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Predictability of Metabolic Risk Factors from Hand and Body Anthropometry in Hausa Ethnic Population of Kano, Nigeria

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Summary: Validity of anthropometric tools of adiposity for metabolic risk factors (MRF) does not exhibit a universal trend, making certain adiposity tools more germane to a particular ethnic/racial group. Adiposity measures are employed to screen MRF by clinicians. The ratio of the second to fourth digit of the hand (2D:4D) has been shown to be a tight correlate of MRF. Attempts to predict MRF from hand anthropometry is relatively a new idea. The present study aims to predict MRF from digit and body anthropometry. The study recruited 266 males and 199 females of Hausa origin. Systematic random sampling was employed. Anthropometric measurements and blood pressure were obtained using standard techniques. Regression analysis was used to predict MRF, SPSS version 20 was used for statistical analyses and $P < 0.05$ was set as level of significance. MRF (serum glucose, total cholesterol, lipoprotein cholesterol, and blood pressure (BP) were predictable from 2D:4D and body anthropometric measures. Waist-to-hip ratio (WHR) was the most consistent MRF predictor. In males, WHR alone predicted TC ($R2 = 0.67$ and $P<0.0001$), HDL-C ($R2 =0.68$ and $P <0.0001$), LDL-C ($R2 = 0.67$ and $P < 0.001$) and diastolic blood pressure (DBP) [$R2 =0.43$ and $P <0.001$)]. The right 2D:4D contributed slightly to the prediction of SBP and FBG increasing the R2 value to 0.62from 0.6 for FBG and from 0.6 to 0.64 for SBP.

Keywords*: Body anthropometry, Digit anthropometry, Metabolic risk, Predictability*

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INTRODUCTION

Dislipidemia, hyperglycemia, hypertension and obesity are leading causes of morbidity and mortality globally and together constitute the metabolic syndrome (MetS) (Moller and Kaufman, 2005). The syndrome was previously thought to be a problem of developed nations, but it's prevalence in the developing nations such as Nigeria is rising alarmingly and has even been shown to assume an epidemic dimension in the last two decades (Mukadas and Misbahu, 2009; Ifeoma et al., 2011).

The use of anthropometric indices of adiposity to estimate and predict the risk of MetS by clinicians and public health physicians is a common practice globally (Tulloch-Reid et al., 2003; Ekezie et al., 2011). This practice is rooted to several documented evidences linking body adipose tissue accumulation to insulin resistance, release of pro-inflamatory cytokines such as interleukins 1 and 6 and reduction in anti-inflamatory cytokines such as adiponectin which are major steps in the pathogenesis of MetS (Kissebah et al., 1982; Lara-castro et al., 2007; Ghantous et al., 2015).

Inspite of the pathologic role of adipose tissue noted, there are convincing evidences that, in addition to the total body adipose tissue reserve, the specific anatomic site of lipid aggregation is critical in the susceptibility of an individual to MetS (Després and Lemieux, 2006; Després et al., 2008). This has recently led endocrinologist to the concept of "fat distribution rather than "fat collection" and consequently a greater attention on central and visceral adiposity (measurable by indices such as waist circumference) rather than generalized adiposity measurable by Body Mass Index (BMI) (Fontana et al., 2007; Amato and Giordano, 2014)

Although the superiority of visceral adiposity over generalized adiposity in their relationships with metabolic risk factors (MRF) has been clearly elucidated in the literature (Amato and Giordano, 2014), however ethnicity and race are documented to modulate these interrelationships such that the predictive potential of a particular adiposity measure does not demonstrate a uniform trend in all racial and ethnic populations (Tulloch-Reid et al., 2003) and may not have the same predictive ability for each MRF (hyperlipidemia, hyperglycemia and hypertension).

