

## African-American Women, Metabolic Syndrome, and National Cholesterol Education Program Criteria: A Pilot Study

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### Abstract

► **Background:** *The association between elevated levels of triglycerides and insulin may be weaker in African-American women than in women of other groups, leading to underdiagnosis of the metabolic syndrome (MetS) in African-American women when using the National Cholesterol Education Program (NCEP) criteria, as that criteria does not include a marker of insulin resistance, using elevated triglycerides to provide an indirect indication of insulin resistance.*

► **Objectives:** *To determine the degree of agreement between two definitions for the MetS, that described by the NCEP and the NCEP criteria with the addition of a marker of insulin resistance in a sample of African-American women.*

► **Method:** *This nonexperimental pilot study took place in the General Clinical Research Center of a major medical center. Thirty-three African-American women 19–45 years of age were screened using the NCEP criteria for MetS, additional markers of insulin resistance, and a 2-hour Oral Glucose Tolerance Test.*

► **Findings:** *Six (18%) women were classified as having the MetS using the NCEP criteria. When one of three markers for insulin resistance (hyperinsulinemia, acanthosis nigricans, or Homeostatic Model Assessment Insulin Resistance) was added to the criteria, 15 (45.5%) to 19 (59.5%) of the women were then identified as having MetS.*

► **Discussion:** *As identified in the literature, the prevalence of cardiovascular disease risk in African-American women may be underestimated based on the sole use of the NCEP criteria. Further, because there is some evidence that insulin resistance develops before many other indicators, the addition of a marker of insulin*

*resistance may assist in earlier identification of African-American women at high risk for cardiovascular disease.*

**Key Words:** *African-American women, CRP, insulin resistance, PAI-1*

*Healthy People 2010 objectives emphasize the need to eradicate racial disparities in the cardiovascular (CV) health of minority populations within the United States (U.S. Department of Health and Human Services, 2000). The number one cause of death in both Caucasian and African-American woman is cardiovascular disease (CVD; American Heart Association [AHA], 2004). The latest data from the AHA identifies African-American women as having the highest prevalence among women for the following: coronary heart disease, myocardial infarction, stroke, angina pectoris, hypertension, obesity, sedentary lifestyle, and type 2 diabetes (AHA, 2000). Type 2 diabetes (T2D), a major risk factor for CVD, is the third leading cause of death among African-American women (AHA, 2000). The age-adjusted prevalence for T2D is more than two times higher among African-American women than in their Caucasian American female counterparts (AHA, 2000). In fact, the state of Alabama, where this study took place, had the highest prevalence of T2D in the nation (10% for Caucasians and 17% for African-Americans) in 2003 (Alabama Department of Public Health), making the study location particularly pertinent for examination of CV risk.*

*Adults with diabetes are two to four times more likely to die of either coronary heart disease or stroke (Alaba-*

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ma Department of Public Health, 2003; AHA, 2004). According to the AHA, the national age-adjusted prevalence for stroke among African-American women 20 years of age and older is 3.2%, which has now outpaced the 2.5% prevalence in African-American men (AHA, 2004), and is the highest age-adjusted prevalence of stroke in the United States. In short, African-American women living in Alabama are at high risk for T2D and CV events such as myocardial infarction or stroke. One reason for these metabolic and CV health disparities may relate to the findings that African-American women have higher levels of body mass index (BMI) and lower levels of insulin sensitivity (i.e., hyperinsulinemia) than their Caucasian counterparts (Gower, Weinsier, Jordan, Hunter, & Desmond, 2002).

