Fasting Triglyceride and the Triglyceride–HDL Cholesterol Ratio Are Not Markers of Insulin Resistance in African Americans

Anne E. Sumner, MD; Karl B. Finley, MD; David J. Genovese, RN; Michael H. Criqui, MD, MPH; Raymond C. Boston, PhD

Background: The “lipid criteria” consist of a triglyceride (TG) level of 130 mg/dL (1.47 mmol/L) or greater and a ratio of TG to high-density lipoprotein cholesterol (HDL-C) of 3 or greater. In Caucasians, the lipid criteria predict insulin resistance in individuals with a body mass index (BMI) of 25 kg/m² or greater. Our goal was to determine whether TG levels or TG–HDL-C ratio predicted insulin resistance in African Americans with a BMI of 25 kg/m² or more.

Methods: Of 125 African Americans, the 98 with a BMI of 25 kg/m² or more participated. All subjects had frequently sampled intravenous glucose tolerance tests with insulin resistance determined by the insulin sensitivity index. Subjects were divided into the following tertiles by insulin sensitivity: 12.8 to 4.3, 4.2 to 2.3, and 2.2 to 0.2 mU/L per minute. Insulin resistance was defined as being in the third tertile. Across tertiles, the distribution of variables was compared by 1-way analysis of variance. Areas under the receiver operating characteristic curve were determined to identify variables that predicted insulin resistance.

Results: Fasting insulin level, BMI, and waist circumference increased across tertiles (all P<.01), but TG levels and TG–HDL-C ratio did not (all P≥.3). The mean±SE areas under the curves for fasting insulin, BMI, and waist circumference were 0.85±0.04, 0.72±0.05, and 0.71±0.05, respectively. For TG level and TG–HDL-C ratio, the areas under the curves were 0.55±0.06 and 0.56±0.06, respectively, meaning that the true-positive rate was nearly equal to the false-positive rate. Therefore, they could not be used as markers of insulin resistance. Furthermore, 17 subjects met the lipid criteria but only 7 were in the insulin-resistant tertile, making the sensitivity of these criteria to identify insulin resistance only 17%.

Conclusion: In African Americans, TG levels and TG–HDL-C ratio are not reliable markers of insulin resistance.

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The National Cholesterol Education Program Adult Treatment Panel III designed the criteria for metabolic syndrome to identify individuals at high risk for cardiovascular events. Five criteria are used to diagnose the metabolic syndrome: elevated triglyceride (TG) levels, low levels of high-density lipoprotein cholesterol (HDL-C), impaired fasting glucose, central obesity, and hypertension. For diagnosis of the metabolic syndrome, 3 of the 5 criteria must be present. Surprisingly, the prevalence of the metabolic syndrome is lower in African Americans than in Caucasians. This is unexpected because the prevalence of heart disease and insulin resistance is higher in African Americans than in Caucasians. The reason for underdiagnosis of the metabolic syndrome in African Americans is attributed to the reliance of the metabolic syndrome criteria on lipids, particularly TG levels. Of the 5 metabolic syndrome criteria, African Americans are least likely to fulfill the TG criteria. In an attempt to overcome this problem and fully identify the metabolic syndrome in African American children, race-adjusted TG levels have been reported.

The metabolic syndrome is often used synonymously with insulin resistance, but the metabolic syndrome is not a diagnostic test for insulin resistance. Tests designed to measure insulin resistance include the glucose clamp, the insulin suppression test, and the frequently sampled intravenous glucose tolerance test.

