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Metabolic Syndrome in Blacks: Are the Criteria Right?

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Abstract Blacks have a lower prevalence of metabolic syndrome (MetS) that can be partly ascribed to the lower prevalent rates of some major components of MetS, namely the lower serum triglycerides and higher high-density lipoprotein cholesterol levels in blacks when compared with whites. Blacks manifest greater insulin resistance, the pivotal lesion underpinning MetS than whites. However, the relationships among insulin resistance and cardiovascular disease (CVD) risk factors are weaker in blacks than whites. The international bodies have recommended the use of European-based cutoff points for MetS for blacks. However, with the emerging inconsistencies in the association of insulin resistance and CVD risk factors in blacks, the use of these definitions and the cutoff points for MetS have become problematic. Therefore, it is important to review the limitations in the use of the current criteria and cutoff points of MetS in blacks to lessen the CVD risk burden in blacks.

Keywords Metabolic syndrome · HDL cholesterol · Triglycerides · Insulin resistance · Blood pressure and obesity · Blacks

Introduction

Recent studies have shown that the pattern of cardiovascular risk factors exhibits substantial heterogeneity and is significantly influenced by ethnicity/race, gender, socioeconomic status, and geographical locations. Blacks living in diverse geographical locations continue to experience higher cardiovascular morbidity and mortality [1, 2••, 3–5•]. Hypertension, congestive heart failure, and socioeconomic status as well as low physical activity have been implicated. Furthermore, recent studies have demonstrated that insulin resistance is associated with cardiovascular disease (CVD) in whites and some non-white populations including Asian Indians [3–5•, 6–11]. In this regard, we have reported higher serum insulin levels and insulin resistance when compared with whites [12–15]. Despite the greater insulin resistance, black individuals with insulin resistance manifest higher high-density lipoprotein cholesterol (HDLC) and lower serum triglycerides when compared with white counterparts [4, 5•, 6, 7, 12–16••]. Blacks have also greater carotid artery intima-media thickness (CIMT), a surrogate for subclinical atherosclerosis, than their white counterparts [7–10, 17]. Recent studies have found lack of significant or weak associations among insulin resistance and serum HDLC-triglycerides ratio [12–16••, 17–24], blood pressure and body composition, [2••, 4, 5•, 6, 12, 14, 22, 25] and CIMT [8–10] in blacks. Thus, current data on MetS are unable to explain the higher and excess CVD deaths in blacks than whites [1, 2••, 3–5•].

The metabolic syndrome (MetS) is defined as the constellation of lipids and lipoprotein, blood pressure and body composition [3, 5•, 11, 14–16••, 17, 18, 25]. Several international bodies have defined the MetS among different ethnic and racial populations with varying prevalent rates [3, 5•, 9, 26–36]. These findings raise concerns as to whether the current metabolic thresholds or criteria defined by National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) [37], International Diabetes

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Federation (IDF) [38], and World Health Organization (WHO) [39] for MetS are valid. In addition, it questions the etiologic notion that insulin resistance is the pivotal lesion underlying MetS in blacks. Furthermore, it also raises doubt as to whether insulin resistance is a universal and necessary prerequisite for MetS in blacks when compared with whites and other racial and ethnic populations. Similar to other ethnic populations, it is also unknown whether the components of MetS carry similar CVD risk burdens in blacks.

Therefore, in the current review, we have compared and contrast the differences in the insulin sensitivity, individual components, and the prevalence of MetS in blacks and whites. We suggest the need for ethnic-specific, new classification and cutoff points of MetS by international bodies for blacks with common genetic ancestry, who reside in diverse geographical locations in the African Diaspora.

Consideration of Components of MetS as Risk Factors for CVD and Type 2 Diabetes in Blacks

MetS has been associated with increasing risk for developing CVD and type 2 diabetes in several populations including blacks [3–5•, 11, 13, 19, 25–36]. The MetS is defined as a constellation of fasting lipids and lipoproteins, glucose, and blood pressure and waist circumference (WC) parameters (Table 1) [37–39]. Different international bodies use different criteria for the definition of MetS, with discordant prevalent rates among different ethnic and racial populations [37–39]. Several studies have reported differences in the prevalence and incidence of MetS based on the criteria used. Whereas the ATP III requires three or more of the five components, the IDF (Table 1) emphasizes WC as a prerequisite in addition to two or more of the other components to define MetS [38]. In contrast, the WHO (Table 1) recommends insulin resistance or its surrogates (impaired fasting glucose, impaired glucose tolerance, and type 2 diabetes) as the prerequisite in addition to two or more components for the definition of MetS [39]. Furthermore, the IDF has provided ethnic-specific values for WC for central obesity in various populations, but the IDF recommended the use of European cutoff points for the diagnosis of MetS in all blacks including those living in Sub-Saharan Africa. Based on the higher CVD outcomes in blacks despite the lower rates of MetS [3–5•, 6, 7, 16••], we surmised that there are racial/ethnic differences in the impact of the five components of MetS for future CVD and type 2 diabetes. Nevertheless, currently, there are no ethnic-specific criteria or cutoff points for MetS in blacks to address the disparity between the prevalence and incidence of MetS and CVD outcomes in blacks.

