Obesity and High Blood Pressure: A Clinical Phenotype for the Insulin Resistance Syndrome in African Americans

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Abstract and Introduction

Abstract

The high prevalence of insulin resistance syndrome in African Americans predisposes this population to higher morbidity and mortality from cardiovascular disease. To test the hypothesis that the combination of obesity and high blood pressure (BP) represents the physical phenotype of insulin resistance syndrome, 337 African-American men and women aged 4 years were examined and classified into four groups (nonobese-normal BP, nonobese-high BP, obese-normal BP, obese-high BP), according to presence or absence of obesity and high BP. Mean values of glucose, insulin, lipids, albumin excretion, and clamp-derived insulin sensitivity were determined for each group. Prevalence of prediabetes (24.4%), diabetes (19.2%), and insulin resistance syndrome (87.2%) were highest in the obese-high BP group (p <0.001). Triglycerides, urinary albumin excretion, fasting glucose, fasting insulin, and insulin resistance were highest in the obese-high BP group (p <0.001). Subjects with both obesity and high BP showed greater expression of lipid and glucose abnormalities and higher urinary albumin excretion, and greater prevalence of prediabetes, undetected type 2 diabetes, and insulin resistance syndrome.

Introduction

Both type 2 diabetes and obesity have been linked with high blood pressure (BP) and dyslipidemia in a clustering of metabolic abnormalities known as the insulin resistance syndrome (IRS). First described by Reaven in the 1988 lecture, IRS has also been known as syndrome X, the metabolic syndrome, and dysmetabolic syndrome. There has been much debate over what criteria are best used for the diagnosis of IRS. In 2001, the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) proposed diagnostic criteria as three or more of the following metabolic abnormalities: waist circumference >102 cm (40 in) for men or >88 cm (34 in) for women, triglycerides >150 mg/dL, high-density lipoprotein (HDL) cholesterol level <40 mg/dL for men or <50 mg/dL for women, BP >/= 130/80 mm Hg, and fasting glucose >/= 110 mg/dL. In a 2003 position statement, the American College of Endocrinology adopted the ATP III guidelines for lipids and BP, but recommended broadening the criteria to include a definition of obesity on body mass index (BMI) and impaired glucose tolerance based on a 2-hour oral glucose tolerance test (OGTT). Analysis of data from the third National Health and Nutrition Examination Survey (NHANES) determined the prevalence of IRS to range from 6.7% among persons aged 20-29 years to 43.5% among persons aged 60-69 years that report, more than 75% of African Americans had at least one metabolic abnormality typical of IRS.[4]

Individuals with IRS experience a higher incidence of both diabetes and cardiovascular disease. Furthermore, IRS has been associated with higher cardiovascular and total mortality.[5] Both hypertension and central adiposity have been shown to contribute to insulin resistance. However, not all patients with hypertension have insulin resistance. The purpose of this study was to test the hypothesis that the combination of obesity with high BP is the physical phenotype of IRS. We predicted that more persons with both obesity and high BP would meet the ATP III criteria for IRS and express more metabolic abnormalities of the IRS than subjects with either high BP alone or obesity alone.

Methods
Population

Participants in this cross-sectional study were drawn from a cohort of African-American men and women enrolled in investigations of BP, insulin sensitivity, and cardiovascular risk. This analysis examined 337 African Americans at average age of 32.1±4.4 years. Written, informed consent was obtained from each participant at the time of enrollment, and an institutionally approved protocol and consent form. Persons with known diabetes were excluded from initial entry. Female participants were evaluated during the follicular phase of the menstrual cycle to limit hormonal variability.

Procedure

Information on health status and current health behaviors was obtained from an enrollment interview. Anthropometric measurements of height and weight were obtained. BMI was calculated as weight (kilograms) divided by height (meters squared). BP measurements were obtained from each subject following a 10-minute rest period in a seated position. Auscultation with a mercury column sphygmomanometer. The average of two determinations of casual systolic (first phase) and diastolic (fifth phase) was used as the BP. An OGTT was conducted after a 12-hour overnight fast. A blood sample was obtained for serum lipids, insulin, and glucose, then 75 g glucose solution (Glucola; Ames Diagnost, Elkhart, IN) was ingested. Blood samples were obtained at 30 minutes, 60 minutes, 90 minutes, and 120 minutes postingestion and were assayed for plasma insulin and glucose concentrations. A timed, overnight urine specimen collected. Urine albumin was measured by radioimmunoassay (Double Antibody Assay KHADL; Diagnostic Product Corporation, Los Angeles, CA) and expressed as micrograms per minute. Urine creatinine was measured and urinary albumin excretion (UAE) was calculated in milligrams per gram creatinine.

