Silver Spring) 2011;**19**(5):1083-89  
38  
39  
40  
41 16. Dhaliwal SS, Welborn TA. Central obesity and multivariable cardiovascular risk as assessed by  
42 the Framingham prediction scores. The American Journal of Cardiology 2009;**103**:1403-07  
43  
44  
45  
46 17. Anderson KM, Odell PM, Wilson PW, Kannel WB. Cardiovascular disease risk profiles. Am.  
47 Heart J. 1991;**121**(1 Part 2):293-98  
48  
49  
50 18. Conroy RM, Pyörälä K, Fitzgerald AP, et al. Estimation of ten-year risk of fatal cardiovascular  
51 disease in Europe: The SCORE project. European Heart Journal 2003;**24**(11):987-1003 doi:  
52 10.1016/s0195-668x(03)00114-3[published Online First: Epub Date]].  
53  
54  
55  
56  
57  
58  
59  
60



- 1  
2  
3 19. D'Agostino RB, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in  
4 primary care - The Framingham Heart Study. *Circulation* 2008;**117**(6):743-53 doi:  
5 10.1161/circulationaha.107.699579[published Online First: Epub Date].  
6  
7  
8  
9 20. Australian Risk Factor Prevalence Study Management Committee. Survey No. 3 1989. Canberra:  
10 National Heart Foundation of Australia and Australia Institute of Health, 1990.  
11  
12 21. Boyle CA, Dobson AJ, Egger G, Benault SA. Waist-to-hip ratios in Australia: A different picture  
13 of obesity. *Australian Journal of Nutrition and Dietetics* 1993;**50**:57-64  
14  
15  
16  
17 22. Alexander H, Dugdale A. Which waist-hip ratio? *Med. J. Aust.* 1990;**153**(6):367-68  
18  
19 23. Cooney MT, Dudina A, De Bacquer D, et al. How much does HDL cholesterol add to risk  
20 estimation? A report from the SCORE investigators. *European Journal of Cardiovascular*  
21 *Prevention & Rehabilitation* 2009;**16**(3):304-14 doi:  
22 10.1097/HJR.0b013e3283213140[published Online First: Epub Date].  
23  
24  
25  
26  
27 24. Neil HAW, Perera R, Armitage JM, Farmer AJ, Mant D, Durrington PN. Estimated 10-year  
28 cardiovascular risk in a British population: results of a national screening project.  
29 *International Journal of Clinical Practice* 2008;**62**(9):1322-31 doi: 10.1111/j.1742-  
30 1241.2008.01828.x[published Online First: Epub Date].  
31  
32  
33  
34  
35 25. Woodward M, Brindle P, Tunstall-Pedoe H. Adding social deprivation and family history to  
36 cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended  
37 Cohort (SHHEC). *Heart* 2007;**93**(2):172-76 doi: 10.1136/hrt.2006.108167[published Online  
38 First: Epub Date].  
39  
40  
41  
42  
43 26. Mosca L, Benjamin EJ, Berra K, et al. Effectiveness-Based Guidelines for the Prevention of  
44 Cardiovascular Disease in Women—2011 Update A Guideline From the American Heart  
45 Association. *J. Am. Coll. Cardiol.* 2011;**57**(12):1404-23 doi:  
46 10.1016/j.jacc.2011.02.005[published Online First: Epub Date].  
47  
48  
49  
50  
51 27. Genest J, McPherson R, Frohlich J, et al. 2009 Canadian Cardiovascular Society/Canadian  
52 guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular  
53 disease in the adult – 2009 recommendations. *The Canadian Journal of Cardiology*  
54 2009;**25**(10):567-79  
55  
56  
57  
58  
59  
60



- 1  
2  
3 28. Wittchen H-U, Balkau B, Massien C, Richard A, Haffner S, Després J-P. International Day for the  
4 Evaluation of Abdominal obesity: rationale and design of a primary care study on the  
5 prevalence of abdominal obesity and associated factors in 63 countries. *Eur. Heart J. Suppl.*  
6 2006;**8**(suppl B):B26-B33 doi: 10.1093/eurheartj/sul005[published Online First: Epub Date]].  
7  
8  
9  
10  
11 29. Balkau B, Deanfield JE, Despres JP, et al. International day for the evaluation of abdominal  
12 obesity (IDEA) - A study of waist circumference, cardiovascular disease, and diabetes  
13 mellitus in 168 000 primary care patients in 63 countries. *Circulation* 2007;**116**(17):1942-51  
14 doi: 10.1161/circulationaha.106.676379[published Online First: Epub Date]].  
15  
16  
17  
18  
19 30. Winter Y, Rohrmann S, Linseisen J, et al. Contribution of Obesity and Abdominal Fat Mass to  
20 Risk of Stroke and Transient Ischemic Attacks. *Stroke* 2008;**39**(12):3145-51 doi:  
21 10.1161/strokeaha.108.523001[published Online First: Epub Date]].  
22  
23  
24  
25 31. Taylor AE, Ebrahim S, Ben-Shlomo Y, et al. Comparison of the associations of body mass index  
26 and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-  
27 cause mortality: a study using data from 4 UK cohorts. *The American Journal of Clinical*  
28 *Nutrition* 2010;**91**(3):547-56 doi: 10.3945/ajcn.2009.28757[published Online First: Epub  
29 Date]].  
30  
31  
32  
33  
34  
35 32. van Dis I, Kromhout D, Geleijnse JM, Boer JMA, Verschuren WMM. Body mass index and waist  
36 circumference predict both 10-year nonfatal and fatal cardiovascular disease risk: study  
37 conducted in 20 000 Dutch men and women aged 20–65 years. *European Journal of*  
38 *Cardiovascular Prevention & Rehabilitation* 2009;**16**(6):729-34 doi:  
39 10.1097/HJR.0b013e328331dfc0[published Online First: Epub Date]].  
40  
41  
42  
43  
44  
45 33. Berg AH, Scherer PE. Adipose Tissue, Inflammation, and Cardiovascular Disease. *Circulation*  
46 *Research* 2005;**96**(9):939-49 doi: 10.1161/01.res.0000163635.62927.34[published Online  
47 First: Epub Date]].  
48  
49  
50  
51 34. Snijder MB, van Dam RM, Visser M, Seidell JC. What aspects of body fat are particularly  
52 hazardous and how do we measure them? *International Journal of Epidemiology*  
53 2006;**35**(1):83-92 doi: 10.1093/ije/dyi253[published Online First: Epub Date]].  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 35. de Koning L, Merchant AT, Pogue J, Anand SS. Waist circumference and waist-to-hip ratio as  
4 predictors of cardiovascular events: meta-regression analysis of prospective studies. *European*  
5 *Heart Journal* 2007;**28**(7):850-56 doi: 10.1093/eurheartj/ehm026[published Online First:  
6 Epub Date]].  
7  
8  
9  
10  
11 36. Dalton M, Cameron AJ, Zimmet PZ, et al. Waist circumference, waist-hip ratio and body mass  
12 index and their correlation with cardiovascular disease risk factors in Australian adults.  
13 *Journal of Internal Medicine* 2003;**254**(6):555-63 doi: 10.1111/j.1365-  
14 2796.2003.01229.x[published Online First: Epub Date]].  
15  
16  
17  
18  
19 37. Antillon D, Towfighi A. No time to 'weight': the link between obesity and stroke in women.  
20 *Women's Health* 2011;**7**(4):453-63 doi: 10.2217/whe.11.36[published Online First: Epub  
21 Date]].  
22  
23  
24  
25 38. Pischon T, Boeing H, Hoffmann K, et al. General and Abdominal Adiposity and Risk of Death in  
26 Europe. *N. Engl. J. Med.* 2008;**359**(20):2105-20 doi: doi:10.1056/NEJMoa0801891[published  
27 Online First: Epub Date]].  
28  
29  
30  
31 39. Li C, Engstrom G, Hedblad B, Calling S, Berglund G, Janzon L. Sex differences in the  
32 relationships between BMI, WHR and incidence of cardiovascular disease: a population-  
33 based cohort study. *International Journal of Obesity* 2006;**30**(12):1775-81 doi:  
34 10.1038/sj.ijo.0803339[published Online First: Epub Date]].  
35  
36  
37  
38  
39 40. Freiberg MS, Pencina MJ, D'Agostino RB, Lanier K, Wilson PWF, Vasan RS. BMI vs. Waist  
40 Circumference for Identifying Vascular Risk. *Obesity* 2008;**16**(2):463-69  
41  
42  
43 41. DiPietro L, Katz LD, Nadel ER. Excess abdominal adiposity remains correlated with altered lipid  
44 concentrations in healthy older women. *International journal of obesity and related metabolic*  
45 *disorders : journal of the International Association for the Study of Obesity* 1999;**23**(4):432-  
46 36  
47  
48  
49  
50  
51 42. Klein S, Allison DB, Heymsfield SB, et al. Waist Circumference and Cardiometabolic Risk: A  
52 Consensus Statement from Shaping America's Health: Association for Weight Management  
53 and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition;  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 and the American Diabetes Association. *Obesity* 2007;**15**(5):1061-67 doi:  
4 10.1038/oby.2007.632[published Online First: Epub Date].  
5  
6  
7 43. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in  
8 women. *JAMA* 1998;**280**(21):1843-48 doi: 10.1001/jama.280.21.1843[published Online  
9 First: Epub Date].  
10  
11  
12 44. Fogelholm M. Physical activity, fitness and fatness: relations to mortality, morbidity and disease  
13 risk factors. A systematic review. *Obesity Reviews* 2010;**11**(3):202-21 doi: 10.1111/j.1467-  
14 789X.2009.00653.x[published Online First: Epub Date].  
15  
16  
17 45. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Accuracy of body mass index in  
18 diagnosing obesity in the adult general population. *Int J Obes* 2008;**32**(6):959-66 doi:  
19 <http://www.nature.com/ijo/journal/v32/n6/supinfo/ijo200811s1.html>[published Online First:  
20 Epub Date].  
21  
22  
23 46. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a  
24 WHO consultation. World Health Organ Tech Rep Ser: WHO, 2000.  
25  
26  
27 47. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis  
28 among different ethnic groups. *International Journal of Obesity* 1998;**22**(12):1164-71  
29  
30  
31 48. Welborn TA, Dhaliwal SS. Being correct about obesity. *Med J Aust* 2011;**194** (8 ):429-30  
32  
33  
34 49. Goh LGH, Dhaliwal SS, Lee AH, Bertolatti D, Della PR. Utility of established cardiovascular  
35 disease risk score models for the 10-year prediction of disease outcomes in women. *Expert*  
36 *Review of Cardiovascular Therapy* 2013;**11**(4):425-35 doi: 10.1586/erc.13.26[published  
37 Online First: Epub Date].  
38  
39  
40 50. The Emerging Risk Factors Collaboration. Separate and combined associations of body-mass  
41 index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58  
42 prospective studies. *The Lancet* 2011;**377**(9771):1085-95 doi:  
43 [http://dx.doi.org/10.1016/S0140-6736\(11\)60105-0](http://dx.doi.org/10.1016/S0140-6736(11)60105-0)[published Online First: Epub Date].  
44  
45  
46 51. Dijk SB, Takken T, Prinsen EC, Wittink H. Different anthropometric adiposity measures and their  
47 association with cardiovascular disease risk factors: a meta-analysis. *Neth Heart J*  
48 2012;**20**(5):208-18 doi: 10.1007/s12471-011-0237-7[published Online First: Epub Date].  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 52. Dobbelsteyn CJ, Joffres MR, MacLean DR, Flowerdew G, The Canadian Heart Health Surveys  
4  
5 Research Group. A comparative evaluation of waist circumference, waist-to-hip ratio and  
6  
7 body mass index as indicators of cardiovascular risk factors. The Canadian Heart Health  
8  
9 Surveys. *International Journal of Obesity* 2001;**25**:652-61  
10  
11 53. Pouliot M-C, Després J-P, Lemieux S, et al. Waist circumference and abdominal sagittal diameter:  
12  
13 Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and  
14  
15 related cardiovascular risk in men and women. *The American Journal of Cardiology*  
16  
17 1994;**73**(7):460-68 doi: [http://dx.doi.org/10.1016/0002-9149\(94\)90676-9](http://dx.doi.org/10.1016/0002-9149(94)90676-9)[published  
18  
19 First: Epub Date]].  
20  
21 54. Turcato E, Bosello O, Di Francesco V, et al. Waist circumference and abdominal sagittal diameter  
22  
23 as surrogates of body fat distribution in the elderly: their relation with cardiovascular risk  
24  
25 factors. *International journal of obesity and related metabolic disorders : journal of the*  
26  
27 *International Association for the Study of Obesity* 2000;**24**(8):1005-10  
28  
29 55. Zhu S, Wang Z, Heshka S, Heo M, Faith MS, Heymsfield SB. Waist circumference and obesity-  
30  
31 associated risk factors among whites in the third National Health and Nutrition Examination  
32  
33 Survey: clinical action thresholds. *The American Journal of Clinical Nutrition* 2002;**76**(4):743  
34  
35 56. Reeder BA, Senthilselvan A, Després JP, et al. The association of cardiovascular disease risk  
36  
37 factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group.  
38  
39 *CMAJ : Canadian Medical Association journal* 1997;**157 Suppl 1**:S39-S45  
40  
41 57. Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjöström L. Distribution of adipose tissue  
42  
43 and risk of cardiovascular disease and death: a 12 year follow up of participants in the  
44  
45 population study of women in Gothenburg, Sweden. *BMJ* 1984;**289**(6454):1257-61 doi:  
46  
47 10.1136/bmj.289.6454.1257[published Online First: Epub Date]].  
48  
49 58. Price GM, Uauy R, Breeze E, Bulpitt CJ, Fletcher AE. Weight, shape, and mortality risk in older  
50  
51 persons: elevated waist-hip ratio, not high body mass index, is associated with a greater risk  
52  
53 of death. *The American Journal of Clinical Nutrition* 2006;**84**(2):449-60  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 59. Lu M, Ye W, Adami HO, Weiderpass E. Prospective study of body size and risk for stroke  
4 amongst women below age 60. *Journal of Internal Medicine* 2006;**260**(5):442-50 doi:  
5 10.1111/j.1365-2796.2006.01706.x[published Online First: Epub Date].  
6  
7  
8  
9 60. Dhaliwal SS, Welborn TA. Central obesity and cigarette smoking are key determinants of  
10 cardiovascular deaths in Australia: A public health perspective. *Preventive Medicine*  
11 2009;**49**(2-3):153-57  
12  
13 61. Welborn TA, Dhaliwal SS. Preferred clinical measures of central obesity for predicting mortality.  
14 *European Journal of Clinical Nutrition* 2007;**61**(12):1373-79  
15  
16 62. Welborn TA, Dhaliwal SS, Bennett SA. Waist-hip ratio is the dominant risk factor predicting  
17 cardiovascular death in Australia. *Med. J. Aust.* 2003;**179**(11-12):580-85  
18  
19 63. Dhaliwal SS, Welborn TA. Measurement error and ethnic comparisons of measures of abdominal  
20 obesity. *Preventive Medicine* 2009;**49**(2-3):148-52 doi:  
21 10.1016/j.ypmed.2009.06.023[published Online First: Epub Date].  
22  
23 64. Goodman-Gruen D, Barrett-Connor E. Sex Differences in Measures of Body Fat and Body Fat  
24 Distribution in the Elderly. *American Journal of Epidemiology* 1996;**143**(9):898-906  
25  
26 65. Ashwell M, Lejeune S. Ratio of waist circumference to height may be better indicator of need for  
27 weight management. *BMJ* 1996;**312**(7027):377 doi: 10.1136/bmj.312.7027.377[published  
28 Online First: Epub Date].  
29  
30 66. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global  
31 indicator for health risks of obesity and how its use could simplify the international public  
32 health message on obesity. *International Journal of Food Sciences and Nutrition*  
33 2005;**56**(5):303-07 doi: doi:10.1080/09637480500195066[published Online First: Epub  
34 Date].  
35  
36 67. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening  
37 tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global  
38 boundary value. *Nutrition Research Reviews* 2010;**23**(02):247-69 doi:  
39 doi:10.1017/S0954422410000144[published Online First: Epub Date].  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 68. Schneider HJ, Glaesmer H, Klotsche J, et al. Accuracy of anthropometric indicators of obesity to  
4 predict cardiovascular risk. *J Clin Endocrinol Metab* 2007;**92**:589-94  
5  
6  
7 69. Okosun IS, Liao Y, Rotimi CN, Prewitt TE, Cooper RS. Abdominal Adiposity and Clustering of  
8 Multiple Metabolic Syndrome in White, Black and Hispanic Americans. *Annals of*  
9 *Epidemiology* 2000;**10**(5):263-70 doi: [http://dx.doi.org/10.1016/S1047-2797\(00\)00045-](http://dx.doi.org/10.1016/S1047-2797(00)00045-4)  
10 [4](http://dx.doi.org/10.1016/S1047-2797(00)00045-4)[published Online First: Epub Date]].  
11  
12  
13 70. Ho SC, Chen YM, Woo JLF, Leunga SSF, Lam THJ, E D. . Association between simple  
14 anthropometric indices and cardiovascular risk factors. *International Journal of Obesity*  
15 2001;**25**(11):1689-97  
16  
17  
18 71. Jeong S-K, Seo M-W, Kim Y-H, Kweon S-S, Nam H-S. Does Waist Indicate Dyslipidemia better  
19 than BMI in Korean Adult Population? *J Korean Med Sci* 2005;**20**(1):7-12  
20  
21  
22 72. Yusuf S, Hawken S, Ôunpuu S, et al. Obesity and the risk of myocardial infarction in 27□000  
23 participants from 52 countries: a case-control study. *The Lancet* 2005;**366**(9497):1640-49 doi:  
24 [http://dx.doi.org/10.1016/S0140-6736\(05\)67663-5](http://dx.doi.org/10.1016/S0140-6736(05)67663-5)[published Online First: Epub Date]].  
25  
26  
27  
28  
29  
30  
31 73. Moore SC. Waist versus weight—which matters more for mortality? *The American Journal of*  
32 *Clinical Nutrition* 2009;**89**(4):1003-04 doi: 10.3945/ajcn.2009.27598[published Online First:  
33 Epub Date]].  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

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
<b>Introduction</b>			
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
<b>Methods</b>			
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.
<b>Results</b>			



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
<b>Discussion</b>			
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
<b>Other information</b>			
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](http://www.strobe-statement.org).





