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Adolescent and Adult African Americans Have Similar Metabolic Dyslipidemia

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Abstract

Background—African Americans (AA) have lower triglycerides (TG) and higher high density lipoprotein-cholesterol (HDL-C) than other ethnic groups yet they also have higher risk for developing diabetes mellitus despite the strong relationship of dyslipidemia with insulin resistance. No studies directly compare adolescents and adults with regard to relationships amongst dyslipidemia, C-reactive protein (hsCRP), and insulin resistance. Here we compare AA adolescents to adults with regard to the relationships of adiposity-related lipid risk markers (TG/HDL ratio and non HDL-C) with body mass index (BMI), waist circumference (WC), homeostasis model of insulin resistance (HOMA), and hsCRP.

Methods—Two cohorts of healthy AA were recruited from the same urban community. Participants in each cohort were stratified by TG/HDL ratio (based on adult tertiles) and non-HDL-C levels. BMI, WC, HOMA and hsCRP were compared in adolescents and adults in the low, middle and high lipid strata.

Results—Prevalence of TG/HDL ratio greater than 2.028 (high group) was 16% (44/283) in adolescents and 33% (161/484) in adults; prevalence of non HDL-C above 145 and 160 respectively was 8% (22/283) in adolescents and 12% (60/484) in adults. HsCRP values were lower and HOMA values were higher in adolescents (both $p < 0.01$). As both TG/HDL ratio and non HDL-C strata increased, BMI, WC, HOMA, and hsCRP increased in both adolescents and adults. In the high TG/HDL and non HDL-C groups, BMI and WC were similar in adolescents vs. adults (BMI 34 kg/m^2 vs 32 kg/m^2; WC 101 cm vs 101 cm). After adjusting for non-HDL-C and other covariates, a 2-fold increase in TG/HDL was associated with increases of 10.4% in hsCRP

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(95% CI: 1.1% – 20.5%) and 24.2% in HOMA (95% CI: 16.4% – 32.6%). Non-HDL-C was not significant in models having TG/HDL.

Conclusions—Elevated TG/HDL ratio is associated with similar inflammation and metabolic risk relationships in adolescent and adult African-Americans.

Keywords

triglycerides; HDL cholesterol; obesity; inflammation; insulin resistance; risk factors

Introduction

Obesity, dyslipidemia, insulin resistance and inflammatory markers such as C-reactive protein (hsCRP) are strongly associated and together increase risk for metabolic and cardiovascular disease. Elevated serum triglyceride (TG) and serum lower high density lipoprotein (HDL) are associated with measures of insulin resistance and both lipid measures are components of metabolic syndrome.[1] The TG/HDL ratio has been shown to be a strong marker for cardiovascular risk and metabolic syndrome in obese children and adults [2–6] Non-HDL cholesterol (non-HDL-C) has also been shown to be strongly associated with the metabolic syndrome in children and reflects the concentration of atherogenic lipoproteins[5, 7]. Non-HDL-C is reported to be the best predictor of adult dyslipidemia and other cardiovascular risks. [8, 9] Insulin resistance is commonly associated with obesity in children and adolescents and has been shown to lead to decreased clearance of TG and Low density lipoprotein (LDL), overproduction of very low density lipoprotein (VLDL), and therefore decreased production of HDL [10–12]. However, direct comparisons between adults and children to determine if there is a difference in the magnitude of association with regard to these traits across the lifespan has not been previously studied.

Health related disparities have been identified in adult ethnic minority populations including African Americans[13, 14]. Ethnic differences in cardiovascular disease outcomes are apparent and important to consider. Compared to Caucasians, African American adults suffer higher rates of obesity and diabetes with disproportionally greater rates of of the premature cardiovascular morbidity and mortality. The Bogalusa Heart Study, which enrolled African American and Caucasian youth, demonstrated that many metabolic parameters, such as obesity, high blood pressure and lipid abnormalities tracked from childhood into adulthood. Although the trends were the same for African Americans and Caucasians, there was a higher prevalence of these risk factors in African Americans [15, 16]. Associations of elevated TG and low-HDL-C exist among both ethnic groups, but the magnitude is different from one ethnic group to another. African Americans have lower TG and higher HDL-C levels, compared to their Caucasian counterparts and this is observed in both children and adults [6, 17, 18]. Nonetheless, African American girls are observed to have higher body mass index (BMI) and greater insulin resistance compared to Caucasian girls of the same age [19]. Despite higher prevalence of insulin resistance, the phenotype of hypertriglyceridemia and low HDL-C are observed less frequently in African Americans of all ages[17].