Prevalence of MetS in Blacks in Diverse Geographic Locations

Using ATP III criteria and the 2000 US census data, 25% (~ 47 million) of US adults had MetS [26–30]. When comparing NHANES (National Health and Nutrition Examination Survey) I and II (1974–1988 vs 1978–1984 data), the prevalence of MetS increased from 23% to 26%, respectively [26]. The NHANES III also revealed that racial/ethnic differences exist in the prevalence and incidence of MetS in the United States. According to NHANES III, the prevalence of MetS was 13.9% for African American men and 20.9% for African American women during the years from 1988 to 1994 [26–29]. The corresponding rates of MetS were 25% in white men and 23% in white women for the same time period.

There is evidence suggesting increasing prevalence and incidence of MetS in blacks residing in Sub-Saharan Africa, but the rates vary significantly among different geographical regions of Africa [5•, 31–36]. The reasons for the racial/ethnic and geographic differences in CVD risk factors and lower MetS in blacks remain unknown. The emerging data available from sub-Saharan Africa on the prevalence and incidence of MetS are limited to rural and urban populations. For example, a study of urban Tanzanian subjects found 38% of their subjects met at least three of the criteria for MetS using the IDF definition [32]. In another study of Botswana hospital health care workers, 34% were found to have MetS using the IDF criteria [33], with a high prevalence of obesity (28.7%) and overweight (27.3%) in their study. In another study by Fezeu et al. [34], MetS varied according to urban (range, 0.2% to 7.3%) versus rural (range, 0% to 1.9%) for various definitions in Cameroon. Kalk et al. [5•] found in type 2 diabetic patients a higher prevalence of MetS using the IDF definition in black and white South Africans (46.5% vs 74.1%, respectively). In a population of overweight and obese South African women, Jennings et al. [31] found the prevalence of MetS to range from 5.3% to 21.9% using the ATP III or IDF definition. Recently, Kelliny et al. [36] found the prevalence of MetS to vary between the three definitions in populations of Africans in the Seychelles Islands of East Africa with 24%, 25%, and 25.1% in men and 32.2%, 24.6%, and 35.4% in women using the ATP III, WHO, and IDF definitions, respectively. In their study, 80% of subjects with diabetes also had the MetS. In population-based cross-sectional study, Tillin et al. [3] studied Europeans, Afro-Caribbeans, and South Asians residing in London, UK. The authors compared the ethnic and sex differences and their association with prevalent coronary heart disease (CHD) using the WHO and NCEP ATP III definitions of MetS. The MetS was highest in South Asians and lowest in Europeans; MetS was threefold

Table 1 Metabolic syndrome definitions

	ATP III [37]	IDF [38]	WHO [39]
Risk factors and defining criteria	> 3 Risk factors	Abdominal obesity (WC) plus any 2 of the following:	Insulin resistance (T2D, IFG, and IGT) plus any 2 of the following:
Abdominal obesity:	WC:	WC:	WC:
Men	> 40 in (> 102 cm)	> 38 in (> 94 cm)	> 40 in (> 102 cm)
Women	> 35 in (> 88 cm)	> 30 in (> 80 cm)	> 35 in (> 88 cm)
Triglycerides	> 150 mg/dL	> 150 mg/dL	> 150 mg/dL
HDLC:	< 40 mg/dL	< 40 mg/dL	< 40 mg/dL
Men	< 50 mg/dL	< 50 mg/dL	< 50 mg/dL
Women			
Blood pressure	> 130/85 mm Hg	> 140/90 mm Hg or drug use	> 140/90 mm Hg or drug use
Fasting glucose	> 100 mg/dL	Insulin resistance (T2D, IGT, and IFG)	
BMI		> 30 kg/m^2 and WHR>0.9 (men) and>0.85 (women)	> 30 kg/m^2 and WHR>0.9 (men) and>0.85 (women)
Urinary albumin		> 20 mg/min: Alb/Cr>30 mg/g	> 20 mg/min: Alb/Cr>30 mg/g