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

Journal:	<i>BMJ Open</i>
Manuscript ID:	bmjopen-2013-004138.R1
Article Type:	Research
Date Submitted by the Author:	20-Nov-2013
Complete List of Authors:	Goh, Louise; Curtin University, School of Public Health Dhaliwal, Satvinder; Curtin University, School of Public Health Welborn, Timothy; Sir Charles Gairdner Hospital, Lee, Andy; Curtin University, School of Public Health Della, Phillip; Curtin University, School of Nursing and Midwifery
<b>Primary Subject Heading</b>:	Public health
Secondary Subject Heading:	Epidemiology
Keywords:	PUBLIC HEALTH, EPIDEMIOLOGY, Cardiology < INTERNAL MEDICINE

SCHOLARONE™  
Manuscripts

1  
2  
3 **Anthropometric measurements of general and central obesity and**  
4  
5  
6 **the prediction of cardiovascular disease risk in women: a cross-**  
7  
8  
9 **sectional study**  
10

14 Correspondence to: Professor Satvinder S Dhaliwal

15 School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
16 U1987, Perth, WA 6845, Australia

17  
18  
19  
20 Tel: +618 9266 2949

21  
22 Fax: +618 9266 2958

23  
24 E-mail: [s.dhaliwal@curtin.edu.au](mailto:s.dhaliwal@curtin.edu.au)  
25  
26  
27

28 Louise GH Goh,<sup>1</sup> Satvinder S Dhaliwal,<sup>1</sup> Timothy A Welborn,<sup>2</sup> Andy H Lee,<sup>1</sup> Phillip R Della<sup>3</sup>  
29  
30  
31

32 <sup>1</sup>School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
33 U1987, Perth, WA 6845, Australia  
34  
35

36 <sup>2</sup>Sir Charles Gairdner Hospital, Hospital Avenue, Nedlands, Perth, WA 6009, Australia  
37

38 <sup>3</sup>School of Nursing and Midwifery, Curtin Health Innovation Research Institute, Curtin University,  
39 GPO Box U1987, Perth, WA 6845, Australia  
40  
41  
42  
43

44 Anthropometric obesity measures and CVD risk  
45  
46  
47

48 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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.

1  
2  
3 **Conclusions:** Central obesity measures are better predictors of CVD risk compared to general obesity  
4 measures in women. It is equally important to maintain a healthy weight and to prevent central obesity  
5 concurrently.  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

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 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<sup>1.5</sup>, 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

1  
2  
3 risk factors in the Framingham risk score model.[17] More research is thus needed to ascertain which  
4  
5 measures are better correlated with CVD risk factors and subsequent CVD risk in women.  
6  
7

8  
9 We aim to assess the associations between general and central obesity anthropometric measures with  
10  
11 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart  
12  
13 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk  
14  
15 calculated from the Framingham risk score model for 10-year CVD incidence or death,[18] SCORE  
16  
17 risk chart for high-risk regions for 10-year CVD death,[19] general CVD and simplified general CVD  
18  
19 risk score models for 10-year CVD incidence and death[20] would also be assessed. To aid  
20  
21 comparison between obesity indices, which are measured in different units, the incremental shift in  
22  
23 CVD risk with one standard deviation increment in each anthropometric measurement above the mean  
24  
25 would be assessed. Finally, we determined which indices of obesity are most sensitive and specific for  
26  
27 identifying females at increased 10-year CVD risk.  
28  
29

## 30 31 **METHODS**

### 32 33 **Study cohort and measurements**

34  
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)  
38  
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,  
44  
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  
48  
49 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,  
50  
51 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of  
52  
53 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip  
54  
55 circumference were collected according to standardised methodologies[22 23] using two observers.  
56  
57 The waist circumference was measured from the front at the narrowest point between the rib cage and  
58  
59  
60

iliac crest after full expiration while the hip circumference was measured from the side at the maximal extension of buttocks by one observer using a metal tape. A second observer recorded another set of measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of two measurements was taken at each site to the nearest centimetre.

### **Risk score models**

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

### **Statistical analysis**



1  
2  
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.  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42

## 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.  
48  
49  
50  
51  
52  
53  
54

55 The 10-year CVD risk of each participant in the sample was calculated using four risk score models.  
56 The frequency distribution of calculated risks is presented in Table 2. Except for the Framingham  
57  
58  
59  
60

1  
2  
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%.  
7  
8  
9

10  
11  
12 Anthropometric measurements of obesity were positively correlated with age, SBP, total cholesterol  
13 and total cholesterol to HDL cholesterol ratio (all Spearman's  $r \geq 0.195$ ,  $p < 0.001$ ), with HC  
14 recording the lowest correlations. These obesity measures were negatively correlated with HDL  
15 cholesterol (all Spearman's  $r \leq -0.160$ ,  $p < 0.001$ ). Measures of central obesity that included a  
16 measure of waist circumference (WC, WHR and WSR) generally recorded better correlations  
17 compared to measures of general obesity (BMI and BAI).  
18  
19  
20  
21  
22  
23  
24  
25  
26

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  
32  
33  
34  
35  
36  
37  
38  
39

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.  
46  
47  
48  
49  
50  
51  
52  
53

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)  
57  
58  
59  
60

1  
2  
3 and general CVD model for 10-year CVD incidence and death (Figure 1b). Although BMI is included  
4  
5 in the simplified general CVD model, high area under the ROC curve ( $> 0.76$ ) are reported for both  
6  
7 WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements  
8  
9 as compared to general obesity measurement in predicting the increased risk of CVD.  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

For peer review only

**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 <sup>2</sup> )	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.

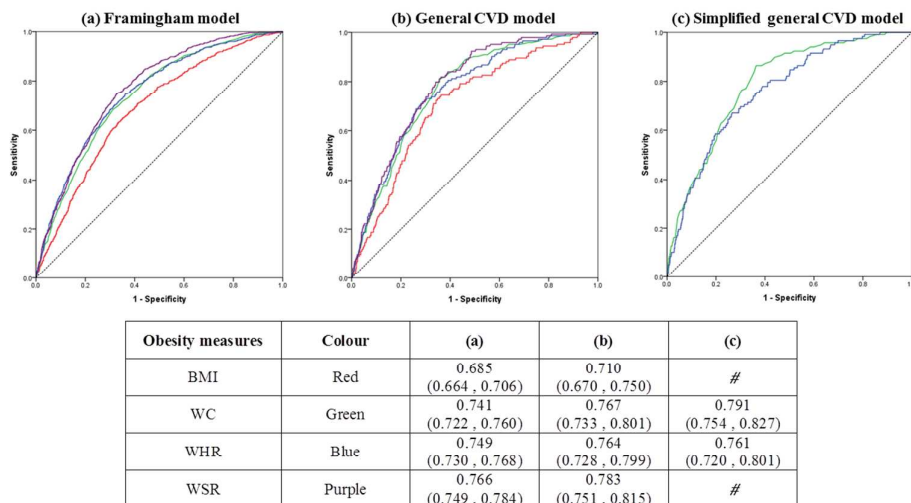
**Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity**

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

1  
2  
3 inflammation which directly contributes to CVD risk.[35] Hence, measures that account for the  
4 accumulation of excess abdominal fat would report stronger associations and are desirable for  
5 assessing adiposity. They would also be more accurate at indicating CVD risk and should be  
6 incorporated into CVD assessment.[36-39] The addition of central obesity measures to BMI has also  
7 been shown to improve the accuracy of stratifying participants into lower and higher risk categories  
8 for mortality[40] and provides incremental value in predicting CVD above and beyond that provided  
9 by general obesity measures.[41-45] BMI is a flawed measure as it does not correctly identify  
10 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does  
11 not account for the effect of age and ethnicity on body fat distribution.[46-50] An increase in muscle  
12 or fat-free mass would, however, be reflected in the central obesity measures.

13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60  
Among central obesity measures, we found their performance to be comparable in our study. It  
remains unclear which measurement should be incorporated into CVD risk score models. A  
collaborative analysis of 58 prospective studies, however, reported that both measures of general and  
central obesity did not improve CVD risk assessment when information is available on SBP, diabetes  
and lipids.[51] Overweight and obesity is nevertheless important in CVD prevention, with one out of  
three of fatal and one out of seven of non-fatal CVD cases attributable to it.[34]

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

1  
2  
3  
4  
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  $\geq 0.88$  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]

25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
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  
54  
55  
56  
57  
58  
59  
60

1  
2  
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.  
8  
9

10  
11  
12  
13  
14  
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.  
20  
21  
22  
23  
24  
25

## 26 27 **CONCLUSIONS**

28  
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.  
33  
34  
35  
36  
37

38  
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]  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
3  
4  
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]  
9  
10  
11  
12

13  
14  
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.  
21  
22  
23  
24  
25  
26  
27  
28

29 **Acknowledgements** Curtin University provided educational support to LGHG through the Curtin  
30 International Postgraduate Research Scholarship.  
31  
32  
33  
34

35 **Contributors** LGHG was involved in drafting the manuscript, interpretation of data and revising the  
36 manuscript critically for important intellectual content. SSD conceived the study, performed the  
37 analysis and data interpretation and revised the manuscript critically for important intellectual content.  
38 TAW participated in the study design, acquired the data and revised the manuscript critically for  
39 important intellectual content. All authors read and approved the final manuscript.  
40  
41  
42  
43  
44  
45  
46  
47

48 **Competing interests** None.  
49  
50

51 **Funding** None.  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
3 **Ethics approval** We have ethics approval for the use of the National Heart Foundation data from the  
4 Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth  
5 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin  
6 University. This study was carried out in accordance with the Declaration of Helsinki.  
7  
8  
9  
10

11  
12  
13 **Data sharing statement** No additional data are available.  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

For peer review only

## REFERENCES

1. World Health Organization. Obesity and overweight. Secondary Obesity and overweight 2012. <http://www.who.int/mediacentre/factsheets/fs311/en/>.
2. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. *Circulation* 2002;**106**(25):3143-421
3. Kannel WB. Metabolic risk factors for coronary heart disease in women: Perspective from the Framingham Study. *Am Heart J* 1987;**114**(2):413-19 doi: [http://dx.doi.org/10.1016/0002-8703\(87\)90511-4](http://dx.doi.org/10.1016/0002-8703(87)90511-4)[published Online First: Epub Date]].
4. Hubert HB, Feinleib M, McNamara PM, et al. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation* 1983;**67**:968-77
5. Manson JE, Colditz GA, Stampfer MJ, et al. A Prospective Study of Obesity and Risk of Coronary Heart Disease in Women. *N Engl J Med* 1990;**322**(13):882-89 doi: doi:10.1056/NEJM199003293221303[published Online First: Epub Date]].
6. Manson JE, Willett WC, Stampfer MJ, et al. Body Weight and Mortality among Women. *N Engl J Med* 1995;**333**(11):677-85 doi: doi:10.1056/NEJM199509143331101[published Online First: Epub Date]].
7. Dorn JM, Schisterman EF, Winkelstein W, et al. Body Mass Index and Mortality in a General Population Sample Women of Men and Women: The Buffalo Health Study. *American Journal of Epidemiology* 1997;**146**(11):919-31
8. Cornier M-A, Després J-P, Davis N, et al. Assessing Adiposity: A Scientific Statement From the American Heart Association. *Circulation* 2011;**124**(18):1996-2019 doi: 10.1161/CIR.0b013e318233bc6a[published Online First: Epub Date]].
9. Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation* 2013



- 1  
2  
3 10. Park YS, Kim J-S. Obesity Phenotype and Coronary Heart Disease Risk as Estimated by the  
4 Framingham Risk Score. *Journal of Korean Medical Science* 2012;**27**(3):243-49  
5  
6  
7 11. Satoh H, Kishi R, Tsutsui H. Body Mass Index can Similarly Predict the Presence of Multiple  
8 Cardiovascular Risk Factors in Middle-aged Japanese Subjects as Waist Circumference.  
9 *Internal Medicine* 2010;**49**(11):977-82  
10  
11  
12  
13 12. Ryan MC, Fenster Farin HM, Abbasi F, et al. Comparison of Waist Circumference Versus Body  
14 Mass Index in Diagnosing Metabolic Syndrome and Identifying Apparently Healthy Subjects  
15 at Increased Risk of Cardiovascular Disease. *The American Journal of Cardiology*  
16 2008;**102**(1):40-46 doi: <http://dx.doi.org/10.1016/j.amjcard.2008.02.096>[published  
17 Online  
18 First: Epub Date]].  
19  
20  
21  
22  
23 13. Ying X, Song Z, Zhao C, et al. Body mass index, waist circumference, and cardiometabolic risk  
24 factors in young and middle-aged Chinese women. *J Zhejiang Univ Sci B* 2010;**11**(9):639-46  
25 doi: 10.1631/jzus.B1000105[published Online First: Epub Date]].  
26  
27  
28  
29 14. Zhu S, Heymsfield SB, Toyoshima H, et al. Race-ethnicity-specific waist circumference cutoffs  
30 for identifying cardiovascular disease risk factors. *The American Journal of Clinical Nutrition*  
31 2005;**81**(2):409-15  
32  
33  
34  
35 15. Huang K-C, Lee M-S, Lee S-D, et al. Obesity in the Elderly and Its Relationship with  
36 Cardiovascular Risk Factors in Taiwan. *Obes Res* 2005;**13**(1):170-78 doi:  
37 10.1038/oby.2005.22[published Online First: Epub Date]].  
38  
39  
40  
41 16. Bergman RN, Stefanovski D, Buchanan TA, et al. A Better Index of Body Adiposity. *Obesity*  
42 (Silver Spring) 2011;**19**(5):1083-89  
43  
44  
45  
46 17. Dhaliwal SS, Welborn TA. Central obesity and multivariable cardiovascular risk as assessed by  
47 the Framingham prediction scores. *Am J Cardiol* 2009;**103**:1403-07  
48  
49  
50 18. Anderson KM, Odell PM, Wilson PW, et al. Cardiovascular disease risk profiles. *Am Heart J*  
51 1991;**121**(1 Part 2):293-98  
52  
53  
54 19. Conroy RM, Pyörälä K, Fitzgerald AP, et al. Estimation of ten-year risk of fatal cardiovascular  
55 disease in Europe: The SCORE project. *European Heart Journal* 2003;**24**(11):987-1003 doi:  
56 10.1016/s0195-668x(03)00114-3[published Online First: Epub Date]].  
57  
58  
59  
60



- 1  
2  
3 20. D'Agostino RB, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in  
4 primary care - The Framingham Heart Study. *Circulation* 2008;**117**(6):743-53  
5  
6  
7 21. Australian Risk Factor Prevalence Study Management Committee. Survey No. 3 1989. Canberra:  
8 National Heart Foundation of Australia and Australia Institute of Health, 1990.  
9  
10  
11 22. Boyle CA, Dobson AJ, Egger G, et al. Waist-to-hip ratios in Australia: A different picture of  
12 obesity. *Aust J Nutr Diet* 1993;**50**:57-64  
13  
14  
15 23. Alexander H, Dugdale A. Which waist-hip ratio? *Med J Aust* 1990;**153**(6):367-68  
16  
17  
18 24. Cooney MT, Dudina A, De Bacquer D, et al. How much does HDL cholesterol add to risk  
19 estimation? A report from the SCORE investigators. *European Journal of Cardiovascular*  
20 *Prevention & Rehabilitation* 2009;**16**(3):304-14 doi:  
21 10.1097/HJR.0b013e3283213140[published Online First: Epub Date].  
22  
23  
24  
25 25. Neil HAW, Perera R, Armitage JM, et al. Estimated 10-year cardiovascular risk in a British  
26 population: results of a national screening project. *Int J Clin Pract* 2008;**62**(9):1322-31  
27  
28  
29 26. Woodward M, Brindle P, Tunstall-Pedoe H. Adding social deprivation and family history to  
30 cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended  
31 Cohort (SHHEC). *Heart* 2007;**93**(2):172-76  
32  
33  
34  
35 27. Mosca L, Benjamin EJ, Berra K, et al. Effectiveness-Based Guidelines for the Prevention of  
36 Cardiovascular Disease in Women—2011 Update A Guideline From the American Heart  
37 Association. *J Am Coll Cardiol* 2011;**57**(12):1404-23  
38  
39  
40  
41 28. Genest J, McPherson R, Frohlich J, et al. 2009 Canadian Cardiovascular Society/Canadian  
42 guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular  
43 disease in the adult – 2009 recommendations. *Can J Cardiol* 2009;**25**(10):567-79  
44  
45  
46  
47 29. Goh LGH, Dhaliwal SS, Lee AH, et al. Utility of established cardiovascular disease risk score  
48 models for the 10-year prediction of disease outcomes in women. *Expert Rev Cardiovasc*  
49 *Ther* 2013;**11**(4):425-35  
50  
51  
52  
53  
54 30. Wittchen H-U, Balkau B, Massien C, et al. International Day for the Evaluation of Abdominal  
55 obesity: rationale and design of a primary care study on the prevalence of abdominal obesity  
56  
57  
58  
59  
60