The euglycemic hyperinsulinemic clamp technique was used to measure insulin-stimulated glucose uptake. In brief, two peripheral venous catheters were placed and sample withdrawal. After determination of fasting plasma glucose concentration, hyperinsulinemia was established with a primed constant infusion of insulin according to the method of Rizza et al. to calculate the primed dose and infusion rate. The primed infusion rate was sufficient to achieve steady state hyperinsulinemia at 80-120 min above fasting insulin levels with the goal of suppressing hepatic glucose production. Hyperinsulinemia was maintained for 120 minutes, during which time euglycemia was achieved using a variable infusion of 20% dextrose in water (20% Dextrose Injection USP; Abbott Laboratory, Abbott Park, IL). Insulin (Novolin R; Eli Lilly, Indianapolis, IN) was added at the concentration of 1000 mU/mL in normal saline. The glucose infusion rate was adjusted by the negative feedback equation of DeFronzo et al. according to plasma glucose sampled every 10 minutes.

The insulin-stimulated glucose uptake was computed as the mean glucose infusion during the final 60 minutes of procedure and expressed as milligrams per kilogram per minute. Higher values of insulin-stimulated glucose uptake indicative of greater insulin sensitivity. Because the level of steady state hyperinsulinemia achieved during the clamp procedure varied slightly among the cases, an index of insulin sensitivity was calculated by dividing the glucose infusion rate by the mean insulin level achieved during the final 60 minutes of steady state hyperinsulinemia. Because muscle tissue is more insulin resistant relative to adipose tissue, the measured insulin-mediated glucose uptake was adjusted for adiposity. Anthropometric measurements were used to estimate the fat-free mass and the insulin-stimulated uptake was calculated in milligrams per kilograms of fat-free mass per minute. The adjusted index of insulin sensitivity also corrected for adiposity and expressed as milligrams per kilograms of fat-free mass per minute divided by milligrams per kilogram per minute.

The fasting serum sample from the OGTT was sent to the lipid research laboratory, where total cholesterol, HDL cholesterol, and triglyceride levels were analyzed with standard enzymatic methods and an automated analyzer (1704; Boehringer-Mannheim Diagnostics, Indianapolis, IN). HDL was isolated according to the method of Bachorik et al. Low-density lipoprotein (LDL) cholesterol was calculated by the Friedewald equation. Plasma glucose concentration was analyzed with the glucose oxidase technique (YS model 27; Glucostat, Yellow Springs, OH). Plasma insulin concentration was determined with a solid phase radioimmunoassay (Coat-a-Count; Diagnostic Products Corp., Los Angeles, CA). Coefficients of variation for inter- and intra- assay variability for glucose, insulin, and lipid assays were less than 5%.

Data Analysis

Subjects were classified into four groups according to the presence or absence of obesity and high BP (nonobese BP, nonobese high BP, obese normal BP, obese high BP). We defined obesity as BMI >/=30. Hypertension was defined as a systolic BP >/=130 mm Hg or diastolic BP >/=85 mm Hg, according to the ATP III criteria. Subjects who were antihypertensive medications were considered to have met the criteria for hypertension, even if their BP was controllably high. Subjects with a history of hypertension were classified as hypertensive and were not included in the analysis. The average age of the participants was 32.1±4.4 years. Written, informed consent was obtained from each participant at the time of enrollment, and an institutionally approved protocol and consent form. Persons with known diabetes were excluded from initial entry. Female participants were evaluated during the follicular phase of the menstrual cycle to limit hormonal variability.