Alb/Cr Albumin/creatinine; *ATP III* Adult Treatment Panel III; *BMI* Body mass index; *HDLC* High-density lipoprotein cholesterol; *IDF* International Diabetes Federation; *IFG* Impaired fasting glucose; *IGT* Impaired glucose tolerance; *T2D* Type 2 diabetes; *WC* Waist circumference; *WHO* World Health Organization; *WHR* Waist-to-hip ratio

higher in Afro-Caribbean women than white women and 1.5 times higher for the men counterparts. Most importantly, MetS was associated with prevalent CHD in whites and South Asians and not in Afro-Caribbeans. The studies in triethnic populations confirmed ethnic and gender differences exist in the prevalence of MetS and their association with MetS. Therefore, these observations call for a careful and systematic study of the current international criteria for MetS in blacks.

Paradox of Insulin Resistance and MetS and Its Components in Blacks

The major premise underpinning MetS is insulin resistance in the general populations. Although the etiology of insulin resistance remains uncertain in most populations, the relationships among several components of MetS and insulin resistance are well established in whites, but remain controversial in blacks [2••, 3–5•, 6, 7, 11–13, 19–24]. The reasons are unclear. In this regard, blacks are more insulin resistant and are more hyperinsulinemic than their white counterparts [4, 5•, 6–8, 10–12, 22, 23]. Most importantly, unlike whites, the relationships between insulin resistance and blood pressure (be it casual or ambulatory) remains weak at best in blacks [2••, 4, 7, 12, 14, 16••, 25, 40]. This relationship is referred to as the "blood pressure and insulin resistance paradox in blacks" [25]. This weak relationship also extends to other lipids and lipoprotein metabolism in blacks. We have referred to this phenomenon as the "insulin resistance and HDLC/triglyceride paradox in blacks" [15, 16••]. These studies show that the association of components of MetS and insulin resistance remains paradoxical, and indeed controversial, in blacks than whites. Thus, we believe understanding the etiologies of the insulin resistance and/or the metabolic pathways of components of MetS could explain in part the ethnic/racial disparities in CVD outcomes among blacks and whites.

Paradox of Insulin Resistance, Body Composition, and MetS in Blacks

Obesity has become epidemic in blacks in the Western world in the United States, the United Kingdom, South Africa, and West Indies [41–43]. This contributes significantly to CVDs in blacks and whites [1, 2••, 3–5•, 6–10]. In particular, obese blacks, especially black women, have two to four times greater rates of CVD morbidity and mortality than white women. These have been attributed partly to higher rates of overweight/obesity, type 2 diabetes, hypertension, strokes and congestive heart failure, and physical inactivity associated with obesity in blacks [1, 2••, 31, 41, 42]. Paradoxically, it is well established that the severity of angiographically documented coronary artery disease (CAD) is less in obese African Americans and Afro-Caribbeans than in whites, despite the higher CVD mortality and morbidity in blacks [3, 9, 10]. The reasons are unclear and deserve further elucidation.

Globally, obesity is a major health problem and on the rise in blacks residing in urban areas in diverse regions including the United States, South Africa, the United

Kingdom, Africa, and West Indies [30, 31, 33–35, 41–45]. Apart from body mass index (BMI), WC is reported to be greater predictor of MetS in several populations, although the cut off points vary according to the IDF criteria [31, 33–35]. In blacks, WC is the most common component of MetS [11, 14–16••, 18]. WC serves as a surrogate for intra-abdominal visceral adipose tissue (VAT) and an important determinant of insulin resistance and its concomitants for CVD in non-black populations [44–47]. However, this concept or hypothesis is more applicable and relevant in several non-black populations, but remains controversial in blacks. For identical BMI, recent studies showed that black adults and youth residing in diverse geographic locations have lower visceral adiposity despite increased insulin resistance when compared with whites [44–47]. These have been confirmed in urban South African blacks [31, 44, 45]. The reasons are uncertain, but paradoxical. Thus, there is a dissociation between insulin resistance, body fat distribution, and composition in blacks consistent with the insulin resistance and VAT paradox in blacks. Whether differences in the VAT explain the racial and ethnic differences in MetS in blacks and whites remain to be investigated.

What Is the Relevance of HDLC Levels and CVD in Blacks?