Since TG/HDL ratio and non-HDL-C are strongly associated with insulin resistance and inflammation, we stratified adolescent and adult African Americans by these measures to determine if associations with BMI, waist circumference (WC), hsCRP, and the homeostasis model of insulin resistance (HOMA) were similar in the two age groups. These comparisons will inform discussions about metabolic risk across the lifespan.

Methods

Cohort

Adolescent and adult studies enrolled African Americans (based on self report) from the same urban community. The adolescent study enrolled participants between ages 13–18 years of age from 2009–2011. The adolescent study enrolled participants for a study designed to compare those with and without high blood pressure (BP; >120/80mmHg) and with and without obesity (defined as BMI >95th percentile) in a 2 × 2 design.[20] The adolescents were recruited from primary care pediatrics and family practices at Thomas Jefferson University and from community primary care practices. Exclusion criteria for adolescent participants were known diabetes, secondary hypertension, stage two hypertension, renal disease and other chronic diseases. This study protocol was approved by the Institutional Review Board of Thomas Jefferson University and the A.I. DuPont Hospital for Children. Written and informed consent was obtained from those 18 years old. Parent or guardian informed consent was obtained for adolescents under age 18.

Adults were between the ages 19–45, recruited from family practices at Thomas Jefferson University and from community primary care practices, and data were collected between 2006 and 20010. All the participants were without chronic health problems with the exception of elevated BP (>130/85mmHg) or receiving antihypertensive medication in approximately half the participants and obesity in half of the participants. Individuals with known diabetes or other chronic diseases were excluded from the adult study. The study protocol was approved by the Institutional Review Board of Thomas Jefferson University. Written informed consent was obtained from each participant at the time of the enrollment.

Study Methods

Similar methods and procedures were applied to both adolescent and adult studies. These methods have been published in other reports.[20, 21]. Data on health status, medication use and health related behaviors were obtained by self-report. Clinical assessment included BP and anthropometric measurements (height, weight and WC). BMI was calculated (weight in kilograms divided by height in meters squared). For the adolescent cohort, obesity was defined as BMI as >95th percentile by CDC criteria (http://www.cdc.gov/obesity/childhood/defining.html).

A fasting blood sample was obtained for glucose, insulin, lipids, and hsCRP. Glucose was measured by the glucose oxidase technique (YS model 27; Glucostat, Yellow Springs, Ohio). Samples of fasting plasma were stored frozen (−80 degrees C) for later assay of insulin and hsCRP. Plasma insulin concentration was assayed using a solid phase radioimmunoassay,(Coat-a-Count; Diagnostic Products Corp, Los Angeles, California).

Assay for hsCRP was performed using an elisa kit from R&D Systems (Minneapolis, MN). Insulin resistance was estimated using HOMA[22]. Fasting lipids were measured including TG, HDL-C, and total cholesterol with LDL-C calculated. Lipids were measured using the Hitachi 704 standard enzymatic method in the Lipid laboratory at Thomas Jefferson University

Statistical Methods

Subjects in each age range were stratified into groups by tertiles of TG/HDL: low (TG/HDL <= 1.136), middle (1.136< TG/HDL <= 2.028) and high (TG/HDL > 2.028). Adolescents were stratified into tertiles according to nonHDL-C strata: low (nonHDL-C <120), middle (120< nonHDL -C< 145) and high (nonHDL-C >=145). Subjects in the adult cohort were also stratified into tertiles according to nonHDL-C strata: low (nonHDL-C <130), middle (130< nonHDL -C< 160) and high (nonHDL-C >=160). The non HDL-C strata were based on ATP III/NHLBI expert guidelines.[23, 24] Adolescents and adults in the low, middle and high strata were compared with regard to BMI, WC, HOMA, and hsCRP.

Study variables were tabled and compared across lipid groups and age groups. Continuous variables are summarized by means with standard deviations or, if substantially skewed, were log transformed and summarized by geometric means with first and third quartiles of the distribution. The distributions of TG/HDL ratio, non-HDL-C, HOMA, and hsCRP were log transformed for testing and modeling. Student's t-tests or ANOVA F-tests were used to evaluate differences in means and Fisher's exact tests were used to evaluate differences in proportions.

