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Obesity, metabolic syndrome and BMI-metabolic-risk sub-phenotypes: A study of an adult Nigerian population

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Obesity and the metabolic syndrome are health care challenges of not only the industrialized nations but also of the developing countries. BMI-metabolic-risk sub-phenotypes separate obesity from its metabolic consequences. These indices have not been duly studied in Nigeria. One hundred and ninety nine adult Nigerians (52.3% females) were studied. Obesity and metabolic syndrome were defined using World Health Organization and US National Cholesterol Education Program Adult Treatment Panel III criteria, respectively. The presence or absence of the metabolic syndrome within the 3 BMI groups (normal, overweight and obese) was used to define 6 BMI-metabolic-risk sub-phenotypes. The results show that 12.1% (13.7% for males and 10.6% for females) of the population were obese. Metabolic syndrome was found in 30.8% (males 34.7%; females 26.9%) of the population. In the obese and overweight subjects, 33.3% and 40.9% respectively were metabolically healthy while 37.6% of the normal weight subjects were metabolically obese. BMI-metabolic-risk sub-phenotypes were found at the rates of 4%-34.2% in the entire population. The results are compared to figures from other studies, and discussed in the light of their implications for a country like Nigeria that is still battling with communicable diseases. Lifestyle modifications that encourage physical exertion and appropriate nutrition are advocated.

Key words: BMI-metabolic-risk sub-phenotypes, metabolic syndrome, obesity

INTRODUCTION

Obesity has become a growing health problem globally, but more importantly in the developing countries where chronic diseases battle with communicable diseases for an often meager healthcare budget (Reddy, 2002; Kengne *et al*., 2005). It confers risk of morbidity and mortality from type 2 diabetes and atherosclerotic cardiovascular disease (CVD) and other chronic diseases (Flegal *et al.,* 2005; Meigs *et al.,* 2006). The measurement of BMI as a universal criterion of overweight (BMI≥25, but <30) and obesity (BMI≥30) has been recommended by the World Health Organization (WHO, 2000).

Visceral fat accumulation which often accompanies obesity, leads to a cascade of metabolic disturbances, often referred to as the metabolic syndromes (Mokdad *et al.,* 2003; Carr and Brunzell, 2004). The US National Cholesterol Education Program (NCEP) Adult Treatment Panel 3 (ATP III) defines the metabolic syndrome as a cluster of three or more of the following (1) abdominal obesity (waist circumference >102 cm in men and >88 cm in women) (2) concentration of triglycerides ≥150 mg/dl (3) concentration of HDL-cholesterol < 40 mg/dl in men and <50 mg/dl in women (4) blood pressure ≥130/85 mmHg and fasting glucose ≥110 mg/dl (NCEP, 2001). Other definitions of the syndrome, with slight variations are also available (Ford, 2005a). The etiology of the metabolic syndrome is still largely unknown, but it is thought to represent a complex interaction among genetic, metabolic and environmental factors (Groop, 2000; Lidfeldt *et al.,* 2003). Though BMI is known to be related to the metabolic syndrome, the relationship may not always be a dose-response relationship (Meigs *et al.,* 2006).

Sub-phenotypes of obesity, that appear to separate obesity from its apparent metabolic consequences have been described. The metabolically obese normal-weight

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(MONW) individual, despite having a normal-weight BMI, still demonstrates metabolic disturbances typical of obese individuals (Ruderman *et al.,* 1998; St. Onge *et al.,* 2004); while the metabolically healthy obese (MHO) individual lacks most of the metabolic abnormalities typical of obesity, yet has a BMI exceeding 30 kg/m^2 (Brochu *et al.,* 2001; Kraelis *et al.,* 2005). Recently, attention has been drawn to the presence of individuals with these BMI-metabolic-risk sub-phenotypes in Nigeria (Ejike *et al.,* 2009).

The present study aims to describe the prevalence and characteristics of obesity, metabolic syndrome and BMI-metabolic-risk sub-phenotypes in Umuahia, Nigeria. The results, hopefully, would be useful in public health policy formulation and action at least in Nigeria.

MATERIALS AND METHODS

Subjects

Adults (22-84 years old) living in Umuahia were studied. A total of 199 subjects (95 males and 104 females) participated in the study. Participants were randomly approached and the study explained to them individually. Those who orally consented to participation and who had no overt signs of ill-health or who were not pregnant (for women) were allowed to participate in the study. The ethics committee of the Federal Medical Centre Umuahia and the board of the Department of Biochemistry, Michael Okpara University of Agriculture, Umudike, both in Abia state, Nigeria, approved the study and its design.

