OBJECTIVE — To examine sex-specific black/white differences in lipoprotein profile and the role of visceral adiposity and to assess the relationship between insulin sensitivity and lipoprotein profiles in each group.

RESEARCH DESIGN AND METHODS — Fasting lipoprotein particle size and concentration and visceral adipose tissue (VAT) were determined in 226 children (117 black, 101 male) aged 8 to <18 years. The relationship between lipoproteins and insulin sensitivity was evaluated in a subset of 194 children (100 black, 88 male) who underwent a hyperinsulinemic-euglycemic clamp.

RESULTS — Black male children had smaller VLDL and black female children had larger HDL size than their white counterparts. Overall, blacks had larger LDL size with no sex-specific race differences. After adjusting for VAT and sex, only VLDL size and concentrations remained significantly favorable in blacks. Analysis of lipoprotein particle size and concentration across insulin sensitivity quartiles revealed that in both racial groups, the most insulin-resistant children had higher concentrations of small dense LDL, small HDL, and large VLDL and smaller LDL and HDL sizes than their more insulin-sensitive counterparts.

CONCLUSIONS — The previously reported favorable lipoprotein profiles in black versus white children is partly due to race differences in VAT. In both groups, however, the most insulin-resistant youths have a high-risk atherogenic profile of small dense LDL, small HDL, and large VLDL, akin to the atherogenic lipoprotein pattern in adults with coronary artery disease.

Diabetes Care 32:2087–2093, 2009
Lipoproteins and insulin sensitivity in youth

Table 1—Physical characteristics of the participants

|          | Male            | Female           | Black vs. white | P     |
|----------|-----------------|------------------|-----------------|-------|
|          | Blacks | Whites | Blacks | Whites | Male | Female |                   |
| n        | 49     | 52     | 68     | 57     | 0.033| NS      |                   |
| Age (years) | 12.7 ± 0.3 | 13.5 ± 0.3 | 12.8 ± 0.3 | 12.6 ± 0.3 |       |         |                   |
| Tanner stage |    |        |        |        |        |         |                   |
| I        | 9      | 6      | 10     | 8      | NS    | NS      |                   |
| II–III   | 22     | 26     | 15     | 15     | NS    | NS      |                   |
| IV–V     | 18     | 20     | 43     | 34     | NS    | NS      |                   |
| Height (cm) | 158.9 ± 2.0 | 164.5 ± 1.8 | 154.8 ± 1.3 | 154.8 ± 1.6 |       |         |                   |
| Weight (kg) | 70.3 ± 3.9 | 76.1 ± 4.6 | 70.6 ± 3.4 | 69.5 ± 3.7 |       |         |                   |
| BMI (kg/m²) | 27.0 ± 1.1 | 27.1 ± 1.2 | 28.4 ± 1.1 | 28.2 ± 1.2 |       |         |                   |
| BMI percentile | 83.0 ± 3.1 | 79.3 ± 4.0 | 82.7 ± 3.0 | 84.2 ± 2.9 |       |         |                   |
| Fat mass (kg) | 20.8 ± 2.2 | 21.5 ± 2.3 | 26.8 ± 2.0 | 25.7 ± 2.1 |       |         |                   |
| Fat-free mass (kg) | 43.9 ± 1.8 | 44.7 ± 1.9 | 40.0 ± 1.4 | 37.1 ± 1.3 |       |         |                   |
| Body fat (%) | 27.9 ± 2.1 | 27.7 ± 1.8 | 35.3 ± 1.4 | 36.2 ± 1.5 |       |         |                   |
| Waist circumference (cm) | 85.6 ± 3.0 | 90.8 ± 3.3 | 83.0 ± 2.9 | 80.1 ± 2.9 |       |         |                   |
| VAT (cm²) | 39.4 ± 5.4 | 56.1 ± 6.7 | 37.3 ± 3.6 | 52.9 ± 5.4 | 0.055| 0.018   |                   |
| SAT (cm²) | 245.8 ± 32.3 | 303.9 ± 37.3 | 327.3 ± 29.0 | 339.2 ± 31.6 | NS   | NS      |                   |

Data are means ± SEM. Tanner stages compared using χ². All other variables were compared using independent t tests. SAT, subcutaneous adipose tissue.