Currently, there are ongoing global efforts to explore other sensitive anthropometric variables to compliment adiposity tools in the risk estimation and prediction of MetS (Asuku et al., 2016; Ranvider and Manju, 2016). On this note, the ratio of the second-to-fourth ratio of the hand (2D:4D), a prenatally determined sexually dimorphic variable has recently gained attention having demonstrated strong correlation with anthropometric indices of body adiposity among Europeans (Fink et al., 2003; Fink et al., 2006), Ugandans (Abba et al., 2012) and among Nigerians (Danborno et al., 2008; Oyeyemi et al., 2016). The recent studies of Asuku et al. (2016) and that of Ranvider and Manju, 2016 revealing a strong correlation between 2D:4D and components of MetS call for a more keen attention on the probable role of 2D:4D in the prediction of MRF. The aim of the present study was therefore to pull together 2D:4D and the anthropometric indices of adiposity (BMI, NC, WC, HC, WHR, WHtR, BAI) in a linear regression model to estimate the contribution of each variable in the prediction of MRF among the Hausa ethnic group in Kano, Nigeria. This study gains its uniqueness from the paucity of data globally and among Nigerians on the role of 2D:4D in predicting MRF and scarcity of studies on the precise contribution of each anthropometric adiposity tool in predicting different MRF.

MATERIALS AND METHODS

Four hundred and sixty-five subjects who are Hausa indigenes of Kano were selected using Systematic sampling technique. Participants were recruited from outpatient units of Murtala Muhammad specialist Hospital, Khadija Memorial Hospital, the old campus of Bayero University, Kano, SU clinic Gabasawa, General Hospital Dawakin – Tofa.

The study included only subjects in the age range of 18 years to 68 years. Subjects with congenital and/or acquired digit deformity and those on medications that could interfere with any components of MetS were excluded. Ethical approval was obtained from Kano state hospitals management board and written informed consent obtained from the subjects.

Anthropometric Methods: Height was measured to the nearest 0.1cm as the vertical distance between the standing surface and the vertex of the head while the subject was standing erect in the frank forth plane and without shoes using a stadiometer. The weight was measured in kilograms using a digital weighing scale while the subject is in light clothes.

Finger Length Measurements: Digit lengths was measured on the ventral surface of the hand from the basal crease of the digit to the tip of the finger using a digital sliding caliper (MicroMak, USA) measuring to 0.01mm and reported on questionnaire. This measurement has been reported to have high degree of repeatability (Manning et al., 1998; Danborno and Danborno, 2015).

Blood pressure: A mercury sphygmomanometer was used for measuring blood pressure. Two measurements were taken, and at least 2 minutes was allowed between readings. While the diastolic reading was taken at the level when sounds disappear (Korotkoff phase V), the systolic was taken at the level when it appears. (Haffner et al., 1992).

Serum Analytical Methods

Serum glucose was measured using enzymatic method of Trinder (1969). Glucose oxidase converts glucose to gluconic acid while peroxidase converts the hydrogen peroxide to water and oxygen which also oxidizes the chromogen (4-aminophenazone) to a pink coloured complex which is measured colorimetrically at 510 nm.

$$\text{Glucose} \xrightarrow{\text{Glucose oxidase}} \text{Gluconic acid} + H_2O_2$$

$$H_2O_2 + \text{phenol} \xrightarrow{\text{Hydrogen peroxidase}} H_2O + \text{Oxidised Chromogen}$$

Test tubes labeled, blank, standard and test, 1ml of glucose reagent was placed. Into the test tubes 10µl of distil water, standard solution and test serum was added to the test tubes respectively. These was then mixed and incubated at 37 ^{0}C for 10 minutes, after which the absorbance (Optical Density) of the test solution and standard was read at 505nm using the blank solution to zero the spectrophotometer

$$\text{glucose conc.} = \frac{\text{Absorbance of Test} \quad \text{x} \quad \text{Concentration of standard}}{\text{Absorbance of STD}}$$

Where the concentration of the glucose standard is 5.55 mmol/L.

