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## Scaling waist girth for differences in body size reveals a new improved index associated with cardiometabolic risk

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Running head: Scaling waist girth and cardiometabolic risk

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## Abstract

Our aim was to examine whether a new ratio, waist divided by height<sup>0.542</sup> (~~WHT<sup>4/2</sup>~~ RWHT.5R), is both independent of stature and a stronger predictor of cardiometabolic risk (CMR) than other anthropometric indices.

Subjects (4117 males and 646 females), aged 20-69 years, were assessed for stature (cm), mass (kg), waist and hip girths (cm) from which, body mass index (BMI), Waist to Hip ratio (WHR), Waist to Height ratio (~~WHT<sup>4/2</sup>~~ RWHT.5R) and two new indices, a body shape index (ABSI) and ~~WHT<sup>4/2</sup>~~ RWHT.5R were determined. We used the allometric power law,  $W = a \cdot HT^b$ , to obtain a simple body-shape index for waist girth (W) to be independent of stature (~~H<sup>4/2</sup>~~ HHT). Physical activity was determined using self-report and physical fitness was determined using the Bruce protocol. Glucose, total cholesterol, low density lipoprotein, high density lipoprotein, triglycerides, and TC/HDL ratio were determined from fasting venous blood samples. A single CMR composite score was derived from log transformed z-scores of: Triglycerides + average blood pressure ((diastolic + systolic)/2) + glucose + HDL (\*-1).

Results confirmed ~~WHT<sup>4/2</sup>~~ RWHT.5R to be independent of stature and the strongest predictor of CMR, compared to BMI, WC, WHR, ABSI and ~~WHT<sup>4/2</sup>~~ RWHT.5R. We also found that CMR scores decline significantly with increasing fitness and physical activity, confirming that being fit and active can compensate for the adverse effects of being fat as measured by all other anthropometric indices.

In conclusion, ~~WHT<sup>4/2</sup>~~ RWHT.5R was the best anthropometric index associated with CMR, and being both physically fit and active has a protective effect on CMR, irrespective of weight status.

**Key words:** Waist-to-Height<sup>0.542</sup> Ratio; Allometric power law; ~~Body Size Index~~; Waist-to-Height Ratio; Centralised Obesity

## Introduction

Although body mass index (BMI) remains a frequently used proxy for obesity, in recent years, measures of abdominal obesity, notably waist circumference (WC) and waist to height ratio (~~WHtR~~~~WHtR~~), have increasingly been linked with cardiometabolic risk in cross sectional and prospective studies (Ashwell, Gunn and Gibson, 2012). Meta-analytic (Lee et al., 2008) and systematic review data (Browning et al., 2010) has suggested that centralised measures of obesity are superior to body mass index (BMI) in detecting cardiometabolic risk (CMR). In the case of Browning et al (2010), both WC and ~~WHtR~~~~WHtR~~ were identified as superior to BMI as cardiometabolic risk predictors. More recently, Ashwell et al (2012) conducted a systematic review and meta-analysis with data representing over 300,000 adults and concluded that ~~WHtR~~~~WHtR~~ was superior to both WC and BMI for detecting cardiometabolic risk in both males and females. They subsequently suggested that ~~WHtR~~~~WHtR~~ should be considered as a standardised screening tool for adult cardiometabolic risk.

The mechanism as to why ~~WHtR~~~~WHtR~~ may be superior to other anthropometric measures in predicting cardiometabolic risk has been considered by previous authors. These proposed mechanisms largely relate to the higher metabolic and inflammatory activity of visceral fat depots within the abdominal cavity (Kuk et al., 2006) compared to subcutaneous adipose depots in other parts of the body (Manolopoulos et al., 2010). Such a suggestion is plausible in explaining why abdominal measures of obesity (WC), more reflective of visceral fat, are better than BMI for identifying cardiometabolic risk. However, there is less clarity as to why WC divided by height would be superior to WC alone in identifying cardiometabolic risk. Stature has been shown to have inverse associations with cardiometabolic morbidity and mortality (Langenberg et al., 2005). This is potentially because, in addition to a genetic component, stature can reflect early life exposures such as inadequate or malnutrition (Barker et al., 1990). Data from Chilean participants has also proposed that adverse environmental exposures in critical growth periods earlier in life 'programme' short stature and a predisposition to abdominal obesity and cardiometabolic risk factors in adult life (Koch et al., 2010). The authors suggest this offers a biologically plausible explanation as to why ~~WHtR~~~~WHtR~~ is superior than other anthropometric measures in explaining cardiometabolic risk in adults. Despite this, there is still debate as to the utility of ~~WHtR~~~~WHtR~~, over BMI or WC, in explaining cardiometabolic risk.