HDLC is a well-established antiatherogenic lipoprotein in several racial and ethnic populations. These CVD outcomes studies were mostly in white and non-black populations, but remain uncertain in blacks. Serum HDLC levels are generally greater in blacks than whites [4, 7, 11, 19–24]. Thus, it can be theoretically assumed that blacks should have lower rates as well as severity of CAD; however, this is not the case. Based on our previous study and those in the literature, it is tempting to conclude that to achieve the presumed antiatherogenic effects of HDLC, blacks may require HDLC levels far in excess of 50 mg/dL. However, because the HDLC isoforms and function were not often studied in the previous CVD outcome studies, it is unknown whether the measured serum HDLC levels are truly cardioprotective. Theoretically, it also can be speculated that HDL may be dysfunctional and less cardioprotective/antiatherogenic in blacks. In this regard, investigating not only the quantity, but the quality (eg, antioxidant, anti-inflammatory properties, paraoxonase enzyme activity) of HDL function is warranted in blacks. Finally, if HDLC levels are not cardioprotective, it is possible that other factors (eg, lipoprotein [a], low-density lipoprotein [LDL] subfractions, oxidative stress burden, proinflammatory cytokines) might mitigate against the potential CVD beneficial effects of HDLC in blacks [48–50].

What Is the Relevance of Serum Triglycerides in CVD in Blacks?

Generally, high serum triglycerides are common in insulin-resistant, obese humans with and without type 2 diabetes than in nondiabetic, insulin-sensitive subjects. Some previous pharmacologic intervention studies targeting reduction of higher serum triglycerides (with concomitant rise in HDLC) were very beneficial to the CVD outcomes (including morbidity and mortality). However, such studies have not been performed exclusively in large samples of blacks with hypertriglyceridemia. We [12–16••] and others [18–23] have previously shown that blacks manifest greater insulin resistance and hyperinsulinemia when compared with whites. Despite the insulin resistance, blacks paradoxically have relatively lower serum triglyceride levels when compared with their white counterparts [12–16••, 17–23]. Recently, Fezeu et al. [34] examined the metabolic components of MetS in native Africans residing in Cameroon. The authors concluded that hypertriglyceridemia was nonexistent in their patients. In addition, there was no rural and urban gradient in the levels of triglycerides in their study. Previously studies have reported the association of postprandial glucose and triglyceride with CAD. With respect to blacks, Ntyintyane et al. [48] examined the role of postprandial lipemia in blacks with and without CAD. The population was also classified according to the presence of MetS. Black patients with CAD, with and without MetS, had similar fasting postprandial and lipid responses to oral fat tolerance test. However, MetS with fasting triglycerides greater than 1.7 mmol/L (136 mg/dL) had greater postprandial responses and higher area under the curve (AUC) than patients with fasting triglycerides less than 1.7 mmol/L (136 mg/dL). But postprandial AUC for lipemia was associated with the presence of CAD. These studies suggest that postprandial lipemia may be a better predictor of CAD than fasting triglycerides, independent of MetS in blacks. Unlike the fasting state, the postprandial lipemia was higher in blacks with CHD than in the controls. Therefore, we have postulated that postprandial lipemia appears to be a better predictor of CHD. This interesting concept needs further investigation in a large sample of blacks. Although the reasons for the low serum triglycerides in blacks remain unclear, it has been postulated that hepatic lipase activity may be increased in blacks [22]. However, the mechanism(s) of the apparently lower serum triglycerides levels and their CVD outcomes remain elusive in blacks.

What Is the Relevance of Blood Pressure in MetS in Blacks?

Hypertension is a well-established independent risk factor for CVD morbidity and mortality in blacks residing in

diverse geographical locations. In addition, there are gender, racial/ethnic, and geographical variations in the prevalence of hypertension. The NHANES III reported that over 65 million US adults have hypertension, with African Americans being disproportionally affected when compared with Caucasians [2••, 26–29]. The prevalence of hypertension is approximately 40% in blacks and 22% in whites [2••, 27]. In addition, African Americans with hypertension have a 4.6-fold increased risk of CVD and chronic kidney disease–related deaths compared with their normotensive counterparts.