Serum TC concentrations were measured using enzymatic method by Wybenga *et al.* (1970).
Total Cholesterol (TC) was measured enzymatically in serum in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol. One of the reaction by-products, H_2O_2 is measured quantitatively in a peroxidase catalyzed reaction that produces a colour. Absorbance is measured at 500 nm. The colour intensity is proportional to cholesterol concentration.

$$\text{Cholesterol ester} \xrightarrow{\text{cholesterol Esterase}} \text{Cholesterol} + \text{Fatty Acids}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol Oxidase}} \text{Cholesterol} - 4\text{en} - 3\text{one} + H_2O_2$$

$$2H_2O_2 + 4\text{AAP} + \text{phenol} \xrightarrow{\text{Peroxides}} \text{Quinoneimine} + 4H_2O\ \text{(endpoint) RED}$$

Three test tubes labeled as test, standard and blank were earmarked and to each 1000 µl of the reagent R1 was added. 10µl sample was added to test and 10 µl standard to standard tube and 10µl distilled water to blank. The content was then mixed well and incubated at room temperature for 15mins. Reading was taken at 530 nm

$$\text{TC conc.} = \frac{\text{Absorbance of Test}}{\text{Absorbance of standard}} \times \text{Conc. of standard}$$

Where concentration of the total cholesterol standard is 5.17 mmol/L.

Serum HDL-C concentrations were measured using enzymetic method of Wybenga *et al.* (1970). This method is

based on the principle that serum chylomicrons, LDL and VLDL are precipitated in the presence of phosphotungustic acid and magnesium chloride and the supernatant is treated as cholesterol. Into a clean test tube 0.5ml serum and 0.5 ml HDL reagent were taken, mixed and allowed to stand for 10 minutes. It was then centrifuged for 20 minutes at 2000rpm. Cholesterol reagent, 1ml was dispensed in to three cleaned test tubes labeled blank, standard and sample. Supernatant, 50µl was dispensed in to sample tube, 50µl of standard was dispensed into standard tube and 50µl of distilled water dispensed in to blank tube. All were mixed and incubated at room temperature for 5 min and read at 530 nm.

$$\text{Conc. of HDL} = \frac{\text{Absorbance of Test} \quad \text{x} \quad \text{Concentration of standard}}{\text{Absorbance of STD}}$$

Where concentration of the total cholesterol standard is 5.17 mmol/L

Serum LDL-Cholesterol was estimated using Friedewald equation (Friedewald *et al.*, 1972). LDL-cholesterol concentrations were calculated from measured values of total cholesterol, triglycerides and HDL-cholesterol according to the Friedewald's formula:
LDL-Cholesterol =TC - (HDL-C + Triglycerides/2.2) mmol/L.

Serum TG concentrations were measured using enzymetic method of Wybenga *et al.* (1970).
TG was measured enzymatically using a series of coupled reactions in which triglycerides are hydrolyzed to produce glycerol. Glycerol is then oxidized using glycerol oxidase and H_2O_2, one of the reaction products was then measured as described above for cholesterol. Absorbance was measured at 500 nm.

$$\text{Triglycerides} + H_2O_2 \xrightarrow{\text{Lipoprotein lipase}} \text{Glycerol} + \text{Fatty acid}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{Glycerol Kinase}} \text{Glycerol} - 3 - \text{phosphate} + \text{ADP}$$

$$\text{Glycerol} - 3 - PO_4 + O_2 \xrightarrow{\text{GPO}} \text{DHAP} + H_2O_2$$

$$H_2O_2 + 4\text{AAP} \xrightarrow{\text{POD}} \text{P} - \text{Chlorophenol Red quinoneimine}$$

Three test tubes were arranged as test, standard and blank tubes. 100 µl of triglyceride was added into each test tube and 10 µl of sample was added into sample tube while 10 µl of standard was added into standard tube. The content was mixed and incubated for 5 minutes at 37^0C. The absorbance was read at 520 nm. Serum TG concentration was calculated as follows:

$$\text{Concentration of test} = \frac{\text{Absorbance of Test} \quad \text{x} \quad \text{Concentration of standard}}{\text{Absorbance of Standard}}$$

Where concentration of the triglycerides standard is 2.28 mmol/l

Data obtained from anthropometric measurements and serum samples of participants were described using mean and standard deviation. Stepwise multiple linear regression analyses were used for prediction of MRF (BP, TC, FBS, HDL-C, LDL-C and VAI) from 2D:4D and body adiposity measures. SPSS version 20 (IBM Corporation, NY) software was used for statistical analyses and $P < 0.05$ was set as level of significance.

