Published in final edited form as:
*Metabolism*. 2007 July ; 56(7): 954–960.

# Plasma Lipid Concentrations in Non-Diabetic, African American Adults:

## Associations with Insulin Resistance and the Metabolic Syndrome

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## Abstract

**OBJECTIVE—**Despite higher rates of cardiovascular disease, African Americans have a more favorable lipid profile. The purpose of the study was to examine the association between plasma lipid concentrations and insulin resistance in African Americans and to determine if insulin resistance is present at a lower triglyceride (TG) threshold than is used for metabolic syndrome criteria.

**METHODS—**Data were examined on 185 non-diabetic African American men (N=61) and women (N=124), mean 39.8 years. Measurements included blood pressure, anthropometrics, oral glucose tolerance test, and insulin sensitivity (M), by insulin clamp. The relationship between lipids and insulin sensitivity was analyzed by correlation analysis and by comparing triglyceride levels among tertiles of M.

**RESULTS—**Despite relatively low mean TG (87.8 ± 55.2 mg/dL), there were statistically significant correlations of M with TG (r = -.23, P<0.002), high density lipoprotein cholesterol (HDL-C; r =.19, P < 0.01)), and TG/HDL-C ratio (r = -.23, P<0.002). The correlations were strongest in men. Subjects with TG in an intermediate range (110-149 mg/dL) had insulin resistance equivalent to the high TG group (≥ 150 mg/dL).

**CONCLUSIONS—**In African Americans, triglyceride levels below the current metabolic syndrome threshold criterion are associated with insulin resistance.

### Keywords

Insulin; Metabolic Syndrome; Insulin Resistance; Triglycerides; High Density Lipoprotein

Insulin resistance (IR), defined as impaired insulin-mediated glucose uptake, has been identified as a pathogenic factor leading to cardiovascular disease (CVD) and diabetes mellitus (DM).[1-3] Compared with Caucasians, African Americans have greater insulin resistance,[4-6] which could contribute to a higher prevalence of CVD and DM in this group.[7] A leading theory to explain the mechanism underlying the detrimental effect of insulin resistance on cardiovascular injury is the association of insulin resistance with atherosclerotic dyslipidemia.

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[8] Insulin resistance, or impaired insulin sensitivity, is difficult to quantify clinically, and the concept of the metabolic syndrome has developed as a strategy to identify individuals with the multiple CVD risk factors that co-segregate with insulin resistance. Despite greater insulin resistance, African Americans have more favorable lipid profiles than Caucasians, including lower triglycerides (TG) and higher high-density lipoprotein cholesterol (HDL-C) concentrations.[9-11] Prior studies in African Americans yielded conflicting results regarding the association of insulin resistance with elevated TG and decreased HDL concentrations.[9, 10, 12-14] Some reports describe a significant association between measures of insulin resistance and plasma lipid concentrations in African Americans, despite more favorable lipid profiles.[10] Others have not detected a relationship of insulin resistance with TG,[13] possibly due to higher levels of lipoprotein lipase among African Americans.[9]

In attempt to identify individuals at risk for CVD and DM due to underlying insulin resistance, clinical criteria have been developed for a condition termed the Metabolic Syndrome, which is a clustering of cardiovascular risk factors linked with underlying insulin resistance.[15, 16] According to these definitions, the clinical diagnosis of the metabolic syndrome requires the presence of several cardiovascular risk factors within an individual, although no one specific risk factor is required. Although varying definitions of the metabolic syndrome exist, the risk parameters that are consistently included are high TG, low HDL, central obesity, high blood pressure, and elevated plasma glucose.[15, 17, 18] Epidemiologic studies that apply these clinical criteria to population data have reported a lower prevalence of metabolic syndrome in African Americans compared to Caucasians despite greater adiposity among African Americans.[19, 20] Due to lower TG concentrations among African Americans, compared to other race groups, African Americans may less frequently meet the metabolic syndrome criterion of TG criterion of ≥150 mg/dL.[20] However, the same relationship between insulin resistance and TG may exist in African Americans but at a lower TG threshold. The purpose of this study was to determine if there is a significant relationship between plasma lipid concentrations and insulin resistance in African Americans; and to examine the utility of different TG threshold levels for the detection of underlying insulin resistance in this racial population.