Instruments and Measures

Blood pressure was measured on a single visit, using sphygmomanometry and appropriate cuff sizes, with the subject in a sitting position, and having rested for at least 10 minutes. Three separate readings were taken per subject, after two minutes intervals and the average of the second and third readings recorded. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken at the 1st and 5th Korotkoff sounds respectively. The same trained personnel took all blood pressure measurements. Weights and heights of participants were taken, with participants dressed in light clothing, and BMI calculated as the weight (kg) divided by the square of the subjects' height (m).

Self-reported age at the last birthday was recorded per participant. Based on their ages, and taking into consideration that age (≥45 years for men, and ≥55 years for women) is one of the risk factors for coronary artery disease (CAD) listed by NCEP (NCEP, 2001), we grouped subjects into age ranges as follows: 22-44 years, 45-54 years, 55-64 years and ≥65 years.

Fasting blood samples (4 ml) was drawn from each participant, and a drop used to measure the concentration of fasting blood glucose by the glucose oxidase method (Washako and Rice, 1961). The rest was allowed to stand at ambient temperature until clotting took place, and the serum separated by centrifugation for 5 minutes at 1000 x g. From the serum, total cholesterol, HDL-Cholesterol and triglycerides were measured by enzymatic colorimetric methods (Allain *et al.,* 1974; Lopes-Virella *et al.,* 1977; Tietz, 1990). LDL-Cholesterol was measured by difference (Friedwald *et al.,* 1972).

Definitions

We defined normal weight as BMI>18.5 but <25, overweight as BMI between 25 and 29.9, and obese as BMI≥30 (WHO, 1995). We defined metabolic syndrome according to the NCEP ATP III definition (NCEP, 2001) but used BMI cut-offs recommended by Schneider et al (2007) (BMI ≥ 26.5 for males and BMI ≥ 25.8 for females) in place of waist circumference as a measure of obesity.

Subjects with the metabolic syndrome were classified as metabolically obese while those without the syndrome were classified as metabolically healthy. Combining these with the 3 BMI groups (normal weight, overweight and obese) 6 BMI-metabolic-risk sub-phenotypes were defined as: Metabolically Healthy Normal Weight (MHNW); Metabolically Obese Normal Weight (MONW); Metabolically Healthy Over-Weight (MHOW); Metabolically Obese Over-Weight (MOOW); Metabolically Healthy Obese (MHO); and Metabolically Obese Obese (MOO).

Statistical analyses

Descriptive statistics on all the data generated was done (age and sex-wise or BMI and metabolic syndrome-wise) and reported as means ± standard deviations. Differences between means were separated by post hoc tests with the least significant difference fixed at 0.05. We calculated the prevalence of the different disorders as the number of such cases divided by the number of subjects in that category, and the answer multiplied by 100. Descriptive statistics and group comparisons were done using SPSS for windows version 11.0 (SPSS Inc Chicago IL) while bar charts were generated using Microsoft Excel 2003 (Microsoft Corporation US).

RESULTS

The characteristics (clinical and anthropometric) of the studied population are displayed in Table 1. A total of 54.8% (49.5% for males and 59.6% for females) had a normal BMI, while 12.1% (13.7% for males and 10.6% for females) were obese (Figure 1). More males, compared to females, had metabolic syndrome (Figure 2). In both sexes, the proportion of population with metabolic syndrome increased with increasing age. For males, it increased more than 5-folds from age 22-44 years (9.7%) to age ≥ 65 (50.0%), while for females, it increased more than 2-folds from age 22-44 years (18.2%) to age ≥ 65 (41.7%).

Figure 3 shows that as much as 37.6% (36.2% for males and 38.7% for females) who had a normal weight were metabolically obese. This however represents 20.6% (17.9% for males and 23.1% for females) of the entire population (Figure 4). Again Figure 3 shows that 33.3% (23.1% for males and 45.5% for females) who were obese actually had a healthy metabolic profile. However, this represents only 4.0% (3.2% for males and 4.8% for females) of the entire population. Females clearly had better metabolic profiles than males. The characteristics of the population within the 6 BMI-metabolic-risk sub-phenotypes are presented in Table 2.