Ordinary least squares regression models were used to analyze HOMA and hsCRP as they respectively relate to TG/HDL and non-HDL-C, particularly in adults vs. adolescents, while adjusting for gender, WC, BMI, systolic BP, and hypertension medications use. First, we tested interaction terms between age groups (adult was the reference level) and TG/HDL in HOMA and hsCRP models. Then we tested interaction terms between cohort and non-HDL-C in HOMA and hsCRP models. If determined not statistically significant, the interaction terms would be dropped from the models and both TG/HDL and non-HDL-C would be entered into the same models of HOMA and hsCRP.

The significance level for all hypothesis testing was set at $\alpha < 0.05$. All statistical analyses were conducted using SAS v9.4 (SAS Institute, Cary, NC, USA).

Results

Complete data were available for analysis on 283 adolescents and 484 adults. Table 1 provides summary data on the adolescent and adult cohorts and compares their BMI, WC, HOMA and hsCRP. The adolescents and adults had similar prevalence of obesity (50.2% and 51.4%, respectively). HOMA was significantly higher among adolescents than adults (1.86 versus 1.55, $p < 0.01$) and hsCRP was significantly lower among adolescents than adults (0.78 versus 1.67, $p < 0.01$).

Table 2 provides the TG/HDL ratio groups defined by adult tertiles of TG/HDL ratio and compares BMI, WC, HOMA and hsCRP across these tertile-based groups in both adolescents and adults. Forty-four of the 283 adolescents (16%) had a TG/HDL ratio above 2.028 (high adult tertile). In both adolescents and adults, BMI and WC significantly increased as TG/HDL ratio increased. The BMI in the high TG/HDL ratio groups was comparable with a geometric mean of 34.0 kg/m^2 in the adolescents and 32.2 kg/m^2 in the adults ($p = 0.18$). Similarly, the waist circumference geometric mean was 100.4 cm in adolescents and of 100.9 cm in adults ($p = 0.75$). Higher TG/HDL ratio was significantly related to higher hsCRP in both adolescents ($p < 0.01$) and adults ($p = 0.01$). The geometric means of hsCRP were not similar when comparing adolescents and adults in the high TG/HDL ratio groups (2.04 mg/dl versus 1.53 mg/dl), but the difference was not statistically significant ($p = 0.12$). Higher TG/HDL ratio was significantly related to higher HOMA in both adolescents ($p = 0.01$) and adults ($p < 0.01$). The geometric means of HOMA were significantly higher in the high TG/HDL adolescent cohort compared to the adults (3.36 mg/dl versus 1.99 mg/dl, $p < 0.01$).

Table 3 shows the non-HDL-C groups defined by adolescent and adult guidelines and compares the study variables of BMI, WC, HOMA and hsCRP by non-HDL-C group. Prevalence of elevated non-HDL-C was 22/283 (8%) in adolescents and 60/484 (12%) in adults. BMI and WC significantly increased as non-HDL-C increased in both cohorts. The BMI geometric means in the high non-HDL-C groups were comparable in adolescents and adults (33.1 kg/m^2 versus 32.0 kg/m^2, respectively). Similarly, the WC geometric means in the high non-HDL-C groups were comparably elevated in adolescents versus adults (100.8 cm versus 101.7 cm, respectively). HsCRP tended to be higher with higher non-HDL-C in both adolescents and adults, but not statistically significantly ($p = 0.09$ and $p = 0.07$, respectively). HOMA also was higher with higher non-HDL-C in adolescents and adults ($p = 0.03$ and $p = 0.048$).

To further explore these relationships, we fit four exploratory regression models. We regressed log transformed hsCRP and log transformed HOMA, respectively, on log transformed TG/HDL and log transformed non-HDL-C, respectively, an age group indicator variable (adult was the reference), and a term for the interaction with age group while adjusting for potential confounding variables. We found that TG/HDL and non-HDL-C do not have statistically significant interactions with either hsCRP (TG/HDL-cohort interaction $p = 0.16$; non-HDL-C-cohort interaction $p = 0.64$) or HOMA (TG/HDL-cohort interaction $p = 0.77$; non-HDL-C-cohort interaction $p = 0.62$). We then fit two more adjusted regression models, one for hsCRP and one for HOMA, including both log transformed dyslipidemia markers. In adjusted regression models having TG/HDL, non-HDL-C is not important or statistically significant for predicting hsCRP and HOMA ($p = 0.84$ and $p = 0.45$, respectively). See figure 1 which depicts the unadjusted relationships between log TG/HDL and log hsCRP (panel A.) and log HOMA (panel B.). After adjusting for non-HDL-C and other covariates, our models suggest that among these adolescents and adults, a 2-fold higher TG/HDL ratio was associated with statistically significant higher hsCRP (10.4%; 95% CI: 1.1% – 20.5%) and HOMA (24.2%: 95% CI: 16.4% – 32.6%).