RESULTS

A total of 266 males (57%) and 199 females (43%) were studied. The participants had a mean age of 34.45 years and 32.06 years for males and females respectively.

Table 1:
Description of age, blood pressure, body and digit anthropometric measures of participants

	Male (n=266)		**Female (n= 199)**	
Variables	**Mean ± SD**	**Min-max**	**Mean ± SD**	**Min-max**
Age(years)	34.45 ± 13.52	18-68	32.06 ±15.18	18-65
BMI (kg/m^2)	21.98 ±3.93	14.52-34.33	22.19 ± 4.70	12.96-39.15
WC (cm)	77.28 ±11.17	57-111	76.02± 13.00	51-118.5
HC (cm)	87.01± 7.80	72.1-109.9	88.96± 9.86	65.6-136
NC (cm)	34.99± 2.29	30-42	31.58± 2.46	26.5-39.5
WHR	0.89± 0.08	0.71-1.11	0.85± 0.11	0.65-1.25
W/Ht	0.46 ± 0.06	0.34-0.65	0.48± 0.08	0.30-0.72
BAI	21.60 ± 3.71	13.88-33.90	26.61± 4.62	15.38-45.58
Height (cm)	169.15 ± 6.27	142-182.3	158.53 ± 6.83	136.9-175
Weight (Kg)	63.03 ±12.28	40.5-98.3	55.86± 12.99	36-108.9
DBP (mmHg)	82.59 ± 12.37	54-120	84.50± 12.99	60-120
SBP (mmHg)	128.07 ± 20.09	90-200	130.66 ± 21.87	95-205
RI (mm)	74.22 ± 5.45	61.17-90.46	67.97± 5.02	53.06-79.06
RII (mm)	72.56 ± 5.09	60.19-87.02	68.94± 4.48	55.42-82.09
RIII (mm)	80.12 ± 5.44	64.17-97.56	75.53± 4.98	63.13-94.26
RIV (mm)	75.63 ± 5.29	62.84-89.32	69.94± 4.51	55.41-85.35
RV (mm)	62.11 ± 5.31	47.17-85.87	57.60± 4.26	44.97-67.32
R2D:4D	0.96 ± 0.03	0.79-1.05	0.99± 0.03	0.86-1.07
LI (mm)	74.05 ± 5.36	60.33-87.47	67.77± 4.49	55.1-78.83
LII (mm)	73.32 ± 4.85	60.04-85.81	69.08± 4.40	57.19-80.44
LIII (mm)	80.50 ± 5.61	66.12-96.55	76.23± 5.56	50.09-98.92
LIV (mm)	76.03 ± 4.91	62.92-87.81	70.10 ± 4.71	57.45-82.26
LV (mm)	62.21 ± 5.09	47.46-74.36	57.69 ± 4.88	43.14-75.71
L2D:4D	0.96 ± 0.03	0.85-1.10	0.99± 0.03	0.92-1.09

DBP: diastolic blood pressure, SBP: systolic blood pressure, I: first digit, II: second digit, III: third digit, IV: fourth digit, V: fifth digit, R: right hand, L: left hand, 2D:4D: second to fourth digit ratio, BMI: body mass index, WC: waist circumference, HC: hip circumference, NC: neck circumference, WHR: waist-to-hip ratio, WHt: waist-to-height ratio, BAI: body adiposity index

Table 2
Serum indices of metabolic risk and visceral adiposity of study participants