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glucose >/=140 mg/dL. The proportion of subjects with IRS, prediabetes, and undiagnosed diabetes was compared among the four BMI-BP groups and analyzed using the Pearson χ² test.

Because ATP III criteria for BP and obesity were used to define the BMI-BP groups, the analysis was repeated for metabolic abnormalities of dyslipidemia and glucose intolerance, excluding obesity and high BP. Analysis of varia tional models (SAS PROC CATMOD, version 8.2; SAS Institute Inc., Cary, NC) was used to compare the differences in metabolic abnormalities among the BMI-BP groups.

Results

Data was available from 337 young adult African Americans. Subjects included 117 men and 220 women. Mean ± standard deviation age was 32.1±4.1 years. This analysis included 133 nonobese subjects with normal BP, 48 nonobese subjects with high BP, 78 obese subjects with normal BP, and 78 obese subjects with high BP. As determined by the OGTT, 104 subjects (20.8%) had prediabetes (impaired fasting glucose or impaired glucose tolerance) and 20 subjects (5.9%) undiagnosed type 2 diabetes. Of the 337 subjects, 115 (34.1%) met the ATP III criteria for diagnosis of IRS.

Table I provides the gender distribution and mean age, BMI, and BP parameters for each of the four subject groups. Subjects with high BP (>130/85 mm Hg) tended to be older. Mean age among subjects with high BP was 33 years among subjects with normal BP, a difference that was statistically significant at this sample size (p<0.001). The nonobese persons averaged 24.6 and 25.2 in the normal and high BP groups respectively. Even in the absence of obesity, many of these subjects were overweight, with a BMI >25. Because the groups were defined by the presence or absence of obesity and high BP, both BMI and BP were statistically different among the groups. BMI was higher in obese groups (37 vs. 25), and both systolic and diastolic BP was higher in the high BP groups (all p<0.001). How ever, was not statistically different between the two obese groups, and systolic or diastolic BP was not statistically different between the two high BP groups (all p>0.05). The gender distribution was skewed with a higher percentage of women in the two obese groups (p<0.001). This reflects the higher prevalence of obesity among African-American women compared to men.

Table II provides the mean values for lipids and OGTT measurements in each of the four groups. There was no statistically significant difference in total cholesterol between the four groups. There was a statistically significant trend for linearity in LDL measurements, with higher LDL measurements in the obese with high BP group and lowest values in the nonobese normal BP group (p=0.04). HDL was lower among obese subjects with or without high BP compared with nonobese subjects (p=0.001). Triglycerides were lowest in the nonobese with normal BP group. The presence of either high BP or obesity resulted in proportionate increases in triglyceride levels. When both obesity and high BP were present, the triglycerides were nearly 20 mg/dL higher than when either obesity or high BP was present alone. This difference in triglyceride measurements reached statistical significance (p<0.001), with a significant trend for linearity (p<0.001).

Both fasting and 2-hour glucose measurements from the OGTT were statistically different between groups (both p<0.001). Fasting glucose was significantly higher in the groups with high BP compared with those with normal BP in both men and nonobese subjects (p=0.03). The 2-hour glucose measurements rose from 111.9 mg/dL in the nonobese with normal BP to 144.4 mg/dL in the obese with high BP group (p<0.001), with a statistically significant linear trend (p<0.001). Lipid concentrations also showed a significant difference in triglyceride levels. The presence of obesity and high BP resulted in proportionate increases in triglyceride levels. When both obesity and high BP were present, the triglycerides were nearly 20 mg/dL higher than when either obesity or high BP was present alone. This difference in triglyceride measurements reached statistical significance (p<0.001), with a significant trend for linearity (p<0.001).

IRS was absent in all nonobese subjects with normal BP. The presence of high BP alone was associated with 25% prevalence of IRS, and obesity alone was associated with 44.9% prevalence of the IRS. However, 87.2% of subjects had both high BP and obesity at least one of the metabolic abnormalities and therefore fulfill the diagnostic criteria for IRS.