- 1  
2  
3 and associated factors in 63 countries. *Eur Heart J Suppl* 2006;**8**(suppl B):B26-B33 doi:  
4 10.1093/eurheartj/sul005[published Online First: Epub Date].  
5  
6  
7 31. Balkau B, Deanfield JE, Despres JP, et al. International day for the evaluation of abdominal  
8 obesity (IDEA) - A study of waist circumference, cardiovascular disease, and diabetes  
9 mellitus in 168 000 primary care patients in 63 countries. *Circulation* 2007;**116**(17):1942-51  
10 doi: 10.1161/circulationaha.106.676379[published Online First: Epub Date].  
11  
12  
13 32. Winter Y, Rohrmann S, Linseisen J, et al. Contribution of Obesity and Abdominal Fat Mass to  
14 Risk of Stroke and Transient Ischemic Attacks. *Stroke* 2008;**39**(12):3145-51 doi:  
15 10.1161/strokeaha.108.523001[published Online First: Epub Date].  
16  
17  
18 33. Taylor AE, Ebrahim S, Ben-Shlomo Y, et al. Comparison of the associations of body mass index  
19 and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-  
20 cause mortality: a study using data from 4 UK cohorts. *The American Journal of Clinical*  
21 *Nutrition* 2010;**91**(3):547-56 doi: 10.3945/ajcn.2009.28757[published Online First: Epub  
22 Date].  
23  
24  
25 34. van Dis I, Kromhout D, Geleijnse JM, et al. Body mass index and waist circumference predict  
26 both 10-year nonfatal and fatal cardiovascular disease risk: study conducted in 20 000 Dutch  
27 men and women aged 20–65 years. *European Journal of Cardiovascular Prevention &*  
28 *Rehabilitation* 2009;**16**(6):729-34 doi: 10.1097/HJR.0b013e328331dfc0[published Online  
29 First: Epub Date].  
30  
31  
32 35. Berg AH, Scherer PE. Adipose Tissue, Inflammation, and Cardiovascular Disease. *Circulation*  
33 *Research* 2005;**96**(9):939-49 doi: 10.1161/01.res.0000163635.62927.34[published Online  
34 First: Epub Date].  
35  
36  
37 36. Snijder MB, van Dam RM, Visser M, et al. What aspects of body fat are particularly hazardous  
38 and how do we measure them? *International Journal of Epidemiology* 2006;**35**(1):83-92 doi:  
39 10.1093/ije/dyi253[published Online First: Epub Date].  
40  
41  
42 37. de Koning L, Merchant AT, Pogue J, et al. Waist circumference and waist-to-hip ratio as  
43 predictors of cardiovascular events: meta-regression analysis of prospective studies. *Eur Heart*  
44 *J* 2007;**28**(7):850-56  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 38. Dalton M, Cameron AJ, Zimmet PZ, et al. Waist circumference, waist-hip ratio and body mass  
4 index and their correlation with cardiovascular disease risk factors in Australian adults.  
5 Journal of Internal Medicine 2003;**254**(6):555-63 doi: 10.1111/j.1365-  
6 2796.2003.01229.x[published Online First: Epub Date]].  
7  
8  
9  
10  
11 39. Antillon D, Towfighi A. No time to 'weight': the link between obesity and stroke in women.  
12 Women's Health 2011;**7**(4):453-63 doi: 10.2217/whe.11.36[published Online First: Epub  
13 Date]].  
14  
15  
16  
17 40. Pischon T, Boeing H, Hoffmann K, et al. General and Abdominal Adiposity and Risk of Death in  
18 Europe. N Engl J Med 2008;**359**(20):2105-20 doi: doi:10.1056/NEJMoa0801891[published  
19 Online First: Epub Date]].  
20  
21  
22  
23 41. Li C, Engstrom G, Hedblad B, et al. Sex differences in the relationships between BMI, WHR and  
24 incidence of cardiovascular disease: a population-based cohort study. International Journal of  
25 Obesity 2006;**30**(12):1775-81 doi: 10.1038/sj.ijo.0803339[published Online First: Epub  
26 Date]].  
27  
28  
29  
30  
31 42. Freiberg MS, Pencina MJ, D'Agostino RB, et al. BMI vs. Waist Circumference for Identifying  
32 Vascular Risk. Obesity 2008;**16**(2):463-69  
33  
34  
35  
36 43. DiPietro L, Katz LD, Nadel ER. Excess abdominal adiposity remains correlated with altered lipid  
37 concentrations in healthy older women. International journal of obesity and related metabolic  
38 disorders : journal of the International Association for the Study of Obesity 1999;**23**(4):432-  
39 36  
40  
41  
42  
43 44. Klein S, Allison DB, Heymsfield SB, et al. Waist Circumference and Cardiometabolic Risk: A  
44 Consensus Statement from Shaping America's Health: Association for Weight Management  
45 and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition;  
46 and the American Diabetes Association. Obesity 2007;**15**(5):1061-67 doi:  
47 10.1038/oby.2007.632[published Online First: Epub Date]].  
48  
49  
50  
51  
52  
53  
54 45. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in  
55 women. JAMA 1998;**280**(21):1843-48 doi: 10.1001/jama.280.21.1843[published Online  
56 First: Epub Date]].  
57  
58  
59  
60

- 1  
2  
3 46. Fogelholm M. Physical activity, fitness and fatness: relations to mortality, morbidity and disease  
4 risk factors. A systematic review. *Obesity Reviews* 2010;**11**(3):202-21 doi: 10.1111/j.1467-  
5 789X.2009.00653.x[published Online First: Epub Date]].  
6  
7  
8  
9 47. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Accuracy of body mass index in  
10 diagnosing obesity in the adult general population. *Int J Obes* 2008;**32**(6):959-66 doi:  
11 <http://www.nature.com/ijo/journal/v32/n6/suppinfo/ijo200811s1.html>[published  
12 Online First:  
13 Epub Date]].  
14  
15  
16  
17 48. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a  
18 WHO consultation. World Health Organ Tech Rep Ser: WHO, 2000.  
19  
20  
21 49. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis  
22 among different ethnic groups. *International Journal of Obesity* 1998;**22**(12):1164-71  
23  
24  
25 50. Welborn TA, Dhaliwal SS. Being correct about obesity. *Med J Aust* 2011;**194** (8 ):429-30  
26  
27  
28 51. The Emerging Risk Factors Collaboration. Separate and combined associations of body-mass  
29 index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58  
30 prospective studies. *The Lancet* 2011;**377**(9771):1085-95 doi:  
31 [http://dx.doi.org/10.1016/S0140-6736\(11\)60105-0](http://dx.doi.org/10.1016/S0140-6736(11)60105-0)[published Online First: Epub Date]].  
32  
33  
34  
35 52. Dijk SB, Takken T, Prinsen EC, et al. Different anthropometric adiposity measures and their  
36 association with cardiovascular disease risk factors: a meta-analysis. *Neth Heart J*  
37 2012;**20**(5):208-18 doi: 10.1007/s12471-011-0237-7[published Online First: Epub Date]].  
38  
39  
40  
41 53. Dobbelsteyn CJ, Joffres MR, MacLean DR, et al. A comparative evaluation of waist  
42 circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk  
43 factors. The Canadian Heart Health Surveys. *International Journal of Obesity* 2001;**25**:652-61  
44  
45  
46  
47 54. Pouliot M-C, Després J-P, Lemieux S, et al. Waist circumference and abdominal sagittal diameter:  
48 Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and  
49 related cardiovascular risk in men and women. *The American Journal of Cardiology*  
50 1994;**73**(7):460-68 doi: [http://dx.doi.org/10.1016/0002-9149\(94\)90676-9](http://dx.doi.org/10.1016/0002-9149(94)90676-9)[published  
51 Online  
52 First: Epub Date]].  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 55. Turcato E, Bosello O, Di Francesco V, et al. Waist circumference and abdominal sagittal diameter  
4 as surrogates of body fat distribution in the elderly: their relation with cardiovascular risk  
5 factors. *Int J Obes Relat Metab Disord* 2000;**24**(8):1005-10  
6  
7  
8  
9 56. Zhu S, Wang Z, Heshka S, et al. Waist circumference and obesity-associated risk factors among  
10 whites in the third National Health and Nutrition Examination Survey: clinical action  
11 thresholds. *The American Journal of Clinical Nutrition* 2002;**76**(4):743  
12  
13  
14  
15 57. Reeder BA, Senthilselvan A, Després JP, et al. The association of cardiovascular disease risk  
16 factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group.  
17 *CMAJ : Canadian Medical Association journal* 1997;**157 Suppl 1**:S39-S45  
18  
19  
20  
21 58. Lapidus L, Bengtsson C, Larsson B, et al. Distribution of adipose tissue and risk of cardiovascular  
22 disease and death: a 12 year follow up of participants in the population study of women in  
23 Gothenburg, Sweden. *BMJ* 1984;**289**(6454):1257-61 doi:  
24 10.1136/bmj.289.6454.1257[published Online First: Epub Date].  
25  
26  
27  
28  
29 59. Price GM, Uauy R, Breeze E, et al. Weight, shape, and mortality risk in older persons: elevated  
30 waist-hip ratio, not high body mass index, is associated with a greater risk of death. *The*  
31 *American Journal of Clinical Nutrition* 2006;**84**(2):449-60  
32  
33  
34  
35 60. Lu M, Ye W, Adami HO, et al. Prospective study of body size and risk for stroke amongst women  
36 below age 60. *Journal of Internal Medicine* 2006;**260**(5):442-50 doi: 10.1111/j.1365-  
37 2796.2006.01706.x[published Online First: Epub Date].  
38  
39  
40  
41 61. Dhaliwal SS, Welborn TA. Central obesity and cigarette smoking are key determinants of  
42 cardiovascular deaths in Australia: A public health perspective. *Preventive Medicine*  
43 2009;**49**(2-3):153-57  
44  
45  
46  
47 62. Welborn TA, Dhaliwal SS. Preferred clinical measures of central obesity for predicting mortality.  
48 *Eur J Clin Nutr* 2007;**61**(12):1373-79  
49  
50  
51 63. Welborn TA, Dhaliwal SS, Bennett SA. Waist-hip ratio is the dominant risk factor predicting  
52 cardiovascular death in Australia. *Med J Aust* 2003;**179**(11-12):580-85  
53  
54  
55 64. Dhaliwal SS, Welborn TA. Measurement error and ethnic comparisons of measures of abdominal  
56 obesity. *Prev Med* 2009;**49**(2-3):148-52  
57  
58  
59  
60

- 1  
2  
3 65. Goodman-Gruen D, Barrett-Connor E. Sex Differences in Measures of Body Fat and Body Fat  
4 Distribution in the Elderly. *Am J Epidemiol* 1996;**143**(9):898-906  
5  
6  
7 66. Ashwell M, Lejeune S. Ratio of waist circumference to height may be better indicator of need for  
8 weight management. *BMJ* 1996;**312**(7027):377 doi: 10.1136/bmj.312.7027.377[published  
9 Online First: Epub Date]].  
10  
11  
12  
13 67. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global  
14 indicator for health risks of obesity and how its use could simplify the international public  
15 health message on obesity. *International Journal of Food Sciences and Nutrition*  
16 2005;**56**(5):303-07 doi: doi:10.1080/09637480500195066[published Online First: Epub  
17 Date]].  
18  
19  
20  
21  
22  
23 68. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening  
24 tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global  
25 boundary value. *Nutrition Research Reviews* 2010;**23**(02):247-69 doi:  
26 doi:10.1017/S0954422410000144[published Online First: Epub Date]].  
27  
28  
29  
30  
31 69. Schneider HJ, Glaesmer H, Klotsche J, et al. Accuracy of anthropometric indicators of obesity to  
32 predict cardiovascular risk. *J Clin Endocrinol Metab* 2007;**92**:589-94  
33  
34  
35  
36 70. Okosun IS, Liao Y, Rotimi CN, et al. Abdominal Adiposity and Clustering of Multiple Metabolic  
37 Syndrome in White, Black and Hispanic Americans. *Annals of Epidemiology*  
38 2000;**10**(5):263-70 doi: [http://dx.doi.org/10.1016/S1047-2797\(00\)00045-4](http://dx.doi.org/10.1016/S1047-2797(00)00045-4)[published  
39 Online  
40 First: Epub Date]].  
41  
42  
43  
44 71. Ho SC, Chen YM, Woo JLF, et al. Association between simple anthropometric indices and  
45 cardiovascular risk factors. *Int J Obes Relat Metab Disord* 2001;**25**(11):1689-97  
46  
47  
48 72. Jeong S-K, Seo M-W, Kim Y-H, et al. Does Waist Indicate Dyslipidemia better than BMI in  
49 Korean Adult Population? *J Korean Med Sci* 2005;**20**(1):7-12  
50  
51  
52 73. Yusuf S, Hawken S, Ôunpuu S, et al. Obesity and the risk of myocardial infarction in 27□000  
53 participants from 52 countries: a case-control study. *Lancet* 2005;**366**(9497):1640-49  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 74. Moore SC. Waist versus weight—which matters more for mortality? The American Journal of  
4  
5 Clinical Nutrition 2009;**89**(4):1003-04 doi: 10.3945/ajcn.2009.27598[published Online First:  
6  
7 Epub Date]].  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

For peer review only



1  
2  
3  
4  
5  
6  
7 **Anthropometric measurements of general and central obesity and**  
8  
9 **the prediction of cardiovascular disease risk in women: a cross-**  
10  
11 **sectional study**  
12  
13

14  
15  
16 Correspondence to: Professor Satvinder S Dhaliwal

17 School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
18 U1987, Perth, WA 6845, Australia  
19

20  
21 Tel: +618 9266 2949

22  
23 Fax: +618 9266 2958

24  
25 E-mail: [s.dhaliwal@curtin.edu.au](mailto:s.dhaliwal@curtin.edu.au)  
26

27  
28 Louise GH Goh,<sup>1</sup> Satvinder S Dhaliwal,<sup>1</sup> Timothy A Welborn,<sup>2</sup> Andy H Lee,<sup>1</sup> Phillip R Della<sup>3</sup>  
29  
30

31  
32 <sup>1</sup>School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
33 U1987, Perth, WA 6845, Australia  
34

35 <sup>2</sup>Sir Charles Gairdner Hospital, Hospital Avenue, Nedlands, Perth, WA 6009, Australia  
36

37 <sup>3</sup>School of Nursing and Midwifery, Curtin Health Innovation Research Institute, Curtin University,  
38 GPO Box U1987, Perth, WA 6845, Australia  
39

40  
41  
42 Anthropometric obesity measures and CVD risk  
43  
44

45  
46 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60



## 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.

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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.

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] [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<sup>1.5</sup>, 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](#)

1  
2  
3  
4  
5  
6  
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.  
10  
11

12  
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.  
23  
24  
25  
26  
27  
28  
29  
30  
31

## 32 33 METHODS

### 34 35 Study cohort and measurements

36 We selected 4487 women aged 20-69 years with no history of heart disease, diabetes or stroke from  
37 the population representative sample of 4727 women from the National Heart Foundation (NHF)  
38 Risk Factor Prevalence Study.[21] Participants taking medications to lower their CVD risk factors  
39 were also excluded. The participants of the NHF study consisted of residents on the federal electoral  
40 rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart,  
41 Darwin and Canberra in a systematic probability sampling by sex and 5-year age groups. Information  
42 on demographic characteristics was collected using a self-administered questionnaire and  
43 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,  
44 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of  
45 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip  
46 circumference were collected according to standardised methodologies[22 23] using two observers.  
47  
48  
49  
50  
51  
52  
53  
54  
55

1  
2  
3  
4  
5  
6  
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  
11  
12  
13  
14 two measurements was taken at each site to the nearest centimetre.