When the metabolic syndrome is used as a test for insulin resistance, missed diagnoses occur. In fact, Cheal et al determined that the sensitivity of the metabolic syndrome to detect insulin resistance was only 46%. In another investigation,
these same investigators found that in subjects with a body mass index (BMI) of 25 kg/m² or more (calculated as weight in kilograms divided by the square of height in meters), the lipid criteria, specifically TG levels and TG–HDL-C ratio, were sensitive markers of insulin resistance.8 They found that the cutoffs for the lipid criteria that were most predictive of insulin resistance were TG level of 130 mg/dL (1.47 mmol/L) or more and/or TG–HDL-C ratio of 3 (1.8) or more. However, the population they studied was only 1% African American. We undertook this investigation because we considered that the use of these 2 criteria might lead to underdiagnosis of insulin resistance in African Americans. The basis for our concern is that African Americans are more insulin resistant than Caucasians are, but have lower TG levels.4,10 Therefore, TG levels that predict the presence of insulin resistance in Caucasians may not apply to African Americans. The consequence of underdiagnosis of insulin resistance in African Americans is that the opportunity for intervention to prevent cardiovascular disease and diabetes could be lost. Our goal was to determine whether the lipid criteria, specifically TG levels or the TG–HDL-C ratio, can be used to identify insulin resistance in African Americans with a BMI of 25 kg/m² or greater.

**METHODS**

One hundred twenty-five African Americans (65 men and 60 women), with a mean±SD age of 35±8 years (range, 20-50 years) enrolled in the cross-sectional Triglyceride and Cardiovascular Risk in African Americans (TARA) study at the National Institutes of Health (NIH). Subjects were recruited from the Washington, DC, area by flyers and the NIH Web site. The women were premenopausal. None of the women was receiving exogenous estrogens. Subjects did not have diabetes, liver disease, or kidney disease and took no medication. Similar to data from the National Health and Nutrition Examination Survey, 42% of the subjects were obese, 22% were glucose intolerant, and 20% were hypertensive.11-13 Seventy-five percent of subjects either had less than 1 drink a week or denied all alcohol intake. Nineteen percent had a drink once a week, and 6% of subjects had more than 1 drink per week. The study was approved by the institutional review board of the NIH, and subjects gave informed consent.

**MEASURING INSULIN RESISTANCE**

Insulin resistance was determined from the insulin sensitivity index (SI) calculated from the minimal model equations using data from the insulin-modified frequently sampled intravenous glucose tolerance test14 (MinMOD Millenium version 6.01, MinMOD Inc, Los Angeles, Calif). The SI is a dynamic measure of insulin sensitivity that is highly correlated with insulin resistance determined by the steady-state glucose clamp technique.14

The frequently sampled intravenous glucose tolerance test was performed at 8 AM after a 12-hour fast. At time 0, glucose (0.3 g/kg) was injected. From 20 to 25 minutes, an insulin infusion was administered (4 mU/kg per minute). Glucose and insulin concentrations were determined at −10, −1, 0, 1, 2, 3, 4, 5, 6, 7, 8, 10, 12, 14, 16, 19, 22, 23, 24, 25, 27, 30, 40, 50, 60, 70, 80, 90, 100, 120, 150, and 180 minutes.

The 125 subjects were divided into tertiles by SI; these tertiles were 12.8 to 4.3, 4.2 to 2.3, and 2.2 to 0.2 mU/L per minute. The lower the value of SI, the greater the degree of insulin resistance. Insulin resistance was defined as being in the third tertile. Insulin resistance is well captured in this third tertile because conditions associated with insulin resistance such as obesity, glucose intolerance, and type 2 diabetes typically have SI values below 2.3 mU/L per minute.15,16 Normal SI is 5.0 mU/L per minute.15

We followed the study design of McLaughlin et al8 the initial investigators who described that TG level and TG–HDL-C ratio were useful predictors of insulin resistance in Caucasians with a BMI of 25 kg/m² or greater. From our population of 125 subjects, 98 had a BMI at that level. To determine whether any of the variables measured could be used to predict the presence of insulin resistance in African Americans with a BMI of 25 kg/m² or greater, we analyzed the distribution of these 98 subjects across the SI tertiles determined by the population of 125.

