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Ethnicity and the association between anthropometric indices of obesity and cardiovascular risk in women: a cross-sectional study

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ABSTRACT

Objectives: The objectives of this study were to determine whether the cross-sectional associations between anthropometric obesity measures, body mass index (BMI), waist circumference (WC) and waist-to-hip ratio (WHR), and calculated 10-year cardiovascular disease (CVD) risk using the Framingham and general CVD risk score models, is the same for women of Australian, United Kingdom and Ireland, North European, South European and Asian descent. This study would investigate which anthropometric obesity measure is most predictive at identifying women at increased CVD risk in each ethnic group.

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

Setting: Population-based survey in Australia.

Participants: 4354 women aged 20-69 years with no previous history of heart disease, diabetes or stroke. Most participants were of Australian, United Kingdom and Ireland, North European, South European or Asian descent (97%).

Outcome measures: Anthropometric obesity measures that demonstrated stronger predictive ability of identifying women at increased CVD risk and likelihood of being above the promulgated treatment thresholds of various risk score models.

Results: Central obesity measures, WC, WHR, were better predictors of cardiovascular risk. WHR reported stronger predictive ability than WC and BMI in Caucasian women. In Northern European women, BMI was a better indicator of risk using the general CVD (10% threshold) and Framingham (20% threshold) risk score models. WC was the most predictive of cardiovascular risk among Asian women.

Conclusions: Ethnicity should be incorporated into CVD assessment. The same anthropometric obesity measure cannot be used across all ethnic groups. Ethnic-specific CVD prevention and treatment strategies need to be further developed.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study confirms that ethnicity influences the association between anthropometric obesity measures and CVD risk.
- Central obesity measures such as WC and WHR are better indicators of CVD risk compared to BMI across ethnic groups.
- The treatment threshold used for a risk score model affects the predictive ability of anthropometric obesity measures and the same cut-point may not be suitable across ethnic groups.
- It is a cross-sectional study of the Australian female population in 1989 and these results require confirmation from prospective studies.
- Due to a sample size of about 200 for the Asian population, different regions in Asia could not be compared.
- The CVD risk was estimated using risk score models in order to stratify individuals above and below the respective treatment thresholds and not actual CVD events.

INTRODUCTION

In Australia, approximately 63% of adults were overweight and obese in 2011-2012.[1] The proportion of the Australian population who are overweight and obese is expected to increase to approximately 66% in the next five years.[2] The National Health and Medical Research Council have developed Clinical Practice Guidelines for the Management of Overweight and Obesity for Adults, Adolescents and Children in Australia to provide guidance on assessing and managing obesity.[3]

Overweight and obesity affects all socioeconomic groups in Australia, but it is more prevalent in some ethnic groups.[4,5] Variations exist in the associations between excess weight and obesity-related conditions among different racial and ethnic groups. Ethnicity significantly affects the associations between anthropometric indices used to assess adiposity such as body mass index (BMI) and waist circumference (WC), and cardiovascular disease (CVD) risk factors.[6]

Previous epidemiological studies which assessed the associations between anthropometric indices of obesity and CVD were mostly conducted in Western societies.[7] It is thus not clear which anthropometric obesity measures are more strongly associated with CVD risk in different ethnic groups.[8] To address this, it is necessary to examine the relationship between anthropometric obesity measures and CVD risk by ethnicity and this has been proposed in previous studies as well.[9-11] These fundamental issues need to be addressed in order to recommend effective weight management and disease prevention strategies to reduce the burden associated with overweight and obesity in all population groups.

The objectives of this study were to determine whether the cross-sectional associations between anthropometric obesity measures (BMI, WC and waist-to-hip ratio) and calculated 10-year CVD risk using the Framingham and general CVD risk score models, is the same for women of Australian, United Kingdom and Ireland, North European, South European and Asian descent. This study would

investigate which anthropometric obesity measure is most predictive at identifying women at increased CVD risk in each ethnic group.

METHODS

Study participants

Participants were selected from the third Risk Factor Prevalence Study [12] conducted by the National Heart Foundation (NHF) of Australia in 1989. Residents on the federal electoral rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart, Darwin and Canberra were recruited for the Risk Factor Prevalence Study by systemic probability sampling of sex and 5-year age groups. Country of birth was used as a surrogate for ethnicity and grouped into regions .[12] We selected a representative sample of 4354 women aged 20-69 years with no previous history of heart disease, diabetes or stroke for analysis. Most participants were of Australian, United Kingdom and Ireland, North European, South European or Asian descent (97%).

Ethics statement

Ethical approval for the NHF data was obtained in advance from the Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth Privacy Commissioner. Participation was entirely voluntary. Those who participated signed an informed consent form.[12] Participant information was anonymized prior to analysis. This study was approved by the Human Research Ethics Committee at Curtin University, and complies with the Declaration of Helsinki.

Anthropometry

A single record of height (to the nearest centimetre) and weight (to the nearest 10^{th} of a kilogram) was taken in light summer clothes without shoes. BMI was calculated based on weight in kilograms divided by square of height in meters. Waist and hip circumferences were measured according to standardized methodologies.[13,14] The WC was measured from the front at the narrowest point between the rib cage and iliac crest after full expiration while the hip circumference was measured from the side at the maximal extension of buttocks by one observer using a metal tape. A second

observer recorded another set of measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of two measurements was taken at each site to the nearest centimetre. The waist-to-hip ratio (WHR) was calculated based on WC divided by the hip circumference. Information on demographic characteristics, medical conditions and smoking behaviour were collected. Mercury sphygmomanometers were used to record blood pressure levels on the right arm of seated participants five minutes apart. [12] Two readings were taken and the average was used in the analysis. Fasting blood samples were 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 cholesterol levels to be assayed.

Risk score models

The Framingham risk score model [15] predicts the 10-year CVD incidence. It was developed from the American Framingham Heart Study using participants aged 30-74 years who were free of CVD and cancer. Risk variables used to calculate the 10-year risk include, age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status, diabetes status and ECG-left ventricular hypertrophy.[15] The most commonly used treatment threshold for the Framingham model was 20%,[16] this denotes that an individual who has a risk score of more than 20% is considered to be at increased risk of experiencing a CVD event within the next 10 years and should be targeted for treatment.

Although the general CVD risk score model for predicting the 10-year CVD incidence and death was also developed based on data from the American Framingham Heart Study, it was developed from a larger cohort and consisted of participants without CVD only.[17] The general CVD risk score model contains these variables, age, total cholesterol level, HDL cholesterol level, SBP, current antihypertensive treatment, smoking status and diabetes status.[17] Treatment thresholds of 10% and 20% were reported for this model.[17,18]

Statistical analysis

Demographic and clinical characteristics of the sample were described using mean ± standard deviation for continuous variables, while counts (percentages) were used for categorical variables. Comparisons between means of continuous variables were conducted using Analysis of Variance, with age as a covariate, and with Bonferroni adjustment for multiple comparisons. Means with different superscripts were significantly different at the 5% level of significance. Non-parametric Spearman's rank correlation was used to assess the associations between BMI, WC and WHR and the 10-year predicted CVD risk calculated using Framingham and general CVD risk score models by ethnicity, due to the skewness in the distribution of risk variables. These measures were also converted to z-scores (original value subtracted by the mean and the result divided by the standard deviation) to represent the number of standard deviations above and below the mean of each anthropometric obesity measure for each individual. Logistic regression was used to assess the effects of each standardised obesity measure of being above the recommended treatment threshold for the respective risk score models (10% and 20%), as a result of a one standard deviation increment above the mean of each measure of obesity, by ethnicity. These effects were represented using odds-ratios and associated 95% confidence intervals. The predictive ability of these anthropometric obesity measures to identify individuals from different ethnic groups above the treatment threshold of 20% for the Framingham model for 10-year CVD incidence, and 10% and 20% for the general CVD risk score model for 10-year CVD incidence and death was assessed using the area under the receiver operating characteristic (ROC) curve. Ethnic-specific cut-off values of the anthropometric obesity measures and associated level of specificity to predict increased risk of CVD at 70% and 80% sensitivity were also presented. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses were performed with IBM SPSS Statistics Version 21.

RESULTS

The demographic and clinical characteristics of the multi-ethnic sample of 4354 women without heart disease, diabetes or stroke were presented in Table 1. Southern European women generally had

higher BMI, WC and WHR compared to other ethnic groups, and Asian women had lower anthropometric obesity measures.

All Spearman's rank correlations were statistically significant ($p < 0.0005$). Overall, WC was most strongly associated with the 10-year predicted risk calculated using the general CVD and Framingham risk score models across all ethnic groups except in European women (Table 2). BMI was more strongly correlated with CVD risk calculated using both models in Northern European women while WHR was more strongly correlated with the predicted risk in Southern European women.

The recommended treatment thresholds for the general CVD at 10% and 20%, and the Framingham risk score model at 20% were identified from a review of the literature. Table 3a presented the effects of a one standard deviation increment in BMI, WC and WHR above the mean on the likelihood of being above the recommended threshold in each ethnic group. Increase in anthropometric measurements was generally associated with an increased likelihood of being above the treatment thresholds for all models. A one standard deviation change in all obesity measures in Asian women did not have a significant effect on the CVD risk as calculated using the general CVD model both at the 10% and 20% threshold. BMI was not effective in predicting the likelihood of being above the treatment threshold across all models for Southern European women.

Table 3b summarised the results in Table 3a by presenting only statistically significant anthropometric obesity measures which increased the likelihood of individuals being above the treatment threshold, with measures of obesity ordered corresponding to odds-ratios, from the highest to lowest. WHR generally recorded higher odds-ratios than WC and BMI and increased the likelihood of individuals of different ethnicity being above the respective treatment thresholds of the respective models. Only BMI presented higher odds-ratios and increased the likelihood of Northern European women being indicated for treatment based on the predicted risk calculated from the general CVD model at the 10% threshold but not 20% threshold and Framingham model at the 20% threshold. WC recorded higher odds-ratios in Asian women using the Framingham model at the 20% threshold.

Higher area under the ROC curve, sensitivity and specificity were recorded with WHR in predicting the 10-year CVD risk calculated using the general CVD and Framingham risk score models across most ethnic groups (Table 4). The highest area under the ROC curve and specificity value at 80% sensitivity for WHR was 0.866 and 84.9% for Northern European women with the general CVD model at the 20% threshold.

In Northern European women, BMI was a better predictor of CVD risk calculated using the general CVD risk score model at the 10% threshold but not 20% threshold and the Framingham risk score model at the 20% threshold, compared with WC and WHR. WHR, however, was the better indicator of CVD risk using the general CVD risk score model with a 20% threshold, in Northern European women. In Asian women, WC reported consistently higher area under the ROC curve, sensitivity and specificity across all CVD models and thresholds. The area under the ROC curve values ranged from 0.630 to 0.688 and specificity values ranged from 50.5% to 53.3% at 80% sensitivity in Asian women. The cut-off values for BMI, WC and WHR were also presented in Table 4. A WHR value of 0.75 would indicate increased CVD risk for women from Australia and United Kingdom and Ireland while a value of 0.78 would indicate increased risk for Southern European women. In Asian women, a WC of 71.8 cm would indicate increased CVD risk. A BMI of 24.4 kg/m^2 would indicate increased risk in Northern European women. The diagnostic abilities of the various anthropometric obesity measures to identify women as being above the threshold and hence identified for treatment varies according to ethnic groups.