Moreover, WC is highly correlated with BMI making it difficult to separate the two as independent epidemiological risk factors (Moore, 2009). In an attempt to address this issue Krakauer and Krakauer (2012) developed a body shape index (ABSI,  $WC/(BMI^{2/3} \times height^{1/2})$ ) based on WC that is independent of height and body mass and compared its ability to predict mortality risk in a sample of over 14000 adults alongside WC and BMI. They found that ABSI was not correlated with BMI and was a better predictor of mortality risk than WC or BMI and suggested that it would be beneficial to examine the utility of ABSI in predicting health risk in other data sets. Few studies to date appear to have acted on this suggestion.

The aim of the current study was to explore whether recent assertions by Ashwell et al (2012) that the waist-to-height ratio is the strongest predictor of cardiometabolic risk in adults, or whether a new ratio, waist divided by height<sup>0.54+2</sup> (~~WHt<sup>1/2</sup>~~ RWHT.5R), is both independent of stature ~~but and~~ also a stronger predictor of CMR. We shall also attempt to extend the extant body of literature on this topic by providing an explanation as to why this new ratio (~~WHt<sup>1/2</sup>~~ RWHT.5R) may be superior to other anthropometric indices

## Methods

### *Participants*

Following institutional ethics approval and informed consent, participants (n = 4763, 4117 males, 646 females), aged 20-69 years (Mean age  $\pm$  SD = 48.6  $\pm$  8.2 years) attended one of five Health & Wellbeing clinics around England for a three-hour health assessment between 2000 and 2009. Prior to participation, participants ~~were instructed in their~~ ~~received an~~ information pack to avoid any form of vigorous physical activity, alcohol and/or caffeinated beverages within the 24 hours prior to their assessment. Participants reported their sex, age, date of birth, and current home postcode. Date of birth was used to calculate age.

## Procedures

### *Anthropometry*

Body mass was measured using digital scales (Marsden, UK) and recorded to the nearest 0.1 kg. Clothing was worn but shoes and belts were removed, and participants evacuated their bladder before stepping onto the scales. Scales were calibrated daily with a known weight and bi-annually by the manufacturer. Stature was measured using a stadiometer (Seca, Hamburg, Germany) and recorded to the nearest 0.1 cm. Participants removed their shoes, stood on the platform with feet together, and head in the Frankfort plane. Buttocks and scapulae were in contact with the back of the stadiometer, shoulders relaxed with hands and arms loosely at the sides, the measurement was taken on full inhalation. WC was measured with participants standing with feet shoulder width apart using a standard, non-elastic anthropometric tape measure (Seca, Birmingham, UK). WC measures were ~~taken end tidal~~ ~~made at minimal inspiration~~ to the nearest 0.1 cm, midway between the lowest rib and the iliac crest, which corresponded with the level of the umbilicus. Hip circumference was measured at the iliac crest (men) and by identifying the widest point of the pelvic region (women). Repeat measurements were made and the mean value of two measures which agreed to within 0.5 cm was used (WHO 1998).

The anthropometric indices of weight status were calculated as follows: Body mass index (BMI) was calculated as weight (kg)/height<sup>2</sup> (m<sup>2</sup>). WHR was calculated by dividing WC by HC, and ~~WHR~~ ~~WHTR~~, by dividing WC by height. A body shape index (ABSI) was calculated using formula:  $ABSI = WC / (BMI^{2/3} \times height^{1/2})$  (see Krakauer and Krakauer, 2012). Finally, a new waist-to-height ratio (~~WHT~~ ~~RWHT~~ ~~5R~~), independent of height, was calculated by dividing WC by height<sup>0.54/2</sup> (for an explanation and justification, see statistical methods and results).