Figure 1 depicts the prevalence of prediabetes and type 2 diabetes in each of the BMI-BP groups. The prevalence of abnormal glucose tolerance in the form of diabetes or prediabetes was 15.8% in the nonobese with normal BP group, with the addition of high BP, the prevalence rose to 20.8%. In the obese population, the prevalence of abnormal glucose tolerance went from 32.1% in those with normal BP to 43.6% in those with high BP. The combination of obesity and high BP was associated with the highest prevalence of prediabetes or type 2 diabetes (p<0.001). The prevalence of prediabetes and type 2 diabetes was lowest in the nonobese with normal BP group and increases significantly with addition of obesity or high BP. The prevalence of type 2 diabetes in the obese with high BP group was 19.2%, compared with 0%-2.6% in all other groups (p<0.001).
Figure 1. Prevalence of prediabetes and undiagnosed type 2 diabetes in each of the four body mass index-blood pressure (BP) groups.

Of the 337 subjects, 283 returned and underwent a euglycemic hyperinsulinemic clamp. Figure 2 shows the mean sensitivity as measured by the euglycemic hyperinsulinemic clamp. M was lower in the obese groups compared with nonobese groups \((p<0.001)\). When corrected for both adiposity and the steady state insulin level achieved, the difference becomes even more striking. In addition, high BP in the absence of obesity results in decreased insulin sensitivity \(14.3 \text{ mg/kg fat-free mass/min}/\mu \text{U insulin x 1000}\), a difference that is statistically significant \((p=0.02)\). The insulin sensitivity is further reduced in both groups with obesity to 6.5 with normal BP and 6.6 with high BP \((p<0.001)\).

Figure 2. Mean insulin sensitivity as measured by euglycemic hyperinsulinemic clamp in each body mass index-blood pressure (BP) group. M=mean ± standard error for measured glucose uptake (in mg/kg/min); \(M'/Ic\)=glucose uptake corrected for adiposity and level of steady-state clamp hyperinsulinemia (mg/kg fat-free mass/min/\mu U).

Figure 3 shows the mean urine albumin excretion per gram creatinine in each group from an overnight urine collection.
performed at the time of the clamp procedure. Nonobese subjects with normal BP had the lowest UAE. In the pre-high BP, the mean UAE increased to 7.1 mg/g creatinine, a difference that was not statistically significant at this size \( (p=0.16) \). In the presence of both obesity and high BP, UAE increased to 17.6 mg/g creatinine, a statistically significant difference compared with nonobese and obese subjects with normal BP \( (p<0.001, p=0.006) \), respective

Figure 3. Bars represent the mean ± standard error of urinary albumin excretion (UAE) in mg/g creatinine each body mass index-blood pressure (BP) group.

The distribution of subjects within each group by number of coexisting metabolic abnormalities (low HDL level, high triglycerides, or abnormal glucose tolerance) is presented in Figure 4. The distribution of metabolic abnormalities significantly skewed with a greater number of abnormalities in the subjects with obesity \( (p<0.001) \) and/or high BP \( (p=0.006) \). There was no significant interaction between obesity and high BP \( (p=0.40) \) and thus the combination of obesity and high BP does not seem to cause a negative synergistic effect, but rather seems to be the combined linear effect of each factor of obesity and high BP.

Figure 4. Distribution of subjects by number of insulin resistance syndrome-associated metabolic abnormalities present (body mass index and blood pressure [BP] excluded). White bar=percentage of subjects with no metabolic abnormalities decreases in subjects with high BP, obesity, or both; gray bar=percentage of subjects in each body mass index-BP group with one metabolic abnormality; black
bar=percentage of subjects with two metabolic abnormalities increases in subjects with high BP, obesity, or both; striped bar=percentage of subjects with three metabolic abnormalities is greatest in the obese with high BP group.

Discussion

The results of this study show a strong association between the phenotype of obesity plus high BP with the metabolic abnormalities of IRS. HDL is lower in the obese groups with and without high BP, while both LDL and triglycerides are highest in subjects with both obesity and high BP. Both fasting and 2-hour glucose by OGTT were highest in the subjects with both obesity and high BP. Subjects in the group with obesity and high BP had significantly greater prevalence of glucose intolerance and previously undetected diabetes. Microalbuminuria is a trait considered by some to be another characteristic of IRS.13-15 Our data also show significantly greater UAE in subjects with both obesity and high BP. Subjects with both obesity and high BP tended to have more concurrent metabolic abnormalities consistent with IRS. Therefore, according to ATP III criteria, approximately 87% of subjects with obesity and high BP had at least one metabolic abnormality and fulfilled the criteria for IRS.