### Contribution of Metabolic Syndrome as a Risk Factor

Preceding the development of either T2D or CVD, there is a well-described pathway, referred to as the metabolic syndrome (MetS), which leads the downward spiral toward vascular injury or CVD. The components of MetS consist of: abdominal central obesity; hyperinsulinemia; dyslipidemia; hypertension; dysfibrinolysis, as indicated by elevated plasma levels of plasminogen activator inhibitor-1 (PAI-1); and inflammation, as designated by elevated levels of C-reactive protein (CRP; Einhorn et al., 2003; Ford, Giles, & Dietz, 2002; National Cholesterol Education Program [NCEP], 2001; Ridker, Buring, Cook, & Rifai, 2003). Additional information provided by the authors expanding this article is on the Editor's Web site at <http://www.nursing-research-editor.com>. Individuals in the early stages of MetS have normal levels of plasma glucose. As the syndrome progresses, hyperglycemia may develop and lead to T2D. It is common to find that as glucose levels increase, the syndrome may progress to impaired fasting glucose (i.e., fasting glucose levels > 110 mg/dl or impaired glucose tolerance [IGT] with serum glucose levels of 140-199 mg/dl 2 hours after a glucose challenge), and ultimately hyperglycemia to the extent that the criteria for T2D are met (i.e., fasting glucose levels > 126 mg/dl or glucose levels > 200 mg/dl after a 2-hour glucose challenge, on two or more separate occasions; ADA, 2002). Although not everyone who has MetS will manifest T2D, those who exhibit both MetS and T2D are at the highest risk for development of CVD. In fact, NCEP considers those with T2D as having a risk for a cardiac event equal to someone who has already had a myocardial infarction (NCEP).

Individuals with elevated plasma PAI-1 levels are at great risk for developing thrombus or plaque within their vasculature leading to coronary heart disease, myocardial infarction, stroke, or peripheral vascular disease (Juhan-Vague, Alessi, & Morange, 2000). CV risk among individuals with elevated levels of PAI-1 may be underestimated by conventional risk factors, especially among African-American women (Alessi & Juhan-Vague, 2004). Comparatively, little is known about how MetS and levels of PAI-1 are manifested in young to middle-aged African-American women. Epidemiological studies have shown a strong association between plasma PAI-1 levels

and fasting insulin (Amowitz, Ridker, Rifai, Loughrey, & Komaroff, 2004). The mechanisms that regulate PAI-1 expression are still not well understood, and the role of insulin has potentially far-reaching clinical consequences with respect to developing premature CVD (Juhan-Vague, Morange, et al., 2003). Likewise, a better understanding of factors (individual, familial, lifestyle, and environmental) that predispose one to central obesity are needed (Devaraj, Chan, & Jialal, 2002; Murohara et al., 2004).

It was reported in recent studies that CV risk based on elevated CRP was due to more than simply inflammation. Ridker et al. (2002) reported that CRP was superior to low-density lipoprotein cholesterol (LDL-C) as an independent indicator of risk for CVD (Ridker et al., (2002). Various authors (Devaraj, Xu, & Jialal, 2003; Ridker et al., 2003) have reported that having an elevated CRP level may be harmful in its own right, as well as being an indicator for CV risk. One potential mechanism for CRP's harmful effects stems from its ability to enhance the release of PAI-1 from human endothelial cells (Devaraj et al., 2003). Elevated plasma levels of PAI-1 are associated with dysfibrinolysis, an abnormality of the clotting system, causing a worsened prognosis in the presence of a myocardial infarction (Alessi & Juhan-Vague, 2004). A higher incidence of thrombosis, impaired vascular remodeling, and atherosclerosis all have been associated with elevated levels of PAI-1 (Alessi & Juhan-Vague, 2004).

Further consequences of the deep visceral abdominal adipocytes metabolic activity stem from the release of deleterious substrates into the systemic circulation. It has been determined that enlarged abdominal adipocytes excrete angiotensin (Engeli et al., 1999; Rakugi & Ogihara, 2002). Release of angiotensin by the adipocytes has been associated with the hypertension found within the MetS. A second deleterious substrate released from the enlarged abdominal adipocyte is PAI-1 (Alessi & Juhan-Vague, 2004). Increased plasma levels of PAI-1 have been highly correlated with vascular injury and CV events (i.e., stroke, myocardial infarction; Juhan-Vague, Alessi, Marvri & Morange, 2003). The combination of hyperinsulinemia, the presence of hypertriglyceridemia, or both have been positively correlated with the release of PAI-1 (Alessi & Juhan-Vague, 2004). Elevated levels of either insulin or triglycerides have been reported to stimulate the enlarged abdominal adipocytes to excrete increased levels of PAI-1 (Alessi & Juhan-Vague, 2004), placing centrally obese individuals at greater risk for CV-related events.