Discussion

While studies in both adults and children show strong relationships among dyslipidemia, obesity, inflammation, and insulin resistance, no prior study has directly compared the quantitative relationships in different age groups. Our data show that when African-Americans are stratified by TG/HDL ratio or non HDL-C values, there are similar levels of metabolic risk in dyslipidemic adolescent and adult African Americans. TG/HDL ratio appears to be the main driver of these relationships. Both adolescents and adults demonstrate comparable BMI and waist circumference at similar levels of dyslipidemia. HsCRP and HOMA were also elevated in adolescents and adults with elevated TG/HDL ratio and non-HDL-C, but when placed in the same model, only TG/HDL was predictive of hsCRP and HOMA. The relationship between HOMA and dyslipidemia appears to be somewhat stronger in adolescent versus adult African Americans, while the relationship of hsCRP with dyslipidemia may be stronger in adults versus adolescents, but this effect modification was not statistically significant in these data.

Metabolic syndrome is a cluster of metabolic and hemodynamic risk factors within individuals that markedly increase risk for adverse cardiovascular outcomes. The core abnormality that links the risk factors is insulin resistance, or impaired insulin mediated glucose uptake,[25, 26]. Insulin resistance, or impaired tissue sensitivity to insulin action, is difficult to quantify clinically. The concept of metabolic syndrome has been developed as a strategy to identify individuals with multiple cardiovascular disease risk factors that are linked with insulin resistance [27, 28]. The clinical utility of the TG/HDL-C ratio in predicting insulin resistance and metabolic syndrome has been recently described. A TG/HDL ratio of 3.5 or above has been shown to be a simple marker for metabolic syndrome and probable cardiovascular disease in adults. Several studies have demonstrated that children with a TG/HDL ratio > 3 had significantly higher BMI and waist circumference. These authors also noted a racial difference between African American and Caucasian children, with a TG/HDL ratio of 2.5 being as accurate in African American children as 3 was in Caucasions.[2–5]

The 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. Several reports describe greater insulin resistance in African Americans compared to Caucasians, including children as well as adults..[29–31] Despite having greater insulin resistance, African Americans have more favorable lipid profiles compared to Caucasians, with TG and HDL-C concentrations compared to Caucasians.[32, 33] Consequently, because metabolic syndrome is determined based on set thresholds for elevated TG and low HDL-C, the reported prevalence of metabolic syndrome is lower in African Americans compared to Caucasians.[34]

Although the more favorable lipid profile observed in African Americans compared to Caucasians would suggest lower atherogenic risk, additional studies indicate significant metabolic risk among African Americans despite somewhat lower TG and higher HDL-C. In a study on young adult African Americans, age 30–45 years, significant correlations were found for TG, HDL-C, and TG/HDL-C ratio with insulin resistance, quantified by the

insulin clamp procedure. Despite obesity in 50% of that sample, only 10% of participants had plasma TG levels ≥150 mg/dL, a level that was a criterion for metabolic syndrome. Participants with TG levels from 110 to 149 mg/dL had measures of insulin resistance comparable to those with TG >150 mg/dL.[35] Despite a more favorable lipid profile among African Americans, it is possible that they have a different threshold for adverse effects of relative dyslipidemia. Lipid mediated vascular injury could be mediated through an oxidative stress pathway. This theory was investigated by Lopes et al. who investigated the effect of acute hyperlipidemia in African Americans and Caucasians. [36] In both African American and Caucasian groups, a comparable increase in plasma TG concentration occurred following an infusion of Intralipid and heparin. However, F2-isoprostanes, a biomarker of oxidative stress in humans, increased significantly more in African Americans compared to Caucasians. Although this report is based on a short-term rise in TG, the results suggest that African Americans could have greater sensitivity to increases in TG. Some reports on metabolic syndrome prevalence, based on studies that include various race groups, question the validity of applying the same criteria for metabolic syndrome to all race and ethnic groups[37, 38]. In both our adult and adolescent cohorts, the TG/HDL ratio, which captures modest increases in TG and modest decreases in HDL-C, may be a better indicator of insulin resistance, metabolic syndrome, and heightened atherogenic cardiovascular risk in African Americans than considering only TG or HDL-C.