Table1. Characteristics of the population, stratified by age and gender

Age (Years)	BMI		Chol (mg/dl)	LDL (mg/dl)	TAG (mg/dl)	HDL (mg/dl)	FBG (mg/dl)	SBP (mmHg)	DBP (mmHg)
22-44 years									
Total	34.6 ± 6.0	23.7 ± 4.0	180.9 ± 71.0	86.6 ± 65.2	147.4 ± 75.8	66.1 ± 33.6	91.2 ± 47.5	118.1 ± 6.2	77.9 ± 9.7
Males	36.9 ± 5.7	23.6 ± 3.5	190.1 ± 64.9	90.7 ± 63.4	144.3 ± 62.5	71.3 ± 32.0	95.1 ± 46.3	123.3 ± 17.7	80.2 ± 9.7
Females	32.9 ± 5.7	23.8 ± 4.4	174.4 ± 75.1	83.6 ± 67.1	149.5 ± 84.6	62.4 ± 34.6	88.4 ± 48.8	114.3 ± 14.2	76.4 ± 9.4
45-54 years									
Total	49.2 ± 2.8	25.0 ± 4.4	236.5 ± 83.0	113.6 ± 81.7	166.7 ± 74.0	88.1 ± 22.1	168.3 ± 98.9	127.4 ± 26.6	77.8 ± 14.6
Males	49.2 ± 3.1	25.3 ± 4.2	219.9 ± 76.8	96.4 ± 82.5	162.3 ± 77.4	91.2 ± 27.7	194.4±115.7	129.7 ± 27.7	80.9 ± 17.2
Females	49.2 ± 2.4	24.8 ± 4.6	252.4 ± 87.1	130.1 ± 79.1	171.0 ± 72.0	85.2 ± 14.9	143.3 ± 73.5	125.3 ± 25.9	74.8 ± 11.0
55-64 years									
Total	59.9 ± 2.8	25.0 ± 4.4	243.1 ± 89.5	128.2 ± 87.2	171.4 ± 68.1	81.2 ± 31.4	144.4 ± 79.1	133.0 ± 25.0	80.9 ± 13.9
Males	59.7 ± 3.1	25.7 ± 4.2	258.2±101.5	140.1 ± 98.2	175.2 ± 51.7	84.9 ± 30.5	166.6 ± 92.4	138.2 ± 21.0	84.6 ± 13.3
Females	60.2 ± 2.5	24.3 ± 4.6	228.6 ± 75.6	116.8 ± 75.5	167.8 ± 81.7	77.6 ± 32.5	123.1 ± 58.2	128.1 ± 27.9	77.3 ± 13.9
≥ 65 years									
Total	68.3 ± 7.1	26.1 ± 3.7	258.0 ± 90.2	127.4 ± 95.4	168.9 ± 68.5	94.4 ± 22.9	144.7 ± 86.2	142.8 ± 26.6	85.5 ± 21.4
Males	69.3 ± 6.0	26.4 ± 3.9	260.0±108.0	131.3± 110.1	157.4 ± 75.7	96.6 ± 25.0	169.5 ± 95.7	146.6 ± 27.7	90.1 ± 25.3
Females	66.8 ± 8.6	25.7 ± 3.6	255.1 ± 58.3	121.5 ± 72.0	186.2 ± 54.7	91.2 ± 20.0	107.6 ± 54.2	137.1 ± 25.0	78.6 ± 11.4

BMI, Chol, LDL, TAG, HDL, FBG, SBP and DBP represent Body Mass Index, Total Cholesterol, Low density lipoprotein cholesterol, Triacylglycerol, High density lipoprotein cholesterol, Fasting blood glucose, Systolic blood pressure and Diastolic blood pressure respectively.

Only fasting blood glucose concentration, SBP and DBP were significantly p<0.05) higher in the 3 BMI groups with metabolic syndrome (metabolically obese phenotypes) compared to their respective BMI groups without metabolic syndrome. BMI, HDL-Cholesterol concentration and LDL-Cholesterol concentration were similar (p>0.05) in subjects with and without metabolic syndrome, within the same BMI group. Triglycerides concentration was significantly (p<0.05) lower in the MHNW group compared to the that of MONW group, while the other groups had similar (p>0.05) values of triglycerides. Total cholesterol concentration was similar (p>0.05) between those with and without metabolic syndrome in all BMI groups except the obese group where MHO individuals had significantly (p<0.05) lower values of this attribute compared to those of MOO subjects. Age of subjects was similar (p>0.05) only in the 2 overweight groups studied.