IRS consists of a cluster of metabolic and physiologic abnormalities that cosegregate within an individual. The original definition of the syndrome, first called syndrome X by Reaven in 1988,[1] included hypertension, glucose intolerance, central adiposity, dyslipidemia, insulin resistance, hyperuricemia, fibrinolysis, hemostasis, inflammation, and microalbuminuria. Presence of IRS is associated with increased mortality and morbidity from coronary heart disease and cardiovascular disease.[5,15-17] Furthermore, risk of coronary heart disease and cardiovascular disease are elevated in the presence of only one to two risk factors for IRS.[17]

Multiple studies have used factor analysis to investigate the clustering of variables considered to be part of IRS.[14] These studies have included subjects of multiple races ranging in age from adolescence to late adulthood. While results vary slightly according to the study population, all analyses have found two or more independent factors of abnormalities. Insulin resistance, obesity, elevated plasma glucose, and dyslipidemia have generally been found to be one factor, while hypertension and insulin resistance have been found to load onto a separate factor of these studies, insulin measurements contribute across multiple separate factors, which suggests that insulin resistance could be the core abnormality of IRS. A recent study by Ford[25] of 6868 subjects, of which 1834 were African American, identified three separate factors contributing to the insulin resistance syndrome in subjects aged 20-44 years. In this study, triglycerides, insulin, and waist circumference were found to load together onto Factor 1; systolic blood pressure, waist circumference, and uric acid were found to load onto Factor 2; and glucose and UAE were found to load on Factor 3. Factor analysis conducted by Hanley et al.[18] identified two factors, a metabolic factor that loaded with BMI, glucose, insulin sensitivity, HDL cholesterol level, and triglyceride level and a BP factor that loaded with systolic and diastolic blood pressure. These studies all suggest that BP acts separately from the metabolic factors to contribute to IRS.

Triglyceride levels have been reported to be lower in both male and female African Americans compared with age-matched whites.[22,23] Similarly, HDL levels have been reported to be higher in African Americans than whites.[22] African Americans still have higher rates of heart disease and mortality from cardiovascular disease.[24] While high triglyceride and low HDL levels are less prevalent, hypertension, obesity, and type 2 diabetes are more prevalent in African Americans than whites.[25-27] This suggests that obesity, diabetes, insulin resistance, and high BP rather than factors may be the major determinants of IRS in African Americans. Alternatively, the relatively low prevalence of elevated triglycerides and low HDL cholesterol levels raise the question of whether the threshold for the effects of dyslipidemia is lower in African Americans.

National population data show that the prevalence of type 2 diabetes increased from 8.9% among participants in the 1980 NHANES to 12.3% among participants in the 1988-1994 NHANES.[25] In addition, the prevalence of obesity increasing. The 1988-1994 NHANES found a 22.8% prevalence of obesity, which increased to 30.5% by 1999-2002. In our study, we found the prevalence of prediabetes ranged from 12.9% in the nonobese normal BP group to 29.5% in the obese normal BP group. In the subjects with obesity and high BP, the prevalence of prediabetes was 24.4%. However, this group the prevalence of undiagnosed diabetes was 19.9%, which is much higher than the prevalence in the general population. Subjects with both obesity and high BP had undiagnosed diabetes almost as frequently as prediabetes. Persons with IRS frequently have an elevated 2-hour glucose on OGTT in the presence of normal fasting glucose levels.

Screening for diabetes or prediabetes with the measurement of only fasting plasma glucose measurement fails to identify many persons with impaired glucose tolerance. For this reason, the American College of Endocrinology currently recommends OGTT for screening for IRS.[9]

Due to the increasing rates of obesity and diabetes, identification of IRS is important for management of premature cardiovascular events. Lifestyle and pharmacologic interventions can improve insulin sensitivity and improve the metabolic components of IRS. Both high BP and obesity are traits easily determined in a physician's office. We fo
87% of our African-American subjects with both obesity and high BP possessed at least one other metabolic abnormality and met the ATP III criteria for diagnosis of IRS. Subjects with the clinical phenotype of obesity plus high BP also had the highest rates of previously undetected diabetes. Because these data are only on African Americans, generalizability may be limited. However, this clinical phenotype is easily identified, and subjects with both obesity and high BP from other groups may have increased rates of treatable metabolic abnormalities.