Table 1 Characteristics of the sample of 4354 women without heart disease, diabetes or stroke by ethnicity

	Statistics	Australia	UK and Ireland	Northern Europe	Southern Europe	Asia
Count	N	3329	416	180	234	195
Age (years)	Mean ± SD	41.9 ± 13.5	45.7 ± 12.5	49.0 ± 11.7	47.8 ± 10.6	40.5 ± 10.9
Current smoker (Yes)	n (%)	751 (22.6%)	91 (21.9%)	39 (21.7%)	32 (13.7%)	19 (9.7%)
Weight (kg)	Mean ± SD	65.4 ± 12.6[a]	65.2 ± 12.0[a]	66.5 ± 12.6[a]	66.9 ± 11.8[a]	58.6 ± 11.6[b]
Height (cm)	Mean ± SD	162.8 ± 6.0[a]	162.3 ± 6.2[a]	161.9 ± 6.2[a]	156.8 ± 6.1[b]	156.7 ± 5.7[b]
BMI (kg/m^2)	Mean ± SD	24.7 ± 4.8[b]	24.7 ± 4.2[b,c]	25.4 ± 4.6[b,d]	27.2 ± 4.4[a]	23.8 ± 4.3[c,d]
WC (cm)	Mean ± SD	75.9 ± 11.0[b]	76.2 ± 10.5[b]	78.4 ± 11.9[b]	81.2 ± 11.0[a]	73.9 ± 10.4[b]
WHR	Mean ± SD	0.76 ± 0.06[c]	0.76 ± 0.06[c]	0.77 ± 0.07[b,c]	0.79 ± 0.06[a]	0.77 ± 0.06[a,b]
SBP (mmHg)	Mean ± SD	122 ± 18[a]	123 ± 18[b,c]	126 ± 19[a,b,c]	127 ± 19[a,b]	116 ± 19[c]
HDL (mmol/L)	Mean ± SD	1.5 ± 0.4[a]	1.5 ± 0.4[a]	1.5 ± 0.4[a]	1.4 ± 0.3[b]	1.4 ± 0.4[a,b]
TC (mmol/L)	Mean ± SD	5.4 ± 1.1	5.6 ± 1.2	5.7 ± 1.3	5.7 ± 1.1	5.2 ± 1.0
Ratio: HDL to TC	Mean ± SD	3.9 ± 1.3[b]	4.0 ± 1.4[a,b]	4.0 ± 1.4[b]	4.3 ± 1.4[a]	3.9 ± 1.2[a,b]

[a,b,c,d] Means with different superscripts were significantly different at the 5% level of significance, after adjusting for age.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; SBP, systolic blood pressure; HDL, high-density lipoprotein cholesterol; TC, total cholesterol.

Table 2 Non-parametric correlations between anthropometric measurements of general and central obesity and 10-year predicted risk of CVD incidence and mortality by ethnicity in 4354 women

Ethnicity	BMI	WC	WHR
General CVD 10-year predicted risk for CVD incidence and death			
Australia	0.372	0.443	0.402
UK and Ireland	0.360	0.406	0.365
Northern Europe	0.504	0.462	0.435
Southern Europe	0.356	0.479	0.485
Asia	0.306	0.396	0.308
Overall	0.384	0.451	0.408
Framingham 10-year predicted risk for CVD incidence			
Australia	0.366	0.440	0.405
UK and Ireland	0.349	0.399	0.361
Northern Europe	0.500	0.464	0.445
Southern Europe	0.358	0.483	0.491
Asia	0.311	0.402	0.308
Overall	0.380	0.449	0.412

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

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

Table 3a Odds-ratios and associated 95% confidence intervals of being above the recommended treatment threshold for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity by ethnicity

Ethnicity	BMI	WC	WHR
General CVD 10-year predicted risk for CVD incidence and death (threshold = 10%) [18]			
Australia	1.69*** (1.55 - 1.85)	2.16*** (1.96 - 2.38)	2.36*** (2.13 - 2.62)
UK and Ireland	1.71*** (1.29 - 2.25)	1.86*** (1.42 - 2.43)	2.09*** (1.58 - 2.75)
Northern Europe	2.50*** (1.67 - 3.74)	2.28*** (1.61 - 3.24)	2.23*** (1.55 - 3.21)
Southern Europe	1.37 (0.97 - 1.94)	1.64** (1.18 - 2.28)	1.89** (1.32 - 2.70)
Asia	1.14 (0.62 - 2.09)	1.57 (0.97 - 2.56)	1.48 (0.88 - 2.47)
General CVD 10-year predicted risk for CVD incidence and death (threshold = 20%) [17,19]			
Australia	1.65*** (1.43 - 1.91)	2.07*** (1.78 - 2.41)	2.11*** (1.80 - 2.47)
UK and Ireland	1.12 (0.64 - 1.96)	1.22 (0.73 - 2.05)	1.68* (1.05 - 2.69)
Northern Europe	2.60** (1.44 - 4.70)	2.76*** (1.58 - 4.80)	3.23*** (1.74 - 5.97)
Southern Europe	1.17 (0.58 - 2.35)	1.77 (0.96 - 3.28)	2.15* (1.11 - 4.18)
Asia	0.96 (0.19 - 4.94)	1.15 (0.29 - 4.57)	0.71 (0.13 - 3.92)
Framingham 10-year predicted risk for CVD incidence (threshold = 20%) [20,21]			
Australia	1.67*** (1.52 - 1.82)	2.13*** (1.94 - 2.34)	2.37*** (2.14 - 2.63)
UK and Ireland	1.71*** (1.30 - 2.25)	1.88*** (1.45 - 2.45)	2.16*** (1.64 - 2.85)
Northern Europe	2.55*** (1.70 - 3.85)	2.27*** (1.59 - 3.23)	2.33*** (1.60 - 3.40)
Southern Europe	1.32 (0.94 - 1.84)	1.67** (1.21 - 2.30)	2.07*** (1.45 - 2.95)
Asia	1.65$^{\#}$ (0.99 - 2.76)	1.89** (1.20 - 2.97)	1.63* (1.02 - 2.61)

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, $^{\#}$ $p = 0.054$

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

Table 3b Significant anthropometric measurements of general and central obesity and 10-year predicted risk of CVD incidence and mortality by ethnicity

Ethnicity	General CVD (threshold = 10%)	General CVD (threshold = 20%)	Framingham (threshold = 20%)
Odds-ratio criterion			
Australia	WHR, WC, BMI	WHR, WC, BMI	WHR, WC, BMI
UK and Ireland	WHR, WC, BMI	WHR	WHR, WC, BMI
Northern Europe	BMI, WC, WHR	WHR, WC, BMI	BMI, WHR, WC
Southern Europe	WHR, WC	WHR	WHR, WC
Asia	WC[#]	-	WC, WHR

Each cell represents statistically significant anthropometric measures of obesity ordered corresponding to odds-ratios, from the highest to lowest. [#]p = 0.054

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

Table 4 Area under the curve and cut-points for anthropometric measurements of general and central obesity to predict increased risk of CVD using risk score models at different thresholds for various levels of sensitivity and specificity by ethnicity

	AUC	Sensitivity = 70%	Sensitivity = 80%
General CVD 10-year predicted risk for CVD incidence and death (threshold = 10%)			
Australia			
BMI	0.691 (0.666 , 0.716)	24.2 (60.1%)	23.0 (46.1%)
WC	0.750 (0.727 , 0.772)	77.3 (69.6%)	74.3 (57.9%)
WHR	0.759 (0.736 , 0.783)	0.77 (70.1%)	0.75 (58.0%)
UK and Ireland			
BMI	0.655 (0.584 , 0.726)	23.7 (50.6%)	22.8 (41.2%)
WC	0.676 (0.611 , 0.741)	75.3 (58.5%)	73.3 (51.2%)
WHR	0.729 (0.671 , 0.787)	0.77 (65.6%)	0.75 (52.4%)
Northern Europe			
BMI	0.770 (0.695 , 0.845)	25.8 (71.4%)	24.4 (58.7%)
WC	0.761 (0.682 , 0.840)	77.8 (66.7%)	75.3 (57.1%)
WHR	0.730 (0.642 , 0.817)	0.77 (59.5%)	0.75 (50.8%)
Southern Europe			
BMI	0.618 (0.536 , 0.699)	26.5 (52.8%)	25.5 (44.9%)
WC	0.686 (0.604 , 0.768)	81.8 (62.4%)	78.8 (53.4%)
WHR	0.702 (0.619 , 0.785)	0.80 (61.8%)	0.79 (57.9%)
Asia			
BMI	0.564 (0.411 , 0.717)	21.9 (38.2%)	21.8 (37.6%)
WC	0.651 (0.524 , 0.778)	73.3 (60.0%)	71.8 (52.4%)
WHR	0.614 (0.490 , 0.739)	0.76 (56.5%)	0.76 (54.7%)
General CVD 10-year predicted risk for CVD incidence and death (threshold = 20%)			
Australia			
BMI	0.725 (0.677 , 0.772)	25.5 (68.8%)	24.3 (58.1%)
WC	0.782 (0.743 , 0.821)	79.8 (72.3%)	77.8 (66.4%)
WHR	0.784 (0.745 , 0.823)	0.79 (76.3%)	0.77 (65.7%)
UK and Ireland			
BMI	0.550 (0.414 , 0.685)	23.0 (40.2%)	21.7 (25.4%)
WC	0.589 (0.472 , 0.706)	74.8 (52.2%)	73.8 (48.3%)
WHR	0.682 (0.572 , 0.791)	0.77 (61.3%)	0.75 (47.3%)
Northern Europe			
BMI	0.818 (0.727 , 0.908)	28.7 (82.4%)	26.3 (67.3%)
WC	0.861 (0.785 , 0.936)	85.3 (81.1%)	84.3 (79.2%)
WHR	0.866 (0.784 , 0.947)	0.84 (86.8%)	0.83 (84.9%)
Southern Europe			
BMI	0.578 (0.437 , 0.719)	26.8 (51.9%)	26.7 (50.9%)
WC	0.711 (0.562 , 0.859)	84.8 (69.6%)	84.8 (69.6%)
WHR	0.725 (0.553 , 0.897)	0.80 (62.1%)	0.79 (55.6%)
Asia			
BMI	0.555 (0.303 , 0.807)	25.4 (73.1%)	21.9 (37.9%)
WC	0.630 (0.466 , 0.795)	78.3 (73.6%)	71.8 (50.5%)

WHR	0.440 (0.306 , 0.573)	0.76 (52.2%)	0.74 (35.7%)
Framingham 10-year predicted risk for CVD incidence (threshold = 20%)			
Australia			
BMI	0.682 (0.657 , 0.707)	24.0 (57.9%)	22.9 (43.8%)
WC	0.745 (0.723 , 0.768)	76.8 (67.5%)	73.8 (55.8%)
WHR	0.759 (0.736 , 0.781)	0.77 (69.7%)	0.75 (58.1%)
UK and Ireland			
BMI	0.656 (0.586 , 0.726)	23.7 (50.6%)	22.5 (37.5%)
WC	0.682 (0.620 , 0.745)	75.3 (58.6%)	73.3 (51.8%)
WHR	0.735 (0.679 , 0.791)	0.77 (65.8%)	0.75 (54.2%)
Northern Europe			
BMI	0.783 (0.710 , 0.856)	26.3 (75.2%)	24.9 (65.1%)
WC	0.770 (0.691 , 0.850)	78.8 (71.3%)	76.3 (60.5%)
WHR	0.742 (0.652 , 0.832)	0.77 (62.8%)	0.75 (51.2%)
Southern Europe			
BMI	0.597 (0.514 , 0.680)	25.8 (47.1%)	25.1 (40.1%)
WC	0.680 (0.601 , 0.760)	80.8 (57.6%)	78.3 (53.5%)
WHR	0.711 (0.633 , 0.789)	0.79 (61.6%)	0.78 (51.7%)
Asia			
BMI	0.647 (0.524 , 0.770)	23.5 (55.1%)	21.9 (39.5%)
WC	0.688 (0.586 , 0.790)	73.3 (60.5%)	71.8 (53.3%)
WHR	0.645 (0.530 , 0.759)	0.76 (56.9%)	0.75 (44.3%)

Abbreviations: AUC, area under the curve; BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio.