DISCUSSION

In this study,we sought to describe the prevalence and characteristics of obesity, metabolic syndrome and BMI-metabolic-risk sub-phenotypes. We found that more than half of the studied population had normal weight BMI while about 45% had undesirable BMI. More males were obese and overweight, compared to the females. These figures agree with a recent study in another part of Nigeria (Ejike *et al.,* 2009) that found obesity in 12.7% of the studied population, and also reported a higher prevalence of obesity in males, compared to that in females. The Nigeria demographic and health survey, 2003

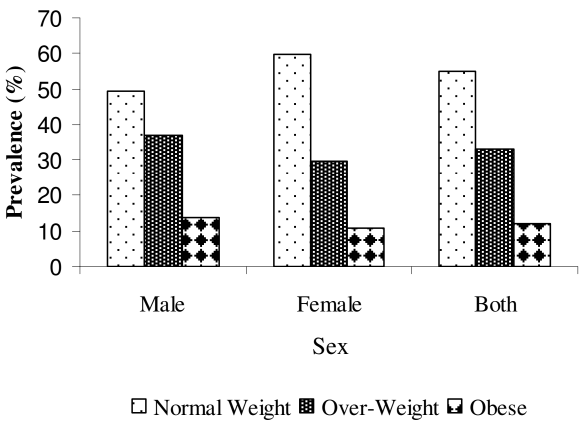

Figure 1. Distribution of BMI in the population.

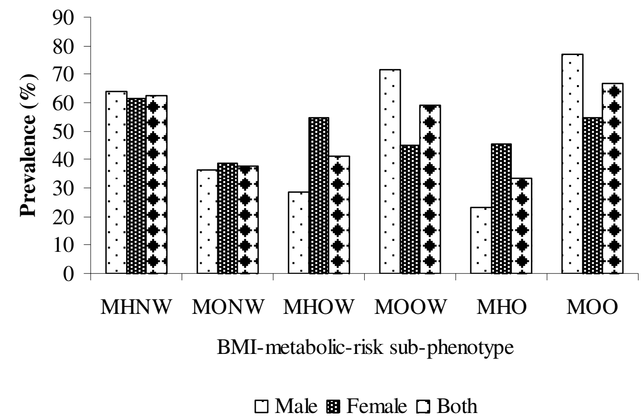

Figure 3. Prevalence of BMI-metabolic-risk sub-phenotypes within the 3 BMI groups.

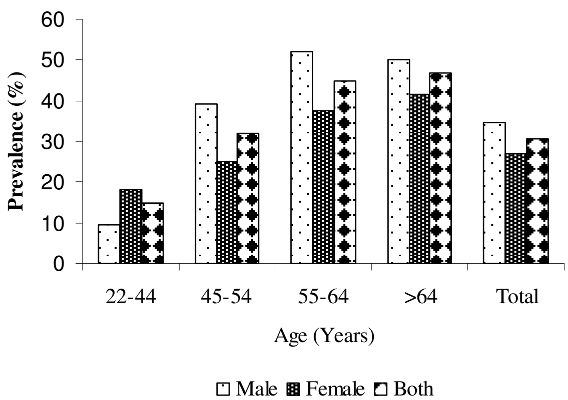

Figure 2. Prevalence of the metabolic syndrome as defined by (NCEP ATPIII) in the population.

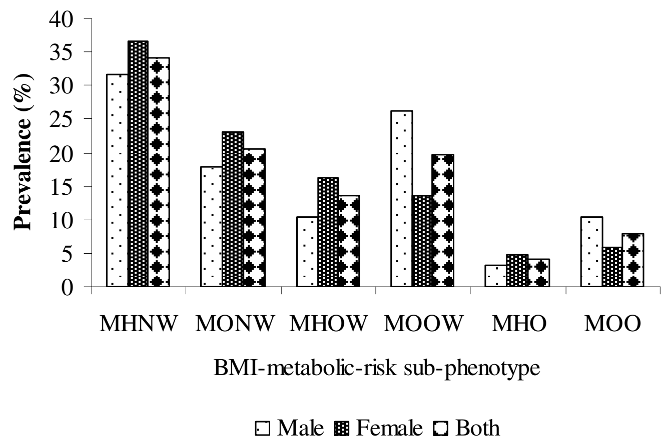

Figure 4. Prevalence of BMI-metabolic-risk sub-phenotypes in the entire population.