With increasing rates of obesity globally, the prevalence of hypertension is increasing in blacks in African Diaspora. We [12, 14, 25] and others [2••, 40, 51–53] have shown that blood pressure is the second most common predictor of MetS in blacks. In this regard, the prevalence of hypertension ranges from 25% to 40% in blacks in diverse geographic locations with a rural-urban gradient. Regarding black immigrants, Agyemang and Bhopal [40] found that African-Surinamese men and women were two to four times more likely than their white-Dutch counterparts to have hypertension. Previous studies from the United Kingdom examining the prevalence of hypertension have consistently found higher rates of hypertension in both immigrant Africans [12, 13] and African Caribbean [3, 9], South Africans [5•, 31, 44, 45] and urban Tanzanian men and women [28]. Further, the prevalence of hypertension is higher in native Cameroonians residing in the urban versus rural areas [30, 42].

The cause of hypertension in blacks is unknown. It has been attributed to genetic and environmental factors. Blood pressure is a major component of MetS defined by most international bodies. Recently, there has been increased interest in the association between insulin resistance and blood pressure in blacks [2••, 5•, 10–13, 25]. However, unlike Europeans, we [12–16••] and others [21–23, 31, 40, 44, 45, 51, 52] have reported that the relationship between insulin resistance and blood pressure is very weak in blacks. This has also been found in South Africans [5•, 24, 31, 44, 45] and native Ghanaians [42, 53], Nigerians [54], urban Tanzanians [32] and African Caribbeans in the United Kingdom [6, 9, 10]. Furthermore, Fezeu et al. [34] found that the associations of homeostasis model assessment of insulin resistance (an index of insulin resistance) with systolic and diastolic blood pressure were not significant after adjusting for BMI in Cameroons. In this regard, it has been shown that MetS is associated with greater echocardiographic left ventricular mass in blacks. This can be attributed in part to the higher prevalence of blood pressure in the MetS group consistent with our recent observation [25], NHANES [26–30], and ARIC (Atherosclerosis Risk in Communities) [7, 17, 51]. Therefore, the conventional notion that insulin resistance is pivotal for hypertensive events does not appear to be supported in blacks, in contrast with those of white populations. Thus, we have termed the dissociation between insulin resistance and blood pressure as an "insulin resistance blood pressure paradox in blacks" [25].

What Is the Relevance of the Components in MetS in Blacks?

The international bodies defining MetS have recommended the use of European definition and cutoff points for blacks including those residing in Sub-Saharan Africa. Using these recommendations, blacks often have higher levels of HDLC than their white counterparts [5•, 32, 36]. Thus, theoretically, blacks should have lower rates of CVD and CHD based on traditional lipids and lipoprotein risk factor estimation than whites; however, this is not the case. Preliminary studies have shown that MetS is not associated with CHD in blacks [3, 5•, 6–11, 19–22, 33]. In the face of insulin resistance, blacks have paradoxically lower serum triglycerides and higher HDLC levels when compared with whites [12, 13, 19–24]. Thus, the extraordinarily lower levels of serum triglycerides (in the face of elevated HDLC) in obese, insulin-resistant blacks when compared with whites should theoretically result in decreased CAD and CVD outcomes; however, this is not the case in nondiabetic blacks. We have recently shown that the prevalence of MetS is higher in black females with the first tertile of HDLC (38.1 ± 4.2 mg/dL, 42.3%) and the third tertile of glucose (103 ± 24 mg/dL, 42.3%), and least with the third tertile of serum triglycerides (146±57 mg/dL, 17%) [15].

We [12, 13, 35, 37] and others [4, 5•, 6–17, 19–23, 47] have reported a weaker association between insulin resistance and triglycerides in African Americans. Furthermore, Sumner et al. [22, 23] and Haffner et al. [4, 49] showed that the increasing serum triglycerides are not associated with changes in serum total cholesterol and LDL cholesterol levels in African American women. In a recent pilot study [15], we observed that insulin resistance did not change with increasing serum triglyceride levels from the first to the third tertiles for serum triglyceride levels (albeit within normal limits) in obese African American women. However, we were again surprised by the extremely low levels of serum triglycerides, despite the marked obesity as assessed by BMI (35 kg/m^2) and WC (> 102 cm) and percent body weight (40%) in our nondiabetic African American women. Lower serum triglycerides levels have been reported in insulin-resistant, nondiabetic, obese black South African women when compared with white South African women [31, 44, 45], native Ghanaian women [42, 53], and Cameroonians [43].

Thus, in blacks with insulin resistance, serum triglycerides appear to be a less powerful predictor of MetS. In this regard, we have found that the third tertile of serum triglycerides is associated with only 17% prevalence of MetS in nondiabetic black women in the United States. Thus, abnormalities in serum triglycerides were the least predictors of MetS in nondiabetic blacks [15, 25].