## METHODS

### Subjects

The sample for this study was drawn from a cohort enrolled in a previous study of BP, insulin resistance, and cardiovascular risk. The subjects were all self-identified African Americans recruited from urban Philadelphia, and were tested between January 2001 and April 2006. Caribbean African Americans were not enrolled. The European admixture of this African American cohort has been previously analyzed and found to be 12.7 to 13.6%.[21] The mean participant age at the time of examination was 39.8 ± 3.9 years, with an age range of 28 to 51 years. Individuals with known or newly identified diabetes mellitus were excluded from this analysis. At the time of enrollment, all subjects provided written informed consent for a protocol on a consent form approved by the Institutional Review Board of the Thomas Jefferson University. All women were premenopausal at enrollment, and all procedures were conducted in the prefollicular phase of their menstrual cycles.

### Procedures

Enrollment assessment consisted of anthropometric measurements (height, weight, skinfold thickness), blood pressure (BP) measurement, fasting blood sample, and an oral glucose tolerance test (OGTT) after a 12-hour fast. Anthropometric measurements were used to calculate body mass index (BMI = $kg/m^2$), percent body fat, and fat-free mass.[22] BP was measured using a mercury column sphygmomanometer with the participant in a seated position after 10 minutes of rest. An average of two measurements for systolic BP (SBP) and diastolic

BP (DBP) was determined. For the OGTT, an oral 75-g glucose solution (Glucola; Ames Laboratories, Elkhart, IN) was ingested. Blood samples were drawn pre-glucose load (fasting), and at 30, 60, and 120 minutes post-glucose load. All samples were assayed for plasma insulin and glucose concentrations after storage at -80°C.

The euglycemic hyperinsulinemic clamp procedure was administered to assess insulin-stimulated glucose utilization.[23, 24] For the euglycemic clamp procedure, each participant returned to the clinic at 8 am after a 12-hour fast. The euglycemic clamp procedure was conducted according to methods previously described.[25] In brief, two peripheral venous catheters were placed after the subject rested for at least 20 minutes. Three samples were withdrawn to determine fasting plasma glucose and insulin concentration. Euglycemic hyperinsulinemia was induced with a priming dose and infusion rate of insulin according to the method of Rizza at al.[24] The infused insulin was administered at 1000 mU/mL in normal saline (Novolin R; Elie Lilly, Indianapolis, IN). Using this method, euglycemic hyperinsulinemia was maintained at 80-120μU/mL above fasting insulin concentration for 120 minutes. Glucose was infused as 20% dextrose (Abbott Laboratory, Abbott Park, IL) to maintain euglycemia. The glucose infusion rate was adjusted as a function of the plasma glucose concentrations sampled every 10 minutes, according to the negative feedback equation of Defronzo et al.[23] Insulin-stimulated glucose metabolism, designated as M, was quantified as the mean glucose infusion rate required to maintain euglycemia during the final 60 minutes (clamp period) of the hyperinsulinemic procedure (M in mg • $kg^{-1}$ • $min^{-1}$).

The fasting blood sample from the enrollment assessment was analyzed for serum lipid concentrations (total cholesterol, HDL cholesterol, and triglycerides) by a lipid research laboratory using standard enzymatic methods and an automated analyzer (Hitachi 704; Boehringer-Mannheim Diagnostics, Indianapolis, IN). HDL was isolated using a method previously described.[26] The Friedewald equation was used to calculate low-density lipoprotein (LDL) cholesterol.[27] Coefficients of variation for inter- and intra-assay variability for the lipid assays and the above glucose and insulin assays were <5%.

## Data Analysis

The Adult Treatment Panel III (ATP III) Guidelines were used to define metabolic syndrome,[28] with the exception that BMI ≥ 30 $Kg/m^2$ used in place of waist circumference (>102 cm in men, >88 cm in women).[29] Subjects were considered to have the metabolic syndrome if they met three or more of the following criteria: TG ≥ 150 mg/dL; HDL < 50 mg/dL for women and < 40 mg/dL for men; fasting glucose ≥ 100 mg/dL; BP ≥ 130/85 mm Hg; BMI ≥ 30 kg/m. For a more accurate assessment of elevated fasting glucose, subjects met this criterion if they had fasting glucose concentrations ≥ 100 mg/dL both on the morning of the OGTT and the morning of the euglycemic clamp.