### 15 16 17 **Variables in ~~r~~Risk score models**

18  
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  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60  
The SCORE risk chart was developed by pooling 12 cohort studies to predict the 10-  
year CVD death risk in Europe. The cohorts consisted of participants aged 19-80 years with no  
previous history of heart attack.[19] It was derived from a much larger dataset than the Framingham,  
~~and general CVD and simplified general CVD risk score models.~~ Fewer variables were used in the  
calculation of the 10-year predicted CVD death risk with the SCORE risk chart for high-risk regions  
(Denmark, Finland and Norway).[19 24] these included: age, sex, smoking status, mean total  
cholesterol level, mean HDL cholesterol level and mean SBP. The general CVD risk score model was  
also developed using data from the American Framingham Heart Study but using a larger cohort than  
the Framingham model.[20] Individuals without CVD were used in the development of the general  
CVD risk score model.[20] The simplified general CVD risk score model was developed similarly as  
the general CVD risk score model. It is, however, a simpler CVD risk prediction model which is  
calculated using non-laboratory predictors. Similar ~~r~~Risk variables (age, SBP, current  
antihypertensive treatment, smoking status and diabetes status) were used in both ~~of the the general~~  
~~CVD and simplified general CVD risk score~~ models.[20]- ~~The only difference is, BMI is included in~~  
the simplified general CVD risk score model instead of total and HDL cholesterol which is used in the  
general CVD risk score model. ~~In the simplified general CVD risk score model, however, the total~~

1  
2  
3  
4  
5  
6  
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.~~  
9

## 10 11 **Statistical analysis**

12  
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 21<sup>9</sup>.  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47

## 48 **RESULTS**

49 The sample of 4487 women aged 20-69 years from the NHF Risk Factor Prevalence Study is a  
50 representative sample of the Australian female population, free of heart disease, diabetes and stroke.  
51  
52  
53  
54

1  
2  
3  
4  
5  
6  
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.  
9

10  
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%.  
17  
18  
19  
20  
21  
22  
23

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).  
30  
31  
32  
33  
34  
35

36 The associations between anthropometric measurements of obesity and the 10-year predicted risks  
37 calculated using the four models ~~are~~were presented in Table 3. All Spearman's rank correlations were  
38 statistically significant ( $p < 0.0005$ ). All anthropometric measures of central obesity (WC, WHR and  
39 WSR) generally had consistently higher correlations with the predicted risks calculated using the four  
40 CVD risk score models, as compared to measures of general obesity.  
41  
42  
43  
44  
45  
46

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,  
51  
52  
53  
54



1  
2  
3  
4  
5  
6 WHR and WSR) generally recorded higher odds-ratios than general measures of obesity and they  
7 increased the likelihood of individuals being above the respective treatment thresholds.  
8  
9

10  
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.  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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%)
<b>Ethnicity</b>	
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 <sup>2</sup> )	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

Formatted: Indent: First line: 0", Space After: 0 pt, Line spacing: single

Formatted: Indent: Left: 0.32", Space After: 0 pt, Line spacing: single

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.

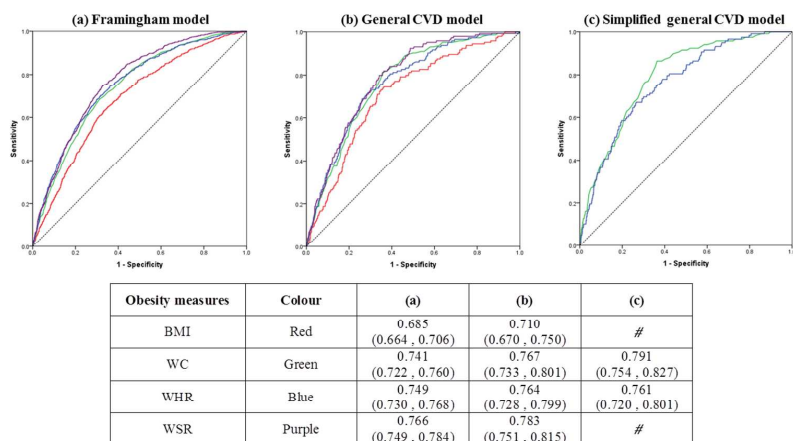
**Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity**

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~~

1  
2  
3  
4  
5  
6  
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]  
9

10  
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.  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35

36 Among central obesity measures, we found their performance to be comparable in our study. It  
37 remains unclear which measurement should be incorporated into CVD risk score models. ~~To date,~~  
38 ~~BMI is included in the simplified general CVD risk score model as an alternative to total and HDL~~  
39 ~~cholesterol level considering its ease of measurement and calculation,[20] and in the QRISK score~~  
40 ~~model.[29]~~ A collaborative analysis of 58 prospective studies, however, reported that both measures  
41 of general and central obesity did not improve CVD risk assessment when information is available on  
42 SBP, diabetes and lipids.[51] Overweight and obesity is nevertheless important in CVD prevention,  
43 with one out of three of fatal and one out of seven of non-fatal CVD cases attributable to it.[34]  
44  
45  
46  
47  
48  
49  
50

51  
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  
54  
55



1  
2  
3  
4  
5  
6  
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]

15  
16  
17  
18  
19  
20  
21  
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  $\geq 0.88$  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

1  
2  
3  
4  
5  
6  
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]  
9

10  
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.  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31

32  
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.  
38  
39  
40  
41  
42

#### 43 CONCLUSIONS

44 ~~The significant and independent effect of obesity measures on CVD risk substantiates its inclusion~~  
45 ~~into risk score models.~~ Central obesity is more strongly associated with CVD risk than general  
46 obesity. The deposition of adipose tissue is associated with systemic inflammation which has a direct  
47 effect on CVD risk. Therefore, increments in central obesity have a more detrimental effect on CVD  
48 risk compared to increments in general obesity.  
49  
50  
51  
52  
53  
54

1  
2  
3  
4  
5  
6  
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]

17  
18  
19  
20  
21  
22  
23  
24  
25  
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]

30  
31  
32  
33  
34  
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.

41  
42  
43  
44  
45  
46  
47 **Acknowledgements** Curtin University provided educational support to LGHG through the Curtin  
48 International Postgraduate Research Scholarship.

49  
50  
51  
52  
53 **Contributors** LGHG was involved in drafting the manuscript, interpretation of data and revising the  
54 manuscript critically for important intellectual content. SSD conceived the study, performed the

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

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.

**Funding** None.

For peer review only

1  
2  
3  
4  
5  
6  
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.  
11  
12

13  
14  
15 **Data sharing statement** No additional data are available.  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

## REFERENCES

1. World Health Organization. Obesity and overweight. Secondary Obesity and overweight 2012. <http://www.who.int/mediacentre/factsheets/fs311/en/>.
2. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. *Circulation* 2002;**106**(25):3143-421
3. Kannel WB. Metabolic risk factors for coronary heart disease in women: Perspective from the Framingham Study. *Am Heart J* 1987;**114**(2):413-19 doi: [http://dx.doi.org/10.1016/0002-8703\(87\)90511-4](http://dx.doi.org/10.1016/0002-8703(87)90511-4)[published Online First: Epub Date]].
4. Hubert HB, Feinleib M, McNamara PM, et al. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation* 1983;**67**:968-77
5. Manson JE, Colditz GA, Stampfer MJ, et al. A Prospective Study of Obesity and Risk of Coronary Heart Disease in Women. *N Engl J Med* 1990;**322**(13):882-89 doi: [doi:10.1056/NEJM199003293221303](https://doi.org/10.1056/NEJM199003293221303)[published Online First: Epub Date]].
6. Manson JE, Willett WC, Stampfer MJ, et al. Body Weight and Mortality among Women. *N Engl J Med* 1995;**333**(11):677-85 doi: [doi:10.1056/NEJM199509143331101](https://doi.org/10.1056/NEJM199509143331101)[published Online First: Epub Date]].
7. Dorn JM, Schisterman EF, Winkelstein W, et al. Body Mass Index and Mortality in a General Population Sample Women of Men and Women: The Buffalo Health Study. *American Journal of Epidemiology* 1997;**146**(11):919-31
8. Cornier M-A, Després J-P, Davis N, et al. Assessing Adiposity: A Scientific Statement From the American Heart Association. *Circulation* 2011;**124**(18):1996-2019 doi: [10.1161/CIR.0b013e318233bc6a](https://doi.org/10.1161/CIR.0b013e318233bc6a)[published Online First: Epub Date]].
9. Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation* 2013

- 1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60
10. Park YS, Kim J-S. Obesity Phenotype and Coronary Heart Disease Risk as Estimated by the Framingham Risk Score. *Journal of Korean Medical Science* 2012;**27**(3):243-49
  11. Satoh H, Kishi R, Tsutsui H. Body Mass Index can Similarly Predict the Presence of Multiple Cardiovascular Risk Factors in Middle-aged Japanese Subjects as Waist Circumference. *Internal Medicine* 2010;**49**(11):977-82
  12. Ryan MC, Fenster Farin HM, Abbasi F, et al. Comparison of Waist Circumference Versus Body Mass Index in Diagnosing Metabolic Syndrome and Identifying Apparently Healthy Subjects at Increased Risk of Cardiovascular Disease. *The American Journal of Cardiology* 2008;**102**(1):40-46 doi: <http://dx.doi.org/10.1016/j.amjcard.2008.02.096>[published Online First: Epub Date].
  13. Ying X, Song Z, Zhao C, et al. Body mass index, waist circumference, and cardiometabolic risk factors in young and middle-aged Chinese women. *J Zhejiang Univ Sci B* 2010;**11**(9):639-46 doi: 10.1631/jzus.B1000105[published Online First: Epub Date].
  14. Zhu S, Heymsfield SB, Toyoshima H, et al. Race-ethnicity-specific waist circumference cutoffs for identifying cardiovascular disease risk factors. *The American Journal of Clinical Nutrition* 2005;**81**(2):409-15
  15. Huang K-C, Lee M-S, Lee S-D, et al. Obesity in the Elderly and Its Relationship with Cardiovascular Risk Factors in Taiwan. *Obes Res* 2005;**13**(1):170-78 doi: 10.1038/oby.2005.22[published Online First: Epub Date].
  16. Bergman RN, Stefanovski D, Buchanan TA, et al. A Better Index of Body Adiposity. *Obesity (Silver Spring)* 2011;**19**(5):1083-89
  17. Dhaliwal SS, Welborn TA. Central obesity and multivariable cardiovascular risk as assessed by the Framingham prediction scores. *Am J Cardiol* 2009;**103**:1403-07
  18. Anderson KM, Odell PM, Wilson PW, et al. Cardiovascular disease risk profiles. *Am Heart J* 1991;**121**(1 Part 2):293-98
  19. Conroy RM, Pyörälä K, Fitzgerald AP, et al. Estimation of ten-year risk of fatal cardiovascular disease in Europe: The SCORE project. *European Heart Journal* 2003;**24**(11):987-1003 doi: 10.1016/s0195-668x(03)00114-3[published Online First: Epub Date].

20. D'Agostino RB, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in primary care - The Framingham Heart Study. *Circulation* 2008;**117**(6):743-53
21. Australian Risk Factor Prevalence Study Management Committee. Survey No. 3 1989. Canberra: National Heart Foundation of Australia and Australia Institute of Health, 1990.
22. Boyle CA, Dobson AJ, Egger G, et al. Waist-to-hip ratios in Australia: A different picture of obesity. *Aust J Nutr Diet* 1993;**50**:57-64
23. Alexander H, Dugdale A. Which waist-hip ratio? *Med J Aust* 1990;**153**(6):367-68
24. Cooney MT, Dudina A, De Bacquer D, et al. How much does HDL cholesterol add to risk estimation? A report from the SCORE investigators. *European Journal of Cardiovascular Prevention & Rehabilitation* 2009;**16**(3):304-14 doi: 10.1097/HJR.0b013e3283213140[published Online First: Epub Date].
25. Neil HAW, Perera R, Armitage JM, et al. Estimated 10-year cardiovascular risk in a British population: results of a national screening project. *Int J Clin Pract* 2008;**62**(9):1322-31
26. Woodward M, Brindle P, Tunstall-Pedoe H. Adding social deprivation and family history to cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended Cohort (SHHEC). *Heart* 2007;**93**(2):172-76
27. Mosca L, Benjamin EJ, Berra K, et al. Effectiveness-Based Guidelines for the Prevention of Cardiovascular Disease in Women—2011 Update A Guideline From the American Heart Association. *J Am Coll Cardiol* 2011;**57**(12):1404-23
28. Genest J, McPherson R, Frohlich J, et al. 2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult – 2009 recommendations. *Can J Cardiol* 2009;**25**(10):567-79
29. Goh LGH, Dhaliwal SS, Lee AH, et al. Utility of established cardiovascular disease risk score models for the 10-year prediction of disease outcomes in women. *Expert Rev Cardiovasc Ther* 2013;**11**(4):425-35
30. Wittchen H-U, Balkau B, Massien C, et al. International Day for the Evaluation of Abdominal obesity: rationale and design of a primary care study on the prevalence of abdominal obesity



- and associated factors in 63 countries. *Eur Heart J Suppl* 2006;**8**(suppl B):B26-B33 doi: 10.1093/eurheartj/sul005[published Online First: Epub Date]].
31. Balkau B, Deanfield JE, Despres JP, et al. International day for the evaluation of abdominal obesity (IDEA) - A study of waist circumference, cardiovascular disease, and diabetes mellitus in 168 000 primary care patients in 63 countries. *Circulation* 2007;**116**(17):1942-51 doi: 10.1161/circulationaha.106.676379[published Online First: Epub Date]].
32. Winter Y, Rohrmann S, Linseisen J, et al. Contribution of Obesity and Abdominal Fat Mass to Risk of Stroke and Transient Ischemic Attacks. *Stroke* 2008;**39**(12):3145-51 doi: 10.1161/strokeaha.108.523001[published Online First: Epub Date]].
33. Taylor AE, Ebrahim S, Ben-Shlomo Y, et al. Comparison of the associations of body mass index and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-cause mortality: a study using data from 4 UK cohorts. *The American Journal of Clinical Nutrition* 2010;**91**(3):547-56 doi: 10.3945/ajcn.2009.28757[published Online First: Epub Date]].
34. van Dis I, Kromhout D, Geleijnse JM, et al. Body mass index and waist circumference predict both 10-year nonfatal and fatal cardiovascular disease risk: study conducted in 20 000 Dutch men and women aged 20–65 years. *European Journal of Cardiovascular Prevention & Rehabilitation* 2009;**16**(6):729-34 doi: 10.1097/HJR.0b013e328331dfc0[published Online First: Epub Date]].
35. Berg AH, Scherer PE. Adipose Tissue, Inflammation, and Cardiovascular Disease. *Circulation Research* 2005;**96**(9):939-49 doi: 10.1161/01.res.0000163635.62927.34[published Online First: Epub Date]].
36. Snijder MB, van Dam RM, Visser M, et al. What aspects of body fat are particularly hazardous and how do we measure them? *International Journal of Epidemiology* 2006;**35**(1):83-92 doi: 10.1093/ije/dyi253[published Online First: Epub Date]].
37. de Koning L, Merchant AT, Pogue J, et al. Waist circumference and waist-to-hip ratio as predictors of cardiovascular events: meta-regression analysis of prospective studies. *Eur Heart J* 2007;**28**(7):850-56