### *Physical Activity*

The level of physical activity (PA) was self-reported by participants during a semi-structured interview. Each participant was asked to report their 'normal' frequency of moderate exercise sessions per week and was informed that moderate exercise was equivalent to brisk walking and that a bout of moderate

exercise should be of at least 30 min duration. This was followed by self-report of strenuous (vigorous) activities using the same method as described above but where participants were told that strenuous activity bouts needed to be 20-25 min and were given the examples of 'going to the gym' or participating in 'sporting activities'. Responses were then categorised based on the recommended guidelines of 150 minutes of moderate activity and/or 75 min of vigorous activity per week (WHO, 2010).

### *Physical Fitness*

$\dot{V}O_{2peak}$  was used as a measure of physical fitness. Each participant was instructed in their pre-assessment information pack to avoid any form of vigorous PA, alcohol and caffeinated beverages within the 24 hours prior to their assessment. After 5 min of rest in the supine position, a 12-lead electrocardiogram (ECG) (Marquette CASE, GE Healthcare, UK) was performed on all participants, which was reviewed by the duty medical officer.

Participants walked on a treadmill (T2100, GE Healthcare Ltd., Buckinghamshire, UK) using the Bruce protocol (3 min incremental stages) (Bruce et al., 1972) and each was discouraged from holding the handrails. At the end of each minute, heart rate was monitored, and recorded every three minutes. Blood pressure was monitored at the second minute of each stage using the automatic Tango stress test BP monitor (Suntech Medical, Oxfordshire, UK). Rating of Perceived Exertion (RPE) was recorded at the end of each stage using the 6-20 Borg Scale (Borg, 1970), and ECG activity was monitored throughout the test. Participants exercised until volitional termination of the test or if they met any of the American College of Sports Medicine (ACSM) test termination criteria (ACSM, 2006). Peak oxygen uptake was estimated and reported in  $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ . Data were divided into 10-year age strata and  $\dot{V}O_{2peak}$  ( $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) was classified as 'Fit' or 'Unfit' based on the Cooper Institute age- and sex-specific cut-offs (1994).

### *Assessment of Cardiometabolic Risk*

In order to determine Cardiometabolic Risk, participants presented in a fasted state (defined as not having eaten for the previous 12 hours) At the start of each assessment. Fasting venous blood samples were obtained using vacutainer tubes and heparinised whole blood was analysed using the Piccolo blood chemistry analyser (Abaxis, USA). The following CMR variables were measured: glucose, total cholesterol (TC), low density lipoprotein (LDL), high density lipoprotein (HDL), triglycerides, and TC/HDL ratio. A single cardiometabolic composite score was then determined using: Triglycerides + average blood pressure ((diastolic + systolic)/2) + glucose + HDL (\*-1). All variables are log transformed before the z-scores are formed and then summed and divided by 4.

\*\*\*Table 1 Here\*\*\*

### Statistical Methods

We developed a simple body shape index for waist girth ( $W$ ) to be independent of stature/height ( $H$ ) using the allometric power law

$$W = a \cdot H^b \cdot HT^\epsilon, \quad (1)$$

where  $a$  and  $b$  are the scaling constant and scaling exponents for the waist girth and  $\epsilon$  is the multiplicative error ratio (Nevill et al. 1992). Note that the multiplicative error ratio ' $\epsilon$ ' assumes that the error will increase in proportion to body size (see Figure 1), a characteristic in data known as heteroscedasticity that can be controlled by taking logarithms, as described below. Age and sex were incorporated into the model by allowing ' $a$ ' to vary for either sex and each age group (age categories 20-29, 30-39, ..., 60+) to accommodate the likelihood that waist girths may rise and then peak sometime during adulthood. The model can be linearized with a log-transformation, and multiple regression/ANCOVA can be used to estimate the stature/height exponent for waist girth having controlled for both age and sex.

\*\*\*Figure 1 Here\*\*\*

To explore the strength of the association between cardiometabolic risk and the six anthropometric indicators of weight status (BMI, Waist circumference, Waist-to-hip ratio, Waist-to-height ratio, ABSI and Waist-to-height<sup>-0.54/2</sup>), we conducted three types of analyses. The first (1), 6 MANOVA's (using the 4 cardiometabolic risk factor variables as multivariate dependent variables) incorporating each anthropometric indicator of weight status as separate covariates, with all 6 analyses incorporating 'age group' (age categories 20-29, 30-39, ..., 60+) and 'sex' as fixed factors. The second set of analyses (2), used ANOVAs to explore the univariate cardiometabolic risk dependent variable, incorporating each of the six anthropometric indicators as separate covariates, again with 'age group' and 'sex' as fixed factors. Finally, the third set of analyses (3), adopted the same 6 ANCOVA analyses as in (2) but incorporated "meeting the PA guidelines" (entered as 0, 1 indicator variable), as well as 'age group' and 'sex' as fixed factors, plus 'VO<sub>2</sub>peak' as an additional covariate.

