CVD Prevention and Control (2010) 5, 75—80

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Low HDL-cholesterol with normal triglyceride levels is the most common lipid pattern in West Africans and African Americans with Metabolic Syndrome: Implications for cardiovascular disease prevention

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Received 18 April 2010; received in revised form 22 July 2010; accepted 26 July 2010

KEYWORDS
Metabolic Syndrome;
Insulin resistance;
Triglycerides;
Dyslipidemia;
African Americans;
West Africans

Summary
Background: Although designed to predict cardiovascular disease and type 2 diabetes mellitus, the Metabolic Syndrome (MetSyn) under-predicts these conditions in African Americans (AA). Failure of MetSyn in AA is often attributed to their relative absence of hypertriglyceridemia. It is unknown if the African experience with MetSyn will be similar or different to that in AA. Focusing on the lipid profile, our goal was to determine in West Africans (WA) and AA the pattern of variables that leads to the diagnosis of the MetSyn.
Methods: Cross-sectional analysis of 1296 subjects (364 WA, 44% male, 932 AA, 46% male). WA were from urban centers in Nigeria and Ghana and enrolled in the Africa America Diabetes Mellitus Study. AA lived in Washington, DC and participated in the Howard University Family Study.
Results: The prevalence of MetSyn was different in WA women and men: 42% vs.19%, $P < 0.001$,

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1875-4570/$ - see front matter Published by Elsevier Ltd. on behalf of World Heart Federation.
doi:10.1016/j.cvdpc.2010.07.003

and in AA women and men: 25% vs.17%, $P < 0.01$. The three variables that most often led to the diagnosis of MetSyn in WA and AA were: low HDL-C, central obesity and hypertension. Less than 40% of AA and less than 25% of WA with the MetSyn had hypertriglyceridemia.
Conclusions: Elevated triglyceride levels were uncommon in both WA and AA with MetSyn. As the relative absence of hypertriglyceridemia is associated with a lack of efficacy of MetSyn in AA, caution is warranted in diagnosing MetSyn in WA, the ancestral population of AA. Prospective studies are necessary to determine if an ethnic-specific reformulation of the MetSyn scoring system for lipids might optimize risk identification in black populations.
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Introduction

Obesity, type 2 diabetes (T2DM), and atherosclerotic cardiovascular disease (CVD) are chronic diseases severely stressing health care systems in Africa [1,2]. In 2006, the International Diabetes Federation (IDF) estimated that 10.8 million Africans had T2DM [1]. By 2025, the number of diabetics in Africa is predicted to be 18.7 million. This 80% increase is much greater than the expected worldwide increase in T2DM of 55% [1]. Additionally, between 2005 and 2030, mortality from CVD is expected to double [2]. Therefore, CVD once rare in Africa, now ranks among the leading causes of death [2].

Due to the triple burden of obesity, T2DM and CVD, effective strategies for prevention are essential. In North America and Europe, the Metabolic Syndrome (MetSyn) was designed as a mathematical construct (3 of 5 factors must be present for a diagnosis) with the intent of achieving early detection of T2DM and CVD. Yet the MetSyn as a distinct entity has generated both acclaim and debate. The American Heart Association, the National Heart Blood and Lung Institute, IDF and the World Health Organization (WHO), citing evidence that the MetSyn carries a 5-fold risk of T2DM and a 2-fold risk of CVD, endorse the use of the MetSyn as a worldwide paradigm to predict the development of CVD and T2DM [3,4]. In contrast, the American Diabetes Association and the European Society for the Study of Diabetes not only dispute the diagnostic effectiveness of the MetSyn, they challenge the validity of the MetSyn concept [5].

Omitted from the debate is a discussion of the controversy regarding the effectiveness of the MetSyn in people of African descent, particularly African Americans (AA). Overall, AA have a higher prevalence of T2DM and CVD than American whites but a lower prevalence of MetSyn [6—11]. This ''Metabolic Syndrome Paradox'' suggests that the MetSyn is less effective in AA than in white Americans in identifying the risk for T2DM and CVD [4,10,12—14]. Similar to the AA experience, the MetSyn is not an effective predictor of CVD in African-Caribbeans [15]. It is unknown if the challenges of the MetSyn in AA and African-Caribbeans, will be predictive of a similar experience of the MetSyn in Africa.