- 1  
2  
3  
4  
5  
6  
7 38. Dalton M, Cameron AJ, Zimmet PZ, et al. Waist circumference, waist-hip ratio and body mass  
8 index and their correlation with cardiovascular disease risk factors in Australian adults.  
9 Journal of Internal Medicine 2003;**254**(6):555-63 doi: 10.1111/j.1365-  
10 2796.2003.01229.x[published Online First: Epub Date]].  
11  
12  
13 39. Antillon D, Towfighi A. No time to 'weight': the link between obesity and stroke in women.  
14 Women's Health 2011;**7**(4):453-63 doi: 10.2217/whe.11.36[published Online First: Epub  
15 Date]].  
16  
17  
18 40. Pischon T, Boeing H, Hoffmann K, et al. General and Abdominal Adiposity and Risk of Death in  
19 Europe. N Engl J Med 2008;**359**(20):2105-20 doi: doi:10.1056/NEJMoa0801891[published  
20 Online First: Epub Date]].  
21  
22  
23 41. Li C, Engstrom G, Hedblad B, et al. Sex differences in the relationships between BMI, WHR and  
24 incidence of cardiovascular disease: a population-based cohort study. International Journal of  
25 Obesity 2006;**30**(12):1775-81 doi: 10.1038/sj.ijo.0803339[published Online First: Epub  
26 Date]].  
27  
28  
29 42. Freiberg MS, Pencina MJ, D'Agostino RB, et al. BMI vs. Waist Circumference for Identifying  
30 Vascular Risk. Obesity 2008;**16**(2):463-69  
31  
32  
33 43. DiPietro L, Katz LD, Nadel ER. Excess abdominal adiposity remains correlated with altered lipid  
34 concentrations in healthy older women. International journal of obesity and related metabolic  
35 disorders : journal of the International Association for the Study of Obesity 1999;**23**(4):432-  
36 36  
37  
38  
39 44. Klein S, Allison DB, Heymsfield SB, et al. Waist Circumference and Cardiometabolic Risk: A  
40 Consensus Statement from Shaping America's Health: Association for Weight Management  
41 and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition;  
42 and the American Diabetes Association. Obesity 2007;**15**(5):1061-67 doi:  
43 10.1038/oby.2007.632[published Online First: Epub Date]].  
44  
45  
46  
47 45. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in  
48 women. JAMA 1998;**280**(21):1843-48 doi: 10.1001/jama.280.21.1843[published Online  
49 First: Epub Date]].  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3  
4  
5  
6  
7 46. Fogelholm M. Physical activity, fitness and fatness: relations to mortality, morbidity and disease  
8 risk factors. A systematic review. *Obesity Reviews* 2010;**11**(3):202-21 doi: 10.1111/j.1467-  
9 789X.2009.00653.x[published Online First: Epub Date]].
- 10  
11 47. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Accuracy of body mass index in  
12 diagnosing obesity in the adult general population. *Int J Obes* 2008;**32**(6):959-66 doi:  
13 <http://www.nature.com/ijo/journal/v32/n6/suppinfo/ijo200811s1.html>[published  
14 Online First:  
15 Epub Date]].
- 16  
17 48. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a  
18 WHO consultation. World Health Organ Tech Rep Ser: WHO, 2000.
- 19  
20 49. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis  
21 among different ethnic groups. *International Journal of Obesity* 1998;**22**(12):1164-71
- 22  
23 50. Welborn TA, Dhaliwal SS. Being correct about obesity. *Med J Aust* 2011;**194** (8 ):429-30
- 24  
25 51. The Emerging Risk Factors Collaboration. Separate and combined associations of body-mass  
26 index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58  
27 prospective studies. *The Lancet* 2011;**377**(9771):1085-95 doi:  
28 [http://dx.doi.org/10.1016/S0140-6736\(11\)60105-0](http://dx.doi.org/10.1016/S0140-6736(11)60105-0)[published Online First: Epub Date]].
- 29  
30 52. Dijk SB, Takken T, Prinsen EC, et al. Different anthropometric adiposity measures and their  
31 association with cardiovascular disease risk factors: a meta-analysis. *Neth Heart J*  
32 2012;**20**(5):208-18 doi: 10.1007/s12471-011-0237-7[published Online First: Epub Date]].
- 33  
34 53. Dobbelsteyn CJ, Joffres MR, MacLean DR, et al. A comparative evaluation of waist  
35 circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk  
36 factors. The Canadian Heart Health Surveys. *International Journal of Obesity* 2001;**25**:652-61
- 37  
38 54. Pouliot M-C, Després J-P, Lemieux S, et al. Waist circumference and abdominal sagittal diameter:  
39 Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and  
40 related cardiovascular risk in men and women. *The American Journal of Cardiology*  
41 1994;**73**(7):460-68 doi: [http://dx.doi.org/10.1016/0002-9149\(94\)90676-9](http://dx.doi.org/10.1016/0002-9149(94)90676-9)[published  
42 Online  
43 First: Epub Date]].
- 44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3  
4  
5  
6  
7 55. Turcato E, Bosello O, Di Francesco V, et al. Waist circumference and abdominal sagittal diameter  
8 as surrogates of body fat distribution in the elderly: their relation with cardiovascular risk  
9 factors. *Int J Obes Relat Metab Disord* 2000;**24**(8):1005-10  
10  
11 56. Zhu S, Wang Z, Heshka S, et al. Waist circumference and obesity-associated risk factors among  
12 whites in the third National Health and Nutrition Examination Survey: clinical action  
13 thresholds. *The American Journal of Clinical Nutrition* 2002;**76**(4):743  
14  
15 57. Reeder BA, Senthilselvan A, Després JP, et al. The association of cardiovascular disease risk  
16 factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group.  
17 *CMAJ : Canadian Medical Association journal* 1997;**157** Suppl 1:S39-S45  
18  
19 58. Lapidus L, Bengtsson C, Larsson B, et al. Distribution of adipose tissue and risk of cardiovascular  
20 disease and death: a 12 year follow up of participants in the population study of women in  
21 Gothenburg, Sweden. *BMJ* 1984;**289**(6454):1257-61 doi:  
22 10.1136/bmj.289.6454.1257[published Online First: Epub Date].  
23  
24 59. Price GM, Uauy R, Breeze E, et al. Weight, shape, and mortality risk in older persons: elevated  
25 waist-hip ratio, not high body mass index, is associated with a greater risk of death. *The*  
26 *American Journal of Clinical Nutrition* 2006;**84**(2):449-60  
27  
28 60. Lu M, Ye W, Adami HO, et al. Prospective study of body size and risk for stroke amongst women  
29 below age 60. *Journal of Internal Medicine* 2006;**260**(5):442-50 doi: 10.1111/j.1365-  
30 2796.2006.01706.x[published Online First: Epub Date].  
31  
32 61. Dhaliwal SS, Welborn TA. Central obesity and cigarette smoking are key determinants of  
33 cardiovascular deaths in Australia: A public health perspective. *Preventive Medicine*  
34 2009;**49**(2-3):153-57  
35  
36 62. Welborn TA, Dhaliwal SS. Preferred clinical measures of central obesity for predicting mortality.  
37 *Eur J Clin Nutr* 2007;**61**(12):1373-79  
38  
39 63. Welborn TA, Dhaliwal SS, Bennett SA. Waist-hip ratio is the dominant risk factor predicting  
40 cardiovascular death in Australia. *Med J Aust* 2003;**179**(11-12):580-85  
41  
42 64. Dhaliwal SS, Welborn TA. Measurement error and ethnic comparisons of measures of abdominal  
43 obesity. *Prev Med* 2009;**49**(2-3):148-52  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55

- 1  
2  
3  
4  
5  
6  
7 65. Goodman-Gruen D, Barrett-Connor E. Sex Differences in Measures of Body Fat and Body Fat  
8 Distribution in the Elderly. *Am J Epidemiol* 1996;**143**(9):898-906  
9
- 10 66. Ashwell M, Lejeune S. Ratio of waist circumference to height may be better indicator of need for  
11 weight management. *BMJ* 1996;**312**(7027):377 doi: 10.1136/bmj.312.7027.377[published  
12 Online First: Epub Date]].  
13
- 14 67. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global  
15 indicator for health risks of obesity and how its use could simplify the international public  
16 health message on obesity. *International Journal of Food Sciences and Nutrition*  
17 2005;**56**(5):303-07 doi: doi:10.1080/09637480500195066[published Online First: Epub  
18 Date]].  
19
- 20 68. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening  
21 tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global  
22 boundary value. *Nutrition Research Reviews* 2010;**23**(02):247-69 doi:  
23 doi:10.1017/S0954422410000144[published Online First: Epub Date]].  
24
- 25 69. Schneider HJ, Glaesmer H, Klotsche J, et al. Accuracy of anthropometric indicators of obesity to  
26 predict cardiovascular risk. *J Clin Endocrinol Metab* 2007;**92**:589-94  
27
- 28 70. Okosun IS, Liao Y, Rotimi CN, et al. Abdominal Adiposity and Clustering of Multiple Metabolic  
29 Syndrome in White, Black and Hispanic Americans. *Annals of Epidemiology*  
30 2000;**10**(5):263-70 doi: [http://dx.doi.org/10.1016/S1047-2797\(00\)00045-4](http://dx.doi.org/10.1016/S1047-2797(00)00045-4)[published  
31 Online First: Epub Date]].  
32
- 33 71. Ho SC, Chen YM, Woo JLF, et al. Association between simple anthropometric indices and  
34 cardiovascular risk factors. *Int J Obes Relat Metab Disord* 2001;**25**(11):1689-97  
35
- 36 72. Jeong S-K, Seo M-W, Kim Y-H, et al. Does Waist Indicate Dyslipidemia better than BMI in  
37 Korean Adult Population? *J Korean Med Sci* 2005;**20**(1):7-12  
38
- 39 73. Yusuf S, Hawken S, Ôunpuu S, et al. Obesity and the risk of myocardial infarction in 27 000  
40 participants from 52 countries: a case-control study. *Lancet* 2005;**366**(9497):1640-49  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

74. Moore SC. Waist versus weight—which matters more for mortality? The American Journal of Clinical Nutrition 2009;**89**(4):1003-04 doi: 10.3945/ajcn.2009.27598[published Online First: Epub Date]].

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
<b>Introduction</b>			
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
<b>Methods</b>			
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.
<b>Results</b>			

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
<b>Discussion</b>			
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
<b>Other information</b>			
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](http://www.strobe-statement.org).





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

Journal:	<i>BMJ Open</i>
Manuscript ID:	bmjopen-2013-004138.R2
Article Type:	Research
Date Submitted by the Author:	23-Dec-2013
Complete List of Authors:	Goh, Louise; Curtin University, School of Public Health Dhaliwal, Satvinder; Curtin University, School of Public Health Welborn, Timothy; Sir Charles Gairdner Hospital, Lee, Andy; Curtin University, School of Public Health Della, Phillip; Curtin University, School of Nursing and Midwifery
<b>Primary Subject Heading</b>:	Public health
Secondary Subject Heading:	Epidemiology, Cardiovascular medicine
Keywords:	PUBLIC HEALTH, EPIDEMIOLOGY, Cardiology < INTERNAL MEDICINE

SCHOLARONE™  
Manuscripts

1  
2  
3 **Anthropometric measurements of general and central obesity and**  
4  
5  
6 **the prediction of cardiovascular disease risk in women: a cross-**  
7  
8  
9 **sectional study**  
10

14 Correspondence to: Professor Satvinder S Dhaliwal

15 School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
16 U1987, Perth, WA 6845, Australia

17  
18  
19 Tel: +618 9266 2949

20  
21  
22 Fax: +618 9266 2958

23  
24 E-mail: [s.dhaliwal@curtin.edu.au](mailto:s.dhaliwal@curtin.edu.au)  
25  
26  
27

28 Louise GH Goh,<sup>1</sup> Satvinder S Dhaliwal,<sup>1</sup> Timothy A Welborn,<sup>2</sup> Andy H Lee,<sup>1</sup> Phillip R Della<sup>3</sup>  
29  
30  
31

32 <sup>1</sup>School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
33 U1987, Perth, WA 6845, Australia  
34  
35

36 <sup>2</sup>Sir Charles Gairdner Hospital, Hospital Avenue, Nedlands, Perth, WA 6009, Australia  
37

38 <sup>3</sup>School of Nursing and Midwifery, Curtin Health Innovation Research Institute, Curtin University,  
39 GPO Box U1987, Perth, WA 6845, Australia  
40  
41  
42  
43

44 Anthropometric obesity measures and CVD risk  
45  
46  
47

48 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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.

1  
2  
3 **Conclusions:** Central obesity measures are better predictors of CVD risk compared to general obesity  
4 measures in women. It is equally important to maintain a healthy weight and to prevent central obesity  
5 concurrently.  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

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

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<sup>1.5</sup>, 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

1  
2  
3 risk factors in the Framingham risk score model.[17] More research is thus needed to ascertain which  
4  
5 measures are better correlated with CVD risk factors and subsequent CVD risk in women.  
6  
7

8  
9 We aim to assess the associations between general and central obesity anthropometric measures with  
10  
11 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart  
12  
13 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk  
14  
15 calculated from the Framingham risk score model for 10-year CVD incidence or death,[18] SCORE  
16  
17 risk chart for high-risk regions for 10-year CVD death,[19] general CVD and simplified general CVD  
18  
19 risk score models for 10-year CVD incidence and death[20] were examined. To aid comparison  
20  
21 between obesity indices, which are measured in different units, the incremental shift in CVD risk with  
22  
23 one standard deviation increment in each anthropometric measurement above the mean would be  
24  
25 assessed. Finally, we determined which indices of obesity are most sensitive and specific for  
26  
27 identifying females at increased 10-year CVD risk.  
28  
29

## 30 31 **METHODS**

### 32 33 **Study cohort and measurements**

34  
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)  
38  
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,  
44  
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  
48  
49 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,  
50  
51 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of  
52  
53 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip  
54  
55 circumference were collected according to standardised methodologies[22 23] using two observers.  
56  
57 The waist circumference was measured from the front at the narrowest point between the rib cage and  
58  
59  
60

iliac crest after full expiration while the hip circumference was measured from the side at the maximal extension of buttocks by one observer using a metal tape. A second observer recorded another set of measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of two measurements was taken at each site to the nearest centimetre. Participants were classified as non-smokers, previous smokers or current smokers.[21] Mercury sphygmomanometers were used to record blood pressure levels on the right arm of seated participants five minutes apart.[21] Two readings were taken and the average was used in the analysis. Fasting blood samples were also collected in EDTA tubes and despatched to the central laboratory at the Division of Clinical Chemistry, Institute of Medical and Veterinary Science, Adelaide each week for lipid levels to be assayed.[21]

### **Risk score models**

The Framingham 10-year predicted risk for CVD incidence or death was developed using data from the American Framingham Heart Study.[18] Participants aged 30-74 years who were free of CVD and cancer were included in the model development. The 10-year risk for CVD incidence or death was calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status. The SCORE risk chart was developed by pooling 12 cohort studies to predict the 10-year CVD death risk in Europe. The cohorts consisted of participants aged 19-80 years with no previous history of heart attack.[19] The SCORE model was derived from a much larger dataset than the Framingham, general CVD and simplified general CVD risk score models. Fewer variables were used in the calculation of the 10-year predicted CVD death risk with the SCORE risk chart for high-risk regions (Denmark, Finland and Norway),[19 24] these included: age, sex, smoking status, mean total cholesterol level, mean HDL cholesterol level and mean SBP. The general CVD risk score model was also developed using data from the American Framingham Heart Study but using a larger cohort than the Framingham model.[20] Individuals without CVD were used in the development of the general CVD risk score model.[20] The simplified general CVD risk score model was developed similarly as the general CVD risk score model. It is, however, a simpler CVD risk prediction model which is



1  
2  
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.  
7  
8  
9

### 10 11 12 13 **Statistical analysis**

14  
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.  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52

### 53 54 55 56 **RESULTS**

1  
2  
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.  
5  
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.  
8  
9

10  
11  
12 The 10-year CVD risk of each participant in the sample was calculated using four risk score models.  
13 The frequency distribution of calculated risks is presented in Table 2. Except for the Framingham  
14 model for CVD incidence, all other models predicted risks of less than 10% for at least 85% of the  
15 sample. The Framingham model for CVD incidence, general CVD model for CVD incidence and  
16 death, and simplified general CVD model for CVD incidence and death, predicted risk values across  
17 the entire range from 0% to greater than 40%.  
18  
19  
20  
21  
22  
23  
24  
25  
26

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).  
33  
34  
35  
36  
37  
38  
39  
40  
41

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  
47  
48  
49  
50  
51  
52  
53

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  
57  
58  
59  
60

1  
2  
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.  
6  
7  
8  
9

10  
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.  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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 <sup>2</sup> )	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



1  
2  
3 inflammation which directly contributes to CVD risk.[35] Hence, measures that account for the  
4 accumulation of excess abdominal fat would report stronger associations and are desirable for  
5 assessing adiposity. They would also be more accurate at indicating CVD risk and should be  
6 incorporated into CVD assessment.[36-39] The addition of central obesity measures to BMI has also  
7 been shown to improve the accuracy of stratifying participants into lower and higher risk categories  
8 for mortality[40] and provides incremental value in predicting CVD above and beyond that provided  
9 by general obesity measures.[41-45] BMI is a flawed measure as it does not correctly identify  
10 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does  
11 not account for the effect of age and ethnicity on body fat distribution.[46-50] An increase in muscle  
12 or fat-free mass would, however, be reflected in the central obesity measures.

13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60  
Among central obesity measures, we found their performance to be comparable in our study. It  
remains unclear which measurement should be incorporated into CVD risk score models. A  
collaborative analysis of 58 prospective studies, however, reported that both measures of general and  
central obesity did not improve CVD risk assessment when information is available on SBP, diabetes  
and lipids.[51] Overweight and obesity is nevertheless important in CVD prevention, with one out of  
three of fatal and one out of seven of non-fatal CVD cases attributable to it.[34]

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

1  
2  
3  
4  
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  $\geq 0.88$  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]

25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
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  
54  
55  
56  
57  
58  
59  
60

1  
2  
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.  
8  
9  
10  
11  
12

13  
14  
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.  
20  
21  
22  
23  
24  
25  
26

## 27 CONCLUSIONS

28  
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.  
33  
34  
35  
36  
37  
38

39  
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]  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
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]  
7  
8  
9

10  
11  
12  
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.  
19  
20  
21  
22  
23  
24  
25  
26

### 27 **Figure legend**

28  
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**  
34  
35

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.  
38

39 Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WSR,  
40 waist-to-stature ratio.  
41  
42  
43  
44  
45  
46  
47

48  
49 **Acknowledgements** Curtin University provided educational support to LGHG through the Curtin  
50 International Postgraduate Research Scholarship.  
51

52  
53  
54 **Contributors** LGHG was involved in drafting the manuscript, interpretation of data and revising the  
55 manuscript critically for important intellectual content. SSD conceived the study, performed the  
56  
57  
58  
59  
60

1  
2  
3 analysis and data interpretation and revised the manuscript critically for important intellectual content.  
4  
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.  
8  
9  
10

11 **Competing interests** None.  
12  
13

14  
15 **Funding** None.  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
3 **Ethics approval** We have ethics approval for the use of the National Heart Foundation data from the  
4 Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth  
5 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin  
6 University. This study was carried out in accordance with the Declaration of Helsinki.  
7  
8  
9  
10

11  
12  
13 **Data sharing statement** No additional data are available.  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

For peer review only

## REFERENCES

1. World Health Organization. Obesity and overweight. Secondary Obesity and overweight 2012. <http://www.who.int/mediacentre/factsheets/fs311/en/>.
2. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. *Circulation* 2002;**106**(25):3143-421
3. Kannel WB. Metabolic risk factors for coronary heart disease in women: Perspective from the Framingham Study. *Am Heart J* 1987;**114**(2):413-19 doi: [http://dx.doi.org/10.1016/0002-8703\(87\)90511-4](http://dx.doi.org/10.1016/0002-8703(87)90511-4)[published Online First: Epub Date]].
4. Hubert HB, Feinleib M, McNamara PM, et al. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation* 1983;**67**:968-77
5. Manson JE, Colditz GA, Stampfer MJ, et al. A Prospective Study of Obesity and Risk of Coronary Heart Disease in Women. *N Engl J Med* 1990;**322**(13):882-89 doi: doi:10.1056/NEJM199003293221303[published Online First: Epub Date]].
6. Manson JE, Willett WC, Stampfer MJ, et al. Body Weight and Mortality among Women. *N Engl J Med* 1995;**333**(11):677-85 doi: doi:10.1056/NEJM199509143331101[published Online First: Epub Date]].
7. Dorn JM, Schisterman EF, Winkelstein W, et al. Body Mass Index and Mortality in a General Population Sample Women of Men and Women: The Buffalo Health Study. *American Journal of Epidemiology* 1997;**146**(11):919-31
8. Cornier M-A, Després J-P, Davis N, et al. Assessing Adiposity: A Scientific Statement From the American Heart Association. *Circulation* 2011;**124**(18):1996-2019 doi: 10.1161/CIR.0b013e318233bc6a[published Online First: Epub Date]].
9. Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation* 2013