(NPC and ORC Marco, 2004) however reported only 15% and 5% overweight and obesity respectively among Nigerian women. BMI is an acceptable measure of nutritional status in adults.

Obesity is largely due to excessive energy intake without a commensurate expenditure rate. The modernization of cultures in Nigeria, and in sub-Saharan Africa, the improving standards of living and less need for physical exertion due to the availability of energy sparing devices, all of which characterize the modern environment, promote behaviors that predispose individuals to obesity (Amoah, 2003). The role of physical exertion in the development of obesity is even made clearer by comparing our BMI data (23.7 ± 4.0 in the youngest age-range and 26.1 ± 3.7 in the oldest age-range) to those of Glew *et al.* (2003) who studied a nomadic Fulani population of Nigeria, subsisting on high saturated fat diets, and yet had a BMI of 20.0 ± 2.2 for men and 20.2 ± 3.0 for women. Though our data show a higher prevalence of obesity in males than in females, the mean BMI of both sexes within the age groups were significantly different (p>0.05). Though BMI is associated with mortality and morbidity, it is important to note that it is excessive body fat that is associated with these health risks, and BMI does not measure fat mass or fat percentage (WHO, 1995).

We found the metabolic syndrome in 30.7% of the population. For the males, the disorder increased 5-folds in prevalence from the low risk for CAD group (22-44 years) to the high risk for CAD group (≥45 years) while for the females the increase in the prevalence of the disorder from the low risk for CAD group (22-54 years) to the high risk for CAD group (≥55 years) was 2-folds. There are, at present, at least 4 different definitions of the metabolic syndrome (NCEP, 2001; Alberti and Zimmet, 1998; Balkau and Charles, 1999; IDF, 2008) and more may still come up. This makes comparison between figures slightly difficult, especially across races and cultures. However, the similarities in all the definitions outweigh the differences (Ford, 2005a). We used the NCEP ATP III definition (NCEP, 2001) and our figure is slightly higher than the 27.8% reported in the Framingham Offspring Study (FOS) (Meigs *et al.,* 2006), but lower than the 34.5% reported in the US National Health and Nutrition

Table 2. Characteristics of the population, stratified by the presence or absence of the metabolic syndrome, within the three BMI categories

	BMI < 25			BMI 25-29.9			BMI ≥ 30		
	No MetS (MHNW)	Yes MetS (MONW)	p	No MetS (MHOW)	Yes MetS (MOOW)	p	No MetS (MHO)	Yes MetS (MOO)	p
Age (Years)	46.2 ± 13.8	*55.3 ± 9.3	0.024	47.3 ± 12.6	53.4 ± 9.3	0.065	44.1 ± 17.2	*59.8 ± 10.2	0.007
BMI	21.5 2.1	22.0 ± 2.1	0.368	27.1 ± 1.4	22.0 ± 2.1	0.325	32.3 ± 2.4	31.9 ± 1.6	0.596
Chol (mg/dl)	208.9 ± 72.1	252.2 ± 66.5	0.098	214.8 ± 105.7	252.2 ± 66.5	0.091	165.2 ± 44.9	*243.0 ± 81.9	0.036
LDL (mg/dl)	100.8 ± 66.4	143.0 ± 81.9	0.083	110.4 ± 100.2	143.0 ± 81.9	0.135	45.7 ± 24.2	98.5 ± 71.2	0.126
TAG (mg/dl)	162.0 ± 73.1	*222.4 ± 66.3	0.006	143.2 ± 68.3	222.4 ± 66.3	0.215	103.1 ± 83.5	163.3 ± 81.8	0.051
HDL (mg/dl)	75.9 ± 30.0	64.7 ± 18.9	0.224	78.0 ± 27.0	64.7 ± 18.9	0.897	92.6 ± 29.7	105.3 ± 29.0	0.328
FBG (mg/dl)	110.2 ± 64.5	*173.4 ± 103.0	0.006	108.3 ± 74.4	*173.4 ± 103.0	0.001	70.1± 31.5	*209.5 ± 93.0	<0.001
SBP (mmHg)	116.5 ± 14.4	*160.2 ± 28.6	<0.001	122.5 ± 21.1	*160.2 ± 28.6	<0.001	121.0 ± 18.3	*150.0 ± 13.3	0.001
DBP (mmHg)	75.3 ± 9.8	*89.8 ± 21.7	<0.001	76.0 ± 11.4	*89.8 ± 21.7	<0.001	79.3 ± 9.0	88.8 ± 7.4	0.093