### Definitions of the Metabolic Syndrome

There are now three different definitions of the MetS. Both the World Health Organization (Balkau & Charles, 1999) and the American College of Endocrinologist (Einhorn et al., 2003) have included some measure of insulin resistance in their definition of the MetS. The definition used most frequently in clinical practice for MetS has been formulated by NCEP (2001). The NCEP Adult Treatment Panel's definition of the MetS does not make any provisions for insulin sensitivity or insulin resistance (NCEP). Instead, the consensus committee maintained that the asso-

**Table 1. Physiological Variables by Waist Category**

Variables	Non- Centrally obese (n = 15)			Centrally Obese (n = 18)			t	P
	M	SD	Range	M	SD	Range		
Pre-PAI-1 (ng/ml)	11.59	9.21	1.00-33.00	33.60	23.01	7.47-83.14	3.51	.002*
Post-PAI-1 (ng/ml)	6.16	4.32	1.0-15.82	22.94	15.76	2.02-56.86	4.21	.001*
SBP (mmHg)	116.58	12.61	100.67-147.33	127.57	14.34	104.00-153.67	2.31	.027*
DBP (mmHg)	71.11	11.62	54.67-100.67	75.35	8.55	61.67-92.67	1.26	.237
BMI( Kg/m <sup>2</sup> )	26.04	2.31	23.08-30.11	35.11	6.01	26.07-50.55	5.90	.001*
Total Cholesterol (mg/dL)	175.60	32.28	123-230	183.44	42.18	132-264	.590	.559
LDL-C (mg/dL)	104.66	27.42	68-150	118.53	38.58	62.40-174.40	1.167	.252
HDL-C (mg/dL)	57.93	18.69	37.0-101.0	48.06	11.62	31.0-67.00	-1.85	.073
Triglycerides (mg/dL)	65.00	24.91	37.0-125.0	84.28	29.31	42.0-153.0	2.01	.053
Pre-Insulin (uU/ml)	15.27	6.27	7.0-30.0	25.17	13.24	10.0-59.0	2.65	.012*
Post-Insulin (uU/ml)	61.43	34.44	27.0-156.0	100.59	56.99	16.0-230.0	2.25	.032*
Fasting Glucose (mg/dL)	84.53	8.04	72.0-99.0	91.89	11.94	77.0-126.0	2.03	.051
2 Hr Post Glucose	98.43	19.39	73.0-138.0	131.18	44.68	74.0-241.0	2.73	.012*
CRP	.395	.448	.04-1.45	.714	.725	.03-2.65	1.47	.152
HOMA <sub>IR</sub>	3.20	1.43	1.40-6.20	5.94	4.11	2.00-18.2	2.65	.015*

Note. PAI-1 = plasminogen activator inhibitor-1; SPB = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass index; LDL = low-density lipoprotein; HDL = high-density lipoprotein; CRP = C-reactive protein; HOMA<sub>IR</sub> = Homeostatic Model Assessment–Insulin Resistance. \*Alpha at .05 level, Statistic: independent t-test

ciation between insulin and dyslipidemia (i.e., elevated triglycerides and low high-density lipoprotein cholesterol [HDL-C]) was strong enough ( $r = .73$ ) or ( $R^2 = .53$ ), explaining 53% of the variance between insulin response to a glucose load and plasma triglyceride levels (Olefsky, Farquhar, & Reaven, 1974), that a separate marker for insulin resistance was not necessary.