DISCUSSION

Our study found anthropometric measures of central obesity (WC and WHR) to be better indicators of CVD risk as they measure ectopic body fat (fat stored in the abdominal region) which is associated with decreased glucose tolerance, reduced insulin sensitivity, adverse lipid profiles and other metabolic abnormalities which are risk factors for CVD and diabetes.[8] Stronger associations were also reported between WC and the 10-year predicted CVD risk calculated using the general CVD and Framingham risk score models across most ethnic groups, while WHR recorded higher odds-ratios than WC and BMI and increased the likelihood of women being above the respective treatment thresholds of the models. WHR also presented higher area under the ROC curve, sensitivity and specificity values. Our findings are consistent with previous studies which have shown that WC and WHR, measures of central adiposity, are superior to BMI in predicting CVD and other obesity-related risk.[22-25] WC has already been incorporated in the diagnosis of the metabolic syndrome, a cluster of risk factors for CVD and diabetes.[26]

WHR should also be incorporated into CVD risk assessment. Our study provided evidence that WHR is a better diagnostic predictor of CVD than BMI and WC. It is also suitable for assessing adiposity and CVD risk in multi-ethnic cohorts as it has low measurement error, high precision, and no bias over a wide range of ethnic groups.[27] Similar cut-off values for WHR could also be applied across ethnic groups; a value of 0.75 and 0.78 would indicate increased CVD risk for women of Australia and United Kingdom and Ireland, and Southern Europe descent, respectively. A study conducted on Latin Americans, non-Hispanic Whites and Blacks and Hispanics to estimate the accuracy and optimal cut-points for BMI, WC and WHR also found that a cut-point of 0.91 for WHR and 94 cm for WC could be used among women of different ethnicity to identify those at high coronary heart disease (CHD) risk.[28] WHR also reported the highest area under the ROC curve across all ethnic groups, ranging from 0.75 to 0.82.[28] It was also the most accurate measure to screen for high CHD risk individuals.[28] Another large case-control study of markers of obesity and myocardial infarction confirmed that WHR is a stronger indicator of myocardial infarction than BMI and increased the population attributable risk of obesity by more than 3-fold in all ethnic groups.[29] The superiority of

WHR over BMI and WC in predicting CVD risk is also demonstrated in prospective studies.[22,30-33]

The measurement of WHR, however, may pose some challenges. For example, it may be inappropriate to measure hip circumference in certain cultures but this can be overcome with same sex observers.[27] Some studies reported that WHR is imprecise while others reported that it is a precise measure.[27,30,34,35] The differing results could be related to the rigour of the techniques used, standardised techniques need to be adopted when measuring WHR.[27] It is not suitable for assessing central adiposity in the elderly [36] due to laxity of their abdominal muscles which would undermine the predictive value of abdominal circumferences.[37] In addition, WHR may remain constant during weight change and is not suitable for monitoring weight loss.[38] Finally, there are technical difficulties in accurately measuring the hip circumference of severely obese individuals (BMI $\geq$ 40 kg/m^2).[27] Measurements may be made in the supine position to overcome this problem.[27] In clinical settings, it may be more feasible to assess adiposity using WC while WHR could be measured in research studies as it is more informative.[30]

Although WHR was the best anthropometric obesity measure in relation to identifying individuals at increased CVD risk, this did not apply to Northern European women. BMI was a better indicator of CVD risk using the general CVD risk score model at the 10% threshold but not 20% threshold and the Framingham risk score model at the 20% threshold, with stronger correlations, higher odds-ratios, higher area under the ROC curves, sensitivity and specificity values presented, compared to WHR. At the 20% threshold of the general CVD risk score model, WHR was the better predictor of CVD risk in Northern European women compared to BMI. This indicates that the predictive ability of anthropometric measures of obesity vary with the treatment thresholds used for the respective risk score models and the same cut-point may not be suitable across ethnic groups.

WC was a better predictor of CVD risk among Asian women compared to BMI and WHR. This was consistent with the results of another cross-sectional population-based survey study on Chinese people

which reported that WC is the best predictor of CVD risk factors in women.[39] It was also the best marker of risk in a 6-year prospective study.[40] A small increment in WC predicted a substantial increase in CHD risk in the Chinese population.[40] A lower WC cut-off for Asians is necessary to avoid underestimating the population at risk.[41,42]

Our study has limitations. It is a cross-sectional study of the Australian female population in 1989 and these results require confirmation from prospective studies. Due to a sample size of about 200 for the Asian population, different regions in Asia could not be compared. Further, the CVD risk was estimated using risk score models in order to stratify individuals above and below the respective treatment thresholds and not actual CVD events.

CONCLUSIONS

Our study confirms that ethnicity influences the association between anthropometric obesity measures and CVD risk. Central obesity measures such as WC and WHR are better indicators of CVD risk compared to BMI across ethnic groups. WHR is the best anthropometric measure for predicting CVD risk in women except Northern European and Asian women. The treatment threshold used for a risk score model affects the predictive ability of anthropometric obesity measures and the same cut-point may not be suitable across ethnic groups.

It is important to incorporate ethnicity in CVD risk assessment. Prevention and treatment efforts should be tailored to meet the needs of each ethnic group.[43] Ethnic-specific CVD prevention strategies need to be developed to promote healthy eating and physical activity to curtail obesity. Continued population-based prospective research is necessary to elucidate the link between obesity and CVD by ethnicity.

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Contributors LGHG was involved in drafting the manuscript, interpretation of data and revising the manuscript critically for important intellectual content. SSD conceived the study, performed the analysis and data interpretation and revised the manuscript critically for important intellectual content. TAW participated in the study design, acquired the data and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

Competing interests None.

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

Data sharing statement No additional data are available.

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

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

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	8-9,11
		(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	8-9,11
		(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	8-16
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	8-16
		(*b*) Report category boundaries when continuous variables were categorized	8-16
		(*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	9,15-16
Discussion			
Key results	18	Summarise key results with reference to study objectives	17
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	19
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	19
Generalisability	21	Discuss the generalisability (external validity) of the study results	19
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	N.A.

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

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

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Ethnicity and the association between anthropometric indices of obesity and cardiovascular risk in women: a cross-sectional study

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Ethnicity and the association between anthropometric indices of obesity and cardiovascular risk in women: a cross-sectional study

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Ethnicity, obesity and cardiovascular risk

Keywords: Obesity, epidemiology, cardiovascular diseases, prevention and women

ABSTRACT

Objectives: The objectives of this study were to determine whether the cross-sectional associations between anthropometric obesity measures, body mass index (BMI), waist circumference (WC) and waist-to-hip ratio (WHR), and calculated 10-year cardiovascular disease (CVD) risk using the Framingham and general CVD risk score models, is the same for women of Australian, United Kingdom and Ireland, North European, South European and Asian descent. This study would investigate which anthropometric obesity measure is most predictive at identifying women at increased CVD risk in each ethnic group.

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

Setting: Population-based survey in Australia.

Participants: 4354 women aged 20-69 years with no previous history of heart disease, diabetes or stroke. Most participants were of Australian, United Kingdom and Ireland, North European, South European or Asian descent (97%).

Outcome measures: Anthropometric obesity measures that demonstrated stronger predictive ability of identifying women at increased CVD risk and likelihood of being above the promulgated treatment thresholds of various risk score models.

Results: Central obesity measures, WC, WHR, were better predictors of cardiovascular risk. WHR reported stronger predictive ability than WC and BMI in Caucasian women. In Northern European women, BMI was a better indicator of risk using the general CVD (10% threshold) and Framingham (20% threshold) risk score models. WC was the most predictive of cardiovascular risk among Asian women.

Conclusions: Ethnicity should be incorporated into CVD assessment. The same anthropometric obesity measure cannot be used across all ethnic groups. Ethnic-specific CVD prevention and treatment strategies need to be further developed.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study confirms that ethnicity influences the association between anthropometric obesity measures and CVD risk.
- Central obesity measures such as WC and WHR are better indicators of CVD risk compared to BMI across ethnic groups.
- The treatment threshold used for a risk score model affects the predictive ability of anthropometric obesity measures and the same cut-point may not be suitable across ethnic groups.
- It is a cross-sectional study of the Australian female population in 1989 and these results require confirmation from prospective studies.
- Due to a sample size of about 200 for the Asian population, different regions in Asia could not be compared.
- The CVD risk was estimated using risk score models in order to stratify individuals above and below the respective treatment thresholds and not actual CVD events.

INTRODUCTION

In Australia, approximately 63% of adults were overweight and obese in 2011-2012.[1] The proportion of the Australian population who are overweight and obese is expected to increase to approximately 66% in the next five years.[2] The National Health and Medical Research Council have developed Clinical Practice Guidelines for the Management of Overweight and Obesity for Adults, Adolescents and Children in Australia to provide guidance on assessing and managing obesity.[3]

Overweight and obesity affects all socioeconomic groups in Australia, but it is more prevalent in some ethnic groups.[4 5] Variations exist in the associations between excess weight and obesity-related conditions among different racial and ethnic groups. Ethnicity significantly affects the associations between anthropometric indices used to assess adiposity such as body mass index (BMI) and waist circumference (WC), and cardiovascular disease (CVD) risk factors.[6]

Previous epidemiological studies which assessed the associations between anthropometric indices of obesity and CVD were mostly conducted in Western societies.[7] It is thus not clear which anthropometric obesity measures are more strongly associated with CVD risk in different ethnic groups.[8] To address this, it is necessary to examine the relationship between anthropometric obesity measures and CVD risk by ethnicity and this has been proposed in previous studies as well.[9-11] These fundamental issues need to be addressed in order to recommend effective weight management and disease prevention strategies to reduce the burden associated with overweight and obesity in all population groups.

The objectives of this study were to determine whether the cross-sectional associations between anthropometric obesity measures (BMI, WC and waist-to-hip ratio) and calculated 10-year CVD risk using the Framingham and general CVD risk score models, is the same for women of Australian, United Kingdom and Ireland, North European, South European and Asian descent. This study would investigate which anthropometric obesity measure is most predictive at identifying women at increased CVD risk in each ethnic group.

METHODS

Study participants

Participants were selected from the third Risk Factor Prevalence Study[12] conducted by the National Heart Foundation (NHF) of Australia in 1989. Residents on the federal electoral rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart, Darwin and Canberra were recruited for the Risk Factor Prevalence Study by systemic probability sampling of sex and 5-year age groups. Complete data were available on 4727 women. Country of birth was used as a surrogate for ethnicity and grouped into regions.[12] Most participants were of Australian, United Kingdom and Ireland, North European, South European or Asian descent (97%). We selected a representative sample of 4354 women aged 20-69 years with no previous history of heart disease, diabetes or stroke for analysis. There were 3329 Australian women, 416 women from the United Kingdom and Ireland, 180 Northern European women, 234 Southern European women and 195 Asian women. Further details have been described in the third Risk Factor Prevalence Study and in a previous study.[12 13]

Ethics statement

Ethical approval for the NHF data was obtained in advance from the Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth Privacy Commissioner. Participation was entirely voluntary. Those who participated signed an informed consent form.[12] Participant information was anonymized prior to analysis. This study was approved by the Human Research Ethics Committee at Curtin University, and complies with the Declaration of Helsinki.

