# Relationships between BMI, waist circumference, hypertension and fasting glucose: Rethinking risk factors in Indigenous diabetes

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# Ethical Approval

The study was approved by the Community Council, the community's Health Action Committee, and the Human Research Ethics Committee at the Australian National University.

## Abstract

*Objective:* To determine whether the body mass index (BMI) threshold defined for obesity (30kg/m<sup>2</sup>) adequately reflects risk in an Aboriginal community with a high rate of Type 2 diabetes.

*Methods:* Data about five diabetes risk factors (age, BMI, waist circumference (WC), hypertension and family history) and fasting glucose (FG) were obtained from a random sample of 117 Aboriginal adults (62 women and 55 men) never diagnosed with diabetes. Linear regression between BMI, WC and FG, and sensitivity and specificity analyses in predicting elevated FG and hypertension were conducted.

*Results:* BMI≥30kg/m<sup>2</sup> and central obesity assessed by WC (women≥88cm; men≥102cm) were strongly and positively associated. Among women, central obesity was near universal, occurring at BMIs below the 'healthy' range of 20-25. WC was linearly associated with other diabetes risk factors. WC≥88cm was more sensitive but less specific than BMI≥30 in predicting elevated FG and hypertension among women, while BMI≥25 among men tended to be both more sensitive and more specific than both BMI≥30 and WC≥102cm.

*Conclusions:* In women, central obesity is a better predictor of diabetes and CVD risk than BMI≥30, which is not a reliable indicator. BMI≥25 was a good predictor in men.

*Implications:* BMI is a useful clinical tool to identify individuals at risk, but to be relevant the guidelines defining risk may need to be reduced for the Aboriginal population. For women, a BMI $\geq$ 25 could more adequately reflect risk, while the current WC of 88cm remains appropriate. For men, a reduction in both BMI to  $\geq$ 25 and WC to 90cm may better reflect diabetes and CVD risk.

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## Introduction

Type 2 diabetes is a serious health burden for many Australian Aboriginal communities; prevalence estimates are as high as 40% in some more urbanised populations [1, 2], more than five times the estimated rate for Australia as a whole [3]. In addition to the morbidity and higher mortality arising directly from diabetes, diabetes also contributes substantially to risk of cardiovascular disease (CVD) and renal disease, which are both significant causes of excess Aboriginal mortality [4]. Diabetes in Indigenous communities causes widespread disability, disrupts family and social life and reduces capacity for work.

There are five risk factors which are currently recognised by Australia's National Health and Medical Research Council (NHMRC) as contributing significantly and independently to diabetes risk (Table 1) [5]. These variables are in reality continuous, but determining certain cut-off points - or thresholds - can be useful in a clinical setting for identifying individuals at risk. With the exception of age, the NHMRC risk criteria are applied uniformly across population groups in Australia.

Table 1. NHMRC guidelines	for independent risk fact	tors relating to Type 2 diabetes
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Risk	Assessment criteria		
Obesity	Body mass index (BMI) ≥30kg/m <sup>2</sup>		
Central obesity	Waist circumference Women: waist ≥88cm; men: ≥102cm		
Hypertension	Systolic pressure >140mmHg and Diastolic Pressure >90mmHg		
Age	≥35 years (≥55 years for non-Aboriginal Australians)		
Family history	At least one first-degree relative has been diagnosed with Type 2 diabetes		

Source: NHMRC (2001) [5]

There is growing evidence, however, that thresholds such as these may not confer uniform risk across different population groups, and therefore applying such definitions across different populations is inappropriate. The appropriate application of body mass index (BMI), in particular, has been questioned in different populations internationally [6-10].

Despite links between obesity and diabetes, BMI does not relate to diabetes in all ethnic groups in the same way it does in people of primarily European descent; the threshold where BMI becomes a significant health risk may be lower or higher. For example, if a BMI of 30kg/m<sup>2</sup> is applied in Japan only 3% of the population are obese, which fails to reflect the rapid adoption of a more Western, energy-dense diet and more sedentary lifestyle in the last 60 years [10]. To adequately reflect the change in BMI that has occurred and the accompanying increase in diseases, Kanazawa and colleagues have recommended that the BMI threshold for obesity in Japan be reduced from 30 to 25 [10]. Similarly in China, the relationship between diabetes and BMI becomes apparent at much lower values than those widely

accepted as indicating obesity [8, 9]. In Asian populations, risk of diabetes at any given BMI may be greater than in Europeans [6].