Mean ±SD of participant characteristics were calculated for men, women, and the total sample. The *t*-test was used to determine whether significant differences existed in mean values between men and women. The Pearson r was used to examine correlations of plasma lipid concentrations and insulin sensitivity (M) with other continuous variables. To further examine the associations with insulin sensitivity, the sample was stratified into tertiles of insulin sensitivity based on total M, with the lower third being most insulin resistant and the upper third being most insulin sensitive. Differences among tertiles were analyzed by 1-way ANOVA. In addition, we stratified the sample by three categories of plasma TG concentration to compare parameters and prevalence of metabolic syndrome in different TG ranges. The mean TG concentration in the most insulin resistant tertile was designated as the upper limit of normal. Thus, the sample was stratified by TG concentrations as normal (<110 mg/dL), intermediate (110-149 mg/dL), and high (≥ 150 mg/dL). To examine the possibility of using TG ≥ 110 as a cut-point for risk in African Americans, 1-way ANOVA was used to compare

differences in means for continuous variables among TG stratifications. The Chi-squared test was used to compare rates of the metabolic syndrome between insulin sensitivity tertiles and in the total population with different TG cut-point applications. To correct for simultaneous multiple comparisons, but which were often correlated, we considered P-values ≤ 0.01 to be statistically significant. However, all observed P-values are provided. Statistical analyses were performed using SAS version 8.2 (SAS Institute Inc, Cary, NC).

## RESULTS

Complete data for this study were available on 185 non-diabetic African American subjects, including 61 men and 124 women. The mean age of this sample was 39.8 ± 3.7 years, and range: 28-51 years. Table 1 provides the anthropometric, BP, and metabolic characteristics. In this sample, 55% were obese and 38% had high BP. Men and women had comparable mean characteristics, with the exception that compared with men, women had significantly higher mean BMI and percent body fat. Despite higher BMI, women had TG and HDL concentrations comparable to men.. Insulin sensitivity (M) was lower in women compared to men, but the difference was not statistically significant (M mean in women= 5.46 ± 2.82 vs. 6.26 ± 2.91 in men; p=0.074). Overall, 19% of this African American sample met the current criteria for the metabolic syndrome. However, all type 2 diabetics, most of whom were previously undetected and found to be diabetic on oral glucose tolerance testing, were excluded from this analysis.

Table 2 provides the correlation coefficients for plasma lipid concentrations with BMI, blood pressure, and metabolic variables. There were significant correlations of both TG and HDL-C with other components of the metabolic syndrome. There were statistically significant correlations of HDL-C with fasting insulin, and M. In addition, there were significant correlations of TG with DBP, fasting glucose, and M. Neither total cholesterol nor LDL had significant correlations with any of the above variables. Although there were no significant difference between men and women in mean values for TG, HDL-C, and M, gender differences were detected when the correlations of triglyceride level with M were examined for men and women separately. Among men, the correlation coefficient for TG with M increased to r = 0.38 (P = 0.003). Among the women the correlation coefficient for TG with M decreased to r = 0.15 (P = 0.10), indicating that when all diabetics were excluded, the significant association of TG with M was largely driven by the men..

Because insulin resistance is the core abnormality of the metabolic syndrome, the relationships of insulin sensitivity (M) with other components of the metabolic syndrome were examined. There were significant correlations of M with fasting insulin concentration (r= -0.30, P <0.0001), SBP (r= -0.20, P=0.01), and DBP (r= -0.20, P=0.01). There were also significant correlations of M with the anthropometric measures of BMI (r= -0.53, P < 0.001) and percent body fat (r= -0.48, P <0.001).

To further examine the relationship of plasma lipid concentrations with insulin sensitivity quantified by the insulin clamp procedure, we divided the sample into tertiles of insulin sensitivity based on M value and compared the relevant variables. These data are provided in Table 3. There were no significant differences between the M tertile groups in LDL, total cholesterol, fasting glucose, or age. There were significant differences in BMI, fasting insulin, 2 hr glucose, HDL-C, TG, and TG-HDL-C across tertiles. The insulin resistant (lowest M) tertile had the highest percent of subjects who met the criteria for the metabolic syndrome (33%), while only 5% of subjects within the insulin sensitive (highest M) tertile met criteria. The mean TG (106.1 ± 68.6 mg/dL) in the most insulin resistant tertile was substantially lower than the threshold metabolic syndrome criterion for hypertriglyceridemia (> 150 mg.dL). Across all tertiles of insulin sensitivity, mean TG/HDL-C ratio was < 3, whereas a ratio >3 is considered to be associated with insulin resistance.[14]