	Male (n=120)		**Female (n= 41)**	
Variable	Mean ± SD	Min-max	Mean ± SD	Min-max
FBG (mg/dl)	84.67 ± 24.73	53.6-187.2	100.63 ± 34.90	54.6-176.4
T.C(mg/dl)	174.35 ± 32.31	123.7-256.10	187.32 ± 43.85	127.3-290.7
HDL–C(mg/dl)	44.10 ± 6.32	28-54.10	47.83 ± 6.71	38.9-60.6
TG(mg/dl)	117.18 ± 31.76	74.3-196.5	121.83 ± 29.25	80.4-165
LDL– C(mg/dl)	106.81 ± 32.44	58.14-192.82	115.12 ± 44.05	54.36-214.46
VAI	3.51 ± 1.71	1.67-9.10	4.46 ± 1.75	2.11-7.56

FBG: fasting blood glucose, T. C: total cholesterol, HDL-C: high density lipoprotein cholesterol, TG: triglyceride, LDL-C: low density lipoprotein cholesterol, VAI: visceral adiposity index.

Table 3:
Stepwise multiple linear regression for prediction of metabolic risk factors from anthropometric measurements in males

Variables	**Model**	**R**	**R^2**	**SEE**	**F**	**P Value**
FBG (mg/dl)	1. FBG= 260.32 (W/H) + (-147.43)	0.77	0.6	15.71	176.83	<0.0001
	2. FBG= 266.07 (W/H) + 106.79(R2D:4D) + (-219.54)	0.78	0.62	15.47	93.59	<0.0001
TC (mg/dl)	1. TC= 358.48 (W/H) + (-145.26)	0.82	0.67	18.74	235.64	<0.0001
	2. TC= 358.53 (W/H) + (-38.83)	0.82	0.68	18.51	122.8	<0.0001
HDL-C (mg/dl)	1. HDL-C =70.74(W/H) + 107.17	0.82	0.68	3.6	248.5	<0.0001
TG (mg/dl)	1.TG= 340.51(W/H) + (-186.41)	0.79	0.62	19.6	194.32	<0.0001
	2.TG= 340.56(W/H) + (0.69)(Height) + (-70.41)	0.8	0.64	19.33	102.11	<0.0001
LDL-C (mg/dl)	1. LDL= 361.12(W/H) + (-215.15)	0.82	0.67	18.69	240.57	<0.0001
VAI	1.VAI= 19.56 (W/H) + (-13.90)	0.844	0.71	0.92	291.81	<0.0001
DBP (mmHg)	1. DBP=104.64 (W/H)+(-10.14)	0.66	0.43	9.34	200.78	<0.0001
SBP (mmHg)	1. SBP= 200.94(W/H) + (-50.00)	0.78	0.6	12.66	403.44	<0.0001
	2. SBP= 173.15(W/H) + 133.66(R2D:4D)+(-153.57)	0.8	0.64	12.02	238.47	<0.0001

FBG: fasting glucose, TC: total cholesterol, HDL-C: high density lipoprotein, TG: triglyceride, LDL-C: low density lipoprotein, VAI; SBP; systolic blood pressure, DBP; systolic blood pressure, W/H: waist-to-hip ratio, R2D:4D; right second to forth digit ratio, L2D:4D; left second-to-forth digit ratio, SEE: standard error of estimate.

Table 4:
Stepwise multiple linear regression for prediction of metabolic risk factors from anthropometric measurements in females