CME Information

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Tables

Table I. Mean Parameters of Each Body Mass Index (BMI)-Blood Pressure (BP) Group

<table>
<thead>
<tr>
<th>Measure</th>
<th>Nonobese Normal BP</th>
<th>Nonobese High BP</th>
<th>Obese Normal BP</th>
<th>Obese High BP</th>
<th>p Value Group</th>
<th>Linear Trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>31.2±4.1</td>
<td>33.2±5.4</td>
<td>31.6±6.0</td>
<td>33.5±4.3</td>
<td>0.15</td>
<td>0.18</td>
</tr>
<tr>
<td>Women (%)</td>
<td>61.7</td>
<td>39.6</td>
<td>78.2</td>
<td>74.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>24.6±3.3</td>
<td>25.2±3.3</td>
<td>36.4±5.4</td>
<td>37.3±6.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>113±9</td>
<td>141±13</td>
<td>118±8</td>
<td>143±13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>72±9</td>
<td>91±14</td>
<td>74±10</td>
<td>92±11</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SBP=systolic blood pressure; DBP=diastolic blood pressure

Table II. Mean Metabolic Measures From Lipid and Oral Glucose Tolerance Testing

<table>
<thead>
<tr>
<th>Measure</th>
<th>Nonobese Normal BP</th>
<th>Nonobese High BP</th>
<th>Obese Normal BP</th>
<th>Obese High BP</th>
<th>p Value Group</th>
<th>Linear Trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol* (mg/dL)</td>
<td>176±39</td>
<td>184±44</td>
<td>174±31</td>
<td>186±40</td>
<td>0.15</td>
<td>0.18</td>
</tr>
<tr>
<td>LDL* (mg/dL)</td>
<td>107±33</td>
<td>113±34</td>
<td>111±28</td>
<td>118±41</td>
<td>0.13</td>
<td>0.04</td>
</tr>
<tr>
<td>HDL* (mg/dL)</td>
<td>52±16</td>
<td>52±23</td>
<td>45±11</td>
<td>46±14</td>
<td>0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglycerides* (mg/dL)</td>
<td>72±29</td>
<td>91±42</td>
<td>91±40</td>
<td>110±65</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting glucose* (mg/dL)</td>
<td>90.7±10.6</td>
<td>94±10.0</td>
<td>97.2±23.9</td>
<td>105.0±21.4</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2-Hour glucose* (mg/dL)</td>
<td>111.9±33.2</td>
<td>116.4±26.4</td>
<td>132.5±47.0</td>
<td>144.4±53.7</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting insulin** (µU/mL)</td>
<td>9.8±9.0</td>
<td>10.1±5.8</td>
<td>16.2±8.7</td>
<td>19.6±18.9</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Prediabetes or diabetes (%)</td>
<td>15.8</td>
<td>20.5</td>
<td>32.1</td>
<td>43.6</td>
<td>&lt;0.001*</td>
<td></td>
</tr>
<tr>
<td>Prevalence: IRS (%)</td>
<td>0.0</td>
<td>25.0</td>
<td>44.9</td>
<td>87.2</td>
<td>&lt;0.001*</td>
<td></td>
</tr>
</tbody>
</table>

BP=blood pressure; LDL=low-density lipoprotein; HDL=high-density lipoprotein; IRS=insulin resistance syndrome; *Pearson’s r test; †to convert total, LDL, and HDL cholesterol in mg/dL to mmol/L, multiply by 0.0259; ‡to convert triglycerides in mg/dL to mmol/L, multiply by 0.0113; ‡to convert glucose in mg/dL to mmol/L, multiply by 0.0555; ‡‡to convert insulin in µU/mL to pmol/L, multiply by 69.

References


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