We then examined the TG threshold for associations with components of the metabolic syndrome. Cases were classified according to TG concentration. A TG concentration that was below the mean value in the most insulin resistant tertile was classified as normal TG (TG < 110 mg/dL); TG ≥ 150 mg/dL classified as high TG; TG value falling between (110 - 149 mg/dL were classified as intermediate TG. Table 4 provides the mean values for the anthropometric, blood pressure and metabolic variables in each TG group. ANOVA on variables across TG groups showed significant differences in fasting insulin, fasting glucose, SBP, DBP, and total cholesterol. There was also a significant difference in insulin sensitivity (M) among TG groups. However, as can be seen in Table 4, the intermediate and high TG groups had nearly identical mean values for both M and HDL-C. There were no significant differences among TG groups in age, BMI, percent body fat, or LDL.

The distribution of cases who meet the criteria for metabolic syndrome with the two different criteria for elevated TG is provided on Table 5. Of the total sample, 23% had TG ≥ 110 mg/dL. When the lower TG concentration of≥ 110 mg/dL was applied as the TG criterion for the metabolic syndrome, the rate of metabolic syndrome increased from 19% to 25% in the total sample. The increase in percent of cases of metabolic syndrome occurred in the most insulin resistant tertile with an increase from 35% to 52%. The mid tertile and high tertiles of insulin sensitivity did not show much change in rates of metabolic syndrome after applying the lower TG criterion.

## DISCUSSION

In this study of non-diabetic primarily third and fourth decade African American adults, plasma triglyceride concentrations were lower and HDL-cholesterol concentrations were higher than lipid concentrations described in Caucasian populations. These results are comparable to previous reports.[19, 30, 31] Although the lipid concentrations in this African American sample were more favorable, there were significant correlations of TG, HDL-C, and TG/HDL-C ratio with insulin resistance measured by the insulin clamp procedure. Despite the association between insulin resistance and TG, only 10% of the sample had elevated TGs according to the ATP III criteria for metabolic syndrome. Subjects with TG concentrations in an intermediate range of 110 to 149 mg/dL had measures of insulin resistance comparable to subjects with elevated TG (>150 mg/dL, and were more insulin resistant than those with TG <110 mg/dL. A TG threshold of ≥ 110 mg/dL increased the detection of the metabolic syndrome in the most insulin resistant M tertile, whereas neither the mid nor high tertiles of insulin sensitivity showed a change in prevalence of metabolic syndrome cases when the lower TG threshold was applied.

Plasma lipid concentrations in African Americans of comparable age and adiposity as the subjects in our study were reported by Sumner et al.[9, 13] They also reported an apparently favorable lipid profile in African Americans, despite obesity and relative insulin resistance, but a significant relationship between TG and insulin resistance was not detected. In contrast, we detected a significant correlation of insulin resistance with TG, HDL-C, and TG/HDL-C. Our study included a somewhat larger sample size, a larger proportion of women, and we quantified insulin sensitivity by the insulin clamp procedure rather than the insulin-modified frequently sampled intravenous glucose tolerance test used by Sumner et al. However, when the association of TG with insulin sensitivity was examined in men and women separately, the correlation increased in men and decreased in women, indicating a significant gender effect on the relationship. Since the women in our study were all pre-menopausal, the gender difference could be due to some salutary effect of endogenous estrogens. Known diabetics were excluded from the analysis. Also excluded from the analysis were subjects, predominately female, in whom diabetes was identified on oral glucose tolerance testing. It is possible that the exclusion of previously undetected diabetics may have amplified the gender differences in

the relationship of TG with insulin sensitivity in this relatively young adult African American sample.

Insulin resistance plays a role in cardiovascular and endothelial damage. Because insulin resistance is difficult to quantify clinically, the constellation of associated CV risk factors designated as the metabolic syndrome serves as a surrogate clinical strategy to optimize detection insulin resistance as an underlying pathogenic condition.[29] In Caucasians, there is a strong correlation of TG with insulin resistance as measured by the insulin suppression test, supporting the inclusion of a TG threshold in the metabolic syndrome definition.[14] The data from this study found that even at lower TG concentrations, the relationship of TG with insulin resistance is present in African Americans. This suggests that a TG criterion of≥ 150 mg/dL may be too high to detect underlying insulin resistance in this high risk ethnic group. These results support lowering the TG cut-point to ≥ 110 mg/dL, as subjects with TG between 110 and 149 mg/dl were just as insulin resistant as subjects with TG > 150 mg/dL.