Without adequate African data, it is logical to study AA. The lower than expected prevalence of MetSyn in AA is often attributed to their relative absence of hypertriglyceridemia [4,11,13,14]. While whites with MetSyn usually have hypertriglyceridemia [9,10], AA with MetSyn usually have normal TG levels [9,10]. The lipid pattern in WA with MetSyn is unknown. Primarily focusing on lipids, our goal was to determine the pattern and prevalence of the MetSyn in WA and AA.

Methods

The participants were 1296 (364 WA, 932 AA) unrelated, non-diabetic individuals participating in either the Africa America Diabetes Mellitus (AADM) Study or the Howard University Family Study (HUFS). No subjects were taking lipid lowering medications.

The AADM recruited West Africans from three urban centers in Nigeria: Enugu, Ibadan and Lagos, and two urban centers in Ghana: Accra and Kumasi [16]. The Nigerian participants were Ibo or Yoruba. The Ghanaians were Akan or Gaa. Initially, the AADM study enrolled sib pairs with T2DM and controls. Subsequently, the AADM project was expanded to include other family members of the affected sibs, unrelated cases of T2DM and unrelated controls. Three-hundred and sixty-four non-diabetic, unrelated Nigerian and Ghanaian participants who enrolled between 2001 and 2005 were analyzed in this study as each underwent extensive lipid and glucose evaluations [16]. The AADM study was approved by Institutional Review Boards at the five participating West African universities and Howard University. All subjects gave informed consent.

HUFS is a population-based study of AA living in Washington, DC. The initial sampling frame included the 120 neighborhoods within eight wards of the District of Columbia [17]. To qualify for selection, the population of individual wards had to be at least 50% AA. Based on the US census data, six (wards 1, 4, 5, 6, 7 and 8) of eight wards qualified. A complete listing of all 85 neighborhoods in the selected wards was generated. Using a random number procedure, 43 of the 85 neighborhoods were selected as the sampling unit. All AA meeting the age requirements within these neighborhoods were eligible. Recruitment was achieved with neighborhood canvassing, newspaper advertisements and booths at community fairs. The 932 AA unrelated non-diabetic participants were recruited between 2001 and 2007. The HUFS was approved by the Howard University Institutional Review Board. Participants gave informed consent.

As the AADM and HUFS had the same Principal Investigator (Charles N. Rotimi), similar procedures were followed for data collection. For both protocols, weight was measured in light clothes on an electronic scale to the nearest 0.1 kg. Height was obtained with a stadiometer to the nearest 0.1 cm. Waist circumference (WC) was measured to the nearest 0.1 cm at the narrowest part of the torso. While

sitting, blood pressure (BP) was taken three times at 5 min intervals with an oscillometric device (Omron). The mean of the last two readings was recorded.

Prior to phlebotomy, all subjects fasted for 8 h. Plasma samples were stored on-site in −80 °C freezers in Washington, DC or West Africa. All West African samples were shipped on dry ice to Howard University. Therefore, analyses for the WA and AA participants were performed in a single laboratory at Howard University. Assays for glucose, total cholesterol (T-CHOL), HDL-C and TG measurements were performed using the Auto-analyzer — COBAS INTEGRA 400 plus (Roche Diagnostics, Indianapolis, IN). Howard University follows Centers for Disease Control Reference methods.

Statistical analyses

Statistical analyses were performed using SAS statistical package (SAS Institute Inc., Cary, NC). Frequency differences were evaluated by chi-square. Sex differences were tested for with unpaired *t*-tests. Variables not normally distributed were transformed by log or square root. Significance was defined as $P \leqslant 0.05$.

Results

Subject characteristics are provided in Table 1. Socioeconomic variables differed by ethnicity. Twenty-eight percent of WA were high school graduates and 23% had at least one year of college education. Ten percent were current or past smokers. Sixty-four percent reported ever drinking alcohol. No data on median family income for the WA were available. For the AA, 59% were high school graduates and 36% had at least one year of college education. Median family income was $30,000. Approximately 60% were current or past smokers. About 72% of AA reported ever drinking alcohol.