The data demonstrating greater insulin resistance in adolescents compared to adults, present across all TG/HDL and non-HDL-C strata may, to some extent, be due to the relative insulin resistance of adolescence. Previous clinical studies in healthy adolescents have demonstrated the presence of a transient increase in insulin resistance that occurs during normal pubertal development.[39–41] The factors that contribute to the changes in insulin action during puberty have not been clearly defined. As in adults, insulin resistance in adolescents is strongly associated with BMI. However, the relative insulin resistance of puberty is not explained by differences in BMI or adiposity.[40]

Obesity-related inflammation has been described in both adults and children. A strong correlation between obesity and CRP was reported among middle-age and elderly African americans in the Jackson Heart Study.[42] We previously reported similar CRP relationships with obesity in our adolescent and young adult African American cohorts. African American adolescents with BMI exceeding 30 Kg/m^2 had levels of CRP that were similar to obese young adult African Americans.[43] [Data from the National Health and Nutrition Examination Survey (NHANES) on children and adolescents document a significant association of plasma hsCRP level with measures of BMI and skinfold thickness.[44] These authors reported significant associations unfavorable changes in metabolic parameters among obese adolescents, including increased Tg/HDL ratio. The Cardiovascular Risk in Young Finns Study, which obtained longitudinal data from childhood through young adulthood, reported that childhood BMI and hsCRP were predictive of adverse health consequences in adulthood.[45] Our data that demonstrate comparable levels of hsCRP in both adolescents and adults with BMI >30 Kg/m^2 suggest the possibility of early adult onset of the adverse health consequences of concurrent exposure inflammation with more atherogenic lipid status.

Study limitations

We examined only African Americans, so the trends observed may not be applicable to other ethnic groups. The study was cross-sectional and did not determine causality. Tanner staging was not done so the impact of stage of puberty on HOMA could not be assessed. The study group was drawn from two primary care practices in the same urban hospital so the results may not be applicable to African Americans in suburban or rural settings.

Conclusions

Our data demonstrate that elevated TG/HDL ratio is associated with equal if not more metabolic risk in adolescents than adult African Americans. Age does not impact adverse metabolic profiles related to obesity in African Americans..

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Highlights

"Adolescent and Adult African Americans Have Similar Metabolic Dyslipidemia"

1. Studies comparing adolescents and adults with regard to metabolic disturbances related to dyslipidemia have not been performed.
2. African Americans experience metabolic disturbances at lower levels of triglycerides than Caucasians.
3. When stratified by triglyceride/HDL-C ratio or by non HDL- C level, adolescent African Americans have similar BMI and waist circumference as adults.
4. As triglyceride/HDL-C ratio increases, HOMA and hs CRP increase, the slope of this relationship is steeper in adolescents compared to adults.
5. Triglyceride/HDL-C ratio is a more important determinant of metabolic risk than non HDL-C.