MetS and Proinflammatory Factors in Blacks

Proinflammatory cytokines are peptides that are generally and predominantly derived from adipose tissues and adipocytes associated with MetS. They are associated with truncal obesity and increased visceral adiposity [44, 45, 55–59]. Most importantly, it is believed that these adipocyte-derived cytokines and peptides could be the link between obesity, insulin resistance, MetS, hypertension, type 2 diabetes, and CVDs. Some studies in blacks have shown higher levels of these fat tissue–derived peptides including tumor necrosis factor-α, resistin, leptin, interleukin-6, C-reactive protein than whites [55–59]. In contrast, serum adiponectin, a 244 aa peptide, also solely derived from adipose tissues, is a potent endogenous insulin sensitizer. Adiponectin has been shown to be associated inversely with insulin resistance, hyperlipidemia, blood pressure, MetS, CAD, and type 2 diabetes and could potentially prevent type 2 diabetes, hypertension, and atherosclerosis [45, 46]. Although these are clearly shown in genetic or knockout mice models, there is evidence that adiponectin prevents type 2 diabetes and hypertension in humans.

Recently, several investigators have reported ethnic/racial differences in adiponectin and its isomers. In this regard, serum adiponectin levels are lower in blacks than whites [44, 52–54]. Whether the lower serum adiponectin levels in blacks contribute to the higher rates of type 2 diabetes, hypertension, and CVD remain unknown. Thus, we hypothesized that adipocytokines, and in particular adiponectin, could play a significant role in the development of CVD and type 2 diabetes in blacks.

Justification for a New Classification and Cutoff Points of the Components of MetS in Blacks

We have attempted to address the inconsistencies and disparities in each of the five components for MetS in blacks of African Diaspora and whites and other ethnic populations. This issue is very important because the current NCEP-ATP III criteria assume that all five ATP III parameters equally identify cardiovascular risk factors or predict CVD outcomes. This is in contrast with the IDF and WHO criteria in which some components are prerequisites. We summarized the cutoff points for MetS of the three international bodies in Table 1. We also provided some references of published data on the components of MetS in blacks to justify the need to reconsider new cutoff points for MetS in blacks (Tables 2 and 3). First, we recently reported that the CVD risk burden associated with the five components of MetS varies in blacks in the United States. We [12–16••, 25] and others [18–24] showed that a decreased HDLC and elevated fasting serum glucose levels are associated with higher MetS, whereas triglycerides are the least predictors in black women (Tables 2 and 3). Furthermore, we showed that blood pressure is an independent determinant of MetS in blacks [12, 25]. Second, given the favorable lipoprotein metabolic profiles in blacks, we suggest that the thresholds or cutoff points of metabolic factors that may be associated with CVD appear to be different in blacks when compared with current NCEP-ATP III/IDF/WHO criteria.

Conclusions

The dissociation of CVD risk factors in the face of insulin resistance has raised several concerns regarding the criteria of MetS and cutoff points for its components in blacks. We suggested that the impact of the various components of MetS and MetS on CVD affects different ethnic/racial groups differently. In blacks of African Diaspora, the components of MetS such as WC, blood pressure, HDLC, and serum glucose appear to carry much greater predictive value, whereas triglycerides are the least predictor for MetS in blacks. These issues are very important when viewed in light of the higher HDLC levels and lower triglyceride levels, which do not appear to be cardioprotective among blacks when compared with whites.

Regarding MetS, we believe the current definitions, criteria, and cutoff points underestimate the burden of components of MetS in blacks. Thus, it is important to reconsider the current classification of MetS by the NCEP, WHO, and IDF in blacks. We are convinced that a new criteria (Tables 2 and 3) as defined by various international bodies in blacks could have significant impact on CVD morbidity and mortality in blacks. To this end, long-term prospective studies to investigate CVD outcomes using a modified criteria versus established criteria and cutoff points for MetS in blacks residing in diverse geographic locations (including Sub-Saharan Africa) are urgently warranted. Such putative new cutoff points could have the potential for early detection and thus prevention of CVD and its outcomes and type 2 diabetes in blacks.