To establish whether waist and hip girth measurements (G) increased in proportion to, or at a greater proportion to mass (M) assumed by geometric similarity, (i.e.,  $M^{0.333}$ ), we adopted the same allometric model as above (see Nevill et al. 2004):

$$G = a \cdot M^b \cdot \varepsilon, \quad (2)$$

where a and b are the scaling constant and scaling exponents for the waist and hip girths and  $\varepsilon$  is the multiplicative error ratio. Age and sex was incorporated into the model as described in (1). The model can be linearized with a log-transformation, and multiple regression/ANCOVA can be used to estimate the mass exponents for both waist and hip girths having controlled for both age and sex, also described in (1).

## Results

The allometric power law model for waist girth (Eq 1), identified the height exponent to be 0.528 (SEE=0.04) having controlled for both age and sex, suggesting that the simple body shape index for waist girth (W) to be independent of stature ( $H/HT$ ) should be  $W \cdot H/HT^{-0.54/2}$ .

The contributions of the six anthropometric covariates to the MANOVA analyses, having controlled for 'age group' and 'sex' are given in Table 2. In all 6 MANOVA analyses, the main effects of 'age group', 'sex' and their interactions were significant ( $P < 0.001$ ). Note that the Wilks lambda

ranges from 0 -1 and the lower the Wilks lambda, the greater the between-group variance or in our case, the stronger the relationship. This was confirmed by the larger F ratios.

\*\*\*Table 2 Here\*\*\*

The contributions of the six anthropometric covariates to the univariate ANOVAs of the Cardiometabolic risk-factor dependent variable are given in Table 3, having controlled for 'age group' and 'sex'

\*\*\*Table 3 Here\*\*\*

When we incorporated the elements of fitness ( $VO_{2peak}$ ) and whether the participants met the physical activity guidelines into the analysis, the contributions of the six anthropometric covariates to the univariate ANOVAs of the Cardiometabolic risk-factor dependent variable, are given in Table 4, having controlled for 'age group' and 'sex' as before.

\*\*\*Table 4 Here \*\*\*

To investigate whether the waist and hip girths were increasing/ expanding at a greater rate than that assumed by geometric similarity, the allometric models (2) were fitted to the data. The results suggest that both waist and hip girths are expanding at a greater rate than that anticipated by geometric similarity ( $M^{0.333}$ ), i.e., the fitted exponents were proportional to body mass,  $M^{0.610}$  and  $M^{0.386}$  respectively, having controlled for both age and sex. Note that the mass exponent SEE's were 0.006 and 0.003 respectively. As anticipated, the level of adiposity as measured by the waist when calculating the waist-to-hip ratio will be partially explained and hence diluted, rendering the ratio less effective at identifying the adiposity of overweight participants.

The ROC analysis (Figure 2, area under the curve = 0.745 [95% CI 0.726 to 0.763]) identified the  $WHH^{1/2}RWHT.5R$  cut-off point to be 0.726- (Sensitivity=0.589 and Specificity=0.761) that would

best discriminate between participants whose cardiometabolic score was greater than 1 standard deviation above the mean (Sardinha et al., 2016). For example, a 1.83m (6' 0") male's waist should not exceed 0.982m (38.7in). Similarly, a 1.70m (5' 7") female's waist should not exceed 0.946m (37.2in).

\*\*\*Figure 2 Here\*\*\*

## Discussion

Our initial findings suggest that, based on the allometric power law (1), in order to obtain a waist girth index that is independent of stature, we need to calculate the ratio  $WH^{1/2}RWHT.5R$  = Waist/Height<sup>0.5+2</sup> rather than the more commonly used  $WH:RWHTR$ .