Conversely, using BMI≥30 as a cut-off may also overestimate associated diabetes risk in some other populations. BMI has been found to be poorly predictive of cardiovascular and diabetes risk in Polynesians, suggesting either that BMI is not an adequate measure of adiposity or that adiposity is not as strongly associated with CVD as in other populations [7]. These apparent contradictions are because BMI does not necessarily reflect the proportion of the body that is fat, and, in particular, average body composition may vary by ethnic group. Lanham and colleagues, for example, found that for lower than national average BMIs, Chinese Australian women carried a higher percentage of fat [11].

Excess abdominal fat is a particular risk for diabetes, indicating insulin resistance arising from the release of free fatty acids [12, 13]. Fat distribution differs by ethnicity, carrying implications for the risk for diabetes and other chronic diseases. Some population groups, such as Indians, Japanese and Australian Aborigines, appear to have a greater propensity than others to store fat abdominally [10, 14-18] and may be at greater risk of disease than others with comparable BMI. In Australia, such a variation in typical adipose distribution has been noted for some years, especially among Aboriginal women [16, 19-22], and also more recently among Aboriginal men [23].

Relationships between 'obesity' (BMI≥30) as defined by current NHMRC guidelines and risk for diabetes and cardiovascular (CVD) should therefore be reassessed in Australia's Indigenous population, as these definitions may be inappropriate for this population. Examining the relationships between diabetes risk factors would illuminate three highly relevant clinical issues. First, whether excess central adiposity is associated with increases in diabetes and CVD risk factors (elevated fasting glucose and hypertension); second, whether the current definitions of general and central obesity are in line with each other in this setting; and, hence, third, whether there are perhaps more appropriate obesity criteria to use in this population to identify individuals at risk. Any assumptions about diabetes risk that use inappropriate thresholds for risk factors may be masking the real risk of diabetes and related morbidities in some communities; the current 'healthy' weight guidelines as recommended by NHMRC (BMI between 20 and 25 kg/m<sup>2</sup>) may need to be revised for Indigenous Australians.

# Methods

Data were collected between September and December 2000, in a large urbanised Aboriginal community (population approximately 1,100) in southeast Queensland, Australia. This community has a high prevalence of diagnosed diabetes (20% among people aged over 18 years) [2].

Participants were selected through random household sampling using a Kish grid. One adult per household who had never been diagnosed with diabetes was asked to take part (pregnant women were excluded). Data were collected on each of the five NHMRC risk factors for Type 2 diabetes [5]. Standing height was measured to the nearest half-centimetre. Weight was measured using digital bathroom-type scales to the nearest 100g. Waist circumference was obtained using a tailor's tape measure at

the level of the umbilicus and measured to the nearest centimetre. Blood pressure was taken after five minutes resting, using an automatic monitor on the participant's non-dominant side. Age in years was determined by the age the participant turned in the calendar year of the study. Positive family history was identified via questionnaire. Twelve-hour fasting blood glucose level was measured, to the nearest 0.1mmol/l using a personal glucose monitor that was calibrated regularly.

Relationships between modifiable risk factors were analysed using linear regression (SPSS for Windows v11.0). Sensitivity and specificity analyses of each modifiable risk factor in predicting elevated fasting glucose and hypertension were also conducted. The results were assessed to determine whether current NHMRC clinical guidelines are appropriate in this population.

## Results

Sixty-two women (aged 18-66, mean = 31.8 years) and 55 men (aged 19-65, mean = 34.5 years) who had never been diagnosed with diabetes took part in the study (response rate 74%). The prevalence of diabetes risk factors among this random sample was extremely high (Table 2). Forty per cent of women and 25% of men had three or more diabetes risk factors. Fasting blood glucose was elevated above the 'normal' level of 5.5mmol in 35% of women and 51% of men (Table 3).

Independent risk factor	Women %( <i>n=</i> 62)	Men %( <i>n</i> =55)
Age ≥35 years	35.5 (22)	45.5 (25)
Overweight or obese: BMI $\geq$ 25 kg/m <sup>2</sup>	59.7 (37)	54.5 (30)
Obesity: BMI ≥30 kg/m²	38.7 (24)	23.6 (13)

72.6 (45)

11.3 (7)

22.6 (14)

8.1 (5)

71.9 (44)

# Table 2. Prevalence of independent diabetes risk factors among adults never diagnosed with diabetes

Family history <sup>c</sup> <sup>a</sup> For women, waist ≥88cm; for men, ≥102cm.