MetS, MHNW, MONW, MHOW, MOOW, MHO and MOO represent Metabolic syndrome, Metabolically Healthy Normal Weight, Metabolically Obese Normal Weight, Metabolically Healthy Overweight, Metabolically Healthy Overweight, Metabolically Healthy Obese and Metabolically Obese Obese. BMI, Chol, LDL, TAG, HDL, FBG, SBP and DBP represent Body Mass Index, Total Cholesterol, Low density lipoprotein cholesterol, Triacylglycerol, High density lipoprotein cholesterol, Fasting blood glucose, Systolic blood pressure and Diastolic blood pressure respectively. * indicates significant mean difference compared to the corresponding metabolically healthy group.

Examination Survey (NHANES) 1999-2002 (Ford, 2005a). The prevalence figure may increase slightly if the International Diabetes Federation (IDF) definition were used, as was the case in the US (Ford, 2005a). The prevalence of the metabolic syndrome in males, but not females, in our study compares with that of Ford (2005a). The prevalence figure from both the FOS and NHANES studies were derived using the NCEP ATP III definition, making comparison with our figures easy. Obesity, elevated blood glucose concentration, hypertension and (to a lesser extent) elevated triglycerides concentration appear to be the major determinants of the metabolic syndrome in our population (see Table 2).

Metabolic syndrome has been shown to be a good surrogate indicator for insulin resistance in predicting the risk and prognosis of cardiovascular diseases (Ford, 2005b; Li *et al.,* 2007) for insulin resistance has been suggested as a possible mechanism for metabolic syndrome (Reaven, 1988). Our data therefore suggests that a large proportion of our population (>30%) are at risk of CVD's – more than 20% of the women at low risk for CAD (age-wise) fall into this group. These figures also indicate the lifestyle patterns of this population, and calls for urgent public health action since the metabolic syndrome is a potentially modifiable risk state for CVD's (McKeown *et al.,* 2004).

Our study confirms the high prevalence of different BMI-metabolic-risk sub-phenotypes in Nigeria. Our data is in tandem with a recent study in Nigeria that found that 33% of the obese population was metabolically healthy (Ejike *et al.,* 2009). The slight difference being that 45.5% of the obese females (as against 40.0% in the said study) and 23.1% of obese males (as against 26.5% in the said study) were metabolically healthy. Our figure of 4.0% for MHO phenotype in the entire population is a lot lower than 11-28% reported in other populations (Ferrannini *et al.,* 1997; Bonora *et al.,* 1998; Kraelis *et al.,* 2004; Iacobellis *et al.,* 2005). Methodological differences however make these comparisons difficult.

We found the MONW phenotype in 37.6% of normal weight subjects, and 20.6% of the entire population. Unlike the earlier study in Nigeria (Ejike *et al.,* 2009), more females (38.7%) than males (36.2%) of the normal weight BMI group were metabolically obese. The 37.6% prevalence of MONW within the normal weight BMI group is stunningly higher than the 8.6% reported by Ejike *et al* (2009). The difference may likely be the definition of metabolic syndrome in both studies which varied markedly. Irrespective of the definition of metabolic syndrome, the prevalence of MONW phenotype in other populations is put at 3-28% (Ferrannini *et al.,* 1997; Bonora *et al.,* 1998; McLaughlin *et al.,* 2004; St. Onge *et al.,* 2004). Our figure of 20.6% for MONW phenotype in the general population falls within this range.

Our study further shows that 40.9% of the overweight BMI group (13.6% of the entire population) were metabolically healthy. It has been proposed that the metabolic consequences of elevated BMI are the chronic diseases associated with fatness (Meigs *et al.,* 2006). The importance of this for our study is that as much as 37.6% of those with normal weight-BMI, who ordinarily appear to have low risk for these chronic diseases are actually at high risk, while 40.9% and 33.3% of those who were overweight and obese respectively actually have a healthy metabolic profile and may not be at high risk for chronic diseases.