However, investigators have reported that African-American women do not to manifest the same patterns of dyslipidemia as other racial groups (Haffner et al., 1999; Keil et al., 1993; Metcalf et al., 1998). The Insulin Resistance Atherosclerosis Study (IRAS) reported that African-American people in general had significantly higher HDL-C ( $p < .001$ ) and lower triglyceride levels ( $p < .001$ ) than either Caucasian or Hispanic people (Haffner et al., 1999). Similar findings regarding racial differences in lipid profiles have appeared in other large epidemiological studies such as the Charleston Heart Study and Atherosclerosis Risk in Communities (ARIC) study (Knapp, Sutherland, Keil, Rust, & Lacklund, 1992; Metcalf et al., 1998). Further, Haffner and colleagues reported that the strong association between triglyceride levels and insulin resistance did not hold in African-American people, suggesting that, absent a separate insulin resistance marker, the NCEP criteria for MetS may underestimate the heart disease risk for African-American individuals (Haffner et al., 1999).

As a result of these differences in physiologic patterns and the high prevalence of morbidity and mortality from CVD in African-American women, further elucidation of the mechanisms involved in development of the MetS trajectory leading to CVD in this population is crucial.

**Methods**

**Purpose**

The purpose of this study was to determine agreement between MetS status using the NCEP and using NCEP criteria plus one of three markers of insulin resistance (NCEP + hyperinsulinemia, acanthosis nigricans [AN], or Homeostatic Model Assessment–Insulin Resistance [HOMA<sub>IR</sub>]), in a sample of African-American women. Research questions were: What is the level of agreement with regards to presence of MetS in a sample of African-American women based on NCEP criteria and NCEP criteria plus a marker of insulin resistance? Does inclusion of a marker of insulin resistance to the NCEP MetS definition lead to larger numbers of women identified as at-risk by virtue of having MetS?

This pilot study was a beginning step to identify how different MetS definitions affect CV risk assessments in healthy African-American women 19–45 years of age, specifically with regard to the addition of a marker of insulin resistance. We hypothesized that inclusion of a marker insulin resistance would result in a greater pro-

**Table 2. Presence of Metabolic Syndrome and Markers of Insulin Resistance Variables by Waist Category**

	Non- Centrally obese (n = 15)		Centrally Obese (n = 18)		$\chi^2$	Level of Significance (p)
	n	%	n	%		
Hyperinsulinemia ( $\geq 17.3$ uIU/ml)	3	20.0 %	12	66.6 %	7.187	.014*
Systolic hypertension* (> 130 mmHg)	2	13.0 %	7	38.8 %	2.69	.134
Diastolic hypertension* (> 85 mmHg)	2	13.0 %	2	11.1 %	.038	1.00
Hypertriglyceridemia* (>150 mg/dL)	0	0.0 %	1	5.5 %	.859	1.00
Low HDL-C* (< 50 mg/dL)	6	40.0 %	11	61.1 %	1.46	.303
IFG (fasting glucose 100-125 mg/dL) †	0	0.0 %	1	5.5 %	.859	1.00
IGT (2-hr post challenge glucose 140-199 mg/dL)	0	0.0 %	7	38.8 %	7.45	.019*
Elevated PAI-1 (> 66 ng/ml)	0	0.0 %	2	11.1 %	1.875	.485
HOMA <sub>IR</sub> > 3	7	46.6 %	15	83.3 %	4.95	.061
AN $\geq 1$	3	20.0 %	13	72.2 %	8.93	.005*
BMI > 25 Kg/ m <sup>2</sup>	8	53.3 %	18	100.0 %	10.66	.002*

Note: IFG = impaired fasting glucose; IGT = impaired glucose tolerance; PAI-1 = plasminogen activator inhibitor-1; HOMA<sub>IR</sub> = Homeostatic Model Assessment—Insulin Resistance; AN = acanthosis nigricans; BMI = body mass index.

Note: None of the participants had IFG using the NCEP criteria of > 110 mg/dl. Therefore, the newer ADA criteria for IFG > 100 mg/dl was utilized.

\* Significance at alpha = .05 level; N = 33 total.

†The following criteria is based on the ATP III Guidelines (NCEP, 2001): Hypertension: either SBP > 130 mmHg or DBP > 85 mmHg or both.

portion of African-American women being identified as having the MetS compared to use of the NCEP criteria without a marker of insulin resistance.