Central Obesity<sup>a</sup>

Systolic blood pressure >140mmHg

Diastolic blood pressure >90mmHg

Systolic and diastolic hypertension <sup>b</sup>

<sup>b</sup> Systolic >140mmHg and diastolic >90mmHg.

<sup>c</sup> Reported that one or more members of their immediate family (first degree relatives: includes parents, offspring, siblings – both full-siblings and half-siblings) had been diagnosed with diabetes

29.1 (16)

27.3 (15)

34.5 (19)

18.2 (10)

45.5 (25)

Fasting glucose	Women %( <i>n</i> =59) <sup>a</sup>	Men %( <i>n</i> =48) <sup>a</sup>
≥7mmol <sup>b</sup>	4.8 (3)	12.7 (7)
5.5-6.9mmol <sup>c</sup>	30.6 (19)	38.2 (21)
<5.5mmol <sup>d</sup>	59.7 (37)	36.4(20)

#### Table 3. Fasting blood glucose levels. Categories from NHMRC

Source: NHMRC (2001) [5]

<sup>a</sup> Fasting glucose was not obtained for three women and seven men.

<sup>b</sup> Diabetes likely.

<sup>c</sup> Diabetes status uncertain.

<sup>d</sup> Diabetes unlikely.

Waist circumference increased linearly with BMI among both women and men (Figure 1), as might be expected. Most women surveyed (73%) had a waist circumference that placed them in the centrally obese category (WC≥88cm), including three out of ten women with BMIs below the 'healthy' range (<20). Overall, nearly one-half of the women with BMIs under 25 were centrally obese under current guidelines. This indicates a dominant pattern of centrally distributed fat even among those whose BMI indicated normal or less than normal bodyweight under current guidelines.

Above a BMI of 25 – the current threshold defining overweight – all women in the present study were centrally obese and might therefore be at increased risk of diabetes and cardiovascular disease. This was not the case among men, as central obesity (WC $\geq$ 102cm) commenced only in the overweight range of 25 to 30, and all men with a BMI over 32 were centrally obese (Figure 1).



Figure 1. Scatter plot of waist circumference and BMI showing linear regression line (mean and 95% confidence interval) for women and men. Horizontal reference line (dotted) is set at 88cm for female waist circumference and 102cm for men, the accepted thresholds for central obesity; filled markers indicate the presence of central obesity, unfilled markers indicate its absence. The vertical reference

lines (dotted) enclose the range of BMIs where central obesity was found to be both present and absent (17.5 to 24 for women, 28 to 32 for men).

#### Linear relationships between risk factors

Waist circumference was positively and significantly associated with increasing blood pressure and fasting blood glucose level among women (linear regression: systolic p=0.018, diastolic p=0.002, fasting glucose p=0.003) (Figure 2). These relationships were not as strong for BMI, so that central fat distribution was a stronger predictor of other risk factors than was overall body mass. The waist circumference threshold of 88cm for women appears to be appropriate given that (with one exception for fasting glucose) the thresholds for blood pressure and fasting glucose level were surpassed only by those who had a waist measurement above 88cm.

The relationships between risk factors appear to be more complex among men than among women. Despite an overall increase in both systolic and diastolic blood pressure and fasting blood glucose with waist circumference (Figure 2), these relationships were not significant (p=0.137 and 0.275 respectively). The only significant association was found for fasting glucose (p=0.017), and even this association was fairly weak (Rsq=0.1169). Hypertension and elevated fasting glucose were also apparent at low waist circumferences, which may suggest that 102cm is too high a definition of central obesity for this group of men.



Figure 2. Scatter plot of systolic and diastolic blood pressure and fasting blood glucose for waist circumference showing linear regression line (mean and 95% confidence interval). Reference lines (dotted) are at 88cm for female and 102cm for male waist circumference, and at 140mmHg for systolic pressure, 90mmHg for diastolic pressure, and 5.5mmol/l for fasting glucose. Filled markers indicate the presence of central obesity, unfilled markers indicate its absence.