Anthropometry

A single record of height (to the nearest centimetre) and weight (to the nearest 10th of a kilogram) was taken in light summer clothes without shoes. BMI was calculated based on weight in kilograms divided by square of height in meters. Waist and hip circumferences were measured according to standardized methodologies by trained anthropometrists.[14 15] The WC was measured from the front at the narrowest point between the rib cage and iliac crest after full expiration while the hip

circumference (HC) was measured from the side at the maximal extension of buttocks by one observer using a metal tape. A second observer recorded another set of measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of two measurements was taken at each site to the nearest centimetre. The waist-to-hip ratio (WHR) was calculated based on WC divided by the HC. Information on demographic characteristics, medical conditions and smoking behaviour were collected. Mercury sphygmomanometers were used to record blood pressure levels on the right arm of seated participants five minutes apart.[12] Two readings were taken and the average was used in the analysis. Fasting blood samples were 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 cholesterol levels to be assayed.

Risk score models

The Framingham risk score model[16] predicts the 10-year CVD incidence. It was developed from the American Framingham Heart Study using participants aged 30-74 years who were free of CVD and cancer. Risk variables used to calculate the 10-year risk include, age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status, diabetes status and electrocardiogram-left ventricular hypertrophy (ECG-LVH).[16] The most commonly used treatment threshold for the Framingham model was 20%,[17] this denotes that an individual who has a risk score of more than 20% is considered to be at increased risk of experiencing a CVD event within the next 10 years and should be targeted for treatment.

Although the general CVD risk score model for predicting the 10-year CVD incidence and death was also developed based on data from the American Framingham Heart Study, it was developed from a larger cohort and consisted of participants without CVD only.[18] The general CVD risk score model contains these variables, age, total cholesterol level, HDL cholesterol level, SBP, current antihypertensive treatment, smoking status and diabetes status.[18] Treatment thresholds of 10% and 20% were reported for this model.[18 19]

Statistical analysis

Demographic and clinical characteristics of the sample were described using mean ± standard deviation for continuous variables, while counts (percentages) were used for categorical variables. Comparisons between means of continuous variables were conducted using Analysis of Variance, with age as a covariate, and with Bonferroni adjustment for multiple comparisons. Means with different superscripts were significantly different at the 5% level of significance. Non-parametric Spearman's rank correlation was used to assess the associations between BMI, WC and WHR and the 10-year predicted CVD risk calculated using Framingham and general CVD risk score models by ethnicity, due to the skewness in the distribution of risk variables. These measures were also converted to z-scores (original value subtracted by the mean and the result divided by the standard deviation) to represent the number of standard deviations above and below the mean of each anthropometric obesity measure for each individual. Logistic regression was used to assess the effects of each standardised obesity measure of being above the recommended treatment threshold for the respective risk score models (10% and 20%), as a result of a one standard deviation increment above the mean of each measure of obesity, by ethnicity. These effects were represented using odds-ratios and associated 95% confidence intervals. The predictive ability of these anthropometric obesity measures to identify individuals from different ethnic groups above the treatment threshold of 20% for the Framingham model for 10-year CVD incidence, and 10% and 20% for the general CVD risk score model for 10-year CVD incidence and death was assessed using the area under the receiver operating characteristic (ROC) curve. Ethnic-specific cut-off values of the anthropometric obesity measures and associated level of specificity to predict increased risk of CVD at 70% and 80% sensitivity were also presented. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses were performed with IBM SPSS Statistics Version 21.

RESULTS

The demographic and clinical characteristics of the multi-ethnic sample of 4354 women without heart disease, diabetes or stroke are presented in Table 1. Southern European women generally had higher

BMI, WC and WHR compared to other ethnic groups, and Asian women had lower anthropometric obesity measures.

All Spearman's rank correlations were statistically significant ($p < 0.0005$). Overall, WC appeared to have a stronger association with the 10-year predicted risk calculated using the general CVD and Framingham risk score models across all ethnic groups except in European women (Table 2). BMI appeared to be more associated with CVD risk calculated using both models in Northern European women while WHR was more associated with the predicted risk in Southern European women.

The recommended treatment thresholds for the general CVD risk score model at 10% and 20%, and the Framingham risk score model at 20% were identified from a review of the literature. Table 3a presents the effects of a one standard deviation increment in BMI, WC and WHR above the mean on the likelihood of being above the recommended threshold in each ethnic group. Increase in anthropometric measurements was generally associated with an increased likelihood of being above the treatment thresholds for all models. A one standard deviation change in all obesity measures in Asian women did not have a significant effect on the CVD risk as calculated using the general CVD model both at the 10% and 20% threshold. BMI was not effective in predicting the likelihood of being above the treatment threshold across all models for Southern European women.

Table 3b summarises the results in Table 3a by presenting only statistically significant anthropometric obesity measures which increase the likelihood of individuals being above the treatment threshold, with measures of obesity ordered corresponding to odds-ratios, from the highest to lowest. WHR generally recorded higher odds-ratios than WC and BMI and increased the likelihood of individuals of different ethnicity being above the respective treatment thresholds of the respective models. Only BMI presented higher odds-ratios and increased the likelihood of Northern European women being indicated for treatment based on the predicted risk calculated from the general CVD model at the 10% threshold but not 20% threshold and Framingham model at the 20% threshold. WC recorded higher odds-ratios in Asian women using the Framingham model at the 20% threshold.

Higher area under the ROC curve, sensitivity and specificity were recorded with WHR in predicting the 10-year CVD risk calculated using the general CVD and Framingham risk score models across most ethnic groups (Table 4). The highest area under the ROC curve and specificity value at 80% sensitivity for WHR was 0.866 and 84.9% for Northern European women with the general CVD model at the 20% threshold.

In Northern European women, BMI was a better predictor of CVD risk calculated using the general CVD risk score model at the 10% threshold but not 20% threshold and the Framingham risk score model at the 20% threshold, compared with WC and WHR. WHR, however, was the better indicator of CVD risk using the general CVD risk score model with a 20% threshold, in Northern European women. In Asian women, WC reported consistently higher area under the ROC curve, sensitivity and specificity across all CVD models and thresholds. The area under the ROC curve values ranged from 0.630 to 0.688 and specificity values ranged from 50.5% to 53.3% at 80% sensitivity in Asian women. The cut-off values for BMI, WC and WHR are also presented in Table 4. A WHR value of 0.75 would indicate increased CVD risk for women from Australia and United Kingdom and Ireland while a value of 0.78 would indicate increased risk for Southern European women. In Asian women, a WC of 71.8 cm would indicate increased CVD risk. A BMI of 24.4 kg/m^2 would indicate increased risk in Northern European women. The diagnostic abilities of the various anthropometric obesity measures to identify women as being above the threshold and hence identified for treatment varies according to ethnic groups.

Table 1 Characteristics of the sample of 4354 women without heart disease, diabetes or stroke by ethnicity

	Statistics	Australia	UK and Ireland	Northern Europe	Southern Europe	Asia
Count	N	3329	416	180	234	195
Age (years)	Mean ± SD	41.9 ± 13.5	45.7 ± 12.5	49.0 ± 11.7	47.8 ± 10.6	40.5 ± 10.9
Current smoker (Yes)	n (%)	751 (22.6%)	91 (21.9%)	39 (21.7%)	32 (13.7%)	19 (9.7%)
Weight (kg)	Mean ± SD	65.4 ± 12.6[a]	65.2 ± 12.0[a]	66.5 ± 12.6[a]	66.9 ± 11.8[a]	58.6 ± 11.6[b]
Height (cm)	Mean ± SD	162.8 ± 6.0[a]	162.3 ± 6.2[a]	161.9 ± 6.2[a]	156.8 ± 6.1[b]	156.7 ± 5.7[b]
BMI (kg/m^2)	Mean ± SD	24.7 ± 4.8[b]	24.7 ± 4.2[b,c]	25.4 ± 4.6[b,d]	27.2 ± 4.4[a]	23.8 ± 4.3[c,d]
WC (cm)	Mean ± SD	75.9 ± 11.0[b]	76.2 ± 10.5[b]	78.4 ± 11.9[b]	81.2 ± 11.0[a]	73.9 ± 10.4[b]
WHR	Mean ± SD	0.76 ± 0.06[c]	0.76 ± 0.06[c]	0.77 ± 0.07[b,c]	0.79 ± 0.06[a]	0.77 ± 0.06[a,b]
SBP (mmHg)	Mean ± SD	122 ± 18[a]	123 ± 18[b,c]	126 ± 19[a,b,c]	127 ± 19[a,b]	116 ± 19[c]
HDL (mmol/L)	Mean ± SD	1.5 ± 0.4[a]	1.5 ± 0.4[a]	1.5 ± 0.4[a]	1.4 ± 0.3[b]	1.4 ± 0.4[a,b]
TC (mmol/L)	Mean ± SD	5.4 ± 1.1	5.6 ± 1.2	5.7 ± 1.3	5.7 ± 1.1	5.2 ± 1.0
Ratio: HDL to TC	Mean ± SD	3.9 ± 1.3[b]	4.0 ± 1.4[a,b]	4.0 ± 1.4[b]	4.3 ± 1.4[a]	3.9 ± 1.2[a,b]

[a,b,c,d] Means with different superscripts were significantly different at the 5% level of significance, after adjusting for age.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; SBP, systolic blood pressure; HDL, high-density lipoprotein cholesterol; TC, total cholesterol.

Table 2 Non-parametric correlations between anthropometric measurements of general and central obesity and 10-year predicted risk of CVD incidence and mortality by ethnicity in 4354 women

Ethnicity	BMI	WC	WHR
General CVD 10-year predicted risk for CVD incidence and death			
Australia	0.372	0.443	0.402
UK and Ireland	0.360	0.406	0.365
Northern Europe	0.504	0.462	0.435
Southern Europe	0.356	0.479	0.485
Asia	0.306	0.396	0.308
Overall	0.384	0.451	0.408
Framingham 10-year predicted risk for CVD incidence			
Australia	0.366	0.440	0.405
UK and Ireland	0.349	0.399	0.361
Northern Europe	0.500	0.464	0.445
Southern Europe	0.358	0.483	0.491
Asia	0.311	0.402	0.308
Overall	0.380	0.449	0.412

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

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

Table 3a Odds-ratios and associated 95% confidence intervals of being above the recommended treatment threshold for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity by ethnicity

Ethnicity	BMI	WC	WHR
General CVD 10-year predicted risk for CVD incidence and death (threshold = 10%) [19]			
Australia	1.69*** (1.55 - 1.85)	2.16*** (1.96 - 2.38)	2.36*** (2.13 - 2.62)
UK and Ireland	1.71*** (1.29 - 2.25)	1.86*** (1.42 - 2.43)	2.09*** (1.58 - 2.75)
Northern Europe	2.50*** (1.67 - 3.74)	2.28*** (1.61 - 3.24)	2.23*** (1.55 - 3.21)
Southern Europe	1.37 (0.97 - 1.94)	1.64** (1.18 - 2.28)	1.89** (1.32 - 2.70)
Asia	1.14 (0.62 - 2.09)	1.57 (0.97 - 2.56)	1.48 (0.88 - 2.47)
General CVD 10-year predicted risk for CVD incidence and death (threshold = 20%) [18 20]			
Australia	1.65*** (1.43 - 1.91)	2.07*** (1.78 - 2.41)	2.11*** (1.80 - 2.47)
UK and Ireland	1.12 (0.64 - 1.96)	1.22 (0.73 - 2.05)	1.68* (1.05 - 2.69)
Northern Europe	2.60** (1.44 - 4.70)	2.76*** (1.58 - 4.80)	3.23*** (1.74 - 5.97)
Southern Europe	1.17 (0.58 - 2.35)	1.77 (0.96 - 3.28)	2.15* (1.11 - 4.18)
Asia	0.96 (0.19 - 4.94)	1.15 (0.29 - 4.57)	0.71 (0.13 - 3.92)
Framingham 10-year predicted risk for CVD incidence (threshold = 20%) [21 22]			
Australia	1.67*** (1.52 - 1.82)	2.13*** (1.94 - 2.34)	2.37*** (2.14 - 2.63)
UK and Ireland	1.71*** (1.30 - 2.25)	1.88*** (1.45 - 2.45)	2.16*** (1.64 - 2.85)
Northern Europe	2.55*** (1.70 - 3.85)	2.27*** (1.59 - 3.23)	2.33*** (1.60 - 3.40)
Southern Europe	1.32 (0.94 - 1.84)	1.67** (1.21 - 2.30)	2.07*** (1.45 - 2.95)
Asia	1.65[#] (0.99 - 2.76)	1.89** (1.20 - 2.97)	1.63* (1.02 - 2.61)