**Setting and Sample**

This pilot study employed a cross-sectional design and took place in the General Clinical Research Center (GCRC) of a major university medical center in the southeastern United States. Premenopausal African-American women 19–45 years of age (N = 33), were recruited via the campus newspaper, local churches, and beauty parlors in the area where the study was conducted. Inclusion criteria consisted of the following: non-pregnant, premenopausal, no recent use of steroids within the past 3 weeks, English-speaking, self-identified as being African-American, able to trace maternal heritage back three generations as all being of main continent United States (i.e., residing on the mainland US vs. a Caribbean island) African descent, and residing in the southern United States.

**Procedures and Measurement of Variables**

The study was approved by the university institutional review board and the Scientific Advisory Committee of the GCRC. The women were asked to fast for 12 hours starting at 8 p.m. the evening before the study and were

requested to arrive by 7:30 a.m. at the GCRC. The research assistant called each participant the evening before their study appointment to remind her to fast, except for intake of plain water, from 8 p.m., the evening prior to the time of the study. If participants took any morning medications, they were asked not to take them until after the study was completed, just before noon. In order to reduce disruption to their prescribed medication routine, they were encouraged to bring those medications with them to the GCRC and to take them at the conclusion of the study when they were served a lunch. All participants were closely monitored for any ill effects of withholding their morning medication(s). Once informed and written consent was obtained, the women were then screened for the MetS and other CV risk markers.

Blood pressure (BP) was measured before any other procedures, using the standard protocol of the GCRC. BP was obtained three times and the three systolic and diastolic measurements were recorded. Averages for systolic (SBP) and diastolic (DBP) were calculated using the three measures. Each measurement was taken after the participant was sitting quietly for 5 minutes. A BP cuff bladder that covered at least two-thirds of the upper right arm and at least half the circumference was used. The BP equipment was calibrated daily according to the unit’s protocol.

While the research assistant was obtaining BP measurements, participants were assessed for AN. AN is manifested as darkening of skin and is a sign of insulin resistance commonly seen among individuals with dark skin pigmentation. AN has been successfully used as a screening tool to identify those at high risk for T2D. AN screenings have been especially useful in the southwestern United States within the Hispanic population (Burke, Hale, Hazuda, & Stern, 1999). The skin of the back of the participant's neck is commonly visualized and rated on a descriptive scale (0-4) for the presence of AN (Appel, Jones, & Kennedy-Malone, 2004). Although AN can result from certain very rare cutaneous cancers, there are not any other known etiologies identified within the literature (i.e., including the effects of sunlight). However, acanthosis is commonly found on areas of the body not exposed to sunlight such as the groin or axillae. AN presents as a smooth and often raised velvet plaque, first noticed at the back of the neck in a single line (+1), in extreme cases (+4) extending to the frontal plane of the

neck and being clearly visible when standing in front of the individual (Burke et al., 1999). Although AN may be found commonly in all body folds (back of neck, axillae, groin, elbows, or knees), the neck is commonly used in research studies, as it is less invasive for the participant and easily recognizable by the researcher. Burke et al. (1999) showed that the AN found on the neck had the highest sensitivity (93 %) for insulin resistance, and it was found on the neck 99% of the time; if AN was present at all it was first found on the back of the neck (Burke et al., 1999, p. 1658). Burke's 0-4 scale was used to rate the severity of AN on the neck. In the analyses, however, AN was dichotomized as either absent (Burke's rating of 0) or present (Burke's rating of 1 or higher); its presence indicated insulin resistance (Burke). The PI and research assistant maintained a 100 % agreement on the presence or absence of AN.