Variables	**Model**	**R**	**R^2**	**SEE**	**F**	**P Value**
FBG (mg/dl)	1. FBG= 326.05 (W/H) + (-191.68)	0.792	0.63	21.59	65.53	<0.0001
TC (mg/dl)	1. TC= 466.70 (W/H) + (-231.10)	0.9	0.81	19.18	170.09	<0.0001
	2. TC= 354.95 (W/H) + 405.41(L2D:4D) (-533.76)	0.93	0.86	16.79	117.34	<0.0001
HDL- (mg/dl)	1. HDL-C =65.30(W/H) + 106.37	0.83	0.68	3.84	82.87	<0.0001
TG (mg/dl)	1. TG= 299.08(W/H) + (-146.30)	0.87	0.75	14.79	117.41	<0.0001
LDL-C	1. LDL-C= 472.19(W/H) + (-308.21)	0.91	0.82	18.66	183.84	<0.0001
(mg/dl)	2. LDL-C= 366.03(W/H) + 385.1(L2D:4D)+ (-595.72)	0.93	0.87	16.47	124.02	<0.0001
VAI	1. VAI= 18.06 (W/H) + (-11.74)	0.875	0.77	0.86	127.37	<0.0001
DBP (mmHg)	1. DBP=81.83 (W/H)+ 14.58	0.68	0.46	9.56	168.22	<0.0001
	2. DBP=59.16 (W/H)+ 0.99(BMI) +12.14	0.74	0.55	8.72	121.38	<0.0001
	3. DBP=56.75 (W/H)+ 0.98(BMI) + 62.06(R2D:4D) + (-46.95)	0.76	0.58	8.52	88.43	<0.0001
	4. DBP=47.18 (W/H)+ 1.46(BMI) + 59.70(R2D:4D) + (-0.54)(BAI)+ (-32.66)	0.77	0.59	8.38	70.24	<0.0001
SBP (mmHg)	1. SBP= 153.98(W/H) + (-0.90)	0.76	0.58	14.29	266.53	<0.0001
	2. SBP= 141.32(W/H) + 158.99(L2D:4D)+(-146.87)	0.79	0.62	13.62	157.3	<0.0001
	3. SBP= 138.48(W/H) + 163.26(L2D:4D)+(-0.44) (Height) (-79.30)	0.8	0.63	13.32	112.89	<0.0001
	4. SBP= 121.99(W/H) + 165.36(L2D:4D)+(-0.45) (Height) + 0.71(BMI)+ (-80.43)	0.81	0.65	13.05	90.6	<0.0001
	5. SBP= 226.38(W/H) + 120.64(L2D:4D) + 7.63(BMI)+ (-2.62) (Weight) + (-7.68)(BAI) + 561.75(W/Ht) + 1.26(NC) + (51.20)	0.84	0.71	12.08	65.39	<0.0001

FBG: fasting glucose, TC: total cholesterol, HDL-C: high density lipoprotein, TG: triglyceride, LDL-C: low density lipoprotein, SBP; systolic blood pressure, DBP; systolic blood pressure, W/H: waist-to-hip ratio, R2D:4D; right second to forth digit ratio, L2D:4D; left second to forth digit ratio, SEE: standard error of estimate.

Stepwise multiple linear regressions (Tables 3 and 4) for predicting MRF shows that WHR was the strongest predictor of all the components of metabolic risks including visceral adiposity but with varying percentages of accuracy. For visceral adipose tissue estimation in the regression equation, WHR had 71% and 77% accuracy in males and females respectively and the SEE of 0.92 and 0.86 for males and females respectively (P<0.001). The regression equation showed the weakest percentage accuracy for DBP in both sexes. In males R^2 = 43%, SEE = 9.3 (P<0.001). However, in females DBP was predictable in a 2-step regression equation and the equation with the strongest prediction strength showed that DBP is predicable from both BMI and WHR with R^2 = 55%, SEE = 8.72 and P <

0.001. For the serum components, the equation with the highest predictive strength which explored only WHR in female was observed for LDL–C and TC (81% and 82% respectively) while the lowest was for FBG in males (63%). For males WHR had the highest estimation ability for HDL-C (68%) and the lowest for FBG (60%). There was however little contribution from R2D:4D in FBG estimation for males and contribution from L2D:4D in LDL-C and TC estimation in females. For BP prediction, there were also contributions from digit ratio, digit length and other anthropometric indices.

Overall VAI, BP and serum indices of metabolic risk were predictable from WHR alone or in addition to other anthropometric measurements in a linear regression model.