Although African Americans appear to have more favorable lipid profiles, it is possible that they have a different threshold for the adverse effects of relative dyslipidemia. One pathway of vascular damage is mediated through oxidative stress. Lopes et al.[32] investigated the effect of acute hyperlipidemia on oxidative stress in both African Americans and Caucasians. Following infusion of Intralipid and heparin, African American and Caucasian subjects experienced a comparable rise in plasma TG concentrations. However, F2-isoprostanes, a biomarker of oxidative stress in humans, increased significantly more in African Americans compared with Caucasians. Although the report by Lopes et al. is based on an acute rise in TG, it does suggest that African Americans could have a heightened sensitivity to increases in TG.

Reports on dyslipidemia in Caucasians describe a significant positive correlation between visceral adiposity and TG concentration.[33] Both central obesity and insulin resistance are associated with TG elevation in Caucasians. However, in an earlier report from the Insulin Resistance Artherosclerosis Study (IRAS),[6] the association between insulin resistance and lipoproteins was independent of waist-hip ratio in African American adults. Data from this study detected no relationship between TG and BMI or percent body fat which is consistent with the IRAS findings. It is possible that the relationship of obesity with insulin resistance is over-emphasized as insulin resistance has been documented in lean individuals.[34]

A limitation of our study may be the small sample size in relation to large-scale epidemiologic reports. However, in comparison to the number of African American subjects in earlier studies of insulin resistance in non-diabetics, the present study had a similar sample size, if not larger. Also, the subjects were representative of the African American population. Among our subjects, 55% were obese, which is similar to the middle-aged African Americans described by the National Health and Nutrition Examination Survey (NHANES).[35] Therefore, the results of our study may apply to non-diabetic African Americans between 30 and 50 years of age.

Since there are presently no quantifiable criteria that designate insulin resistance within individuals, the use of the lowest M tertile as a stratum of insulin resistance could be considered arbitrary. However, the euglycemic hyperinsulinemic clamp procedure used in the present study to quantify insulin-mediated glucose uptake is considered the gold standard in measuring insulin sensitivity.[13] Moreover, clustering of components of the metabolic syndrome segregated in the lowest M tertile, indicate that this designation was reasonable.

Until recently, data on the metabolic syndrome were derived from predominately Caucasian populations. Recent observations from various ethnic and racial groups have questioned the validity of applying the same metabolic syndrome criteria to different populations.[19, 36] Data from the present study demonstrates that the existing TG threshold of ≥ 150 mg/dL may under-detect insulin resistance in African Americans. Reaven has suggested that insulin resistance is

the underlying pathology for developing CVD and that the concept of the metabolic syndrome may be misleading.[37] The diagnosis of the metabolic syndrome, while flawed, can be clinically useful to detect patients with probable insulin resistance. Even if the diagnosis of metabolic syndrome is abandoned, African Americans with characteristics of insulin resistance, including high blood pressure, obesity, and prediabetic blood glucose levels, but with seemingly normal TG and HDL may benefit from risk factor reduction. Further studies are needed to evaluate CVD in insulin resistant African Americans and to determine if lower lipid thresholds contribute to CVD progression.

**ACKNOWLEDGEMENTS**

This work was supported by the following grants from the National Institutes of Health: HL51547, DK046107, and HL007845.