**ANALYTIC METHODS**

Variables measured were fasting glucose, insulin, TG, HDL-C, TG–HDL-C ratio, very low-density lipoprotein particle size (VLDL size), low-density lipoprotein particle size (LDL size), and HDL size. Glucose was determined by the glucose oxidase method (Glucostat; Yellow Springs Instrument, Yellow Springs, Ohio). Insulin was measured with double-antibody chemiluminescent sandwich assays (Diagnostic Products, Los Angeles, Calif). Triglycerides were analyzed by enzymatic methods and an automated analyzer (Hitachi 917; Boehringer Mannheim, Mannheim, Germany). The HDL-C was isolated with dextran sulfate–magnesium17,18 The coefficients of variation for the TG and HDL-C assays were 1.5% and 2.2%, respectively. Lipid particle diameters were determined by nuclear magnetic spectroscopy (LipoScience, Raleigh, NC).18

**STATISTICAL ANALYSIS**

Data are presented as mean±SD. Parameters not normally distributed were transformed. Because results did not vary when adjusted for sex, combined results are presented. Tertile distributions of the 98 subjects with a BMI of 25 kg/m² or greater were compared by 1-way analysis of variance with Bonferroni corrections for multiple comparisons. Categorical data were analyzed with χ² and Fisher exact tests. In addition, areas under the receiver operating characteristic (ROC) curves were determined for each variable to identify which were predictors of insulin resistance. Areas under the ROC curves are provided with standard errors. An ROC curve is a plot of the sensitivity (true positive) vs 1–specificity (false positive) for each potential marker tested.19 The area under the ROC curve is a summary of the overall diagnostic accuracy of the test.19 The best markers have ROC curves that are shifted to the left with areas under the curve near unity. Nondiagnostic markers are represented by diagonals with areas under the ROC curves near 0.5.8,19 Analyses were performed with Stata software, version 8.0 (Stata Corp, College Station, Tex).

**RESULTS**

The demographic characteristics of the 125 participants are provided in Table 1. The tertiles of SI were determined on the basis of this population of 125. Of these subjects, 98 had a BMI of 25 kg/m² or greater. Twenty-seven of the 98 were in the first tertile of SI, 30 were in the second, and 41 were in the third (Table 2). Fasting insulin level, BMI, and waist circumference increased significantly across tertiles; fasting glucose level, TG level,
HDL-C level, TG–HDL-C ratio, VLDL size, LDL size, and HDL size did not.

Relating to the 5 metabolic syndrome criteria, 5 subjects (5%) had TG levels of 150 mg/dL (1.70 mmol/L) or more, but 60 subjects (61%) had central obesity (waist circumference ≥102 cm for men, ≥88 cm for women), 34 (35%) had low HDL-C levels (<40 mg/dL [<1.03 mmol/L] for men, ≤50 mg/dL [<1.29 mmol/L] for women), 24 (25%) had hypertension (blood pressure ≥130/85 mm Hg), and 10 (10%) had impaired fasting glucose levels (≥100 mg/dL [≥5.55 mmol/L]). For the 17 subjects (17%) who met the lipid criteria, 6 (6%) met both criteria (TG ≥130 mg/dL [1.47 mmol/L] and TG–HDL-C ratio ≥3.0 [1.8]), 4 (4%) met only the TG criterion, and 7 (7%) met only the TG/HDL-C ratio criterion.

Fourteen subjects had the metabolic syndrome, but only 12 of these were in the insulin-resistant tertile (Table 2). The sensitivity, specificity, and positive predictive value of the metabolic syndrome to diagnose insulin resistance in African Americans were 30%, 96%, and 86%, respectively. Of the 17 subjects who met the lipid criteria, only 7 were in the insulin-resistant tertile. The sensitivity, specificity, and positive predictive value of the lipid criteria to identify African Americans with insulin resistance were 17%, 83%, and 41%, respectively.

The ROC curve analyses showed that the best marker of insulin resistance was fasting insulin level, with an area under the ROC curve of 0.85 ± 0.04 (Table 3, Figure A). The BMI and waist circumference also discriminated insulin resistance, as they had areas under the ROC curve of 0.72 ± 0.05 and 0.71 ± 0.05, respectively. Level of TG and the TG–HDL-C ratio were not effective markers of insulin resistance. The areas under the ROC curve for these were 0.55 ± 0.06 and 0.56 ± 0.06, respectively (Figure, B and C). In addition, the TG-related variables of VLDL size, LDL size, and HDL size had areas under the ROC curve that were near 0.5.