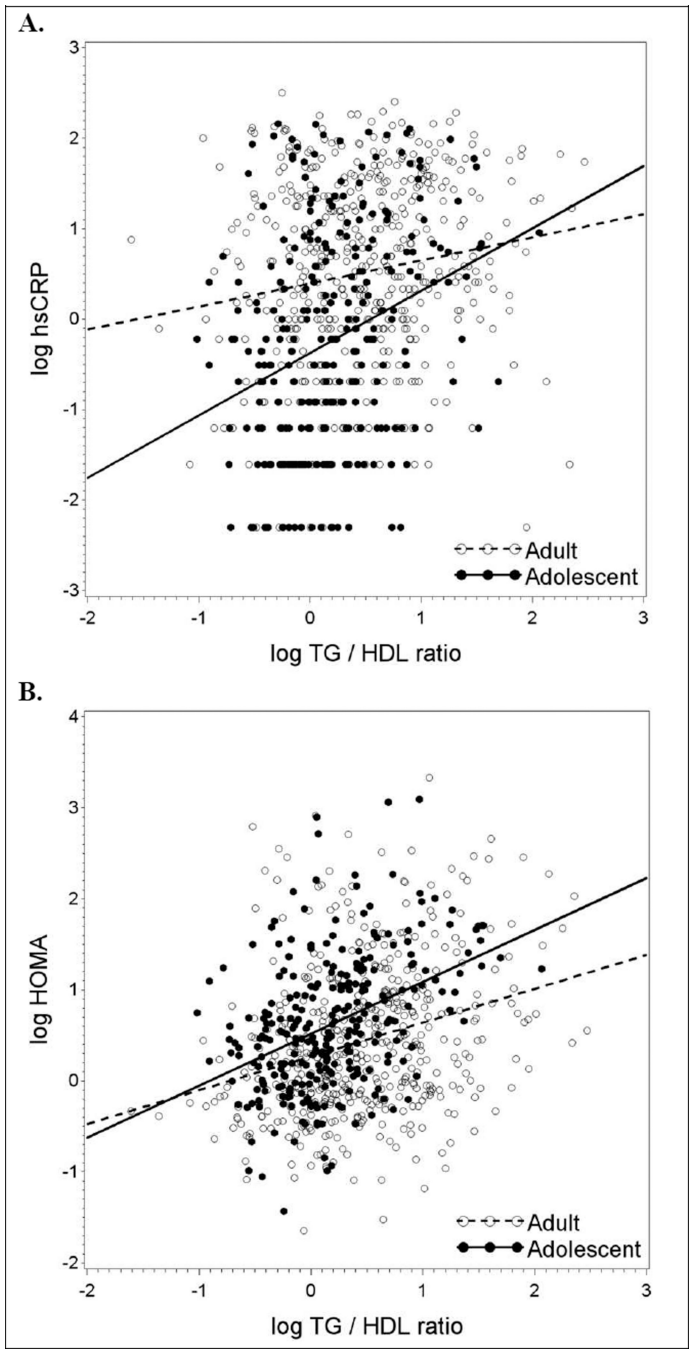

Figure 1.

Log transformed hsCRP (panel A.) and log transformed HOMA (panel B.) by log transformed triglyceride/HDL ratio with least squares regression slopes for adolescents and adults.

Table 1

Descriptive statistics on the adolescent and adult participants summarized with frequencies (percentages), means (SD), or geometric means [1st quartile, 3rd quartile].

Variable	Adult (N=484)	Adolescent (N=283)	p†
Age (years)	37.73 (7.50)	16.19 (1.68)	<.01
Gender, female	242 (50.0%)	136 (48.2%)	0.65
Smoking	300 (62.0%)	9 (3.2%)	<.01
Alcohol use	232 (47.9%)	17 (6.1%)	<.01
BMI (kg/m^2)	30.64 [25.71, 35.42]	28.40 [22.92, 34.66]	<.01
WC (cm)	96.87 [86.00,107.00]	86.74 [74.15,101.65]	<.01
Obesity**	243 (50.2%)	145 (51.4%)	0.77
SBP (mmHg)	120.66 [110.00, 130.0]	112.99 [105.67, 120.67]	<.01
DBP (mmHg)	74.53 [66.00, 82.00]	62.46 [57.67, 67.67]	<.01
Heart rate (bpm)	70.39 [64.00, 78.00]	71.07 [64.00, 78.00]	0.35
Hypertensive medications	169 (34.9%)	N/A	
High BP*	246 (50.8%)	78 (27.7%)	<.01
HDL-C (mg/dl)	47.95 (14.75)	52.63 (12.65)	<.01
LDL-C (mg/dl)	108.50 (29.18)	88.63 (26.70)	<.01
Total cholesterol (mg/dl)	172.77 (32.47)	155.10 (29.89)	<.01
Non-HDL-C (mg/dl)	124.82 (31.33)	102.47 (28.93)	<.01
Triglycerides (mg/dl)	72.34 [51.00, 97.00]	61.72 [47.00, 77.00]	<.01
Triglycerides/HDL-C ratio	1.58 [1.00, 2.38]	1.21 [0.83, 1.60]	<.01
Fasting glucose (mg/dl)	103.66 (18.85)	96.91 (10.46)	<.01
Fasting insulin (mg/dl)	6.14 [3.55, 9.70]	7.84 [4.70, 12.50]	<.01
Metabolic syndrome‡	126 (26.0%)	39 (13.8%)	<.01
hsCRP (mg/dl)	1.67 [0.90, 3.95]	0.78 [0.30, 2.20]	<.01
HOMA (mg/dl)	1.55 [0.85, 2.48]	1.86 [1.12, 2.93]	<.01