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**Table 1**

Participant Characteristics

| | Men N=61 | Women N=124 | Total N=185 | P-Values* |
|---|---|---|---|---|
| Age (years) | 39.7 ± 3.9 | 39.8 ± 3.6 | 39.8 ± 3.7 | 0.86 |
| Weight (kg) | 93.2 ± 23.1 | 90.6 ± 22.2 | 91.5 ± 22.5 | 0.47 |
| BMI (kg/m$^2$) | 29.7 ± 6.8[a] | 33.4 ± 8.0 | 32.2 ± 7.8 | 0.002 |
| Percent Body Fat | 25.4 ± 7.4[b] | 38.4 ± 4.4 | 34.1 ± 8.3 | < 0.001 |
| SBP (mm Hg) | 128.9 ± 15.2 | 124.0 ± 19.4 | 125.6 ± 18.3 | 0.09 |
| DBP (mm HG) | 76.1 ± 11.9 | 72.6 ± 12.1 | 73.8 ± 12.1 | 0.07 |
| Fasting insulin (μU/mL) | 9.5 ± 8.4 | 10.9 ± 9.8 | 10.5 ± 9.4 | 0.02 |
| Fasting glucose (mg/dL) | 101.4 ± 9.6 | 97.9 ± 10.6 | 99.1 ± 10.4 | 0.03 |
| 2 hr Glucose (mg/dL) | 122.5 ± 39.2 | 128.1 ± 33.3 | 126.3 ± 34.4 | 0.31 |
| Total Cholesterol (mg/dL) | 182.0 ± 38.8 | 184.0 ± 35.4 | 183.4 ± 36.4 | 0.72 |
| LDL (mg/dL) | 117.5 ± 39.8 | 118.6 ± 32.4 | 118.2 ± 34.9 | 0.85 |
| HDL-C (mg/dL) | 49.4 ± 20.4 | 49.7 ± 13.6 | 49.6 ± 16.1 | 0.95 |
| Triglycerides (mg/dL) | 95.5 ± 72.2 | 84.0 ± 44.5 | 87.8 ± 55.2 | 0.25 |
| Triglycerides/HDL-C | 2.34 ± 2.65 | 1.86 ± 1.29 | 2.02 ± 1.86 | 0.19 |
| Metabolic Syndrome % | 18% | 21% | 19% | |

* P-values for t-test comparing means of men and women.

**Table 2**

Correlations of Plasma Lipid Concentrations and with Variables

| | BMI | | SBP | | DBP | | Fasting Glucose | | Fasting Insulin | | Total M | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **r** | **P** | **r** | **P** | **r** | **P** | **r** | **P** | **r** | **P** | **r** | **P** |
| Total Cholesterol | 0.002 | 0.98 | -0.08 | 0.28 | -0.01 | 0.94 | 0.07 | 0.36 | -0.04 | 0.56 | -0.08 | 0.29 |
| LDL | 0.03 | 0.69 | -0.05 | 0.54 | 0.04 | 0.63 | 0.06 | 0.36 | -0.02 | 0.81 | -0.12 | 0.10 |
| HDL-C | -0.17 | 0.02 | -0.15 | 0.05 | -0.17 | 0.02 | 0.15 | 0.04 | -0.21 | 0.004 | 0.19 | 0.01 |
| TG | 0.10 | 0.19 | 0.18 | 0.02 | 0.19 | 0.01 | 0.24 | <0.001 | 0.17 | 0.02 | -0.23 | 0.002 |
| TG/HDL-C | 0.11 | 0.13 | 0.19 | 0.01 | 0.21 | 0.004 | 0.23 | <0.001 | 0.23 | 0.02 | -0.23 | 0.002 |

**Table 3**

Characteristics Across Tertiles of Insulin Sensitivity (M)

| | Insulin Resistant (Lowest M) n=60 | Mid Sensitivity (Mid M) n=63 | Insulin Sensitive (Highest M) n=62 | 1-way ANOVA P-value |
|---|---|---|---|---|
| Age (years) | 39.9 ± 3.1 | 39.8 ± 4.05 | 39.6 ± 3.8 | 0.89 |
| Percent body fat | 38.3 ± 5.6 | 35.3 ± 6.4 | 28.9 ± 9.3 | < 0.001 |
| BMI ($kg/m^2$) | 37.6 ± 8.1 | 31.5 ± 6.2 | 27.6 ± 5.5 | < 0.001 |
| SBP (mm Hg) | 130.8 ± 19.0 | 123.8 ± 16.7 | 122.4 ± 18.2 | 0.02 |
| DBP (mm HG) | 76.9 ± 11.7 | 73.6 ± 10.9 | 70.9 ± 13.1 | 0.02 |
| Fasting insulin (μU/mL) | 15.4 ± 12.2 | 9.3 ± 7.7 | 6.9 ± 4.8 | < 0.001 |
| Fasting glucose (mg/dL) | 100.3 ± 12.3 | 98.9 ± 9.6 | 97.9 ± 9.1 | 0.44 |
| 2 hr Glucose (mg/dL) | 140.7 ± 37.7 | 128.3 ± 30.3 | 110.3 ± 31.5 | < 0.001 |
| Total Cholesterol (mg/dL) | 185.9 ± 34.4 | 188.8 ± 38.7 | 175.4 ± 35.1 | 0.09 |
| LDL (mg/dL) | 122.5 ± 33.9 | 125.1 ± 36.8 | 107.1 ± 31.6 | 0.01 |
| HDL-C (mg/dL) | 43.6 ± 9.6 | 52.4 ± 18.2 | 52.5 ± 17.4 | 0.002 |
| Triglycerides (mg/dL) | 108.5 ± 68.3 | 79.0 ± 39.7 | 76.6 ± 49.6 | 0.002 |
| Triglycerides/HDL-C | 2.81 ± 2.63 | 1.61 ± 0.87 | 1.67 ± 1.42 | < 0.001 |
| M ($mg \cdot kg^{-1} \cdot min^{-1}$)* | 2.89 ± 0.61 | 5.26 ± 0.82 | 8.93 ± 2.27 | <0.001 |
| I (clamp) ((μU/mL)** | 92.5 ± 29.7 | 77.7 ± 23.6 | 67.4 ± 24.3 | < 0.001 |
| Metabolic Syndrome % meeting criteria | 35% | 17% | 6% | < 0.001 |