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, [#] $p = 0.054$

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

Table 3b Significant anthropometric measurements of general and central obesity and 10-year predicted risk of CVD incidence and mortality by ethnicity

Ethnicity	General CVD (threshold = 10%)	General CVD (threshold = 20%)	Framingham (threshold = 20%)
Odds-ratio criterion			
Australia	WHR, WC, BMI	WHR, WC, BMI	WHR, WC, BMI
UK and Ireland	WHR, WC, BMI	WHR	WHR, WC, BMI
Northern Europe	BMI, WC, WHR	WHR, WC, BMI	BMI, WHR, WC
Southern Europe	WHR, WC	WHR	WHR, WC
Asia	-	-	WC, WHR, BMI[#]

Each cell represents statistically significant anthropometric measures of obesity ordered corresponding to odds-ratios, from the highest to lowest. [#] $p = 0.054$

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

Table 4 Area under the curve and cut-points for anthropometric measurements of general and central obesity to predict increased risk of CVD using risk score models at different thresholds for various levels of sensitivity and specificity by ethnicity

	AUC	Sensitivity = 70%	Sensitivity = 80%
General CVD 10-year predicted risk for CVD incidence and death (threshold = 10%)			
Australia			
BMI	0.691 (0.666 , 0.716)	24.2 (60.1%)	23.0 (46.1%)
WC	0.750 (0.727 , 0.772)	77.3 (69.6%)	74.3 (57.9%)
WHR	0.759 (0.736 , 0.783)	0.77 (70.1%)	0.75 (58.0%)
UK and Ireland			
BMI	0.655 (0.584 , 0.726)	23.7 (50.6%)	22.8 (41.2%)
WC	0.676 (0.611 , 0.741)	75.3 (58.5%)	73.3 (51.2%)
WHR	0.729 (0.671 , 0.787)	0.77 (65.6%)	0.75 (52.4%)
Northern Europe			
BMI	0.770 (0.695 , 0.845)	25.8 (71.4%)	24.4 (58.7%)
WC	0.761 (0.682 , 0.840)	77.8 (66.7%)	75.3 (57.1%)
WHR	0.730 (0.642 , 0.817)	0.77 (59.5%)	0.75 (50.8%)
Southern Europe			
BMI	0.618 (0.536 , 0.699)	26.5 (52.8%)	25.5 (44.9%)
WC	0.686 (0.604 , 0.768)	81.8 (62.4%)	78.8 (53.4%)
WHR	0.702 (0.619 , 0.785)	0.80 (61.8%)	0.79 (57.9%)
Asia			
BMI	0.564 (0.411 , 0.717)	21.9 (38.2%)	21.8 (37.6%)
WC	0.651 (0.524 , 0.778)	73.3 (60.0%)	71.8 (52.4%)
WHR	0.614 (0.490 , 0.739)	0.76 (56.5%)	0.76 (54.7%)
General CVD 10-year predicted risk for CVD incidence and death (threshold = 20%)			
Australia			
BMI	0.725 (0.677 , 0.772)	25.5 (68.8%)	24.3 (58.1%)
WC	0.782 (0.743 , 0.821)	79.8 (72.3%)	77.8 (66.4%)
WHR	0.784 (0.745 , 0.823)	0.79 (76.3%)	0.77 (65.7%)
UK and Ireland			
BMI	0.550 (0.414 , 0.685)	23.0 (40.2%)	21.7 (25.4%)
WC	0.589 (0.472 , 0.706)	74.8 (52.2%)	73.8 (48.3%)
WHR	0.682 (0.572 , 0.791)	0.77 (61.3%)	0.75 (47.3%)
Northern Europe			
BMI	0.818 (0.727 , 0.908)	28.7 (82.4%)	26.3 (67.3%)
WC	0.861 (0.785 , 0.936)	85.3 (81.1%)	84.3 (79.2%)
WHR	0.866 (0.784 , 0.947)	0.84 (86.8%)	0.83 (84.9%)
Southern Europe			
BMI	0.578 (0.437 , 0.719)	26.8 (51.9%)	26.7 (50.9%)
WC	0.711 (0.562 , 0.859)	84.8 (69.6%)	84.8 (69.6%)
WHR	0.725 (0.553 , 0.897)	0.80 (62.1%)	0.79 (55.6%)
Asia			
BMI	0.555 (0.303 , 0.807)	25.4 (73.1%)	21.9 (37.9%)
WC	0.630 (0.466 , 0.795)	78.3 (73.6%)	71.8 (50.5%)

WHR	0.440 (0.306 , 0.573)	0.76 (52.2%)	0.74 (35.7%)
Framingham 10-year predicted risk for CVD incidence (threshold = 20%)			
Australia			
BMI	0.682 (0.657 , 0.707)	24.0 (57.9%)	22.9 (43.8%)
WC	0.745 (0.723 , 0.768)	76.8 (67.5%)	73.8 (55.8%)
WHR	0.759 (0.736 , 0.781)	0.77 (69.7%)	0.75 (58.1%)
UK and Ireland			
BMI	0.656 (0.586 , 0.726)	23.7 (50.6%)	22.5 (37.5%)
WC	0.682 (0.620 , 0.745)	75.3 (58.6%)	73.3 (51.8%)
WHR	0.735 (0.679 , 0.791)	0.77 (65.8%)	0.75 (54.2%)
Northern Europe			
BMI	0.783 (0.710 , 0.856)	26.3 (75.2%)	24.9 (65.1%)
WC	0.770 (0.691 , 0.850)	78.8 (71.3%)	76.3 (60.5%)
WHR	0.742 (0.652 , 0.832)	0.77 (62.8%)	0.75 (51.2%)
Southern Europe			
BMI	0.597 (0.514 , 0.680)	25.8 (47.1%)	25.1 (40.1%)
WC	0.680 (0.601 , 0.760)	80.8 (57.6%)	78.3 (53.5%)
WHR	0.711 (0.633 , 0.789)	0.79 (61.6%)	0.78 (51.7%)
Asia			
BMI	0.647 (0.524 , 0.770)	23.5 (55.1%)	21.9 (39.5%)
WC	0.688 (0.586 , 0.790)	73.3 (60.5%)	71.8 (53.3%)
WHR	0.645 (0.530 , 0.759)	0.76 (56.9%)	0.75 (44.3%)

Abbreviations: AUC, area under the curve; BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio.

DISCUSSION

Our study found anthropometric measures of central obesity (WC and WHR) to be better indicators of CVD risk as they measure ectopic body fat (fat stored in the abdominal region) which is associated with decreased glucose tolerance, reduced insulin sensitivity, adverse lipid profiles and other metabolic abnormalities which are risk factors for CVD and diabetes.[8] Stronger associations were also reported between WC and the 10-year predicted CVD risk calculated using the general CVD and Framingham risk score models compared with BMI and WHR across most ethnic groups, while WHR recorded higher odds-ratios than WC and BMI and increased the likelihood of women being above the respective treatment thresholds of the models. WHR also presented higher area under the ROC curve, sensitivity and specificity values. Our findings are consistent with previous studies which have shown that WC and WHR, measures of central adiposity, are superior to BMI in predicting CVD and other obesity-related risk.[23-26] WC has already been incorporated in the diagnosis of the metabolic syndrome, a cluster of risk factors for CVD and diabetes.[27]

WHR should also be incorporated into CVD risk assessment. Our study provided evidence that WHR is a better diagnostic predictor of CVD than BMI and WC. It is also suitable for assessing adiposity and CVD risk in multi-ethnic cohorts as it has low measurement error, high precision, and no bias over a wide range of ethnic groups.[13] Equivalence tests across ethnic groups showed WHR to be independent of ethnicity.[13] Similar cut-off values for WHR could also be applied across ethnic groups; a value of 0.75 and 0.78 would indicate increased CVD risk for women of Australia and United Kingdom and Ireland, and Southern Europe descent, respectively. A study conducted on Latin Americans, non-Hispanic Whites and Blacks and Hispanics to estimate the accuracy and optimal cut-points for BMI, WC and WHR also found that a cut-point of 0.91 for WHR and 94 cm for WC could be used among women of different ethnicity to identify those at high coronary heart disease (CHD) risk.[28] WHR also reported the highest area under the ROC curve across all ethnic groups, ranging from 0.75 to 0.82.[28] It was also the most accurate measure to screen for high CHD risk individuals.[28] Another large case-control study of markers of obesity and myocardial infarction confirmed that WHR is a stronger indicator of myocardial infarction than BMI and increased the population

attributable risk of obesity by more than 3-fold in all ethnic groups.[29] The superiority of WHR over BMI and WC in predicting CVD risk is also demonstrated in prospective studies.[23 30-33]

The measurement of WHR, however, may pose some challenges. For example, it may be inappropriate to measure HC in certain cultures but this can be overcome with same sex observers.[13] Some studies reported that WHR is imprecise while others reported that it is a precise measure.[13 30 34 35] The differing results could be related to the rigour of the techniques used, standardised techniques need to be adopted when measuring WHR.[13] A study which evaluated the precision of measuring WHR, WC and HC with comparison across ethnic groups using data from the third Risk Factor Prevalence Study found that the coefficients of variation were 0.91% for WHR, 0.78% for WC and 0.57% for HC, less than 1%, indicating good precision in females.[13] The measurement error was 0.02 for WHR, 1.66 cm for WC and 1.59 cm for HC between two successive measurements in females.[13] In addition, the absolute difference between two WHR measurements for females was not significantly associated with the size of the participants.[13] WHR is not suitable for assessing central adiposity in the elderly[36] due to laxity of their abdominal muscles which would undermine the predictive value of abdominal circumferences.[37] In addition, WHR may remain constant during weight change and is not suitable for monitoring weight loss.[38] Finally, there are technical difficulties in accurately measuring the HC of severely obese individuals (BMI $\geq$ 40 kg/m^2).[13] Measurements may be made in the supine position to overcome this problem.[13] In clinical settings, it may be more feasible to assess adiposity using WC while WHR could be measured in research studies as it is more informative.[30]

Although WHR was the best anthropometric obesity measure in relation to identifying individuals at increased CVD risk, this did not apply to Northern European women. BMI was a better indicator of CVD risk using the general CVD risk score model at the 10% threshold but not 20% threshold and the Framingham risk score model at the 20% threshold, with higher correlations, higher odds-ratios, higher area under the ROC curves, sensitivity and specificity values presented, compared with WHR. At the 20% threshold of the general CVD risk score model, WHR was the better predictor of CVD

risk in Northern European women compared to BMI. This indicates that the predictive ability of anthropometric measures of obesity vary with the treatment thresholds used for the respective risk score models and the same cut-point may not be suitable across ethnic groups.