Fasting laboratory values were obtained for glucose, lipids, insulin, hemoglobin A1C, CRP, and PAI-1 from a vein using minimal tourniquet time so as not to lyse the

**Table 3. Bi-variate correlations**

Bi-variate	Waist	HDL-C	Trig.	SBP	DBP	Pre-glucose	2-hr Post-glucose	Pre-Insulin	2-hr Post-Insulin	PAI-1	CRP	BMI	HOMA <sub>IR</sub>
Waist	1												
HDL-C	-.381 .029*	1											
Trig.	.223 .212	-.062 .733	1										
SBP	.435 .011*	-.091 .615	.091 .613	1									
DBP	.257 .149	-.006 .976	-.029 .872	.716 .001**	1								
Pre-Glucose	.416 .016*	-.433 .012	.257 .149	.217 .226	.042 .817	1							
2hr Post Glucose	.406 .023*	-.203 .273	.162 .384	.265 .150	.069 .713	.380 .035*	1						
Pre Insulin	.451 .008**	-.281 .114	.258 .147	.032 .858	.077 .669	.720 .001**	.236 .201	1					
2hr Post Insulin	.320 .080	-.276 .132	.315 .084	-.042 .824	.212 .253	.481 .006**	.309 .091	.724 .001**	1				
Pai-1	.725 .001*	-.335 .071	.221 .240	.408 .025*	.063 .740	.40 .014	.339 .078	.405 .026*	.403 .034*	1			
CRP	.345 .053	.104 .570	.051 .783	.152 .405	.033 .859	-.050 .784	.638 .001**	-.006 .974	.181 .329	.503 .005**	1		
BMI	.819 .001**	-.308 .081	.211 .238	.482 .004**	.121 .503	.391 .024*	.452 .011*	.414 .017*	.381 .034*	.892 .001**	.484 .005**	1	
HOMA <sub>IR</sub>	.420 .015*	-.291 .101	.258 .148	.057 .754	.097 .590	.803 .001**	.322 .077	.981 .001**	.689 .001**	.421 .021*	.001 .994	.419 .015*	1

\* Correlation is significant at the .05 level (2-tailed)

\*\* Correlation is significant at the .01 level (2-tailed)

blood cells. Appropriate blood tubes were utilized according to specific laboratory protocols for the core laboratory on-campus. The blood for PAI-1 was the only test conducted off-campus. Due to the diurnal variation of PAI-1 levels, which increase through the day (Declerck & Collen, 1990), all samples were collected 8:30–9:30 a.m. Due to the short half-life of free PAI-1, the University of Vermont recommends a 30-minute window for processing of those samples. The research assistant processed each PAI-1 sample within the 30-minute window, after which they were then snap frozen, stored in a –80 Celsius freezer until the study was completed, and later sent for overnight delivery packed in dry ice for final processing by the laboratory at the University of Vermont.

An oral glucose tolerance test was performed by administering 75 g of glucose orally. Blood glucose and insulin levels were measured 2 hours after glucose administration. Participants were asked during this 2-hour period to remain in the GCRC, staying seated as much as possible and performing only sedentary activities (watching TV, reading, or doing handwork such as needlepoint or knitting). Sedentary activities were encouraged to maintain a similar metabolic rate among participants. During the 2-hour waiting period, the GCRC’s nutritionist obtained height, weight, and waist circumference for every participant. Upon completion of the study, a HOMA<sub>IR</sub> was computed as one proxy for insulin resistance. The formula used for HOMA<sub>IR</sub> was: Fasting Plasma Insulin × Fasting Plasma Glucose/22.5 (Matthews, 1985). Because glucose was measured in mg/dl and the insulin in μU/ml, glucose was first converted from mg/dl to mmol/L, multiplying by 0.0551.

Data were analyzed using SPSS; all data were double-entered and compared for any discrepancy. Continuous physiological variables were compared by waist category using *t* test (Table 1). Distribution of MetS and categorical variable markers of insulin resistance was compared between waist categories using the chi-square statistic (Table 2). Pearson’s correlations were examined among all pairs of continuous variables (Table 3). Kappa coefficients were calculated to determine the level of agreement with regard to presence of MetS based on NCEP criteria and presence of MetS using NCEP criteria plus one of three markers of insulin resistance, in this sample of African-American women (Table 4).

**Results**

Central obesity was present in 18 (54.5%) and 15 (45.4%) were found to be noncentrally obese. Descriptively, the most prevalent risk factor was central obesity; with 18 (54.5%) of women identified as at-risk due to being centrally obese. Similarly, 17 (51.5%) of the participants were classified as being at-risk based on having a BMI classified within the overweight-to-obese categories (BMI > 25 kg/m<sup>2</sup>).