* M= mean glucose infusion rate required to maintain euglycemia during the final 60 minutes (clamp period) of the hyperinsulinemic procedure

** I (clamp)= mean insulin concentration collected during the hyperinsulinemic clamp procedure

## Table 4

Stratification by TG Concentration

| | Low TG <110 n= 143 | Intermediate TG 110-149 n= 23 | High TG ≥ 150 n= 19 | 1-way ANOVA P-value |
|---|---|---|---|---|
| Total M | 6.09 ± 2.97 | 4.40 ± 2.28 | 4.62 ± 1.83 | 0.01 |
| Age (yrs) | 39.9 ± 3.6 | 38.8 ± 3.0 | 40.4 ± 4.7 | 0.31 |
| Weight (kg) | 89.7 ± 23.0 | 98.3 ± 21.3 | 96.3 ± 18.1 | 0.14 |
| Percent body fat | 33.8 ± 8.6 | 34.7 ± 7.9 | 35.3 ± 5.6 | 0.73 |
| BMI ($kg/m^2$) | 28.3 ± 12.8 | 34.2 ± 6.9 | 33.6 ± 6. 9 | 0.24 |
| SBP (mm Hg) | 124.3 ± 16.5 | 122.7 ± 14.4 | 139.4 ± 30.0 | 0.002 |
| DBP (mm HG) | 73.1 ± 11.8 | 71.2 ± 9.5 | 81.8 ± 14.4 | 0.01 |
| Fasting insulin (μU/mL) | 9.2 ± 7.7 | 11.8 ± 6.6 | 17.9 ± 17.6 | < 0.001 |
| Fasting glucose (mg/dL) | 97.7 ± 9.4 | 101.9 ± 10.8 | 105.7 ± 14.1 | 0.002 |
| 2 hr Glucose (mg/dL) | 124.4 ± 34.9 | 129.0 ± 40.5 | 137.1 ± 31.3 | 0.32 |
| Total Cholesterol (mg/dL) | 179.1 ± 33.2 | 194.3 ± 37.2 | 202.3 ± 50.1 | 0.01 |
| LDL (mg/dL) | 116.6 ± 31. 9 | 122.2 ± 38.6 | 125.7 ± 50.1 | 0.48 |
| HDL-C (mg/dL) | 51.3 ± 16.5 | 45.4 ± 10.0 | 41.4 ± 15.7 | 0.02 |
| Triglycerides (mg/dL) | 66.1 ± 19.4 | 124.6 ± 12.7 | 205.9 ± 88.6 | < 0.001 |
| Triglycerides/HDL-C | 1.40 ± 0.594 | 2.84 ± 0.591 | 5.68 ± 3.75 | < 0.001 |

**Table 5**

Metabolic Syndrome Rate with TG criteria > 150 mg/dL and >110 mg/dL

| | Metabolic Syndrome Rate with TG criterion > 150 mg/dL | Metabolic Syndrome Rate with TG criterion > 110 mg/dL |
|---|---|---|
| Total Population | 19% | 25% |
| Insulin Sensitivity Tertiles | | |
| Insulin Resistant | 35% | 52% |
| Intermediate | 17% | 21% |
| Insulin Sensistive | 6% | 6% |