Caution must be exercised in interpreting these data especially for the MHOW and MHO phenotypes, as the measurement of sub-clinical inflammation, endothelial dysfunction or adiponectin might reveal that they have less than healthy metabolic states (Festa *et al.,* 2000; Kraelis *et al.,* 2005; Meigs *et al.,* 2004). Meigs *et al* (2006) also suggested that follow-up longer than 7-11 years might be required to be certain that obese subjects without metabolic risk factors are indeed at low risk. Our small sample size also implies limited statistical power to make appropriate inferrals. The small sample size was because we studied people in a community that culturally view blood as synonymous with life, and as such resist, often vehemently, to cooperate with researchers that require their blood. Our detailed and standard measures and studying a clearly un-studied population (with respect to the studied metabolic disturbances) are the strengths of this study.

Conclusion

In conclusion, we found obesity in 12.1% of the population, metabolic syndrome in 30.7% of the population and BMI-metabolic-risk sub-phenotypes at prevalence rates of 4.0-34.2% of the population. Lifestyle modifications that emphasize good nutrition and physical exertion to check these trends in a rapidly modernizing society like ours are advocated.

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REFERENCES

Alberti KG, Zimmet PZ (1998). Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Provisional report of a WHO consultation. Diabet. Med. **15**:539-553

Allain CC, Poon LS, Chan CSC, Richmond W, Fu PC (1974). Enzymatic colorimetric method for cholesterol estimation. Clin. Chem. 20:470-475

Amoah AGB (2003). Socio-demographic variations in obesity among Ghanaian adults. Public Health Nutr. 6: 751-757

Balkau B, Charles MA (1999). Comment on the provisional report from the WHO consultation: European Group for the study of Insulin Resistance (EGIR). Diabet. Med. 16: 442-443

Bonora E, Kiechi S, Willet J, Oberhollenzer F, Egger G, Targher G, Alberiche M, Bonadonna RC, Muggeo M (1998). Prevalence of insulin resistance in metabolic disorders. Diabetes 47:1643-9

Brochu M, Tchernof A, Dionne IJ, Sites CK, Eltabbakh GH, Sims EA and Poehlman ET (2001). What are the physical characteristics associated with normal metabolic profile despite having a high level of obesity in post menopausal women? J. Clin. Endocrinol. Metab. 86:1020-25

Carr MC, Brunzell JD (2004). Abdominal obesity and dyslipidemia in the metabolic syndrome: importance of type 2 diabetes and familial combined hyperlipidemias in coronary artery disease risk. J. Clin. Endocrinol. Metab. 89: 2601-2607

Ejike CECC Ugwu CE, Ezeanyika LUS (2009). Nutritional status, prevalence of some metabolic risk factors for cardiovascular disease and BMI-metabolic-risk sub-phenotypes in an adult Nigerian population. Biokemistri 21:17-24

Ferrannini E, Natali A, Bell P, Cavallo-Perin P, Lalic N, Mingrove G (1997). Insulin resistance and hypersecretion in obesity. European Group for the study of Insulin Resistance (EGIR). J. Clin. Invest. 100: 1166-1173

Festa A, D'Agostino Jr R, Howard G, Mykkanen L, Tracy RP, Haffner SM (2000). Chronic subclinical inflammation as part of the insulin resistance syndrome: the Insulin Resistance Atherosclerosis Study (IRAS). Circ. 102: 42-7

Flegal KM, Graubard BI, Williamson DF, Gail MH (2005). Excess deaths associated with underweight, overweight and obesity. JAMA 2931861-1867

Ford ES (2005a). Prevalence of the metabolic syndrome defined by the International Diabetes Federation among adults in the US. Diabetes Care 28:2745-2749

Ford ES (2005b). Risks for all-cause mortality, cardiovascular disease, and diabetes associated with the metabolic syndrome; a summary of the evidence. Diabetes Care 28:1769-1778

Friedewald WT, Levy RI, Fredickson DS (1972). Estimation of the concentration of LDL cholesterol in plasma, without use of the preparative ultracentrifuge. Clin. Chem. 18:499-502

Glew RH, Willams M, Conn CA, Cadena SM, Croseey M, Okolo SN and VanderJagt DJ (2003). Cardiovascular disease risk factors and diet of Fulani pastoralists of northern Nigeria. *Am J Clin Nutr.* **74:** 730-6

Groop I (2000). Genetics of the metabolic syndrome. Br. J. Nutr. 83(Suppl 1): S39-S48

Iacobellis G, Ribaudo MC, Zappaterreno A, Iannucci CU, Leonetti F (2005). Prevalence of uncomplicated obesity in an Italian obese population. Obesity Res **13**: 1116-1122