By the NCEP-ATPIII definition, the difference in prevalence of MetSyn in WA women and men was: 42% vs.19%, $P < 0.001$, and in AA women and men was: 25% vs.17%, $P < 0.01$. The three factors most likely to lead to the diagnosis of MetSyn in WA and AA were: central obesity, hypertension and low HDL-C (Fig. 1). The least common criteria were: hypertriglyceridemia and fasting hyperglycemia (Fig. 1).

Independent of the presence of MetSyn, low HDL-C levels were more common in WA than AA. In every BMI category, mean HDL-C levels in WA were below established thresholds (i.e. men <40 mg/dL, women <50 mg/dL) (Fig. 2). In AA, even though mean HDL-C declined with increasing BMI category, mean HDL-C remained above established risk thresholds (Fig. 2).

Discussion

Due to the relative absence of elevated TG levels in AA, MetSyn under-predicts risk for T2DM and CVD [4,11,13,14]. For the first time, we have demonstrated that WA and AA with the MetSyn are likely to have the same three characteristics, specifically low HDL-C, central obesity and hypertension. Therefore, WA with the MetSyn, like their AA counterparts, are not likely to have hypertriglyceridemia. The MetSyn was designed with the expectation that it would predict the development of T2DM and CVD [4]. As AA have a higher prevalence of T2DM and CVD than whites, but a lower prevalence of the MetSyn, the MetSyn is not optimally diagnosed in AA [6,7,10]. Due to the strong similarity in the distribution

Table 1 Characteristics of the West African and African American participants.[a]

	West Africans			African Americans		
	Total	Men	Women	Total	Men	Women
Variables	*n* = 364	*n* = 159	*n* = 205	*n* = 932	*n* = 428	*n* = 504
Age (years)	46 ± 14	45 ± 16	46 ± 14	45 ± 13	46 ± 13	45 ± 13
Weight (kg)	73.1 ± 15.5	72.5 ± 14.0	73.6 ± 16.6	85.4 ± 21.1	86.9 ± 20.4	84.0 ± 21.5‡
Height (m)	1.64 ± 0.10	1.70 ± 0.08	1.60 ± 0.08‡	1.69 ± 0.09	1.76 ± 0.07	1.64 ± 0.07‡
BMI (kg/m^2)	27.2 ± 5.9	25.2 ± 4.6	28.9 ± 6.3‡	29.9 ± 7.3	28.1 ± 6.2	31.4 ± 7.8‡
% BMI ⩾ 30	29%	15%	40%‡	43%	30%	53%‡
Waist circ. (cm)	91 ± 13	88 ± 12	93 ± 13†	94 ± 15	94 ± 15	95 ± 16
SysBP (mm Hg)	135 ± 23	134 ± 23	135 ± 23	129 ± 21	130 ± 21	128 ± 21
DiaBP (mm Hg)	81 ± 15	81 ± 15	82 ± 14	81 ± 13	82 ± 14	80 ± 13
Glucose (mg/dL)	86 ± 12	86 ± 13	85 ± 11	86 ± 11	87 ± 11	84 ± 11‡
TG (mg/dL)	90 ± 37	93 ± 39	87 ± 35	102 ± 61	105 ± 59	99 ± 62*
HDL-C (mg/dL)	35 ± 15	35 ± 15	36 ± 15	53 ± 17	52 ± 1	55 ± 17†
CHOL (mg/dL)	181 ± 50	171 ± 53	189 ± 46‡	191 ± 42	186 ± 4	195 ± 42†
CHOL/HDL-C ratio	6.1 ± 3.4	5.8 ± 3.0	6.4 ± 3.6	3.9 ± 1.7	4.0 ± 1.5	3.9 ± 1.8
%MetSyn NCEP-ATPIII	32%	19%	42%‡	21%	17%	25%†
%MetSyn IDF	38%	26%	48%‡	24%	21%	27%*

P-value are for comparison of WA men vs. WA women and AA men vs. AA women.