## Sensitivity and specificity analyses

Central obesity among women was a highly sensitive predictor for elevated fasting blood glucose (both at diabetes diagnostic level of  $\geq$ 7mmol and elevated 'at risk' level  $\geq$ 5mmol) (Figure 3). It was, however, not a very specific predictor, leading to a high proportion of 'false positives'. Obesity was more specific (fewer false positives) but less sensitive. Obesity was a poor predictor of hypertension in women (low sensitivity, moderate specificity), while central obesity was highly sensitive but again yielded a high false positive rate. As a predictor of hypertension, overweight approached central obesity for sensitivity, and was more specific than both central obesity and general obesity. All measures had fairly low positive predictive values, but high negative predictive values, with overweight and central obesity performing better than obesity. A BMI of 25 or greater was a better predictor of central obesity than a BMI of 30 or more (Figure 4), having both greater sensitivity and negative predictive value.

Among men, overweight was more sensitive in predicting elevated fasting glucose and hypertension, but less specific than both obesity and central obesity. Overweight also had higher negative predictive value than obesity and central obesity, but lower positive predictive value than central obesity for hypertension and both obesity and central obesity for elevated fasting glucose. A reduced waist circumference threshold (≥90cm) in men was much more sensitive and generally had higher negative predictive value than the current central obesity threshold in predicting elevated blood glucose and hypertension, but was less specific and had slightly lower positive predictive value (Table 4).



Figure 3. Sensitivity and specificity analyses comparing current guidelines marking obesity (BMI ≥30), overweight (BMI ≥25) and central obesity (waist circumference ≥88cm for women and ≥102cm for men) in predicting elevated blood glucose and hypertension.



Figure 4. Sensitivity and specificity analyses comparing current thresholds marking obesity (BMI ≥30) and overweight (BMI ≥25) in predicting central obesity (waist circumference (≥88cm) in women.

	Fasting glucose ≥7mmol	Fasting glucose≥5.5mol	Systolic hypertension	Diastolic hypertension	
Sensitivity	1.00	0.93	0.85	0.89	
Specificity	0.24	0.40	0.21	0.26	
Positive predictive value	0.18	0.68	0.30	0.46	
Negative predictive value	1.00	0.80	0.78	0.78	

Table 4. Sensitivity analyses for waist circumference ≥90cm in predicting elevated fasting glucose and hypertension among men

The unmodifiable risks, age  $\geq$ 35 years and positive family history of diabetes, were not significantly associated with either waist circumference or BMI among women or men (Table 5), and were not influencing the observed relationships between these and other diabetes risks. Younger women had lower BMIs than the older women, but their waist circumferences were greater. These differences were not significant, but raise questions about a possible secular trend of increasing central obesity, producing a more 'at risk' body shape in younger women.

		Women			Men			
	Age (years) Family history		Age (years)		Family history			
	≥35	<35	Yes	No	≥35	<35	Yes	No
Waist circumference	106.90 (13.1)	110.17 (18.8)	103.79 (17.7)	99.76 (16.0)	101.48 (17.2)	98.76 (14.3)	101.55 (15.4)	98.81 (16.1)
(cm) (SD)	p=0.162		p=0.424		p=0.554		p=0.552	
BMI (kg/m²)	30.48 (6.6)	28.51 (9.2)	29.90 (8.5)	27.40 (7.9)	27.69 (6.4)	27.13 (5.0)	27.40 (5.5)	27.39 (5.9)
(50)	p=0	.396	p=0	.302	p=0.	.736	p=0.	.997

Table 5. Means (SD) and significance levels (independent <i>t</i> -test) for waist
circumference and BMI for age and family history

# **Conclusions and implications**

The positive relationship between BMI and waist circumference was linear and strong among both women and men. These variables are in reality continuous, but thresholds are useful in a clinical setting in identifying individuals at risk. Many women with BMIs conventionally considered normal, or even underweight, were found to be centrally obese by current NHMRC thresholds, and all women with a BMI of 25 or greater were centrally obese. Both BMI≥25 and WC≥88cm were found to be fairly good predictors of elevated fasting glucose and hypertension in women. Among men, BMI ≥25 and WC ≥90cm were fairly good predictors of elevated FG and hypertension.

Given the relationship between central obesity and BMI, either the risk threshold for central obesity for women in this population could be raised, or the threshold for obesity could be lowered to more adequately reflect diabetes and CVD risk. As WC≥88cm was more sensitive than BMI≥30 in predicting elevated fasting glucose and hypertension, reducing the BMI threshold rather than increasing the waist circumference threshold is a consideration that should be investigated further. The threshold for overweight, BMI≥25, was not only strongly predictive of central obesity but was both more sensitive than BMI≥30 and more specific than central obesity in predicting elevated fasting glucose and hypertension.