The goal of this study was to determine whether lipid criteria, specifically the TG level or the TG–HDL-C ratio, were markers of insulin resistance in African Americans. As both the TG level and the TG–HDL-C ratio had areas under the ROC curve that approached 0.5, neither was a useful marker of insulin resistance in African Americans. The inability of the TG level or the TG–HDL-C ratio to be an effective marker of insulin resistance is reinforced by 2 observations: first, the TG level and the TG–HDL-C ratio did not increase across SI tertiles; second, VLDL size, LDL size, and HDL size did not change significantly across tertiles and had areas under the ROC curve that approached 0.5. The size of these lipid particles relates to TG levels because if TG had increased significantly across SI tertiles, the size of the particles should have decreased significantly.

Because neither TG level nor TG–HDL-C ratio was a marker of insulin resistance in African Americans, no threshold value or cutoff could be calculated. According to McLaughlin et al., cutoff levels that are diagnostic of insulin resistance in Caucasians are 130 mg/dL (1.47 mmol/L) or greater for TG level and 3 (1.8) or greater for TG–HDL-C ratio. When we applied these cutoffs to African Americans, we found that only 17 of 98 subjects met one of these criteria. Of these 17 subjects, only 7 were in the insulin-resistant tertile. Therefore, the lipid criteria had a sensitivity of only 17% in African Americans when used to diagnose insulin resistance.

In contrast to TG level and TG–HDL-C ratio, fasting insulin, BMI, and waist circumference were excellent markers of insulin resistance. Hence, African Americans have the expected relationship between insulin resistance and increased body size. However, studies have demonstrated that the strength of the correlation between obesity and TG levels is lower in African Americans than in Caucasians. Clearly, in African Americans the interrelationship between insulin resistance, obesity, and TG is not yet fully defined and cannot be extrapolated from studies in Caucasians.

As the metabolic syndrome does not rely solely on lipid measures, we postulated that the sensitivity of the metabolic syndrome to diagnose insulin resistance may be superior to that of the lipid criteria. Indeed, the sensitivity and positive predictive value of the metabolic syndrome were 30% and 86%, respectively, compared with 17% and 41% for the lipid criteria. Therefore, in African Americans the metabolic syndrome is somewhat superior to the lipid criteria in the identification of subjects with insulin resistance. Nonetheless, the metabolic syndrome had a sensitivity of only 30%. The sensitivity for the metabolic syndrome to diagnose insulin resistance is still relatively low at 30% because there were 29 of 41 subjects in the third SI tertile who had insulin resistance but were undiagnosed because they did not carry the metabolic syndrome label. Therefore, as found by McLaughlin et al. in Caucasians, relying on the diagnosis of the metabolic syndrome to identify insulin resistance leads to underdiagnosis of insulin resistance. For African Americans, the poor relationship

### Table 1. Demographic and Metabolic Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>35 ± 8</td>
<td>20-50</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>30.9 ± 7.6</td>
<td>18.5-54.7</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>98 ± 18</td>
<td>69-173</td>
</tr>
<tr>
<td>Fasting glucose, mg/dL</td>
<td>86 ± 8</td>
<td>70-114</td>
</tr>
<tr>
<td>Blood pressure, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>117 ± 14</td>
<td>92-168</td>
</tr>
<tr>
<td>Diastolic</td>
<td>70 ± 9</td>
<td>50-94</td>
</tr>
<tr>
<td>TG, mg/dL</td>
<td>77 ± 39</td>
<td>22-224</td>
</tr>
<tr>
<td>HDL-C, mg/dL</td>
<td>48 ± 10</td>
<td>29-83</td>
</tr>
<tr>
<td>TG–HDL-C ratio</td>
<td>1.55 ± 1.04</td>
<td>0.44-6.88</td>
</tr>
</tbody>
</table>

SI conversion factors: To convert glucose to millimoles per liter, multiply by 0.0555; HDL-C to millimoles per liter, multiply by 0.0259; TG to millimoles per liter, multiply by 0.0113; TG–HDL-C ratio, multiply by 0.6.