DISCUSSION

This observational study was conducted to investigate the predictive strength of digit ratio (2D:4D) and anthropometric measures of adiposity for visceral adiposity reserve, blood pressure and serum indices of metabolic risks among the Hausa ethnic group of Kano, Nigeria. The idea of predicting MRF from anthropometric measures of adiposity is rooted to the widely documented strong correlations between body adiposity reserves and MRF (Mathieu et al., 2009; Whitlock et al., 2009; Eckel et al., 2010; Simmons et al., 2010; Okamkpa et al., 2016). This is thought to be mediated by the role of adipose tissue in insulin resistance theory which is a major step in the pathogenesis of adverse metabolic indicators (Fujioka et al., 1987; Lara-castro et al., 2007; Ghantous et al., 2015).

However, 2D:4D on the other hand is a prenatally determined anthropometric variable whose development is essentially influenced by sex hormone (estrogen and progesterone) and genetic factors has only been shown recently to demonstrate strong and significant correlations with body adiposity measures (Asuku et al., 2018) and determinants of metabolic risk (Asuku et al., 2019; Ranvider and Manju, 2016). It is currently speculated that the genetic and hormonal determinants of 2D:4D may similarly be implicated in the development of body adiposity phenotypes. This may therefore explain why 2D:4D have demonstrated some predictive potential for MRF as observed in the current study.

The current study is therefore unique as it attempts to come up with a novel idea of pulling all the adiposity measures that have been documented to show strong relationships with MRF together with 2D:4D in a linear regression equation to predict BP, FBS, TC, LDL-C, HDL-C and VAI

The observation from the present study that WHR was the most consistent anthropometric predictor of MRF in the predictive equation may be explained from the point of view that the various anthropometric measures of adiposity shows different discriminatory powers for MRF (Pischon et al., 2008; MacKay et al., 2009). Even though wide ethnic and racial variations have been documented on the usefulness of different measures of body adiposity in estimating metabolic risks (Mbanya et al., 2015), more recent studies (Asuku et al., 2016; 2017; 2018) suggest that in most ethnic/racial groups, the anthropometric indices of central adiposity exemplified by WHR, WC and WHtR are the most implicated in the development of MRF. This is theoretically believed to be due to the close association between the amount of visceral adipose tissue reserve (a major factor in the development of insulin resistance) and values obtained from anthropometric measurements of central adiposity. This therefore implies that, WHR is the most relevant anthropometric measure of adiposity among the Hausa ethnic group of Kano which is in keeping with the findings of similar investigators amongst other racial and ethnic groups (Mbanya et al., 2015). This also agrees with the findings of Asuku et al. (2018) which revealed that WHR shows the strongest correlation with metabolic syndrome indices among the Hausa ethnic group. This finding is also strengthened by the observation in this study that WHR alone demonstrated a good predictive power for VAI which is the hallmark of metabolic syndrome phenotype,

Contrary to the findings of the current study, there are a few reports indicating that WHtR and other newer anthropometric measures of body adiposity such as BAI are more useful to some ethnic population (Bergman et al., 2011a). This suggests that ethnic specific factors significantly influences the pathophysiologic interplay between body adipose tissue reserve and MRF and also strengthens the current global recommendation (Tulloch-Reid et al., 2003) that every ethnic/racial population should strive to identify its own germane anthropometric determinants of MRF.

Interestingly, the present study identified 2D:4D as a useful anthropometric tool that contributes to the adiposity measures in MRF prediction. Since 2D:4D is established in utero by both genetic and hormonal factors and remains unchanged throughout life (Çelik et al., 2010; Umut et al., 2015), it is likely that certain phenotypes of digit ratio may have similar genetic determinants with those that predisposes to MRF. This finding on the contribution of 2D:4D to MRF prediction has added to the wide pool of body traits with which digit ratio has been documented to strongly correlate with and has identified 2D:4D as a simple, easily measurable anthropometric tool that may be useful to clinicians in predicting metabolic risks.

The variations observed in the predictive power of WHR for different measures of metabolic risk may suggest that even though WHR is superior to other adiposity tools in MRF prediction, it however demonstrates a discriminatory potential for different indices of metabolic risks. Thus the observations from this study that 2D:4D alone has strongest prediction for LDL-C in females (R^2 = 82%) and weakest prediction for DBP in males (R^2 = 47%).

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