[a] Data are mean ± SD.

* $P \leqslant 0.05$.

† $P \leqslant 0.01$.

‡ $P \leqslant 0.001$.

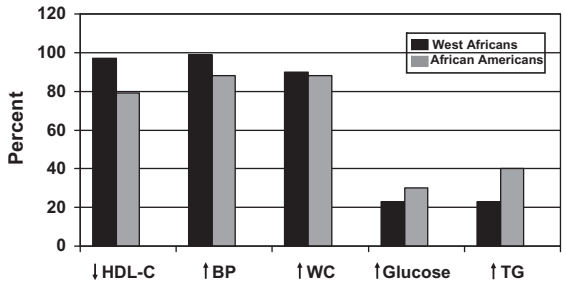

Figure 1 Prevalence of diagnostic variables in West Africans and African Americans with the Metabolic Syndrome.

of variables that lead to the diagnosis of the MetSyn in both WA and AA, it is possible that the MetSyn will also under-predict T2DM and CVD in WA.

Importantly, even with cultural and environmental differences, the presentation of the MetSyn was similar in WA and AA. For example, WA and AA participants did not have the same level of education, alcohol intake or exposure to smoking. Nevertheless, the same three factors, low HDL-C, central obesity and hypertension led to the diagnosis of MetSyn in both groups. As WA are the ancestral population of AA with as much as 70% of the AA gene pool of West African origin [18], the role of genetics must be considered in understanding the lipid profile in people of African descent.

The combination of high TG and low HDL-C levels, are known as the *dyslipidemia of insulin resistance* or the *dyslipidemia of Metabolic Syndrome* [4]. Therefore the TG/HDL-C ratio has been recommended as a marker of insulin resistance [19]. Yet due to ethnic differences in TG levels, the TG/HDL-C ratio is not effective as a marker of insulin resistance in African Americans [20,21]. Further, we report that the majority of WA and AA had only ''half'' of the dyslipidemia of insulin resistance, specifically normal TG and low HDL levels [14]. This pattern of normal TG levels with low HDL levels has been observed even in WA diabetics. Okafor et al. reported that 60% of Nigerian diabetics had low HDL levels, while only 25% had elevated TG levels [22].

Due to the observation that low HDL-C and normal TG levels are characteristic of the MetSyn in WA and AA, we demonstrate the need to reconsider the universality of the concept of the dyslipidemia of Metabolic Syndrome as the combination of low HDL-C and high TG levels. According to well defined pathways, high TG and low HDL-C occur together because the two enzymes that clear these lipoproteins are reciprocally regulated. In the presence of insulin resistance, lipoprotein lipase (LPL) activity is impaired and hepatic lipase activity is enhanced [23]. However, these pathways do not explain the finding of normal TG and low HDL-C in black populations. The term ''black populations'' is used because normal TG levels in the presence of insulin resistance has also been described in African-Caribbeans [24]. Normal TG levels in insulin-resistant AA may be explained by: high levels of LPL activity, LPL activity unimpaired by insulin resistance and low apolipoprotein CIII levels [25–27].

HDL-C levels

Low HDL-C was one of the three most frequent characteristics of the MetSyn in WA and AA. Our investigation demonstrates that even in the absence of MetSyn, low HDL-C levels in WA are common. In fact, over the last 40 years, a decline in