† Fishers exact test (categorical) or Students t-test (continuous);

* High BP: SBP ≥120/80 mmHg (adolescents) or ≥130/85 or HTN Rx (adults);

** Obesity: >=95th percentile (adolescents), >=30 BMI (adults);

‡ Metabolic Syndrome: 3 or more of following: Waist circumference ≥102 cm (males) or ≥88 (females), SBP ≥120/80 mmHg (adolescents) or ≥130/85 or hypertension medications use (adults), HDL <40 mg/dl (males) or <50 (females), Triglycerides ≥110 mg/dl (adolescents) or ≥150 (adults), Fasting glucose ≥110 mg/dl.

Abbreviations
SD = Standard Deviation
BMI = body mass index
WC = waist circumference
SBP = systolic blood pressure
DBP = diastolic blood pressure
HDL-C = high density lipoprotein cholesterol
LDL-C = low density lipoprotein cholesterol
hsCRP = high sensitivity c-reactive protein
HOMA = homeostasis model of insulin resistance

Table 2

Selected study variables by age group and triglyceride/HDL ratio groups summarized with means (SD) or geometric means [1st quartile, 3rd quartile].

	Adults				Adolescents			
Variable	**Low (Tri/HDL <= 1.14) (N = 160)**	**Middle (1.14<Tri/HDL<=2.03) (N = 163)**	**High (Tri/HDL > 2.03) (N = 161)**	**p†**	**Low (Tri/HDL <= 1.14) (N = 136)**	**Middle (1.14<Tri/HDL<=2.03) (N = 103)**	**High (Tri/HDL > 2.028) (N = 44)**	**p†**
Waist Circumference (cm)	92.93 [82.00,102.0]	96.95 [83.00,110.0]	100.87 [91.00,110.0]	<.01	80.42 [70.00,90.00]	90.56 [77.50,105.0]	100.44 [86.50,117.0]	<.01
BMI (kg/m2)	29.22 [24.75,32.90]	30.60 [25.30,37.32]	32.16 [27.66,36.67]	0.02	25.93 [21.20,30.09]	29.75 [24.71,35.69]	34.04 [29.19,40.07]	<.01
SBP (mmHg)	120.40 [110.00, 129.50]	119.49 [109.00, 130.00]	122.12 [112.00, 130.00]	0.37	111.68 [104.33, 118.33]	112.65 [105.33, 121.00]	117.71 [110.58, 125.39]	0.01
DBP (mmHg)	74.34 [65.50, 83.00]	73.56 [66.00, 80.00]	75.70 [68.00, 82.00]	0.30	62.25 [58.00, 67.83]	61.66 [57.00, 66.22]	64.90 [58.67, 72.00]	0.06
HDL-C (mg/dl)	58.99 (14.66)	47.29 (11.37)	37.64 (9.10)	<.01	59.61 (11.54)	48.91 (9.74)	39.23 (6.66)	<.01
LDL-C (mg/dl)	105.07 (26.95)	106.53 (28.80)	113.91 (31.04)	0.03	83.58 (25.21)	92.13 (27.27)	97.09 (27.65)	0.01
Total Cholesterol (mg/dl)	173.54 (29.34)	168.15 (32.06)	176.67 (35.33)	0.10	152.79 (29.55)	155.54 (29.74)	161.95 (30.96)	0.29
non-HDL-Cholesterol	114.55 (27.31)	120.86 (28.94)	139.03 (32.40)	<.01	93.18 (25.69)	106.63 (27.57)	122.73 (30.57)	<.01
Triglycerides (mg/dl)	45.77 [40.00, 54.00]	70.18 [60.00, 82.00]	117.58 [92.00, 145.00]	<.01	46.27 [39.00, 55.00]	69.63 [60.00, 78.00]	115.22 [88.00, 137.50]	<.01
Fasting Glucose (mg/dl)	100.26 (16.99)	102.53 (19.78)	108.19 (18.87)	<.01	95.32 (8.35)	97.77 (12.88)	100.16 (9.21)	0.01
hsCRP (mg/dl)	1.41 [0.60, 3.75]	1.60 [0.80, 3.80]	2.04 [1.10, 4.70]	0.01	0.60 [0.20, 1.50]	0.84 [0.30, 2.20]	1.53 [0.65, 4.23]	<.01
HOMA (mg/dl)	1.25 [0.74, 1.70]	1.50 [0.83, 2.55]	1.99 [0.98, 3.39]	<.01	1.45 [0.91, 1.98]	2.06 [1.31, 3.23]	3.36 [2.18, 5.28]	<.01