WC was a better predictor of CVD risk among Asian women compared to BMI and WHR. This was consistent with the results of another cross-sectional population-based survey study on Chinese people which reported that WC is the best predictor of CVD risk factors in women.[39] It was also the best marker of risk in a 6-year prospective study.[40] A small increment in WC predicted a substantial increase in CHD risk in the Chinese population.[40] It has been suggested that ethnicity influences specific fat depots, possibly explaining the relationship between ethnicity, adiposity and CVD risk.[41] A lower WC cut-off for Asians is necessary to avoid underestimating the population at risk.[41 42]

Our study has limitations. It is a cross-sectional study of the Australian female population in 1989 and these results require confirmation from prospective studies. In addition, it is limited by the use of country of birth as a proxy for ethnicity.[43] Although country of birth is a good proxy for ethnicity in older age minority groups and is of intrinsic interest in distinguishing environmental and genetic differences, it is no longer an appropriate proxy as it does not consider the diversity of country of origin of the individual. The measurement or assignment of ethnicity is difficult and the way forward is possibly to enable people to identify themselves.[44] Due to a sample size of about 200 for the Asian population, different regions in Asia could not be compared. Menopausal status which is associated with increased central obesity has not been assessed in our study and has not been incorporated into these risk score models.[45] Further, the CVD risk was estimated using risk score models in order to stratify individuals above and below the respective treatment thresholds and not actual CVD events. Only the Framingham and general CVD risk score models were assessed in our study. Other risk score models were excluded either because they could not be determined due to requirement for variables not assessed in our study (QRISK)[46] or, due to low number of participants above the respective recommended treatment thresholds (SCORE)[47]. Finally, the 10-year CVD risk for young adults is very rarely elevated, even in the presence of significant risk factors.[48]

CONCLUSIONS

Our study confirms that ethnicity influences the association between anthropometric obesity measures and CVD risk. Central obesity measures such as WC and WHR are better indicators of CVD risk compared to BMI across ethnic groups. WHR is the best anthropometric measure for predicting CVD risk in women except Northern European and Asian women. The treatment threshold used for a risk score model affects the predictive ability of anthropometric obesity measures and the same cut-point may not be suitable across ethnic groups.

It is important to incorporate ethnicity in CVD risk assessment. Prevention and treatment efforts should be tailored to meet the needs of each ethnic group.[49] Ethnic-specific CVD prevention strategies need to be developed to promote healthy eating and physical activity to curtail obesity. Continued population-based prospective research is necessary to elucidate the link between obesity and CVD by ethnicity.

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Contributors LGHG was involved in drafting the manuscript, performing the analysis, interpretation of data and revising the manuscript critically for important intellectual content. SSD conceived the study, performed the analysis and data interpretation and revised the manuscript critically for important intellectual content. TAW participated in the study design, acquired the data and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

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Ethnicity and the association between anthropometric indices of obesity and cardiovascular risk in women: a cross-sectional study

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Ethnicity, obesity and cardiovascular risk

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ABSTRACT

Objectives: The objectives of this study were to determine whether the cross-sectional associations between anthropometric obesity measures, body mass index (BMI), waist circumference (WC) and waist-to-hip ratio (WHR), and calculated 10-year cardiovascular disease (CVD) risk using the Framingham and general CVD risk score models, is the same for women of Australian, United Kingdom and Ireland, North European, South European and Asian descent. This study would investigate which anthropometric obesity measure is most predictive at identifying women at increased CVD risk in each ethnic group.

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

Setting: Population-based survey in Australia.

Participants: 4354 women aged 20-69 years with no previous history of heart disease, diabetes or stroke. Most participants were of Australian, United Kingdom and Ireland, North European, South European or Asian descent (97%).

Outcome measures: Anthropometric obesity measures that demonstrated stronger predictive ability of identifying women at increased CVD risk and likelihood of being above the promulgated treatment thresholds of various risk score models.

Results: Central obesity measures, WC, WHR, were better predictors of cardiovascular risk. WHR reported stronger predictive ability than WC and BMI in Caucasian women. In Northern European women, BMI was a better indicator of risk using the general CVD (10% threshold) and Framingham (20% threshold) risk score models. WC was the most predictive of cardiovascular risk among Asian women.

Conclusions: Ethnicity should be incorporated into CVD assessment. The same anthropometric obesity measure cannot be used across all ethnic groups. Ethnic-specific CVD prevention and treatment strategies need to be further developed.

ARTICLE SUMMARY

Strengths and limitations of this study

- This study confirms that ethnicity influences the association between anthropometric obesity measures and CVD risk.
- Central obesity measures such as WC and WHR are better indicators of CVD risk compared to BMI across ethnic groups.
- The treatment threshold used for a risk score model affects the predictive ability of anthropometric obesity measures and the same cut-point may not be suitable across ethnic groups.
- It is a cross-sectional study of the Australian female population in 1989 and these results require confirmation from prospective studies.
- Due to a sample size of about 200 for the Asian population, different regions in Asia could not be compared.
- The CVD risk was estimated using risk score models in order to stratify individuals above and below the respective treatment thresholds and not actual CVD events.

INTRODUCTION

In Australia, approximately 63% of adults were overweight and obese in 2011-2012.[1] The proportion of the Australian population who are overweight and obese is expected to increase to approximately 66% in the next five years.[2] The National Health and Medical Research Council have developed Clinical Practice Guidelines for the Management of Overweight and Obesity for Adults, Adolescents and Children in Australia to provide guidance on assessing and managing obesity.[3]

Overweight and obesity affects all socioeconomic groups in Australia, but it is more prevalent in some ethnic groups.[4 5] Variations exist in the associations between excess weight and obesity-related conditions among different racial and ethnic groups. Ethnicity significantly affects the associations between anthropometric indices used to assess adiposity such as body mass index (BMI) and waist circumference (WC), and cardiovascular disease (CVD) risk factors.[6]

Previous epidemiological studies which assessed the associations between anthropometric indices of obesity and CVD were mostly conducted in Western societies.[7] It is thus not clear which anthropometric obesity measures are more strongly associated with CVD risk in different ethnic groups.[8] To address this, it is necessary to examine the relationship between anthropometric obesity measures and CVD risk by ethnicity and this has been proposed in previous studies as well.[9-11] These fundamental issues need to be addressed in order to recommend effective weight management and disease prevention strategies to reduce the burden associated with overweight and obesity in all population groups.

The objectives of this study were to determine whether the cross-sectional associations between anthropometric obesity measures (BMI, WC and waist-to-hip ratio) and calculated 10-year CVD risk using the Framingham and general CVD risk score models, is the same for women of Australian, United Kingdom and Ireland, North European, South European and Asian descent. This study would investigate which anthropometric obesity measure is most predictive at identifying women at increased CVD risk in each ethnic group.

METHODS

Study participants

Participants were selected from the third Risk Factor Prevalence Study[12] conducted by the National Heart Foundation (NHF) of Australia in 1989. Residents on the federal electoral rolls of December 1988 in North and South Sydney, Melbourne, Brisbane, Adelaide, Perth, Hobart, Darwin and Canberra were recruited for the Risk Factor Prevalence Study by systemic probability sampling of sex and 5-year age groups. Complete data were available on 4727 women. Country of birth was used as a surrogate for ethnicity and grouped into regions.[12] Most participants were of Australian, United Kingdom and Ireland, North European, South European or Asian descent (97%). We selected a representative sample of 4354 women aged 20-69 years with no previous history of heart disease, diabetes or stroke for analysis. There were 3329 Australian women, 416 women from the United Kingdom and Ireland, 180 Northern European women, 234 Southern European women and 195 Asian women. Further details have been described in the third Risk Factor Prevalence Study and in a previous study.[12 13]

Ethics statement

Ethical approval for the NHF data was obtained in advance from the Australian Institute of Health Interim Ethics Committee, after consultation with the Commonwealth Privacy Commissioner. Participation was entirely voluntary. Those who participated signed an informed consent form.[12] Participant information was anonymized prior to analysis. This study was approved by the Human Research Ethics Committee at Curtin University, and complies with the Declaration of Helsinki.

Anthropometry

A single record of height (to the nearest centimetre) and weight (to the nearest 10th of a kilogram) was taken in light summer clothes without shoes. BMI was calculated based on weight in kilograms divided by square of height in meters. Waist and hip circumferences were measured according to standardized methodologies by trained anthropometrists.[14 15] The WC was measured from the front at the narrowest point between the rib cage and iliac crest after full expiration while the hip

circumference (HC) was measured from the side at the maximal extension of buttocks by one observer using a metal tape. A second observer recorded another set of measurements and ensured that the metal tape was kept strictly horizontal at all times. The mean of two measurements was taken at each site to the nearest centimetre. The waist-to-hip ratio (WHR) was calculated based on WC divided by the HC. Information on demographic characteristics, medical conditions and smoking behaviour were collected. Mercury sphygmomanometers were used to record blood pressure levels on the right arm of seated participants five minutes apart.[12] Two readings were taken and the average was used in the analysis. Fasting blood samples were 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 cholesterol levels to be assayed.

Risk score models

The Framingham risk score model[16] predicts the 10-year CVD incidence. It was developed from the American Framingham Heart Study using participants aged 30-74 years who were free of CVD and cancer. Risk variables used to calculate the 10-year risk include, age, sex, systolic blood pressure (SBP), diastolic blood pressure, total cholesterol level, high-density lipoprotein (HDL) cholesterol level, smoking status, diabetes status and electrocardiogram-left ventricular hypertrophy (ECG-LVH).[16] The most commonly used treatment threshold for the Framingham model was 20%,[17] this denotes that an individual who has a risk score of more than 20% is considered to be at increased risk of experiencing a CVD event within the next 10 years and should be targeted for treatment.

Although the general CVD risk score model for predicting the 10-year CVD incidence and death was also developed based on data from the American Framingham Heart Study, it was developed from a larger cohort and consisted of participants without CVD only.[18] The general CVD risk score model contains these variables, age, total cholesterol level, HDL cholesterol level, SBP, current antihypertensive treatment, smoking status and diabetes status.[18] Treatment thresholds of 10% and 20% were reported for this model.[18 19]

Statistical analysis

Demographic and clinical characteristics of the sample were described using mean ± standard deviation for continuous variables, while counts (percentages) were used for categorical variables. Comparisons between means of continuous variables were conducted using Analysis of Variance, with age as a covariate, and with Bonferroni adjustment for multiple comparisons. Means with different superscripts were significantly different at the 5% level of significance. Non-parametric Spearman's rank correlation was used to assess the associations between BMI, WC and WHR and the 10-year predicted CVD risk calculated using Framingham and general CVD risk score models by ethnicity, due to the skewness in the distribution of risk variables. These measures were also converted to z-scores (original value subtracted by the mean and the result divided by the standard deviation) to represent the number of standard deviations above and below the mean of each anthropometric obesity measure for each individual. Logistic regression was used to assess the effects of each standardised obesity measure of being above the recommended treatment threshold for the respective risk score models (10% and 20%), as a result of a one standard deviation increment above the mean of each measure of obesity, by ethnicity. These effects were represented using odds-ratios and associated 95% confidence intervals. The predictive ability of these anthropometric obesity measures to identify individuals from different ethnic groups above the treatment threshold of 20% for the Framingham model for 10-year CVD incidence, and 10% and 20% for the general CVD risk score model for 10-year CVD incidence and death was assessed using the area under the receiver operating characteristic (ROC) curve. Ethnic-specific cut-off values of the anthropometric obesity measures and associated level of specificity to predict increased risk of CVD at 70% and 80% sensitivity were also presented. P-values of less than 0.05 were considered to be statistically significant. All statistical analyses were performed with IBM SPSS Statistics Version 21.