Table 2 Percentage of blacks with MetS from various studies

Study (population)	% with MetS	Triglycerides (≥ 150 mg/dL), %	Low HDLC (M<40 mg/dL, W<50 mg/dL), %	Glucose (≥ 100 mg/dL), %	WC (M > 102 cm, W>88 cm), %	HTN (> 130/85 mm Hg), %
Park et al. [27]	M: 13.9	M: 25.3	M: 22.3	M: 11.2	M: 23.5	M: 50.1
	W: 20.9	W: 15.3	W: 40.6	W: 14.1	W: 68.6	W: 40.5
Ford et al. [28]	M: 16.4 ± 1.1	M: 21.4	M: 22.6	M: 14.5	M: 23.3	M: 49.6
	W: 25.7 ± 1.3	W: 14.4	W: 34.0	W: 15.5	W: 62.1	W: 43.3
St-Ong et al. [18] (BMI, 25.0–26.9 kg/m^2)	17.5	M: 24.6	M: 27.2	M: 9.1	M: 4.0	M: 41.5
		W: 21.9	W: 31.9	W: 11.5	W: 47.7	W: 41.0
McNeill et al. [51]	M: 26.9	M: 16.5	M: 25.9	M: 13.7	M: 24.9	M: 61.6
	W: 17.8	W: 10.9	W: 30.7	W: 16.1	W: 72.3	W: 61.1
Burchfiel et al. [17]	M: 13.1	M: 39	M: 60	M: 21	Not reported	66.5
	W: 14.5	W: 38	W: 50	W: 24		71.8
Taylor et al. [35]	M: 32.7	M: 20.1	M: 33.1	M: 29.9	M: 43.7	M: 69.9
	W: 43.3	W: 14.5	W: 39.5	W: 29.9	W: 76.5	W: 70.7
Gaillard et al. [15] (women)	35.5	17	42.3	42.3	71	SBP: 29.8
						DBP: 25
Meis et al. [14]	32.3	2	M: 20	2	M: 16	SBP: 8
			W: 49		W: 58	DBP: 10
Tillin et al. [3]	M: 26.7	Dyslipidemia:		DM: IGT:	M: 73.1	M: 38.1
African-Caribbean	W: 26. 4	M: 22.3		M: 18.4 45.7	W: 60.3	W: 47.8
Europeans	M: 14.6	W: 13.8		W: 18.8 50.6	M: 73.9	M: 24.2
	W: 6.8	M: 38.7		M: 6.9 27.3	W: 30.1	W: 20.7
		W: 25.9		W: 4.2 21.3		
Jennings et al. [31]	ATP: 10.4	6.7	64.9	6.8	ATP: 75.4	25
South African	IDF: 12.9				IDF: 84.9	
Black women (3rd tertile)						
Njelekela et al. [32] (Africa)	38	84	38	6	23	57
Kelliny et al. [36] (Seychelles Islands, East Africa)	ATP: 24.0	12.6	38.2	33.3	35.2	51.7
	IDF: 25.1			33.9	55.8	

ATP III Adult Treatment Panel III; *BMI* Body mass index; *DBP* Diastolic blood pressure; *DM* Diabetes mellitus; *HDLC* High-density lipoprotein cholesterol; *HTN* Hypertension; *IDF* International Diabetes Federation; *IGT* Impaired glucose tolerance; *M* men; *MetS* metabolic syndrome; *SBP* systolic blood pressure; *W* women; *WC* waist circumference

Table 3 Means±SD values of metabolic syndrome components in blacks from various studies