†ANOVA F-test (continuous variables)

Abbreviations
HDL-C = high density lipoprotein cholesterol
SD = Standard Deviation
BMI = body mass index
SBP = systolic blood pressure
DBP = diastolic blood pressure
LDL-C = low density lipoprotein cholesterol
hsCRP = high sensitivity c-reactive protein
HOMA = homeostasis model of insulin resistance
ANOVA = analysis of variance

Table 3

Selected study variables by age group and Non-HDL groups summarized with means (SD) or geometric means [1st quartile, 3rd quartile].

Variable	Adults				Adolescents			
	Low (nonHDL < 130) (N = 274)	Middle (130<= nonHDL<160) (N = 150)	High (nonHDL >= 160) (N = 60)	p†	Low (nonHDL < 120) (N = 223)	Middle (120<=nonHDL<145) (N = 38)	High (nonHDL >= 145) (N = 22)	p†
Waist Circumference (cm)	94.92 [83.00,105.0]	98.57 [89.00,111.0]	101.69 [93.50,110.0]	0.01	84.63 [71.40,98.00]	93.09 [80.00,111.2]	100.83 [89.00,116.9]	<.01
BMI (kg/m2)	29.87 [24.96,34.65]	31.54 [27.32,36.67]	32.00 [27.77,36.45]	0.04	27.54 [21.97,33.22]	31.43 [26.17,37.86]	33.09 [29.17,37.96]	<.01
SBP (mmHg)	120.28 [110.00, 130.00]	121.06 [110.00, 131.00]	121.44 [111.00, 130.00]	0.89	112.57 [105.33, 120.33]	116.10 [110.67, 125.78]	111.51 [103.33, 121.00]	0.28
DBP (mmHg)	74.30 [66.00, 82.00]	74.36 [65.00, 83.00]	75.96 [70.50, 82.00]	0.69	61.91 [57.67, 67.00]	64.49 [59.67, 69.00]	64.36 [58.00, 71.33]	0.11
HDL-C (mg/dl)	49.16 (15.96)	47.41 (13.10)	43.80 (12.00)	0.04	53.42 (12.39)	48.87 (11.97)	50.05 (15.91)	0.14
LDL-C (mg/dl)	89.15 (17.67)	125.05 (13.56)	155.50 (19.44)	<.01	78.49 (16.35)	115.16 (14.05)	147.64 (21.29)	<.01
Total Cholesterol (mg/dl)	152.14 (22.02)	190.47 (15.47)	222.73 (21.66)	<.01	144.43 (19.95)	181.89 (13.12)	218.55 (27.72)	<.01
Triglycerides (mg/dl)	62.61 [47.00, 83.00]	80.31 [58.00, 111.00]	107.80 [83.49, 144.50]	<.01	57.71 [45.00, 72.00]	74.68 [60.00, 103.00]	90.61 [54.00, 136.00]	<.01
Trig/HDL ratio	1.34 [0.88, 1.96]	1.76 [1.11, 2.51]	2.55 [1.68, 3.67]	<.01	1.11 [0.79, 1.50]	1.57 [1.13, 2.15]	1.89 [1.15, 3.63]	<.01
Fasting Glucose (mg/dl)	101.67 (17.82)	105.72 (17.60)	107.63 (24.74)	0.07	96.20 (8.49)	97.95 (6.74)	102.77 (24.17)	0.28
hsCRP (mg/dl)	1.50 [0.70, 3.80]	1.87 [1.00, 4.60]	2.02 [1.05, 4.45]	0.07	0.73 [0.30, 2.00]	0.81 [0.30, 2.20]	1.54 [0.50, 3.80]	0.09
HOMA (mg/dl)	1.42 [0.80, 2.22]	1.69 [0.90, 2.99]	1.86 [1.13, 2.69]	0.05	1.74 [1.06, 2.80]	2.06 [1.21, 3.34]	3.39 [2.11, 5.43]	0.03

†ANOVA F-test (continuous variables)

Abbreviations
HDL-C = high density lipoprotein cholesterol
SD = Standard Deviation
BMI = body mass index
SBP = systolic blood pressure
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