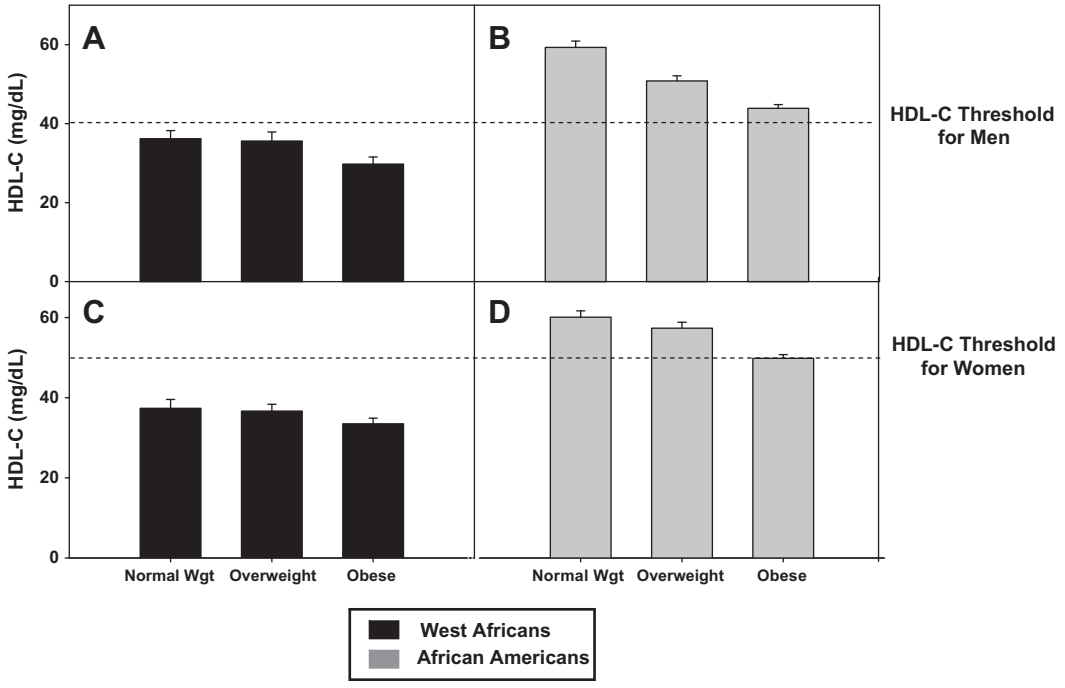

Figure 2 HDL-C concentrations by sex, ethnicity and BMI. Data are presented as mean ± SE. Sex-specific thresholds for HDL are denoted on each panel. Data for West Africans are in Panels A and C. Data for African Americans are in Panels B and D; for men in top panels and women in bottom panels.

HDL-C levels has been gradually occurring in Africa. In 1975 Walker and Walker reported HDL-C levels in Africans between 60 and 70 mg/dL [28]. In the 1980's and 1990's reports appeared revealing that HDL-C levels in Africans were between 40 and 60 mg/dL [29,30]. Then in 2006, Akpa et al. reported that mean HDL-C levels in Nigerians were 35 mg/dL [31]. This fall in HDL-C levels in Africa, documented over a four decade time-span, can be attributed to the urbanization of African populations and the attendant changes in diet and exercise. Our investigation confirms this trend of declining HDL-C levels in urban West Africans. We enrolled Africans from urban centers in Nigeria and Ghana and found that mean HDL-C levels in normal weight, overweight and obese WA were all below threshold (i.e. <40 mg/dL in men and <50 mg/dL in women). Yet in AA, mean HDL-C levels were above these thresholds. As HDL-C has a key role in reverse cholesterol transport as well as anti-thrombotic, anti-inflammatory and anti-oxidant properties, HDL-C is cardioprotective [32]. Therefore, low HDL-C levels in WA may represent a substantial and evolving cardiovascular risk.

However, low HDL-C levels in WA without the MetSyn could be secondary to low total cholesterol levels (T-CHOL). To correct for low T-CHOL levels, the T-CHOL/HDL-C ratio was calculated (data not shown). An elevated T-CHOL/HDL-C ratio is a marker of cardiovascular disease risk, which increases substantially when the ratio is above five [33]. While AA had normal T-CHOL/HDL-C ratios, these ratios were elevated in WA. Concern about high T-CHOL/HDL-C ratios in WA arises because of the findings of INTERHEART Africa Study, a case—control, multi-ethnic, cross-sectional investigation of Africans from nine countries who have experienced an acute myocardial infarction [34]. The INTERHEART Africa investigators measured apolipoprotein B/apolipoprotein A1 (apoB/apoA1) ratios. ApoB and apoA1 are the apolipoproteins on LDL and HDL particles, respectively. ApoB/apoA1 and T-CHOL/HDL-C ratios provide equivalent information about CVD risk. The INTERHEART Africa investigators report a strong association between high apoB/apoA1 ratios and myocardial infarction.