RESULTS

The demographic and clinical characteristics of the multi-ethnic sample of 4354 women without heart disease, diabetes or stroke are presented in Table 1. Southern European women generally had higher

BMI, WC and WHR compared to other ethnic groups, and Asian women had lower anthropometric obesity measures.

All Spearman's rank correlations were statistically significant ($p < 0.0005$). Overall, WC ~~was most~~ appeared to have a stronger association ~~strongly associated~~ with the 10-year predicted risk calculated using the general CVD and Framingham risk score models across all ethnic groups except in European women (Table 2). BMI ~~was~~ appeared to be more ~~strongly correlated~~associated with CVD risk calculated using both models in Northern European women while WHR was ~~more strongly correlated~~more associated with the predicted risk in Southern European women.

The recommended treatment thresholds for the general CVD risk score model at 10% and 20%, and the Framingham risk score model at 20% were identified from a review of the literature. Table 3a presents the effects of a one standard deviation increment in BMI, WC and WHR above the mean on the likelihood of being above the recommended threshold in each ethnic group. Increase in anthropometric measurements was generally associated with an increased likelihood of being above the treatment thresholds for all models. A one standard deviation change in all obesity measures in Asian women did not have a significant effect on the CVD risk as calculated using the general CVD model both at the 10% and 20% threshold. BMI was not effective in predicting the likelihood of being above the treatment threshold across all models for Southern European women.

Table 3b summarises the results in Table 3a by presenting only statistically significant anthropometric obesity measures which increase the likelihood of individuals being above the treatment threshold, with measures of obesity ordered corresponding to odds-ratios, from the highest to lowest. WHR generally recorded higher odds-ratios than WC and BMI and increased the likelihood of individuals of different ethnicity being above the respective treatment thresholds of the respective models. Only BMI presented higher odds-ratios and increased the likelihood of Northern European women being indicated for treatment based on the predicted risk calculated from the general CVD model at the 10%

threshold but not 20% threshold and Framingham model at the 20% threshold. WC recorded higher odds-ratios in Asian women using the Framingham model at the 20% threshold.

Higher area under the ROC curve, sensitivity and specificity were recorded with WHR in predicting the 10-year CVD risk calculated using the general CVD and Framingham risk score models across most ethnic groups (Table 4). The highest area under the ROC curve and specificity value at 80% sensitivity for WHR was 0.866 and 84.9% for Northern European women with the general CVD model at the 20% threshold.

In Northern European women, BMI was a better predictor of CVD risk calculated using the general CVD risk score model at the 10% threshold but not 20% threshold and the Framingham risk score model at the 20% threshold, compared with WC and WHR. WHR, however, was the better indicator of CVD risk using the general CVD risk score model with a 20% threshold, in Northern European women. In Asian women, WC reported consistently higher area under the ROC curve, sensitivity and specificity across all CVD models and thresholds. The area under the ROC curve values ranged from 0.630 to 0.688 and specificity values ranged from 50.5% to 53.3% at 80% sensitivity in Asian women. The cut-off values for BMI, WC and WHR are also presented in Table 4. A WHR value of 0.75 would indicate increased CVD risk for women from Australia and United Kingdom and Ireland while a value of 0.78 would indicate increased risk for Southern European women. In Asian women, a WC of 71.8 cm would indicate increased CVD risk. A BMI of 24.4 kg/m^2 would indicate increased risk in Northern European women. The diagnostic abilities of the various anthropometric obesity measures to identify women as being above the threshold and hence identified for treatment varies according to ethnic groups.

Table 1 Characteristics of the sample of 4354 women without heart disease, diabetes or stroke by ethnicity

	Statistics	Australia	UK and Ireland	Northern Europe	Southern Europe	Asia
Count	N	3329	416	180	234	195
Age (years)	Mean ± SD	41.9 ± 13.5	45.7 ± 12.5	49.0 ± 11.7	47.8 ± 10.6	40.5 ± 10.9
Current smoker (Yes)	n (%)	751 (22.6%)	91 (21.9%)	39 (21.7%)	32 (13.7%)	19 (9.7%)
Weight (kg)	Mean ± SD	65.4 ± 12.6[a]	65.2 ± 12.0[a]	66.5 ± 12.6[a]	66.9 ± 11.8[a]	58.6 ± 11.6[b]
Height (cm)	Mean ± SD	162.8 ± 6.0[a]	162.3 ± 6.2[a]	161.9 ± 6.2[a]	156.8 ± 6.1[b]	156.7 ± 5.7[b]
BMI (kg/m^2)	Mean ± SD	24.7 ± 4.8[b]	24.7 ± 4.2[b,c]	25.4 ± 4.6[b,d]	27.2 ± 4.4[a]	23.8 ± 4.3[c,d]
WC (cm)	Mean ± SD	75.9 ± 11.0[b]	76.2 ± 10.5[b]	78.4 ± 11.9[b]	81.2 ± 11.0[a]	73.9 ± 10.4[b]
WHR	Mean ± SD	0.76 ± 0.06[c]	0.76 ± 0.06[c]	0.77 ± 0.07[b,c]	0.79 ± 0.06[a]	0.77 ± 0.06[a,b]
SBP (mmHg)	Mean ± SD	122 ± 18[a]	123 ± 18[b,c]	126 ± 19[a,b,c]	127 ± 19[a,b]	116 ± 19[c]
HDL (mmol/L)	Mean ± SD	1.5 ± 0.4[a]	1.5 ± 0.4[a]	1.5 ± 0.4[a]	1.4 ± 0.3[b]	1.4 ± 0.4[a,b]
TC (mmol/L)	Mean ± SD	5.4 ± 1.1	5.6 ± 1.2	5.7 ± 1.3	5.7 ± 1.1	5.2 ± 1.0
Ratio: HDL to TC	Mean ± SD	3.9 ± 1.3[b]	4.0 ± 1.4[a,b]	4.0 ± 1.4[b]	4.3 ± 1.4[a]	3.9 ± 1.2[a,b]

[a,b,c,d] Means with different superscripts were significantly different at the 5% level of significance, after adjusting for age.

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; SBP, systolic blood pressure; HDL, high-density lipoprotein cholesterol; TC, total cholesterol.

Table 2 Non-parametric correlations between anthropometric measurements of general and central obesity and 10-year predicted risk of CVD incidence and mortality by ethnicity in 4354 women

Ethnicity	BMI	WC	WHR
General CVD 10-year predicted risk for CVD incidence and death			
Australia	0.372	0.443	0.402
UK and Ireland	0.360	0.406	0.365
Northern Europe	0.504	0.462	0.435
Southern Europe	0.356	0.479	0.485
Asia	0.306	0.396	0.308
Overall	0.384	0.451	0.408
Framingham 10-year predicted risk for CVD incidence			
Australia	0.366	0.440	0.405
UK and Ireland	0.349	0.399	0.361
Northern Europe	0.500	0.464	0.445
Southern Europe	0.358	0.483	0.491
Asia	0.311	0.402	0.308
Overall	0.380	0.449	0.412

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

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

Table 3a Odds-ratios and associated 95% confidence intervals of being above the recommended treatment threshold for various risk score models as a result of a 1 standard deviation increment above the mean for each anthropometric measure of obesity by ethnicity

Ethnicity	BMI	WC	WHR
General CVD 10-year predicted risk for CVD incidence and death (threshold = 10%) [19]			
Australia	1.69*** (1.55 - 1.85)	2.16*** (1.96 - 2.38)	2.36*** (2.13 - 2.62)
UK and Ireland	1.71*** (1.29 - 2.25)	1.86*** (1.42 - 2.43)	2.09*** (1.58 - 2.75)
Northern Europe	2.50*** (1.67 - 3.74)	2.28*** (1.61 - 3.24)	2.23*** (1.55 - 3.21)
Southern Europe	1.37 (0.97 - 1.94)	1.64** (1.18 - 2.28)	1.89** (1.32 - 2.70)
Asia	1.14 (0.62 - 2.09)	1.57 (0.97 - 2.56)	1.48 (0.88 - 2.47)
General CVD 10-year predicted risk for CVD incidence and death (threshold = 20%) [18 20]			
Australia	1.65*** (1.43 - 1.91)	2.07*** (1.78 - 2.41)	2.11*** (1.80 - 2.47)
UK and Ireland	1.12 (0.64 - 1.96)	1.22 (0.73 - 2.05)	1.68* (1.05 - 2.69)
Northern Europe	2.60** (1.44 - 4.70)	2.76*** (1.58 - 4.80)	3.23*** (1.74 - 5.97)
Southern Europe	1.17 (0.58 - 2.35)	1.77 (0.96 - 3.28)	2.15* (1.11 - 4.18)
Asia	0.96 (0.19 - 4.94)	1.15 (0.29 - 4.57)	0.71 (0.13 - 3.92)
Framingham 10-year predicted risk for CVD incidence (threshold = 20%) [21 22]			
Australia	1.67*** (1.52 - 1.82)	2.13*** (1.94 - 2.34)	2.37*** (2.14 - 2.63)
UK and Ireland	1.71*** (1.30 - 2.25)	1.88*** (1.45 - 2.45)	2.16*** (1.64 - 2.85)
Northern Europe	2.55*** (1.70 - 3.85)	2.27*** (1.59 - 3.23)	2.33*** (1.60 - 3.40)
Southern Europe	1.32 (0.94 - 1.84)	1.67** (1.21 - 2.30)	2.07*** (1.45 - 2.95)
Asia	1.65[#] (0.99 - 2.76)	1.89** (1.20 - 2.97)	1.63* (1.02 - 2.61)

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, [#] $p = 0.054$

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

Table 3b Significant anthropometric measurements of general and central obesity and 10-year predicted risk of CVD incidence and mortality by ethnicity

Ethnicity	General CVD (threshold = 10%)	General CVD (threshold = 20%)	Framingham (threshold = 20%)
Odds-ratio criterion			
Australia	WHR, WC, BMI	WHR, WC, BMI	WHR, WC, BMI
UK and Ireland	WHR, WC, BMI	WHR	WHR, WC, BMI
Northern Europe	BMI, WC, WHR	WHR, WC, BMI	BMI, WHR, WC
Southern Europe	WHR, WC	WHR	WHR, WC
Asia	-	-	WC, WHR, BMI[#]

Each cell represents statistically significant anthropometric measures of obesity ordered corresponding to odds-ratios, from the highest to lowest. [#] $p = 0.054$

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

Table 4 Area under the curve and cut-points for anthropometric measurements of general and central obesity to predict increased risk of CVD using risk score models at different thresholds for various levels of sensitivity and specificity by ethnicity