- 1  
2  
3 10. Park YS, Kim J-S. Obesity Phenotype and Coronary Heart Disease Risk as Estimated by the  
4 Framingham Risk Score. *Journal of Korean Medical Science* 2012;**27**(3):243-49  
5  
6  
7 11. Satoh H, Kishi R, Tsutsui H. Body Mass Index can Similarly Predict the Presence of Multiple  
8 Cardiovascular Risk Factors in Middle-aged Japanese Subjects as Waist Circumference.  
9 *Internal Medicine* 2010;**49**(11):977-82  
10  
11  
12  
13 12. Ryan MC, Fenster Farin HM, Abbasi F, et al. Comparison of Waist Circumference Versus Body  
14 Mass Index in Diagnosing Metabolic Syndrome and Identifying Apparently Healthy Subjects  
15 at Increased Risk of Cardiovascular Disease. *The American Journal of Cardiology*  
16 2008;**102**(1):40-46 doi: <http://dx.doi.org/10.1016/j.amjcard.2008.02.096>[published  
17 Online  
18 First: Epub Date]].  
19  
20  
21  
22  
23 13. Ying X, Song Z, Zhao C, et al. Body mass index, waist circumference, and cardiometabolic risk  
24 factors in young and middle-aged Chinese women. *J Zhejiang Univ Sci B* 2010;**11**(9):639-46  
25 doi: 10.1631/jzus.B1000105[published Online First: Epub Date]].  
26  
27  
28  
29 14. Zhu S, Heymsfield SB, Toyoshima H, et al. Race-ethnicity-specific waist circumference cutoffs  
30 for identifying cardiovascular disease risk factors. *The American Journal of Clinical Nutrition*  
31 2005;**81**(2):409-15  
32  
33  
34  
35 15. Huang K-C, Lee M-S, Lee S-D, et al. Obesity in the Elderly and Its Relationship with  
36 Cardiovascular Risk Factors in Taiwan. *Obes Res* 2005;**13**(1):170-78 doi:  
37 10.1038/oby.2005.22[published Online First: Epub Date]].  
38  
39  
40  
41 16. Bergman RN, Stefanovski D, Buchanan TA, et al. A Better Index of Body Adiposity. *Obesity*  
42 (Silver Spring) 2011;**19**(5):1083-89  
43  
44  
45  
46 17. Dhaliwal SS, Welborn TA. Central obesity and multivariable cardiovascular risk as assessed by  
47 the Framingham prediction scores. *Am J Cardiol* 2009;**103**:1403-07  
48  
49  
50 18. Anderson KM, Odell PM, Wilson PW, et al. Cardiovascular disease risk profiles. *Am Heart J*  
51 1991;**121**(1 Part 2):293-98  
52  
53  
54 19. Conroy RM, Pyörälä K, Fitzgerald AP, et al. Estimation of ten-year risk of fatal cardiovascular  
55 disease in Europe: The SCORE project. *European Heart Journal* 2003;**24**(11):987-1003 doi:  
56 10.1016/s0195-668x(03)00114-3[published Online First: Epub Date]].  
57  
58  
59  
60



- 1  
2  
3 20. D'Agostino RB, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in  
4 primary care - The Framingham Heart Study. *Circulation* 2008;**117**(6):743-53  
5  
6  
7 21. Australian Risk Factor Prevalence Study Management Committee. Survey No. 3 1989. Canberra:  
8 National Heart Foundation of Australia and Australia Institute of Health, 1990.  
9  
10  
11 22. Boyle CA, Dobson AJ, Egger G, et al. Waist-to-hip ratios in Australia: A different picture of  
12 obesity. *Aust J Nutr Diet* 1993;**50**:57-64  
13  
14  
15 23. Alexander H, Dugdale A. Which waist-hip ratio? *Med J Aust* 1990;**153**(6):367-68  
16  
17  
18 24. Cooney MT, Dudina A, De Bacquer D, et al. How much does HDL cholesterol add to risk  
19 estimation? A report from the SCORE investigators. *European Journal of Cardiovascular*  
20 *Prevention & Rehabilitation* 2009;**16**(3):304-14 doi:  
21 10.1097/HJR.0b013e3283213140[published Online First: Epub Date].  
22  
23  
24 25. Neil HAW, Perera R, Armitage JM, et al. Estimated 10-year cardiovascular risk in a British  
25 population: results of a national screening project. *Int J Clin Pract* 2008;**62**(9):1322-31  
26  
27  
28 26. Woodward M, Brindle P, Tunstall-Pedoe H. Adding social deprivation and family history to  
29 cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended  
30 Cohort (SHHEC). *Heart* 2007;**93**(2):172-76  
31  
32  
33 27. Mosca L, Benjamin EJ, Berra K, et al. Effectiveness-Based Guidelines for the Prevention of  
34 Cardiovascular Disease in Women—2011 Update A Guideline From the American Heart  
35 Association. *J Am Coll Cardiol* 2011;**57**(12):1404-23  
36  
37  
38 28. Genest J, McPherson R, Frohlich J, et al. 2009 Canadian Cardiovascular Society/Canadian  
39 guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular  
40 disease in the adult – 2009 recommendations. *Can J Cardiol* 2009;**25**(10):567-79  
41  
42  
43 29. Goh LGH, Dhaliwal SS, Lee AH, et al. Utility of established cardiovascular disease risk score  
44 models for the 10-year prediction of disease outcomes in women. *Expert Rev Cardiovasc*  
45 *Ther* 2013;**11**(4):425-35  
46  
47  
48 30. Wittchen H-U, Balkau B, Massien C, et al. International Day for the Evaluation of Abdominal  
49 obesity: rationale and design of a primary care study on the prevalence of abdominal obesity  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 and associated factors in 63 countries. *Eur Heart J Suppl* 2006;**8**(suppl B):B26-B33 doi:  
4 10.1093/eurheartj/sul005[published Online First: Epub Date].  
5  
6  
7 31. Balkau B, Deanfield JE, Despres JP, et al. International day for the evaluation of abdominal  
8 obesity (IDEA) - A study of waist circumference, cardiovascular disease, and diabetes  
9 mellitus in 168 000 primary care patients in 63 countries. *Circulation* 2007;**116**(17):1942-51  
10 doi: 10.1161/circulationaha.106.676379[published Online First: Epub Date]].  
11  
12  
13 32. Winter Y, Rohrmann S, Linseisen J, et al. Contribution of Obesity and Abdominal Fat Mass to  
14 Risk of Stroke and Transient Ischemic Attacks. *Stroke* 2008;**39**(12):3145-51 doi:  
15 10.1161/strokeaha.108.523001[published Online First: Epub Date]].  
16  
17  
18 33. Taylor AE, Ebrahim S, Ben-Shlomo Y, et al. Comparison of the associations of body mass index  
19 and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-  
20 cause mortality: a study using data from 4 UK cohorts. *The American Journal of Clinical*  
21 *Nutrition* 2010;**91**(3):547-56 doi: 10.3945/ajcn.2009.28757[published Online First: Epub  
22 Date]].  
23  
24  
25 34. van Dis I, Kromhout D, Geleijnse JM, et al. Body mass index and waist circumference predict  
26 both 10-year nonfatal and fatal cardiovascular disease risk: study conducted in 20 000 Dutch  
27 men and women aged 20–65 years. *European Journal of Cardiovascular Prevention &*  
28 *Rehabilitation* 2009;**16**(6):729-34 doi: 10.1097/HJR.0b013e328331dfc0[published Online  
29 First: Epub Date]].  
30  
31  
32 35. Berg AH, Scherer PE. Adipose Tissue, Inflammation, and Cardiovascular Disease. *Circulation*  
33 *Research* 2005;**96**(9):939-49 doi: 10.1161/01.res.0000163635.62927.34[published Online  
34 First: Epub Date]].  
35  
36  
37 36. Snijder MB, van Dam RM, Visser M, et al. What aspects of body fat are particularly hazardous  
38 and how do we measure them? *International Journal of Epidemiology* 2006;**35**(1):83-92 doi:  
39 10.1093/ije/dyi253[published Online First: Epub Date]].  
40  
41  
42 37. de Koning L, Merchant AT, Pogue J, et al. Waist circumference and waist-to-hip ratio as  
43 predictors of cardiovascular events: meta-regression analysis of prospective studies. *Eur Heart*  
44 *J* 2007;**28**(7):850-56  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 38. Dalton M, Cameron AJ, Zimmet PZ, et al. Waist circumference, waist-hip ratio and body mass  
4 index and their correlation with cardiovascular disease risk factors in Australian adults.  
5 Journal of Internal Medicine 2003;**254**(6):555-63 doi: 10.1111/j.1365-  
6 2796.2003.01229.x[published Online First: Epub Date].  
7  
8  
9  
10  
11 39. Antillon D, Towfighi A. No time to 'weight': the link between obesity and stroke in women.  
12 Women's Health 2011;**7**(4):453-63 doi: 10.2217/whe.11.36[published Online First: Epub  
13 Date]].  
14  
15  
16  
17 40. Pischon T, Boeing H, Hoffmann K, et al. General and Abdominal Adiposity and Risk of Death in  
18 Europe. N Engl J Med 2008;**359**(20):2105-20 doi: doi:10.1056/NEJMoa0801891[published  
19 Online First: Epub Date]].  
20  
21  
22  
23 41. Li C, Engstrom G, Hedblad B, et al. Sex differences in the relationships between BMI, WHR and  
24 incidence of cardiovascular disease: a population-based cohort study. International Journal of  
25 Obesity 2006;**30**(12):1775-81 doi: 10.1038/sj.ijo.0803339[published Online First: Epub  
26 Date]].  
27  
28  
29  
30  
31 42. Freiberg MS, Pencina MJ, D'Agostino RB, et al. BMI vs. Waist Circumference for Identifying  
32 Vascular Risk. Obesity 2008;**16**(2):463-69  
33  
34  
35  
36 43. DiPietro L, Katz LD, Nadel ER. Excess abdominal adiposity remains correlated with altered lipid  
37 concentrations in healthy older women. International journal of obesity and related metabolic  
38 disorders : journal of the International Association for the Study of Obesity 1999;**23**(4):432-  
39 36  
40  
41  
42  
43 44. Klein S, Allison DB, Heymsfield SB, et al. Waist Circumference and Cardiometabolic Risk: A  
44 Consensus Statement from Shaping America's Health: Association for Weight Management  
45 and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition;  
46 and the American Diabetes Association. Obesity 2007;**15**(5):1061-67 doi:  
47 10.1038/oby.2007.632[published Online First: Epub Date]].  
48  
49  
50  
51  
52  
53  
54 45. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in  
55 women. JAMA 1998;**280**(21):1843-48 doi: 10.1001/jama.280.21.1843[published Online  
56 First: Epub Date]].  
57  
58  
59  
60

- 1  
2  
3 46. Fogelholm M. Physical activity, fitness and fatness: relations to mortality, morbidity and disease  
4 risk factors. A systematic review. *Obesity Reviews* 2010;**11**(3):202-21 doi: 10.1111/j.1467-  
5 789X.2009.00653.x[published Online First: Epub Date]].  
6  
7  
8  
9 47. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Accuracy of body mass index in  
10 diagnosing obesity in the adult general population. *Int J Obes* 2008;**32**(6):959-66 doi:  
11 <http://www.nature.com/ijo/journal/v32/n6/supinfo/ijo200811s1.html>[published  
12 Online First:  
13 Epub Date]].  
14  
15  
16 48. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a  
17 WHO consultation. World Health Organ Tech Rep Ser: WHO, 2000.  
18  
19 49. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis  
20 among different ethnic groups. *International Journal of Obesity* 1998;**22**(12):1164-71  
21  
22 50. Welborn TA, Dhaliwal SS. Being correct about obesity. *Med J Aust* 2011;**194** (8 ):429-30  
23  
24 51. The Emerging Risk Factors Collaboration. Separate and combined associations of body-mass  
25 index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58  
26 prospective studies. *The Lancet* 2011;**377**(9771):1085-95 doi:  
27 [http://dx.doi.org/10.1016/S0140-6736\(11\)60105-0](http://dx.doi.org/10.1016/S0140-6736(11)60105-0)[published Online First: Epub Date]].  
28  
29 52. Dijk SB, Takken T, Prinsen EC, et al. Different anthropometric adiposity measures and their  
30 association with cardiovascular disease risk factors: a meta-analysis. *Neth Heart J*  
31 2012;**20**(5):208-18 doi: 10.1007/s12471-011-0237-7[published Online First: Epub Date]].  
32  
33 53. Dobbelsteyn CJ, Joffres MR, MacLean DR, et al. A comparative evaluation of waist  
34 circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk  
35 factors. The Canadian Heart Health Surveys. *International Journal of Obesity* 2001;**25**:652-61  
36  
37 54. Pouliot M-C, Després J-P, Lemieux S, et al. Waist circumference and abdominal sagittal diameter:  
38 Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and  
39 related cardiovascular risk in men and women. *The American Journal of Cardiology*  
40 1994;**73**(7):460-68 doi: [http://dx.doi.org/10.1016/0002-9149\(94\)90676-9](http://dx.doi.org/10.1016/0002-9149(94)90676-9)[published  
41 Online  
42 First: Epub Date]].  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 55. Turcato E, Bosello O, Di Francesco V, et al. Waist circumference and abdominal sagittal diameter  
4 as surrogates of body fat distribution in the elderly: their relation with cardiovascular risk  
5 factors. *Int J Obes Relat Metab Disord* 2000;**24**(8):1005-10  
6  
7  
8  
9 56. Zhu S, Wang Z, Heshka S, et al. Waist circumference and obesity-associated risk factors among  
10 whites in the third National Health and Nutrition Examination Survey: clinical action  
11 thresholds. *The American Journal of Clinical Nutrition* 2002;**76**(4):743  
12  
13  
14 57. Reeder BA, Senthilselvan A, Després JP, et al. The association of cardiovascular disease risk  
15 factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group.  
16 *CMAJ : Canadian Medical Association journal* 1997;**157 Suppl 1**:S39-S45  
17  
18  
19 58. Lapidus L, Bengtsson C, Larsson B, et al. Distribution of adipose tissue and risk of cardiovascular  
20 disease and death: a 12 year follow up of participants in the population study of women in  
21 Gothenburg, Sweden. *BMJ* 1984;**289**(6454):1257-61 doi:  
22 10.1136/bmj.289.6454.1257[published Online First: Epub Date].  
23  
24  
25 59. Price GM, Uauy R, Breeze E, et al. Weight, shape, and mortality risk in older persons: elevated  
26 waist-hip ratio, not high body mass index, is associated with a greater risk of death. *The*  
27 *American Journal of Clinical Nutrition* 2006;**84**(2):449-60  
28  
29  
30 60. Lu M, Ye W, Adami HO, et al. Prospective study of body size and risk for stroke amongst women  
31 below age 60. *Journal of Internal Medicine* 2006;**260**(5):442-50 doi: 10.1111/j.1365-  
32 2796.2006.01706.x[published Online First: Epub Date].  
33  
34  
35 61. Dhaliwal SS, Welborn TA. Central obesity and cigarette smoking are key determinants of  
36 cardiovascular deaths in Australia: A public health perspective. *Preventive Medicine*  
37 2009;**49**(2-3):153-57  
38  
39  
40 62. Welborn TA, Dhaliwal SS. Preferred clinical measures of central obesity for predicting mortality.  
41 *Eur J Clin Nutr* 2007;**61**(12):1373-79  
42  
43  
44 63. Welborn TA, Dhaliwal SS, Bennett SA. Waist-hip ratio is the dominant risk factor predicting  
45 cardiovascular death in Australia. *Med J Aust* 2003;**179**(11-12):580-85  
46  
47  
48 64. Dhaliwal SS, Welborn TA. Measurement error and ethnic comparisons of measures of abdominal  
49 obesity. *Prev Med* 2009;**49**(2-3):148-52  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 65. Goodman-Gruen D, Barrett-Connor E. Sex Differences in Measures of Body Fat and Body Fat  
4  
5 Distribution in the Elderly. *Am J Epidemiol* 1996;**143**(9):898-906  
6  
7 66. Ashwell M, Lejeune S. Ratio of waist circumference to height may be better indicator of need for  
8  
9 weight management. *BMJ* 1996;**312**(7027):377 doi: 10.1136/bmj.312.7027.377[published  
10  
11 Online First: Epub Date]].  
12  
13 67. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global  
14  
15 indicator for health risks of obesity and how its use could simplify the international public  
16  
17 health message on obesity. *International Journal of Food Sciences and Nutrition*  
18  
19 2005;**56**(5):303-07 doi: doi:10.1080/09637480500195066[published Online First: Epub  
20  
21 Date]].  
22  
23 68. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening  
24  
25 tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global  
26  
27 boundary value. *Nutrition Research Reviews* 2010;**23**(02):247-69 doi:  
28  
29 doi:10.1017/S0954422410000144[published Online First: Epub Date]].  
30  
31 69. Schneider HJ, Glaesmer H, Klotsche J, et al. Accuracy of anthropometric indicators of obesity to  
32  
33 predict cardiovascular risk. *J Clin Endocrinol Metab* 2007;**92**:589-94  
34  
35 70. Okosun IS, Liao Y, Rotimi CN, et al. Abdominal Adiposity and Clustering of Multiple Metabolic  
36  
37 Syndrome in White, Black and Hispanic Americans. *Annals of Epidemiology*  
38  
39 2000;**10**(5):263-70 doi: [http://dx.doi.org/10.1016/S1047-2797\(00\)00045-4](http://dx.doi.org/10.1016/S1047-2797(00)00045-4)[published  
40  
41 Online  
42 First: Epub Date]].  
43  
44 71. Ho SC, Chen YM, Woo JLF, et al. Association between simple anthropometric indices and  
45  
46 cardiovascular risk factors. *Int J Obes Relat Metab Disord* 2001;**25**(11):1689-97  
47  
48 72. Jeong S-K, Seo M-W, Kim Y-H, et al. Does Waist Indicate Dyslipidemia better than BMI in  
49  
50 Korean Adult Population? *J Korean Med Sci* 2005;**20**(1):7-12  
51  
52 73. Yusuf S, Hawken S, Ôunpuu S, et al. Obesity and the risk of myocardial infarction in 27□000  
53  
54 participants from 52 countries: a case-control study. *Lancet* 2005;**366**(9497):1640-49  
55  
56  
57  
58  
59  
60