International Diabetes Federation (2008). The IDF consensus world wide definition of the metabolic syndrome. Available online at http://www.idf.org/webdata/docs/Metabolic_syndrome_definition.pdf

Kengne AP, Amoah AGB, Mbanya JC (2005). Cardiovascular complications of diabetes mellitus in sub-Saharan Africa. Circ. 112:3592-601

Kraelis AD, Faraj M, Bastard JP, St. Pierre DH, Brochu M, Prud'homme D, Rabasa-Lhoret R (2005). The metabolically healthy but obese individual presents a favourable inflammation profile. J. Clin. Endocrinol. Metab. 90: 4145-4150

Kraelis AD, St-Pierre DH, Conus F, Rabasa-Lhoret R, Poehlman EJ (2004). Metabolic and body composition factors in subgroups of obesity: what do we know? *J Clin Endocrinol Metab* **89**: 2569-75

Li C, Ford ES, McGuire LC and Mokdad AH (2007). Association of metabolic syndrome and insulin resistance with congestive heart failure: findings from the third national health and nutrition examination survey. J. Epidemiol. Comm. Health 61:67-73

Lidfeldt J, Nyberg P, Nerbrand C, Samsioe G, Schersten B, Agardh CD (2003). Socio-demographic and psychosocial factors are associated with features of the metabolic syndrome: the Women's Health in the Lund Area (WHILA) study. Diabetes Obes. Metab. 5:106-112

Lopes-Virella MF, Stone P, Ellis S (1977). Cholesterol determination in high density lipoprotein separated by three different methods. Clin. Chem. 23:882

McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PWF, Jacques PF (2004). Carbohydrate nutrition, insulin resistance and prevalence of the metabolic syndrome in the Framingham Offspring Cohort. Diabetes Care 27:538-546

McLaughlin J, Allison G, Abbasi F, Lamendola C and Reaven G (2004). Prevalence of insulin resistance and associated cardiovascular disease risk factors among normal weight, overweight and obese individuals. Metab. 53:495-499

Meigs JB, Hu FB, Rifai N, Manson JE (2004). Biomarkers of endothelial dysfunction and risk of type 2 diabetes mellitus. JAMA 291:1978-1986

Meigs JB, Wilson WFP, Fox CS, Vasan RS, Nathan DM, Sullivan LM, D'Agostino RB (2006). Body mass index, metabolic syndrome and risk of type 2 diabetes or cardiovascular disease. J. Clin. Endocrinol. Metab. 91:2900-2912

Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, Marks JS (2003). Prevalence of obesity, diabetes and obesity-related health risk factors, 2001. JAMA 289**:**76-79

National Cholesterol Education Program (NCEP) (2001). Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) JAMA 285:2486-2497

National Population Commission (NPC) [Nigeria] and ORC Macro (2004). Nigeria demographic and Health Survey 2003. Calverton, Maryland pp 21-26.

Reaven GM (1988). Banting lecture 1988. Role of insulin resistance in human disease. Diabetes 37:1595-1607

Reddy KS (2002). Cardiovascular diseases in the developing countries: dimensions, determinants, dynamics and directions for public health action. Public Health Nutr. 5:231–237

Ruderman N, Chisholm D, Pi-Sunyer X, Schneider S (1998). The metabolically-obese normal weight individual revivited. Diabetes 47:699-713

Schneider HJ, Glaesmer H, Klotsche J, DETECT Study Group (2007). Accuracy of anthropometric indicators of obesity to predict cardiovascular risk. J. Clin. Endocrinol. Metab. 92:589-594

St. Onge MP, Janssen I, Heymsfield SB (2004). Metabolic syndrome in normal weight Americans: new definition of the metabolically obese, normal weight individual. Diabetes Care. 27:2222-2228

Tietz NW (1990). Clinical guide to laboratory tests. 2nd edition. WB Saunders Company, Philadelphia, USA. pp. 554-556

Washako ME and Rice EW (1961)**.** Determination of glucose by an improved enzymatic procedure. Clin. Chem 7:542-545

World Health Organization (WHO) (1995). Physical status: the use and interpretation of anthropometry. Report of a WHO expert committee. WHO Tech Rep Ser 854:1-452

World Health Organization (WHO) (2000). Obesity: preventing and managing the global epidemic: report of a WHO consultation. WHO Tech Rep Ser 894:1-253