Subsequently, we identified the strongest predictor of, or association with, cardiometabolic risk was indeed the waist-to-height<sup>0.5+2</sup> ratio, identified in all three analyses (see Tables 2, 3 and 4). Indeed, the strongest through to the weakest anthropometric covariate associated with cardiometabolic risk was found to be the following order, (1) the waist-to-height<sup>0.5+2</sup> ratio, (2) waist-to-height ratio, (3) absolute Waist, (4) BMI, (5) waist-to-hip ratio, and finally (6) ABSI, an order that remained consistent in all three types of analyses, see Tables 2, 3 and 4. The second and third best predictors of cardiometabolic risk were waist-to-height ratio and absolute waist, certainly better than either BMI, waist-to-hip ratio, or ABSI. These results lend some support to the assertions made by Ashwell et al. (2012) based on systematic review and meta-analysis that  $WH:RWHTR$  was more strongly associated with cardiometabolic risk than WC or BMI. The explanation as to why this might be the case is debated in the literature (See Kuk et al., 2006; Manolopoulos et al., 2010; Langenberg et al., 2005). However, the present study offers, as far as we are aware, a novel anthropometric index ( $WH^{1/2}RWHT.5R$ ) that is not only independent of body stature but also consistently predicts cardiometabolic risk with the greatest precision. We offer the following viable and biologically plausible explanations for this consistent rank order in anthropometric indices, with  $WH^{1/2}RWHT.5R$  rather than  $WH:RWHTR$  being the best index when predicting cardiometabolic risk.

Any change in waist girth is likely to reflect changes in adiposity associated with cardiometabolic risk, whereas it is possible that changes in BMI might also reflect changes in muscle mass as well as adiposity, especially in younger (see Nevill and Metsios, 2015) and more athletic

populations (see Nevill et al. 2006). This confirms that central adiposity is more relevant to cardiometabolic risk than being generally overweight as measured by stature-adjusted body mass, i.e., BMI.

The fact that absolute waist was not optimal when predicting cardiometabolic risk highlights the need for the waist girth measurements to be normalized for differences in body size (to be independent of body size), and hence to better reflect the centralized adiposity associated with cardiometabolic risk. However, in the past, the WHR has been thought to do precisely that, i.e., to “normalize” waist girth for individuals of different body size. So why does the waist-to-hip ratio come almost last of our competing anthropometric indices when predicting cardiometabolic risk?

The explanation relies on the fact that not only does waist girths increase/expand in bigger subjects ( $M^{0.61}$ ) at a greater rate than that expected by geometric similarity ( $M^{0.333}$ ), hip girths also expands at a greater rate ( $M^{0.386}$ ) than that assumed by geometric similarity. As such, the level of adiposity as measured by the waist will be partially explained and hence diluted when the waist-to-hip ratio is calculated, a dilution that would be absent by dividing/normalizing by a body-size dimension such as stature/height that is unaffected by changes in adiposity. However, even the  $\frac{WH}{RHTR}$  fails to normalise waist girth entirely. The only waist ratio that is entirely independent of body size (height) and elements of adiposity (e.g., hip girth and BMI) is  $\frac{WH^{1/2}}{RWHT.5R}$ , that might explain why WHR and ABSI are ranked 5<sup>th</sup> and 6<sup>th</sup> in our list of anthropometric indices predicting cardiometabolic risk, and why  $\frac{WH^{1/2}}{RWHT.5R}$  is ranked 1<sup>st</sup>. Note that the ratio ABSI divides waist by  $BMI^{2/3}$  and height<sup>1/2</sup>, the latter term providing some consistency with  $\frac{WH^{1/2}}{RWHT.5R}$ .

We also found evidence of the so called ‘fat and fit’ phenomenon, i.e., in Table 4 we demonstrate that the cardiometabolic score declines significantly with increasing PA and fitness ( $VO_2$ peak) but increases significantly with all measures of adiposity, e.g.,  $\frac{WH^{1/2}}{RWHT.5R}$ , confirming the compensatory nature of being both fat and being fit (and active), that might result in a similar level of cardiometabolic risk to a more lean but less fit person. Previous research has shown that obese individuals with high levels of cardiorespiratory fitness have a lower risk of all-cause mortality and cardiovascular disease compared to lean individuals with low levels of cardiorespiratory fitness (Barry et al., 2014; Lee et al., 1999). Furthermore, overweight/obese individuals with higher cardiorespiratory fitness have reduced visceral adiposity and in turn a more favourable cardiometabolic risk profile (e.g.