- 1  
2  
3 74. Moore SC. Waist versus weight—which matters more for mortality? The American Journal of  
4  
5 Clinical Nutrition 2009;**89**(4):1003-04 doi: 10.3945/ajcn.2009.27598[published Online First:  
6  
7 Epub Date]].  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

For peer review only



1  
2  
3 **Anthropometric measurements of general and central obesity and**  
4  
5  
6 **the prediction of cardiovascular disease risk in women: a cross-**  
7  
8  
9 **sectional study**  
10

14 Correspondence to: Professor Satvinder S Dhaliwal

15 School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
16 U1987, Perth, WA 6845, Australia

17  
18  
19 Tel: +618 9266 2949

20  
21  
22 Fax: +618 9266 2958

23  
24  
25 E-mail: [s.dhaliwal@curtin.edu.au](mailto:s.dhaliwal@curtin.edu.au)  
26  
27

28 Louise GH Goh,<sup>1</sup> Satvinder S Dhaliwal,<sup>1</sup> Timothy A Welborn,<sup>2</sup> Andy H Lee,<sup>1</sup> Phillip R Della<sup>3</sup>  
29  
30  
31

32 <sup>1</sup>School of Public Health, Curtin Health Innovation Research Institute, Curtin University, GPO Box  
33 U1987, Perth, WA 6845, Australia  
34  
35

36 <sup>2</sup>Sir Charles Gairdner Hospital, Hospital Avenue, Nedlands, Perth, WA 6009, Australia  
37

38 <sup>3</sup>School of Nursing and Midwifery, Curtin Health Innovation Research Institute, Curtin University,  
39 GPO Box U1987, Perth, WA 6845, Australia  
40  
41  
42

43  
44 Anthropometric obesity measures and CVD risk  
45  
46  
47

48  
49 Keywords: Cardiovascular disease risk, anthropometric, adiposity, waist circumference and female  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60



**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.

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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.

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 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<sup>1.5</sup>, 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

1  
2  
3 risk factors in the Framingham risk score model.[17] More research is thus needed to ascertain which  
4  
5 measures are better correlated with CVD risk factors and subsequent CVD risk in women.  
6  
7

8  
9 We aim to assess the associations between general and central obesity anthropometric measures with  
10  
11 CVD risk factors, using a representative sample of 4487 females aged 20-69 years without heart  
12  
13 disease, diabetes or stroke. The associations between these indices of obesity with predicted risk  
14  
15 calculated from the Framingham risk score model for 10-year CVD incidence or death,[18] SCORE  
16  
17 risk chart for high-risk regions for 10-year CVD death,[19] general CVD and simplified general CVD  
18  
19 risk score models for 10-year CVD incidence and death[20] ~~would also be assessed~~ were examined.

20  
21 To aid comparison between obesity indices, which are measured in different units, the incremental  
22  
23 shift in CVD risk with one standard deviation increment in each anthropometric measurement above  
24  
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.  
28  
29

## 30 31 **METHODS**

### 32 33 **Study cohort and measurements**

34  
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)  
38  
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,  
44  
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  
48  
49 conventional CVD risk variables recorded in this prevalence study include: anthropometric measures,  
50  
51 smoking status, systolic and diastolic blood pressure, and lipid levels. Physical measurements of  
52  
53 height (to the nearest centimetre), weight (to the nearest 10th of a kilogram), and waist and hip  
54  
55 circumference were collected according to standardised methodologies[22 23] using two observers.  
56  
57 The waist circumference was measured from the front at the narrowest point between the rib cage and  
58  
59  
60

iliac crest after full expiration while the hip circumference was measured from the side at the maximal extension of buttocks by one observer using a metal tape. A second observer recorded another set of measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of two measurements was taken at each site to the nearest centimetre. Participants were classified as non-smokers, previous smokers or current smokers.[21] Mercury sphygmomanometers were used to record blood pressure levels on the right arm of seated participants five minutes apart.[21] Two readings were taken and the average was used in the analysis. Fasting blood samples were also collected in EDTA tubes and despatched to the central laboratory at the Division of Clinical Chemistry, Institute of Medical and Veterinary Science, Adelaide each week for lipid levels to be assayed.[21]

### **Risk score models**

The Framingham 10-year predicted risk for CVD incidence or death was developed using data from the American Framingham Heart Study.[18] Participants aged 30-74 years who were free of CVD and cancer were included in the model development. The 10-year risk for CVD incidence or death was calculated using these variables: age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status and diabetes status. The SCORE risk chart was developed by pooling 12 cohort studies to predict the 10-year CVD death risk in Europe. The cohorts consisted of participants aged 19-80 years with no previous history of heart attack.[19] ~~H~~The SCORE model was derived from a much larger dataset than the Framingham, general CVD and simplified general CVD risk score models. Fewer variables were used in the calculation of the 10-year predicted CVD death risk with the SCORE risk chart for high-risk regions (Denmark, Finland and Norway),[19 24] these included: age, sex, smoking status, mean total cholesterol level, mean HDL cholesterol level and mean SBP. The general CVD risk score model was also developed using data from the American Framingham Heart Study but using a larger cohort than the Framingham model.[20] Individuals without CVD were used in the development of the general CVD risk score model.[20] The simplified general CVD risk score model was developed similarly as the general CVD risk score model. It is, however, a simpler CVD risk prediction model which is

1  
2  
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.  
7  
8  
9

### 10 11 12 13 **Statistical analysis**

14  
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.  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53

### 54 55 56 **RESULTS**

1  
2  
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.  
5  
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.  
8  
9

10  
11  
12 The 10-year CVD risk of each participant in the sample was calculated using four risk score models.  
13 The frequency distribution of calculated risks is presented in Table 2. Except for the Framingham  
14 model for CVD incidence, all other models predicted risks of less than 10% for at least 85% of the  
15 sample. The Framingham model for CVD incidence, general CVD model for CVD incidence and  
16 death, and simplified general CVD model for CVD incidence and death, predicted risk values across  
17 the entire range from 0% to greater than 40%.  
18  
19  
20  
21  
22  
23  
24  
25  
26

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).  
33  
34  
35  
36  
37  
38  
39  
40  
41

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  
47  
48  
49  
50  
51  
52  
53

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  
57  
58  
59  
60



1  
2  
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.  
6  
7  
8  
9

10 Anthropometric measurements of central obesity (WC, WHR and WSR) also recorded higher area  
11 under the ROC curves, higher sensitivity and specificity, than BMI in identifying females above and  
12 below the 20% treatment threshold for the Framingham model for 10-year CVD incidence (Figure 1a)  
13 and general CVD model for 10-year CVD incidence and death (Figure 1b). Although BMI is included  
14 in the simplified general CVD model, high area under the ROC curve ( $> 0.76$ ) are reported for both  
15 WC and WHR (Figure 1c), indicating the independent contribution of central obesity measurements  
16 as compared to general obesity measurement in predicting the increased risk of CVD.  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

**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 <sup>2</sup> )	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.

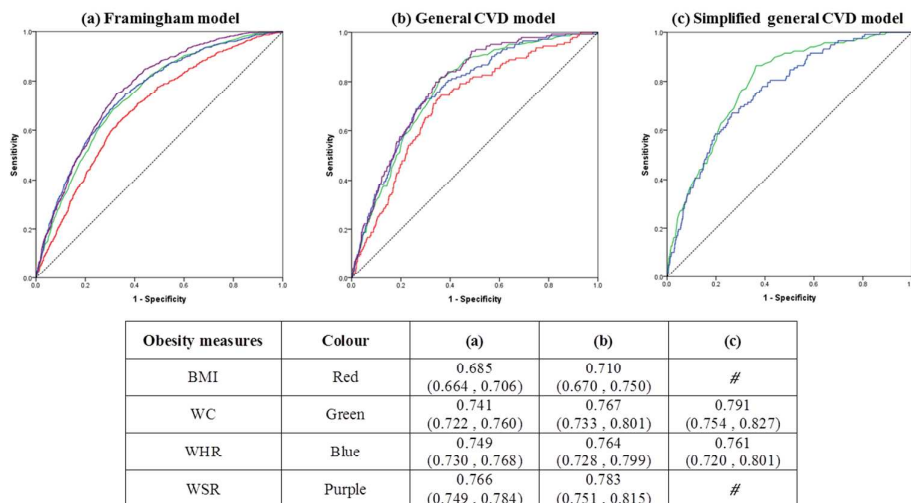
**Table 4 Odds-ratios and associated 95% confidence intervals of being above the recommended treatment thresholds for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity**

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

1  
2  
3 inflammation which directly contributes to CVD risk.[35] Hence, measures that account for the  
4 accumulation of excess abdominal fat would report stronger associations and are desirable for  
5 assessing adiposity. They would also be more accurate at indicating CVD risk and should be  
6 incorporated into CVD assessment.[36-39] The addition of central obesity measures to BMI has also  
7 been shown to improve the accuracy of stratifying participants into lower and higher risk categories  
8 for mortality[40] and provides incremental value in predicting CVD above and beyond that provided  
9 by general obesity measures.[41-45] BMI is a flawed measure as it does not correctly identify  
10 individuals with excess body fat due to its inability to differentiate fat and fat-free mass and it does  
11 not account for the effect of age and ethnicity on body fat distribution.[46-50] An increase in muscle  
12 or fat-free mass would, however, be reflected in the central obesity measures.

13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26 Among central obesity measures, we found their performance to be comparable in our study. It  
27 remains unclear which measurement should be incorporated into CVD risk score models. A  
28 collaborative analysis of 58 prospective studies, however, reported that both measures of general and  
29 central obesity did not improve CVD risk assessment when information is available on SBP, diabetes  
30 and lipids.[51] Overweight and obesity is nevertheless important in CVD prevention, with one out of  
31 three of fatal and one out of seven of non-fatal CVD cases attributable to it.[34]

32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60  
Opinion remains divided as to which is a more appropriate measurement for assessing adiposity and  
its association with CVD risk.[37] Some studies recommended the use of WC in clinical assessment  
and research studies.[52-53] In a systematic review and meta-analysis study of Caucasians without  
CVD, WC was most highly correlated with all CVD risk factors, compared to BMI, WHR, WSR and  
body fat percentage, in women.[52] In other studies, WC was also more closely associated with CVD  
risk factors than other measures of central obesity and BMI in women.[54-57] The advantages of WC  
are, it is easy to measure and interpret, and it is less prone to measurement and calculation error.[53]  
Appropriate sex, age and ethnic-specific WC cut points would need to be established.[44] It would  
also be difficult to use WC in today's multicultural societies due to requirements for different cut  
points.[50]



1  
2  
3  
4  
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  $\geq 0.88$  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]

25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
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  
54  
55  
56  
57  
58  
59  
60

1  
2  
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.  
8  
9

10  
11  
12 Our study has limitations. This study is cross-sectional, however, it is a representative sample of the  
13 Australian female population. There is only one set of baseline measurements recorded for some risk  
14 variables but important variables including anthropometric measures of obesity are measured twice.  
15 Further, the 10-year CVD risks are calculated using risk score models to stratify individuals against  
16 the treatment thresholds of the various models, and are not prospective CVD events.  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26

## 27 CONCLUSIONS

28  
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.  
33  
34  
35  
36  
37  
38

39  
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]  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
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]  
7  
8  
9

10  
11  
12  
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.  
19  
20  
21  
22  
23  
24

25  
26  
27 **Acknowledgements** Curtin University provided educational support to LGHG through the Curtin  
28 International Postgraduate Research Scholarship.  
29  
30  
31

32  
33 **Contributors** LGHG was involved in drafting the manuscript, interpretation of data and revising the  
34 manuscript critically for important intellectual content. SSD conceived the study, performed the  
35 analysis and data interpretation and revised the manuscript critically for important intellectual content.  
36 TAW participated in the study design, acquired the data and revised the manuscript critically for  
37 important intellectual content. All authors read and approved the final manuscript.  
38  
39  
40  
41  
42  
43  
44

45  
46 **Competing interests** None.  
47  
48

49  
50 **Funding** None.  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

1  
2  
3 **Ethics approval** We have ethics approval for the use of the National Heart Foundation data from the  
4 Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth  
5 Privacy Commissioner, and approval from the Human Research Ethics Committee at Curtin  
6 University. This study was carried out in accordance with the Declaration of Helsinki.  
7  
8  
9  
10

11  
12  
13 **Data sharing statement** No additional data are available.  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

For peer review only

## REFERENCES

1. World Health Organization. Obesity and overweight. Secondary Obesity and overweight 2012. <http://www.who.int/mediacentre/factsheets/fs311/en/>.
2. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. *Circulation* 2002;**106**(25):3143-421
3. Kannel WB. Metabolic risk factors for coronary heart disease in women: Perspective from the Framingham Study. *Am Heart J* 1987;**114**(2):413-19 doi: [http://dx.doi.org/10.1016/0002-8703\(87\)90511-4](http://dx.doi.org/10.1016/0002-8703(87)90511-4)[published Online First: Epub Date]].
4. Hubert HB, Feinleib M, McNamara PM, et al. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation* 1983;**67**:968-77
5. Manson JE, Colditz GA, Stampfer MJ, et al. A Prospective Study of Obesity and Risk of Coronary Heart Disease in Women. *N Engl J Med* 1990;**322**(13):882-89 doi: doi:10.1056/NEJM199003293221303[published Online First: Epub Date]].
6. Manson JE, Willett WC, Stampfer MJ, et al. Body Weight and Mortality among Women. *N Engl J Med* 1995;**333**(11):677-85 doi: doi:10.1056/NEJM199509143331101[published Online First: Epub Date]].
7. Dorn JM, Schisterman EF, Winkelstein W, et al. Body Mass Index and Mortality in a General Population Sample Women of Men and Women: The Buffalo Health Study. *American Journal of Epidemiology* 1997;**146**(11):919-31
8. Cornier M-A, Després J-P, Davis N, et al. Assessing Adiposity: A Scientific Statement From the American Heart Association. *Circulation* 2011;**124**(18):1996-2019 doi: 10.1161/CIR.0b013e318233bc6a[published Online First: Epub Date]].
9. Jensen MD, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS Guideline for the Management of Overweight and Obesity in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. *Circulation* 2013

- 1  
2  
3 10. Park YS, Kim J-S. Obesity Phenotype and Coronary Heart Disease Risk as Estimated by the  
4 Framingham Risk Score. *Journal of Korean Medical Science* 2012;**27**(3):243-49  
5  
6  
7 11. Satoh H, Kishi R, Tsutsui H. Body Mass Index can Similarly Predict the Presence of Multiple  
8 Cardiovascular Risk Factors in Middle-aged Japanese Subjects as Waist Circumference.  
9 *Internal Medicine* 2010;**49**(11):977-82  
10  
11  
12  
13 12. Ryan MC, Fenster Farin HM, Abbasi F, et al. Comparison of Waist Circumference Versus Body  
14 Mass Index in Diagnosing Metabolic Syndrome and Identifying Apparently Healthy Subjects  
15 at Increased Risk of Cardiovascular Disease. *The American Journal of Cardiology*  
16 2008;**102**(1):40-46 doi: <http://dx.doi.org/10.1016/j.amjcard.2008.02.096>[published  
17 Online  
18 First: Epub Date]].  
19  
20  
21  
22  
23 13. Ying X, Song Z, Zhao C, et al. Body mass index, waist circumference, and cardiometabolic risk  
24 factors in young and middle-aged Chinese women. *J Zhejiang Univ Sci B* 2010;**11**(9):639-46  
25 doi: 10.1631/jzus.B1000105[published Online First: Epub Date]].  
26  
27  
28  
29 14. Zhu S, Heymsfield SB, Toyoshima H, et al. Race-ethnicity-specific waist circumference cutoffs  
30 for identifying cardiovascular disease risk factors. *The American Journal of Clinical Nutrition*  
31 2005;**81**(2):409-15  
32  
33  
34  
35 15. Huang K-C, Lee M-S, Lee S-D, et al. Obesity in the Elderly and Its Relationship with  
36 Cardiovascular Risk Factors in Taiwan. *Obes Res* 2005;**13**(1):170-78 doi:  
37 10.1038/oby.2005.22[published Online First: Epub Date]].  
38  
39  
40  
41 16. Bergman RN, Stefanovski D, Buchanan TA, et al. A Better Index of Body Adiposity. *Obesity*  
42 (Silver Spring) 2011;**19**(5):1083-89  
43  
44  
45  
46 17. Dhaliwal SS, Welborn TA. Central obesity and multivariable cardiovascular risk as assessed by  
47 the Framingham prediction scores. *Am J Cardiol* 2009;**103**:1403-07  
48  
49  
50 18. Anderson KM, Odell PM, Wilson PW, et al. Cardiovascular disease risk profiles. *Am Heart J*  
51 1991;**121**(1 Part 2):293-98  
52  
53  
54 19. Conroy RM, Pyörälä K, Fitzgerald AP, et al. Estimation of ten-year risk of fatal cardiovascular  
55 disease in Europe: The SCORE project. *European Heart Journal* 2003;**24**(11):987-1003 doi:  
56 10.1016/s0195-668x(03)00114-3[published Online First: Epub Date]].  
57  
58  
59  
60