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); HDL-C, high-density lipoprotein cholesterol; TG, triglyceride.
between insulin resistance and either the metabolic syndrome or the lipid criteria may be due to a low correlation between TG level and insulin resistance.

Racial differences in lipoprotein lipase (LPL) activity may be responsible for racial differences in TG. Lipoprotein lipase is the enzyme responsible for clearing TG-containing lipoproteins from the circulation. The LPL levels are higher in African Americans than in Caucasians. This is the reason most often cited as to why African Americans have lower TG levels than Caucasians. In Caucasians, insulin resistance leads to an impairment of LPL levels and high TG levels. There is some evidence that insulin resistance may not lead to an impairment of LPL activity in African Americans. If that is the case, then TG-containing lipid particles are cleared from the circulation even in the presence of insulin resistance and plasma TG levels do not rise. This could account in part for a weak association between TG levels and insulin resistance in African Americans.

Our conclusions differ from those of McLaughlin et al, but there are 3 major differences between our studies. First, all of our participants were African Americans. In the McLaughlin et al study, 87% of the participants were Caucasian and only 1% were African American. The differences in our results most likely represent important racial differences. Second, McLaughlin et al used the insulin suppression test to measure insulin resistance, while we used $S_2$. As both methods are valid mea-

### Table 2. Characteristics by Insulin Resistance Tertile of Subjects With BMI Greater Than 25 kg/m²

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Tertile 1 (n = 27)</th>
<th>Tertile 2 (n = 30)</th>
<th>Tertile 3 (n = 41)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, No. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>17 (63)</td>
<td>18 (60)</td>
<td>19 (46)</td>
<td>.33‡</td>
</tr>
<tr>
<td>Female</td>
<td>10 (37)</td>
<td>12 (40)</td>
<td>22 (54)</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>35 ± 9</td>
<td>36 ± 8</td>
<td>36 ± 7</td>
<td>.27</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>29.4 ± 3.5</td>
<td>32.1 ± 6.5</td>
<td>36.7 ± 7.8</td>
<td>.006§</td>
</tr>
<tr>
<td>WC, cm</td>
<td>94.6 ± 10.2</td>
<td>101.1 ± 12.5</td>
<td>111.3 ± 18.4</td>
<td>&lt;.001§</td>
</tr>
<tr>
<td>Fasting insulin, µU/mL</td>
<td>5.3 ± 2.8</td>
<td>7.5 ± 2.9</td>
<td>12.4 ± 5.8</td>
<td>&lt;.001§</td>
</tr>
<tr>
<td>Fasting glucose, mg/dL</td>
<td>84 ± 10</td>
<td>86 ± 9</td>
<td>88 ± 11</td>
<td>.67</td>
</tr>
<tr>
<td>TG, mg/dL</td>
<td>67 ± 28</td>
<td>79 ± 39</td>
<td>83 ± 45</td>
<td>.35</td>
</tr>
<tr>
<td>HDL-C, mg/dL</td>
<td>49 ± 11</td>
<td>48 ± 11</td>
<td>46 ± 8</td>
<td>.72</td>
</tr>
<tr>
<td>TG–HDL-C ratio</td>
<td>1.43 ± 0.66</td>
<td>1.80 ± 1.15</td>
<td>1.91 ± 1.21</td>
<td>.28</td>
</tr>
<tr>
<td>Lipid values directly dependent on TG levels</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VLDL size, nm</td>
<td>45.9 ± 7.8</td>
<td>46.5 ± 11.9</td>
<td>47.8 ± 8.0</td>
<td>.58</td>
</tr>
<tr>
<td>LDL size, nm</td>
<td>21.07 ± 0.50</td>
<td>21.09 ± 0.63</td>
<td>21.02 ± 0.66</td>
<td>.91</td>
</tr>
<tr>
<td>HDL size, nm</td>
<td>9.04 ± 0.44</td>
<td>8.87 ± 0.45</td>
<td>8.90 ± 0.44</td>
<td>.32</td>
</tr>
<tr>
<td>Frequency of metabolic syndrome and lipid criteria</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. (%) with metabolic syndrome‡</td>
<td>0</td>
<td>2 (7)</td>
<td>12 (30)</td>
<td>&lt;.001‡</td>
</tr>
<tr>
<td>No. (%) with lipid criteria§</td>
<td>3 (11)</td>
<td>7 (23)</td>
<td>7 (17)</td>
<td>.48‡</td>
</tr>
</tbody>
</table>