improvements in HDL-Cholesterol, triglycerides, blood pressure, and insulin resistance) compared to BMI-matched overweight/obese individuals with low cardiorespiratory fitness (Donovan et al., 2011; Arsenault et al., 2009; Rheaume et al., 2009). Our data were suggestive that with higher levels of cardiorespiratory fitness even with greater adiposity (i.e. higher BMI, WC, WHR, ~~WHR~~<sup>4/2</sup>~~RWHT.R~~, ABSI and ~~WHR~~<sup>4/2</sup>~~RWHT.5R~~) is associated with lower cardiometabolic risk profiles compared to those with the same level of adiposity but lower levels of cardiorespiratory fitness. We also found similar associations between higher levels of physical activity and adiposity and lower cardiometabolic risk, which is in line with the findings of previous studies (Jefferis et al., 2016; Kim et al., 2013).

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The current study is not without limitations and addressing these would provide key future research direction. Central adiposity can differ between individuals from different ethnic backgrounds and this may influence the applicability of WHT.5R across different ethnic groups. A natural progression from the work presented here, given the relatively fit and lean sample we examined, would be to examine the utility of WHT.5R as an anthropometric index associated with cardiometabolic risk in different groups including those of different ethnicity, those who are obese and across the adult lifespan. The anthropometric assessment was also completed with participants wearing light clothing (t-shirt and shorts). This may have contributed to some extra error in any index which used body mass. However, given the nature of the clothing worn any such error is likely to be minimal.

Taken collectively, the results of the present study suggest ~~WHR~~<sup>4/2</sup>~~RWHT.5R~~ may be a more useful anthropometric index associated with cardiometabolic risk compared to either ~~WHR~~<sup>4/2</sup>~~RWHT.R~~, BMI, WHR, ABSI or WC alone and that cardiorespiratory fitness has a protective effect for cardiometabolic risk, even in those who are overweight and obese. For health practitioners and public health professionals the promotion of cardiorespiratory fitness should be a key message in reduction of cardiometabolic risk and the use of ~~WHR~~<sup>4/2</sup>~~RWHT.5R~~ as a diagnostic screening tool should be encouraged over other measures of weight status including ~~WHR~~<sup>4/2</sup>~~RWHT.R~~ itself.

## Perspectives

Recent research by Ashwell et al (2012) suggests that the waist-to-height ratio is the strongest predictor of cardiometabolic risk (CMR) in adults. Here we show that a new ratio, waist divided by height<sup>1/20.5</sup> (~~WHR~~<sup>1/20.5</sup> ~~RWHT.5R~~), is not only independent of stature (using allometry) but also a stronger predictor of CMR compared with a wide range of other anthropometric indices including BMI, waist-to-hip ratio (WHR), waist-to-height ratio (~~WHR~~<sup>1/20.5</sup> ~~RWHT.5R~~), and a new body shape index (ABSI, WC/(BMI<sup>2/3</sup> x height<sup>1/2</sup>)) Krakauer and Krakauer (2012).

The likely explanations are twofold; (1) waist girth is the most sensitive dimension to detect changes in adiposity, certainly better than BMI that might reflect changes in muscle mass as well as adiposity, and (2) using height<sup>0.51/2</sup> to normalize or scale waist girth for individuals of different body size is more suitable, since ~~WHR~~<sup>1/20.5</sup> ~~RWHT.5R~~ will be independent of stature but also height<sup>1/20.5</sup> is unaffected by changes in adiposity, unlike hip girth and BMI, used to normalise WHR and ABSI respectively. We also found that CMR scores decline significantly with increasing fitness (VO<sub>2</sub>peak) and physical activity, confirming that being fit and active can compensate for the adverse effects of being fat as measured by all of the anthropometric indices.

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### Legend to Figures

Figure 1 The relationship between waist circumference (cm) and stature/~~height~~ (cm) for male and female subjects that demonstrates a multiplicative error ratio where the error increase in proportion to body size, a characteristic in data known as heteroscedasticity..

Figure 2. The ROC analysis (area under the curve = 0.745 [95% CI 0.726 to 0.763]) identified the ~~WHR~~<sup>1/2</sup> RWHT.5R cut-off point to be 0.726 (Sensitivity=0.589 and Specificity=0.761) that would best discriminate between participants whose cardiometabolic score was greater than 1 standard deviation above the mean.