Among men, the relationship between BMI and waist circumference suggests that BMI $\geq$ 30 appears to adequately reflect central obesity when the waist circumference threshold is  $\geq$ 102cm. BMIs currently regarded as 'overweight' included both those with and without central obesity, while 'obese' adequately predicted those with central obesity. However, the current waist circumference threshold may be set too high, with  $\geq$ 90cm being a better predictor of elevated fasting glucose and hypertension, and this was associated with BMIs  $\geq$ 25.

The relationships between risk factors are complex, and no single one is sufficient to assess diabetes risk. Linear regressions and sensitivity analyses suggest, however,

that both WC≥88cm and BMI≥25 may be appropriate first stage screening tools for women, and BMI≥25 and possibly WC≥90cm might be more useful for men in this population. While they do yield a high proportion of false positives in elevated fasting glucose and hypertension, these measures are more sensitive than BMI≥30, and will identify more readily those individuals who may be at risk.

The current NHMRC criterion defining central obesity is a very good predictor of the presence or absence of other independent diabetes risk factors among women, including fasting glucose. It is therefore recommended that, if BMI is to be used, then the 'at risk' threshold should be reduced for Australian Aboriginal women. Current NHMRC definitions of overweight and obesity appear to be inappropriate for women in this Aboriginal community, given the relationships between waist circumference and other diabetes risk factors. This finding is likely to be generalisable to other Aboriginal groups. Similar relationships have been observed between BMI and central obesity elsewhere, and waist circumference has been found in other communities to be associated with CVD and diabetes risk [23, 24]. Central obesity is clearly correlated with diabetes risk, and the BMI guidelines for Indigenous people should reflect this risk. This increased risk at lower BMIs is not reflected by the current guidelines for obesity.

A reduced BMI threshold for women by  $5\text{kg/m}^2$  to  $25\text{kg/m}^2$  could be a more appropriate indicator of individual risk. An even lower threshold for Aboriginal populations has been advocated by Daniel and colleagues, who suggest that a BMI  $\geq 22\text{kg/m}^2$  might be appropriate [22]. Among men, reducing waist circumference threshold to  $\geq 90$ cm and BMI to  $25\text{kg/m}^2$  would appear to better reflect risk.

Internationally too, there are precedents for defining population-specific obesity thresholds; lowering the threshold has been proposed for Japanese [10] and Chinese [8, 9, 25] populations, for example. Alternatively, BMI should perhaps not be regarded as a risk factor at all; waist circumference alone is perhaps a better predictor of diabetes and CVD risk in both Indigenous and non-Indigenous populations. Wang and Hoy found waist circumference better than BMI at predicting CVD risk in a remote Aboriginal community[24, 26], as did Welborn and colleagues in a national survey of the Australian population [27]. Dalton and colleagues suggest that waist-hip ratio might be better in a clinical setting than BMI at identifying at risk individuals [28]. BMI is not intrusive to measure and is easy to calculate, and it therefore remains a useful clinical tool. It may also be perceived (and may actually be) more achievable for individuals to reduce their overall body weight, rather than target fat reduction in specific areas. For BMI to be relevant in Indigenous communities, however, the classifications of healthy and at risk need to be reconsidered, particularly among women.

Further, the data presented here demonstrate that even women who are 'underweight' by NHMRC guidelines may still be centrally obese, and therefore at risk of elevated fasting glucose and hypertension. Because of their low BMI, this group of women may be ignored in initial clinical assessments of diabetes risk. Women with low BMIs may need to be targeted specifically for diabetes screening. There may also be a secular trend of increased waist circumference, producing an 'at risk' body shape among younger women.

Waist circumference is much more strongly and significantly associated with other risk factors among women than among men. Given the strong positive linear relationship between waist circumference and BMI, reducing overall obesity among women, especially, could have a substantial impact on diabetes risk by reducing central adiposity.

This paper highlights the problems associated with developing guidelines for one population and applying them to another. Indigenous Australians are now recognised to be at particular risk of diabetes, but the only specific risk factor in the current NHMRC clinical guidelines that recognises this is age.

Current national guidelines on waist circumference in women appear to be appropriate, but the relevance of these guidelines for BMI thresholds should be investigated further. In men, both waist circumference and BMI thresholds could be reduced to better reflect diabetes and CVD risk.

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