	AUC	Sensitivity = 70%	Sensitivity = 80%
General CVD 10-year predicted risk for CVD incidence and death (threshold = 10%)			
Australia			
BMI	0.691 (0.666 , 0.716)	24.2 (60.1%)	23.0 (46.1%)
WC	0.750 (0.727 , 0.772)	77.3 (69.6%)	74.3 (57.9%)
WHR	0.759 (0.736 , 0.783)	0.77 (70.1%)	0.75 (58.0%)
UK and Ireland			
BMI	0.655 (0.584 , 0.726)	23.7 (50.6%)	22.8 (41.2%)
WC	0.676 (0.611 , 0.741)	75.3 (58.5%)	73.3 (51.2%)
WHR	0.729 (0.671 , 0.787)	0.77 (65.6%)	0.75 (52.4%)
Northern Europe			
BMI	0.770 (0.695 , 0.845)	25.8 (71.4%)	24.4 (58.7%)
WC	0.761 (0.682 , 0.840)	77.8 (66.7%)	75.3 (57.1%)
WHR	0.730 (0.642 , 0.817)	0.77 (59.5%)	0.75 (50.8%)
Southern Europe			
BMI	0.618 (0.536 , 0.699)	26.5 (52.8%)	25.5 (44.9%)
WC	0.686 (0.604 , 0.768)	81.8 (62.4%)	78.8 (53.4%)
WHR	0.702 (0.619 , 0.785)	0.80 (61.8%)	0.79 (57.9%)
Asia			
BMI	0.564 (0.411 , 0.717)	21.9 (38.2%)	21.8 (37.6%)
WC	0.651 (0.524 , 0.778)	73.3 (60.0%)	71.8 (52.4%)
WHR	0.614 (0.490 , 0.739)	0.76 (56.5%)	0.76 (54.7%)
General CVD 10-year predicted risk for CVD incidence and death (threshold = 20%)			
Australia			
BMI	0.725 (0.677 , 0.772)	25.5 (68.8%)	24.3 (58.1%)
WC	0.782 (0.743 , 0.821)	79.8 (72.3%)	77.8 (66.4%)
WHR	0.784 (0.745 , 0.823)	0.79 (76.3%)	0.77 (65.7%)
UK and Ireland			
BMI	0.550 (0.414 , 0.685)	23.0 (40.2%)	21.7 (25.4%)
WC	0.589 (0.472 , 0.706)	74.8 (52.2%)	73.8 (48.3%)
WHR	0.682 (0.572 , 0.791)	0.77 (61.3%)	0.75 (47.3%)
Northern Europe			
BMI	0.818 (0.727 , 0.908)	28.7 (82.4%)	26.3 (67.3%)
WC	0.861 (0.785 , 0.936)	85.3 (81.1%)	84.3 (79.2%)
WHR	0.866 (0.784 , 0.947)	0.84 (86.8%)	0.83 (84.9%)
Southern Europe			
BMI	0.578 (0.437 , 0.719)	26.8 (51.9%)	26.7 (50.9%)
WC	0.711 (0.562 , 0.859)	84.8 (69.6%)	84.8 (69.6%)
WHR	0.725 (0.553 , 0.897)	0.80 (62.1%)	0.79 (55.6%)
Asia			
BMI	0.555 (0.303 , 0.807)	25.4 (73.1%)	21.9 (37.9%)
WC	0.630 (0.466 , 0.795)	78.3 (73.6%)	71.8 (50.5%)

WHR	0.440 (0.306 , 0.573)	0.76 (52.2%)	0.74 (35.7%)
Framingham 10-year predicted risk for CVD incidence (threshold = 20%)			
Australia			
BMI	0.682 (0.657 , 0.707)	24.0 (57.9%)	22.9 (43.8%)
WC	0.745 (0.723 , 0.768)	76.8 (67.5%)	73.8 (55.8%)
WHR	0.759 (0.736 , 0.781)	0.77 (69.7%)	0.75 (58.1%)
UK and Ireland			
BMI	0.656 (0.586 , 0.726)	23.7 (50.6%)	22.5 (37.5%)
WC	0.682 (0.620 , 0.745)	75.3 (58.6%)	73.3 (51.8%)
WHR	0.735 (0.679 , 0.791)	0.77 (65.8%)	0.75 (54.2%)
Northern Europe			
BMI	0.783 (0.710 , 0.856)	26.3 (75.2%)	24.9 (65.1%)
WC	0.770 (0.691 , 0.850)	78.8 (71.3%)	76.3 (60.5%)
WHR	0.742 (0.652 , 0.832)	0.77 (62.8%)	0.75 (51.2%)
Southern Europe			
BMI	0.597 (0.514 , 0.680)	25.8 (47.1%)	25.1 (40.1%)
WC	0.680 (0.601 , 0.760)	80.8 (57.6%)	78.3 (53.5%)
WHR	0.711 (0.633 , 0.789)	0.79 (61.6%)	0.78 (51.7%)
Asia			
BMI	0.647 (0.524 , 0.770)	23.5 (55.1%)	21.9 (39.5%)
WC	0.688 (0.586 , 0.790)	73.3 (60.5%)	71.8 (53.3%)
WHR	0.645 (0.530 , 0.759)	0.76 (56.9%)	0.75 (44.3%)

Abbreviations: AUC, area under the curve; BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio.

DISCUSSION

Our study found anthropometric measures of central obesity (WC and WHR) to be better indicators of CVD risk as they measure ectopic body fat (fat stored in the abdominal region) which is associated with decreased glucose tolerance, reduced insulin sensitivity, adverse lipid profiles and other metabolic abnormalities which are risk factors for CVD and diabetes.[8] Stronger associations were also reported between WC and the 10-year predicted CVD risk calculated using the general CVD and Framingham risk score models compared with BMI and WHR across most ethnic groups, while WHR recorded higher odds-ratios than WC and BMI and increased the likelihood of women being above the respective treatment thresholds of the models. WHR also presented higher area under the ROC curve, sensitivity and specificity values. Our findings are consistent with previous studies which have shown that WC and WHR, measures of central adiposity, are superior to BMI in predicting CVD and other obesity-related risk.[23-26] WC has already been incorporated in the diagnosis of the metabolic syndrome, a cluster of risk factors for CVD and diabetes.[27]

WHR should also be incorporated into CVD risk assessment. Our study provided evidence that WHR is a better diagnostic predictor of CVD than BMI and WC. It is also suitable for assessing adiposity and CVD risk in multi-ethnic cohorts as it has low measurement error, high precision, and no bias over a wide range of ethnic groups.[13] Equivalence tests across ethnic groups showed WHR to be independent of ethnicity.[13] Similar cut-off values for WHR could also be applied across ethnic groups; a value of 0.75 and 0.78 would indicate increased CVD risk for women of Australia and United Kingdom and Ireland, and Southern Europe descent, respectively. A study conducted on Latin Americans, non-Hispanic Whites and Blacks and Hispanics to estimate the accuracy and optimal cut-points for BMI, WC and WHR also found that a cut-point of 0.91 for WHR and 94 cm for WC could be used among women of different ethnicity to identify those at high coronary heart disease (CHD) risk.[28] WHR also reported the highest area under the ROC curve across all ethnic groups, ranging from 0.75 to 0.82.[28] It was also the most accurate measure to screen for high CHD risk individuals.[28] Another large case-control study of markers of obesity and myocardial infarction confirmed that WHR is a stronger indicator of myocardial infarction than BMI and increased the population

attributable risk of obesity by more than 3-fold in all ethnic groups.[29] The superiority of WHR over BMI and WC in predicting CVD risk is also demonstrated in prospective studies.[23 30-33]

The measurement of WHR, however, may pose some challenges. For example, it may be inappropriate to measure HC in certain cultures but this can be overcome with same sex observers.[13] Some studies reported that WHR is imprecise while others reported that it is a precise measure.[13 30 34 35] The differing results could be related to the rigour of the techniques used, standardised techniques need to be adopted when measuring WHR.[13] A study which evaluated the precision of measuring WHR, WC and HC with comparison across ethnic groups using data from the third Risk Factor Prevalence Study found that the coefficients of variation were 0.91% for WHR, 0.78% for WC and 0.57% for HC, less than 1%, indicating good precision in females.[13] The measurement error was 0.02 for WHR, 1.66 cm for WC and 1.59 cm for HC between two successive measurements in females.[13] In addition, the absolute difference between two WHR measurements for females was not significantly associated with the size of the participants.[13] WHR is not suitable for assessing central adiposity in the elderly[36] due to laxity of their abdominal muscles which would undermine the predictive value of abdominal circumferences.[37] In addition, WHR may remain constant during weight change and is not suitable for monitoring weight loss.[38] Finally, there are technical difficulties in accurately measuring the HC of severely obese individuals (BMI $\geq$ 40 kg/m^2).[13] Measurements may be made in the supine position to overcome this problem.[13] In clinical settings, it may be more feasible to assess adiposity using WC while WHR could be measured in research studies as it is more informative.[30]

Although WHR was the best anthropometric obesity measure in relation to identifying individuals at increased CVD risk, this did not apply to Northern European women. BMI was a better indicator of CVD risk using the general CVD risk score model at the 10% threshold but not 20% threshold and the Framingham risk score model at the 20% threshold, with ~~stronger~~ higher correlations, higher odds-ratios, higher area under the ROC curves, sensitivity and specificity values presented, compared with WHR. At the 20% threshold of the general CVD risk score model, WHR was the better predictor of

CVD risk in Northern European women compared to BMI. This indicates that the predictive ability of anthropometric measures of obesity vary with the treatment thresholds used for the respective risk score models and the same cut-point may not be suitable across ethnic groups.

WC was a better predictor of CVD risk among Asian women compared to BMI and WHR. This was consistent with the results of another cross-sectional population-based survey study on Chinese people which reported that WC is the best predictor of CVD risk factors in women.[39] It was also the best marker of risk in a 6-year prospective study.[40] A small increment in WC predicted a substantial increase in CHD risk in the Chinese population.[40] It has been suggested that ethnicity influences specific fat depots, possibly explaining the relationship between ethnicity, adiposity and CVD risk.[41] A lower WC cut-off for Asians is necessary to avoid underestimating the population at risk.[41 42]

Our study has limitations. It is a cross-sectional study of the Australian female population in 1989 and these results require confirmation from prospective studies. In addition, it is limited by the use of country of birth as a proxy for ethnicity.[43] Although country of birth is a good proxy for ethnicity in older age minority groups and is of intrinsic interest in distinguishing environmental and genetic differences, it is no longer an appropriate proxy as it does not consider the diversity of country of origin of the individual. The measurement or assignment of ethnicity is difficult and the way forward is possibly to enable people to identify themselves.[44] Due to a sample size of about 200 for the Asian population, different regions in Asia could not be compared. Menopausal status which is associated with increased central obesity has not been assessed in our study and has not been incorporated into these risk score models.[45] Further, the CVD risk was estimated using risk score models in order to stratify individuals above and below the respective treatment thresholds and not actual CVD events. Only the Framingham and general CVD risk score models were assessed in our study. Other risk score models were excluded either because they could not be determined due to requirement for variables not assessed in our study (QRISK)[46] or, due to low number of participants above the respective recommended treatment thresholds (SCORE)[47]. Finally, the 10-year CVD risk for young adults is very rarely elevated, even in the presence of significant risk factors.[48]

CONCLUSIONS

Our study confirms that ethnicity influences the association between anthropometric obesity measures and CVD risk. Central obesity measures such as WC and WHR are better indicators of CVD risk compared to BMI across ethnic groups. WHR is the best anthropometric measure for predicting CVD risk in women except Northern European and Asian women. The treatment threshold used for a risk score model affects the predictive ability of anthropometric obesity measures and the same cut-point may not be suitable across ethnic groups.

It is important to incorporate ethnicity in CVD risk assessment. Prevention and treatment efforts should be tailored to meet the needs of each ethnic group.[49] Ethnic-specific CVD prevention strategies need to be developed to promote healthy eating and physical activity to curtail obesity. Continued population-based prospective research is necessary to elucidate the link between obesity and CVD by ethnicity.

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Contributors LGHG was involved in drafting the manuscript, performing the analysis, interpretation of data and revising the manuscript critically for important intellectual content. SSD conceived the study, performed the analysis and data interpretation and revised the manuscript critically for important intellectual content. TAW participated in the study design, acquired the data and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

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

Data sharing statement No additional data are available.

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

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

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	8-10
		(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	8-9,11
		(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	8-16
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	8-16
		(*b*) Report category boundaries when continuous variables were categorized	8-16
		(*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	8-9,15-16
Discussion			
Key results	18	Summarise key results with reference to study objectives	17
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	20
Generalisability	21	Discuss the generalisability (external validity) of the study results	20
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	N.A.

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

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