### Table 3. Comparison of Areas Under ROC Curves for Potential Markers of Insulin Resistance

<table>
<thead>
<tr>
<th>Variable</th>
<th>Area Under ROC Curve, Mean ± SE</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting insulin</td>
<td>0.85 ± 0.04</td>
<td>0.78-0.92</td>
</tr>
<tr>
<td>BMI</td>
<td>0.72 ± 0.05</td>
<td>0.61-0.82</td>
</tr>
<tr>
<td>WC</td>
<td>0.71 ± 0.05</td>
<td>0.61-0.81</td>
</tr>
<tr>
<td>Fasting glucose</td>
<td>0.58 ± 0.05</td>
<td>0.47-0.69</td>
</tr>
<tr>
<td>TG–HDL-C ratio</td>
<td>0.56 ± 0.06</td>
<td>0.45-0.68</td>
</tr>
<tr>
<td>TG</td>
<td>0.55 ± 0.06</td>
<td>0.43-0.66</td>
</tr>
<tr>
<td>HDL-C</td>
<td>0.47 ± 0.06</td>
<td>0.35-0.58</td>
</tr>
</tbody>
</table>

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by the square of height in meters); HDL, high-density lipoprotein; HDL-C, HDL cholesterol; LDL, low-density lipoprotein; TG, triglyceride; VLDL, very low-density lipoprotein; WC, waist circumference.

SI conversion factors: To convert insulin to picomoles per liter, multiply by 6.945; glucose to millimoles per liter, multiply by 0.0555; TG to millimoles per liter, multiply by 0.0113; HDL-C to millimoles per liter, multiply by 0.0259; TG–HDL-C ratio, multiply by 0.6.

*Data are presented as mean ± SD unless otherwise indicated. Tertile 1 had an insulin resistance value of 6.28 ± 2.03 mU/L per minute (range, 12.75-4.28 mU/L per minute); tertile 2, 3.05 ± 0.64 mU/L per minute (range, 4.24-2.24 mU/L per minute); and tertile 3, 1.40 ± 0.51 mU/L per minute (range, 2.20-0.17 mU/L per minute) (P < .001).

†Unless otherwise stated, analyses were by 1-way analysis of variance with Bonferroni corrections for multiple comparisons.

‡By χ² analysis.

§Significant differences between tertiles 1 vs 2 and 1 vs 3 only.

¶Metabolic syndrome was present if 3 of 5 criteria were met: TG level of 150 mg/dL or greater; HDL-C level of 40 mg/dL or less in men or 50 mg/dL or less in women; WC of 102 cm or greater in men or 88 cm or greater in women; blood pressure of 130/85 mm Hg or greater; and fasting glucose level of 100 mg/dL or greater.

#Lipid criteria were met if TG level was 130 mg/dL or greater or if TG–HDL-C ratio was 3 or greater.
diagnose insulin resistance will lead to an underestimation of risk for disorders related to insulin resistance. Before recommendations are made to use TG level and TG–HDL-C ratio as markers of insulin resistance in African Americans, we urge testing these criteria in large populations of African Americans over a wide age range.

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5. Giannini E, Testa R. The metabolic syndrome: all criteria are equal, but some criteria are more equal than others. Arch Intern Med. 2003;163:2787-2788.


Figure. Receiver operating characteristic curves. Sensitivity represents true-positive results, and 1–specificity, false-positive results. A, Fasting insulin concentration. B, Triglyceride concentration. C, Triglyceride–high-density lipoprotein cholesterol ratio.

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We suggest that using lipid criteria, specifically TG level or the TG–HDL-C ratio, in African Americans to