Based on the NCEP criteria, six (18.0%) of the women were classified as possessing the MetS (Table 4). Using the NCEP+ criteria, wherein a marker of insulin resis-

**Table 4. Comparisons for Agreement**

Variables	κ	Percent Agreement	<i>p</i>
NCEP versus NCEP + Hyperinsulinemia	0.355	66.6	.01*
Acanthosis Nigricans vs. Hyperinsulinemia	0.457	72.7	.008*
Hyperinsulinemia vs. HOMA <sub>IR</sub>	0.742	87.8	>.001*
Acanthosis nigricans vs. HOMA <sub>IR</sub>	0.341	66.6	.031*

Note: NCEP = National Cholesterol Education Program; HOMA<sub>IR</sub> = Homeostatic Model Assessment– Insulin Resistance.

\*Alpha level .05 significance.

tance was added to the NCEP criteria (i.e., hyperinsulinemia: fasting insulin level of > 17 mg/dl), 15 (45.5%) of the women were identified as possessing the MetS (Monzillo & Hamdy, 2003). A kappa coefficient of .355 (*p* = .01) was calculated, with an observed overall 66.6% agreement between the two methods.

Agreement between AN, hyperinsulinemia, and HOMA<sub>IR</sub> as markers of insulin resistance was also examined (Table 4). While all kappas were statistically significant, indicating greater than chance agreement, hyperinsulinemia and HOMA<sub>IR</sub> demonstrated the greatest level of agreement. Using AN, 16 (45.5%) were identified as possessing insulin resistance, whereas hyperinsulinemia alone identified 19 (59.5%) as having insulin resistance, resulting in a kappa of .457 (*p* = .008), with 72.7% agreement between the AN and hyperinsulinemia methods. Applying the HOMA<sub>IR</sub>, 23 (69.7%) of the women were identified as being insulin resistant. A kappa of .742 (*p* = .001), with an 87.8% agreement, was calculated between hyperinsulinemia and HOMA<sub>IR</sub>. HOMA<sub>IR</sub> was then compared with AN, producing a kappa of .341 (*p* = .031), with a 66.6% agreement between the HOMA<sub>IR</sub> and AN.

**Discussion**

As identified in the literature, African-American women’s risk for CVD is likely significantly underestimated based on the sole use of NCEP criteria (Liao et al., 2004). Clinicians should consider definitions of risk containing more components than are contained within the NCEP criteria. The inclusion of markers of inflammation (CRP), and prothrombotic factors (PAI-1), along with measures of insulin resistance, may improve early identification of CV-risk among African-American women.

Additional research is needed to identify whether unique patterns indicative of risk for CVD occur within additional specific populations. It has been known for some time that African-American women usually do not present with elevated triglycerides and commonly have adequate levels of HDL-C which do not appear to be cardioprotective

as in their Caucasian counterparts (Marcovina et al., 1996). Measures such as Lp(a) and LDL particle size (Scanu, 2003) may provide better estimates of CV risk than using solely the lipid criteria from the NCEP. When assessing African-American women's risk for CVD, insulin resistance should be taken into consideration, as it may be a primary manifestation of MetS (Amowitz et al., 2004) in that population. Use of markers of insulin resistance along with emerging bio-markers such as CRP, which have been found to be associated with higher risk for CVD among African-American women, may facilitate early risk identification (Ridker et al., 2003).

Lack of physical activity and diets high in fat with resulting obesity set the stage for insulin resistance. This permits up-regulation of susceptible genes (i.e., PAI-1) and fosters development of the MetS leading to vascular injury (Juhan-Vague et al., 2000; Juhan-Vague, Alessi, et al., 2003). When those at risk for the MetS disease trajectory are identified early, there is more opportunity to modify associated behavioral and lifestyle factors. An improved understanding of these factors within the framework of the MetS and CVD risk may suggest strategies to address CV health disparities among African-American women.

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