- 1  
2  
3 20. D'Agostino RB, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in  
4 primary care - The Framingham Heart Study. *Circulation* 2008;**117**(6):743-53  
5  
6  
7 21. Australian Risk Factor Prevalence Study Management Committee. Survey No. 3 1989. Canberra:  
8 National Heart Foundation of Australia and Australia Institute of Health, 1990.  
9  
10  
11 22. Boyle CA, Dobson AJ, Egger G, et al. Waist-to-hip ratios in Australia: A different picture of  
12 obesity. *Aust J Nutr Diet* 1993;**50**:57-64  
13  
14  
15 23. Alexander H, Dugdale A. Which waist-hip ratio? *Med J Aust* 1990;**153**(6):367-68  
16  
17  
18 24. Cooney MT, Dudina A, De Bacquer D, et al. How much does HDL cholesterol add to risk  
19 estimation? A report from the SCORE investigators. *European Journal of Cardiovascular*  
20 *Prevention & Rehabilitation* 2009;**16**(3):304-14 doi:  
21 10.1097/HJR.0b013e3283213140[published Online First: Epub Date].  
22  
23  
24  
25 25. Neil HAW, Perera R, Armitage JM, et al. Estimated 10-year cardiovascular risk in a British  
26 population: results of a national screening project. *Int J Clin Pract* 2008;**62**(9):1322-31  
27  
28  
29 26. Woodward M, Brindle P, Tunstall-Pedoe H. Adding social deprivation and family history to  
30 cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended  
31 Cohort (SHHEC). *Heart* 2007;**93**(2):172-76  
32  
33  
34  
35 27. Mosca L, Benjamin EJ, Berra K, et al. Effectiveness-Based Guidelines for the Prevention of  
36 Cardiovascular Disease in Women—2011 Update A Guideline From the American Heart  
37 Association. *J Am Coll Cardiol* 2011;**57**(12):1404-23  
38  
39  
40  
41 28. Genest J, McPherson R, Frohlich J, et al. 2009 Canadian Cardiovascular Society/Canadian  
42 guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular  
43 disease in the adult – 2009 recommendations. *Can J Cardiol* 2009;**25**(10):567-79  
44  
45  
46  
47 29. Goh LGH, Dhaliwal SS, Lee AH, et al. Utility of established cardiovascular disease risk score  
48 models for the 10-year prediction of disease outcomes in women. *Expert Rev Cardiovasc*  
49 *Ther* 2013;**11**(4):425-35  
50  
51  
52  
53  
54 30. Wittchen H-U, Balkau B, Massien C, et al. International Day for the Evaluation of Abdominal  
55 obesity: rationale and design of a primary care study on the prevalence of abdominal obesity  
56  
57  
58  
59  
60



- 1  
2  
3 and associated factors in 63 countries. *Eur Heart J Suppl* 2006;**8**(suppl B):B26-B33 doi:  
4 10.1093/eurheartj/sul005[published Online First: Epub Date].  
5  
6  
7 31. Balkau B, Deanfield JE, Despres JP, et al. International day for the evaluation of abdominal  
8 obesity (IDEA) - A study of waist circumference, cardiovascular disease, and diabetes  
9 mellitus in 168 000 primary care patients in 63 countries. *Circulation* 2007;**116**(17):1942-51  
10 doi: 10.1161/circulationaha.106.676379[published Online First: Epub Date].  
11  
12  
13 32. Winter Y, Rohrmann S, Linseisen J, et al. Contribution of Obesity and Abdominal Fat Mass to  
14 Risk of Stroke and Transient Ischemic Attacks. *Stroke* 2008;**39**(12):3145-51 doi:  
15 10.1161/strokeaha.108.523001[published Online First: Epub Date].  
16  
17  
18 33. Taylor AE, Ebrahim S, Ben-Shlomo Y, et al. Comparison of the associations of body mass index  
19 and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-  
20 cause mortality: a study using data from 4 UK cohorts. *The American Journal of Clinical*  
21 *Nutrition* 2010;**91**(3):547-56 doi: 10.3945/ajcn.2009.28757[published Online First: Epub  
22 Date]].  
23  
24  
25 34. van Dis I, Kromhout D, Geleijnse JM, et al. Body mass index and waist circumference predict  
26 both 10-year nonfatal and fatal cardiovascular disease risk: study conducted in 20 000 Dutch  
27 men and women aged 20–65 years. *European Journal of Cardiovascular Prevention &*  
28 *Rehabilitation* 2009;**16**(6):729-34 doi: 10.1097/HJR.0b013e328331dfc0[published Online  
29 First: Epub Date]].  
30  
31  
32 35. Berg AH, Scherer PE. Adipose Tissue, Inflammation, and Cardiovascular Disease. *Circulation*  
33 *Research* 2005;**96**(9):939-49 doi: 10.1161/01.res.0000163635.62927.34[published Online  
34 First: Epub Date]].  
35  
36  
37 36. Snijder MB, van Dam RM, Visser M, et al. What aspects of body fat are particularly hazardous  
38 and how do we measure them? *International Journal of Epidemiology* 2006;**35**(1):83-92 doi:  
39 10.1093/ije/dyi253[published Online First: Epub Date]].  
40  
41  
42 37. de Koning L, Merchant AT, Pogue J, et al. Waist circumference and waist-to-hip ratio as  
43 predictors of cardiovascular events: meta-regression analysis of prospective studies. *Eur Heart*  
44 *J* 2007;**28**(7):850-56  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60



- 1  
2  
3 38. Dalton M, Cameron AJ, Zimmet PZ, et al. Waist circumference, waist-hip ratio and body mass  
4 index and their correlation with cardiovascular disease risk factors in Australian adults.  
5 Journal of Internal Medicine 2003;**254**(6):555-63 doi: 10.1111/j.1365-  
6 2796.2003.01229.x[published Online First: Epub Date]].  
7  
8  
9  
10  
11 39. Antillon D, Towfighi A. No time to 'weight': the link between obesity and stroke in women.  
12 Women's Health 2011;**7**(4):453-63 doi: 10.2217/whe.11.36[published Online First: Epub  
13 Date]].  
14  
15  
16  
17 40. Pischon T, Boeing H, Hoffmann K, et al. General and Abdominal Adiposity and Risk of Death in  
18 Europe. N Engl J Med 2008;**359**(20):2105-20 doi: doi:10.1056/NEJMoa0801891[published  
19 Online First: Epub Date]].  
20  
21  
22  
23 41. Li C, Engstrom G, Hedblad B, et al. Sex differences in the relationships between BMI, WHR and  
24 incidence of cardiovascular disease: a population-based cohort study. International Journal of  
25 Obesity 2006;**30**(12):1775-81 doi: 10.1038/sj.ijo.0803339[published Online First: Epub  
26 Date]].  
27  
28  
29  
30  
31 42. Freiberg MS, Pencina MJ, D'Agostino RB, et al. BMI vs. Waist Circumference for Identifying  
32 Vascular Risk. Obesity 2008;**16**(2):463-69  
33  
34  
35  
36 43. DiPietro L, Katz LD, Nadel ER. Excess abdominal adiposity remains correlated with altered lipid  
37 concentrations in healthy older women. International journal of obesity and related metabolic  
38 disorders : journal of the International Association for the Study of Obesity 1999;**23**(4):432-  
39 36  
40  
41  
42  
43 44. Klein S, Allison DB, Heymsfield SB, et al. Waist Circumference and Cardiometabolic Risk: A  
44 Consensus Statement from Shaping America's Health: Association for Weight Management  
45 and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition;  
46 and the American Diabetes Association. Obesity 2007;**15**(5):1061-67 doi:  
47 10.1038/oby.2007.632[published Online First: Epub Date]].  
48  
49  
50  
51  
52  
53  
54 45. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in  
55 women. JAMA 1998;**280**(21):1843-48 doi: 10.1001/jama.280.21.1843[published Online  
56 First: Epub Date]].  
57  
58  
59  
60

- 1  
2  
3 46. Fogelholm M. Physical activity, fitness and fatness: relations to mortality, morbidity and disease  
4 risk factors. A systematic review. *Obesity Reviews* 2010;**11**(3):202-21 doi: 10.1111/j.1467-  
5 789X.2009.00653.x[published Online First: Epub Date]].  
6  
7  
8  
9 47. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Accuracy of body mass index in  
10 diagnosing obesity in the adult general population. *Int J Obes* 2008;**32**(6):959-66 doi:  
11 <http://www.nature.com/ijo/journal/v32/n6/suppinfo/ijo200811s1.html>[published  
12 Online First:  
13 Epub Date]].  
14  
15  
16  
17 48. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a  
18 WHO consultation. World Health Organ Tech Rep Ser: WHO, 2000.  
19  
20  
21 49. Deurenberg P, Yap M, van Staveren WA. Body mass index and percent body fat: a meta analysis  
22 among different ethnic groups. *International Journal of Obesity* 1998;**22**(12):1164-71  
23  
24  
25  
26 50. Welborn TA, Dhaliwal SS. Being correct about obesity. *Med J Aust* 2011;**194** (8 ):429-30  
27  
28  
29 51. The Emerging Risk Factors Collaboration. Separate and combined associations of body-mass  
30 index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58  
31 prospective studies. *The Lancet* 2011;**377**(9771):1085-95 doi:  
32 [http://dx.doi.org/10.1016/S0140-6736\(11\)60105-0](http://dx.doi.org/10.1016/S0140-6736(11)60105-0)[published Online First: Epub Date]].  
33  
34  
35  
36 52. Dijk SB, Takken T, Prinsen EC, et al. Different anthropometric adiposity measures and their  
37 association with cardiovascular disease risk factors: a meta-analysis. *Neth Heart J*  
38 2012;**20**(5):208-18 doi: 10.1007/s12471-011-0237-7[published Online First: Epub Date]].  
39  
40  
41  
42 53. Dobbelsteyn CJ, Joffres MR, MacLean DR, et al. A comparative evaluation of waist  
43 circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk  
44 factors. The Canadian Heart Health Surveys. *International Journal of Obesity* 2001;**25**:652-61  
45  
46  
47  
48 54. Pouliot M-C, Després J-P, Lemieux S, et al. Waist circumference and abdominal sagittal diameter:  
49 Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and  
50 related cardiovascular risk in men and women. *The American Journal of Cardiology*  
51 1994;**73**(7):460-68 doi: [http://dx.doi.org/10.1016/0002-9149\(94\)90676-9](http://dx.doi.org/10.1016/0002-9149(94)90676-9)[published  
52 Online  
53 First: Epub Date]].  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 55. Turcato E, Bosello O, Di Francesco V, et al. Waist circumference and abdominal sagittal diameter  
4 as surrogates of body fat distribution in the elderly: their relation with cardiovascular risk  
5 factors. *Int J Obes Relat Metab Disord* 2000;**24**(8):1005-10  
6  
7  
8  
9 56. Zhu S, Wang Z, Heshka S, et al. Waist circumference and obesity-associated risk factors among  
10 whites in the third National Health and Nutrition Examination Survey: clinical action  
11 thresholds. *The American Journal of Clinical Nutrition* 2002;**76**(4):743  
12  
13  
14  
15 57. Reeder BA, Senthilselvan A, Després JP, et al. The association of cardiovascular disease risk  
16 factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group.  
17 *CMAJ : Canadian Medical Association journal* 1997;**157 Suppl 1**:S39-S45  
18  
19  
20  
21 58. Lapidus L, Bengtsson C, Larsson B, et al. Distribution of adipose tissue and risk of cardiovascular  
22 disease and death: a 12 year follow up of participants in the population study of women in  
23 Gothenburg, Sweden. *BMJ* 1984;**289**(6454):1257-61 doi:  
24 10.1136/bmj.289.6454.1257[published Online First: Epub Date].  
25  
26  
27  
28  
29 59. Price GM, Uauy R, Breeze E, et al. Weight, shape, and mortality risk in older persons: elevated  
30 waist-hip ratio, not high body mass index, is associated with a greater risk of death. *The*  
31 *American Journal of Clinical Nutrition* 2006;**84**(2):449-60  
32  
33  
34  
35 60. Lu M, Ye W, Adami HO, et al. Prospective study of body size and risk for stroke amongst women  
36 below age 60. *Journal of Internal Medicine* 2006;**260**(5):442-50 doi: 10.1111/j.1365-  
37 2796.2006.01706.x[published Online First: Epub Date].  
38  
39  
40  
41 61. Dhaliwal SS, Welborn TA. Central obesity and cigarette smoking are key determinants of  
42 cardiovascular deaths in Australia: A public health perspective. *Preventive Medicine*  
43 2009;**49**(2-3):153-57  
44  
45  
46  
47 62. Welborn TA, Dhaliwal SS. Preferred clinical measures of central obesity for predicting mortality.  
48 *Eur J Clin Nutr* 2007;**61**(12):1373-79  
49  
50  
51 63. Welborn TA, Dhaliwal SS, Bennett SA. Waist-hip ratio is the dominant risk factor predicting  
52 cardiovascular death in Australia. *Med J Aust* 2003;**179**(11-12):580-85  
53  
54  
55  
56 64. Dhaliwal SS, Welborn TA. Measurement error and ethnic comparisons of measures of abdominal  
57 obesity. *Prev Med* 2009;**49**(2-3):148-52  
58  
59  
60

- 1  
2  
3 65. Goodman-Gruen D, Barrett-Connor E. Sex Differences in Measures of Body Fat and Body Fat  
4 Distribution in the Elderly. *Am J Epidemiol* 1996;**143**(9):898-906  
5  
6  
7 66. Ashwell M, Lejeune S. Ratio of waist circumference to height may be better indicator of need for  
8 weight management. *BMJ* 1996;**312**(7027):377 doi: 10.1136/bmj.312.7027.377[published  
9 Online First: Epub Date]].  
10  
11  
12  
13 67. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global  
14 indicator for health risks of obesity and how its use could simplify the international public  
15 health message on obesity. *International Journal of Food Sciences and Nutrition*  
16 2005;**56**(5):303-07 doi: doi:10.1080/09637480500195066[published Online First: Epub  
17 Date]].  
18  
19  
20  
21  
22  
23 68. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening  
24 tool for the prediction of cardiovascular disease and diabetes: 0.5 could be a suitable global  
25 boundary value. *Nutrition Research Reviews* 2010;**23**(02):247-69 doi:  
26 doi:10.1017/S0954422410000144[published Online First: Epub Date]].  
27  
28  
29  
30  
31 69. Schneider HJ, Glaesmer H, Klotsche J, et al. Accuracy of anthropometric indicators of obesity to  
32 predict cardiovascular risk. *J Clin Endocrinol Metab* 2007;**92**:589-94  
33  
34  
35  
36 70. Okosun IS, Liao Y, Rotimi CN, et al. Abdominal Adiposity and Clustering of Multiple Metabolic  
37 Syndrome in White, Black and Hispanic Americans. *Annals of Epidemiology*  
38 2000;**10**(5):263-70 doi: [http://dx.doi.org/10.1016/S1047-2797\(00\)00045-4](http://dx.doi.org/10.1016/S1047-2797(00)00045-4)[published  
39 Online  
40 First: Epub Date]].  
41  
42  
43  
44 71. Ho SC, Chen YM, Woo JLF, et al. Association between simple anthropometric indices and  
45 cardiovascular risk factors. *Int J Obes Relat Metab Disord* 2001;**25**(11):1689-97  
46  
47  
48 72. Jeong S-K, Seo M-W, Kim Y-H, et al. Does Waist Indicate Dyslipidemia better than BMI in  
49 Korean Adult Population? *J Korean Med Sci* 2005;**20**(1):7-12  
50  
51  
52 73. Yusuf S, Hawken S, Ôunpuu S, et al. Obesity and the risk of myocardial infarction in 27□000  
53 participants from 52 countries: a case-control study. *Lancet* 2005;**366**(9497):1640-49  
54  
55  
56  
57  
58  
59  
60

- 1  
2  
3 74. Moore SC. Waist versus weight—which matters more for mortality? The American Journal of  
4  
5 Clinical Nutrition 2009;**89**(4):1003-04 doi: 10.3945/ajcn.2009.27598[published Online First:  
6  
7 Epub Date]].  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30  
31  
32  
33  
34  
35  
36  
37  
38  
39  
40  
41  
42  
43  
44  
45  
46  
47  
48  
49  
50  
51  
52  
53  
54  
55  
56  
57  
58  
59  
60

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
<b>Introduction</b>			
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
<b>Methods</b>			
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.
<b>Results</b>			

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
<b>Discussion</b>			
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
<b>Other information</b>			
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](http://www.strobe-statement.org).