Study (population)	Age, *y*	Triglycerides, *mg/dL*	HDLC, *mg/dL*	Glucose, *mg/dL*	WC, *cm*	BP, *mm Hg*
Zhu et al. [30]	41.05	NA	M: 52.0 W: 56.9	M: 96.9 W: 96.2	M: 92.3 W: 92.9	M: SBP, 126.4; DBP, 78.0 W: SBP, 121.6; DBP, 73.9
Burchfiel et al. [17]	59±6	M: 121 ± 96 W: 113±61	M: 50 ± 17 W: 60 ± 18	M: 122 ± 58 W: 121 ± 56	M: 99 ± 12 W: 104 ± 16	M: SBP, 131 ± 21; DBP, 79 ± 11 W: SBP, 131 ± 21; DBP, 76 ± 10
Palanippan et al. [29] (28.9% blacks)	54.4±8.5	116.1 ± 70.5	49.5 ± 15.3	96.7 ± 10.4	87.4 ± 1	SBP: 119.8 ± 16 DBP: 76.9±9
Haffner et al. [49]	55.5±0.4	102.1 ± 1.0	47.0±0.8	122.5±3.1	94.0±0.8	SBP: 129.3±0.8 DBP: 79.2 ± 0.5
Diabetes Prevention Program (DPP) [20]	50.3	M: 117 W: 99.2	M: 44.1 W: 51.2	NA	BMI, 32.0 kg/m^2	NA
Carnethon et al. [11]	24.9±3.6	M: 67.8±40.5 W: 61.8 ± 28.7	M: 54.0 ± 13.3 W : 55.6 ± 12.6	M: 83.3 ± 10.0 W: 80.1 ± 17.1	M: 79.9±9.1 W: 76.0 ± 12.5	M: SBP, 115 ± 10.4; DBP, 70.7 ± 10.0 W: SBP, 107.9±9.6; DBP, 67.2 ± 9.3
Tzou et al. [8] (young adults)	32 ± 3	M: 119 ± 123 W: 83±45	M-52 ± 23 W-57 ± 16	M: 80±9 W: 70 ± 10	M: 90 ± 18 W: 89 ± 17	M: SBP, 119 ± 12; DBP, 78 ± 10 W: SBP, 115 ± 15; DBP, 76 ± 11
Sumner et al. [23]	35±8	77±39	48 ± 10	86±8	98 ± 18	SBP: 117 ± 14 DBP: 70±9
Meis et al. [14]	40±8	77±38	M 49 ± 1 W 53 ± 13	78 ± 16	M: 94 ± 15 W: 93 ± 18	SBP: 116 ± 11 DBP: 73±9
Gaillard et al. [15] (women)	42.4±8.4	88±54	50.6 ± 13.2	83.5 ± 20.3	99.3 ± 18.3	SBP: 122 ± 17 DBP: 76 ± 11
Davis et al. [52] (type 2 diabetics)	51 ± 78	88±56	47.6 ± 10	163.8 ± 118	BMI, 27.0 kg/m^2	SBP: 137 ± 19 DBP: 85 ± 10
Kalra et al. [9]	52.6 ± 10.5	88±56	60 ± 16	95.4 ± 19.8	BMI: WHR:	SBP: 130.7 ± 16.3; DBP: 80.1 ± 7.5
UK African-Caribbean	52.1 ± 10.1	96±56	64 ± 16	93.6 ± 21.6	28.8 0.86	SBP: 127.6 ± 20.3; DBP: 77.1 ± 12.5
Jamaican African-Caribbean					27.6 0.85	
Jennings et al. [31] (South African women, 3rd tertile)	26 ± 1	67.2 ± 3.2	48 ± 20	84.6 ± 1.8	100.7 ± 1.8	SBP: 112 ± 2 DBP: 76 ± 1
Kruger et al. [60] (South Africa)	45–54	100.8	96 ± 27.2	86.4 ± 28.4	81.8 ± 13.6	SBP: 137.8 ± 24.5 DBP: 85.7 ± 14.9
Strain et al. [10]	55 ± 1	96±72	53.6 ± 1.6	91.8±84.6	89 ± 1	SBP: 127 ± 2 ; DBP: 79 ± 1
Europeans African-Caribbean	53 ± 1	72 ± 56	50.4 ± 1.2	91.8±86.4	90 ± 1	SBP: 155±3 ; DBP: 87 ± 1
Ntyintyane et al. [48] (South Africans with CAD with MetS)	54.4±9.0	144 ± 20	44.8 ± 10	96.7 ± 13.7	104±7.2	96% on hypertensive medications
Kelliny et al. [36] (Seychelles Islands, East Africa)	42.0 ± 10.8	80±0.0	56±0.5	106.2 ± 2.0	89.5 ± 13	SBP: 127.1 ± 18.6 DBP: 83.3 ± 11.8
Fezeu et al. [34] (African men)	46 ± 14	38.4 ± 1.0	Not measured	73.8 ± 1.8	79.6±0.5	SBP: 118.1 ± 1.1; DBP: 72.9±0.9
Rural	38±9	42.4 ± 1.0		77.4 ± 1.8	84.4±0.4	SBP: 123.3±0.9; DBP: 81.8±0.7
Urban						

BMI Body mass index; *BP* Blood pressure; *CAD* Coronary artery disease; *DBP* Diastolic blood pressure; *HDLC* High-density lipoprotein cholesterol; *M* men; *MetS* metabolic syndrome; *NA* not applicable; *SBP* systolic blood pressure; *SD* standard deviation; *W* women; *WC* waist circumference; *WHR* waist-to-hip ratio

Disclosure Dr. Kwame Osei is a member of the scientific advisory committee for Eli-Lilly and Daiichi Sankyo Pharmaceutical Companies. He is also on the speakers' bureau for Novo Nordisk Pharmaceutical Company.

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- • Of importance
- •• Of major importance

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