In whites with MetSyn, low HDL-C and high TG levels usually occur together [9,10]. However, we report low HDL-C levels with normal TG levels as the most frequent MetSyn lipid pattern in WA and AA. Therefore, we suggest that the scoring system used to diagnose the ''dyslipidemia of the MetSyn'' be re-evaluated. The WHO definition of the MetSyn is the oldest, the most complicated and least used. Nevertheless, in regard to the dyslipidemia of the MetSyn, the WHO system may have the potential to better define risk in people of African descent than the scoring systems of NCEP-ATPIII and IDF.

The WHO gives one point if TG is elevated or HDL-C is low [4]. Therefore the maximum number of points that WHO gives for the presence of dyslipidemia is one. This means that the WHO system gives the same credit for low HDL-C whether or not TG is elevated. In contrast, NCEP-ATPIII and IDF give two points if HDL-C is low and TG is elevated [4].

Prevalence of the Metabolic Syndrome

The prevalence of the MetSyn was higher in women than men. In addition the prevalence of MetSyn was higher with the IDF definition than the NCEP-ATPIII definition. Interestingly, the prevalence of MetSyn in WA and AA men was similar, but the prevalence of the MetSyn was higher in WA than AA women. As the AADM study was not based on a random sample, the high rate of MetSyn in WA women should be interpreted with caution. Nevertheless, even with this difference in prevalence, the specific variables that led to the diagnosis of MetSyn were similar in WA and AA women.

This investigation is a cross-sectional study. In the absence of studies equivalent to NHANES, Framingham or the Jackson Heart Study, this investigation represents a major first step towards getting baseline data of the characteristics of the Metabolic Syndrome in West Africans. With 364 participants, our study is the largest descriptive study of the MetSyn in West Africa. By comparison, only 144 participants in the multi-ethnic INTERHEART study were black Africans [34]. The black African participants were from nine countries. Seven of the nine countries (Benin, Botswana, Cameroon, Mozambique, Nigeria, Seychelles and Zimbabwe) enrolled fewer than 20 subjects each. By comparison, the current investigation has enrolled 364 unrelated, non-diabetic West Africans from urban centers in only two countries (Nigeria and Ghana). Furthermore a key strength of this investigation is that all lipid analyses were performed in a single laboratory. Therefore the lipid profile in the WA and AA can be compared without the confounding factor of different assays.

West Africans are the ancestral population of AA. As obesity, diabetes, cardiovascular disease and MetSyn are of recent onset in Africa, the AA experience with these conditions may forecast the WA experience. Since the presentation of the MetSyn in WA and AA is similar (low HDL-C, central obesity and hypertension) and MetSyn under-predicts risk for CVD and T2DM in AA, this may occur in WA as well. Under-diagnosis of MetSyn does not mean the absence of risk; rather it means the lost opportunity for early diagnosis and intervention. Therefore, before scarce resources in West Africa are invested for widespread screening of MetSyn, prospective studies to determine its efficacy are essential. Furthermore, as low HDL-C levels are especially prominent in urban West African populations, rigorous population-based prospective investigations are necessary to determine the associated risk and identify the best intervention.

Conflict of interest

None declared.

Acknowledgements

The authors would like to thank Bernard V. Miller III, MD, Ugochi J. Ukegbu and Michael G. Knight for their thoughtful reviews of the manuscript. Anne E. Sumner and Omoye E. Imoisili were supported by the intramural program of NIDDK. Charles N. Rotimi and Jie Zhou were supported by the Intramural Research Program of the Center for Research in Genomics and Global Health, NHGRI, NIH. The HUFS was supported by Grant S06GM008016-320107. African American enrollment was carried out at the General Clinical Research

Center supported by NCRR Grant 2M01RR010284 and the National Human Genome Center at Howard University. The AADM study was supported by grants obtained from NCMHD, NHGRI and NIDDK.

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