Studies took place at the Children’s Hospital of Pittsburgh NIH-funded Pediatric Clinical and Translational Research Center after institutional review board approval. Participants and their parents gave written informed consent. Of the 226 youth, 194 had a hyperinsulinemic-euglycemic clamp. Exclusion criteria included diagnosis of diabetes and use of medications that influence glucose, lipid metabolism, or blood pressure. Participants’ health was assessed by medical history, physical examination, and hematological and biochemical tests. Pubertal development was assessed using Tanner criteria.

Body weight and height were measured using standardized equipment. Waist circumference was obtained at the midpoint between the lowest rib and the iliac crest (17). Body composition and abdominal adiposity were assessed by dual-energy X-ray absorptiometry and computed tomography, respectively, as described previously (12). Fasting blood samples were collected from all 226 children for analysis of lipoprotein particle size and concentration.

In vivo insulin sensitivity

A subset of children (100 black and 94 white, including 16 girls with PCOS) underwent a 3-h hyperinsulinemic-euglycemic clamp after 10–12 h of overnight fasting. Briefly, intravenous crystalline insulin (Humulin; Lilly, Indianapolis, IN) was infused at a constant rate of 40 mU·m⁻²·min⁻¹ in normal-weight subjects and 80 mU·m⁻²·min⁻¹ in obese subjects to suppress hepatic glucose production, as described previously (12). Plasma glucose was clamped at 5.6 mmol/l with a variable rate infusion of 20% dextrose based on arterialized plasma glucose determined every 5 min.

Biochemical measurements

Plasma glucose was measured using a glucose analyzer (YSI, Yellow Springs, OH), and insulin concentrations were measured by radioimmunoassay (12). Plasma lipid concentrations were determined using the standards of the Centers for Disease Control and Prevention as described previously (18). Plasma insulin concentrations were determined using nuclear magnetic resonance spectroscopy (LipoScience, Raleigh, NC) (19).

Calculations

Insulin-stimulated glucose disposal was calculated using the average exogenous glucose infusion rate during the final 30 min of the clamp (12). Insulin sensitivity was calculated by dividing the insulin-stimulated glucose disposal rate by steady-state plasma insulin concentrations during the last 30 min of the clamp, as described previously (3,12).

Statistical analysis

Independent t tests or χ² tests for categorical variables were used to examine race-related differences in subject characteristics and lipoprotein particle size and concentration in the group as a whole and stratified by sex. ANCOVA was used to determine the influence of sex and visceral adiposity on race-related differences in lipoprotein particle size and concentration. Black and white subjects were divided into insulin sensitivity quartiles by sex. One-way ANOVA or the nonparametric Kruskal-Wallis test, based on the nonviolation of statistical assumptions, was used to compare differences in lipoprotein particle size and concentration among quartiles. Tukey’s post hoc comparison was used to identify differences among quartiles. Because insulin sensitivity changes with puberty, we analyzed differences among quartiles with an ANCOVA including Tanner stage as a covariate. Data were also analyzed with exclusion of the 16 girls with PCOS to determine whether this condition may have affected our results. Stepwise multiple regression was used to assess the contribution of race, sex, age, insulin sensitivity, and VAT to lipoprotein particle size. Data are presented as means ± SEM with significance at P < 0.05.

RESULTS

Sex-specific, race-related differences

Participant characteristics are summarized in Table 1. Black and white youth had similar body weight, BMI, body composition, and subcutaneous abdominal
adipose tissue. VAT was lower in black than in white girls, with a similar tendency in black versus white boys \((P = 0.055)\).

As a group, black children had lower VLDL, total LDL, and small dense LDL and higher large HDL concentrations and larger HDL and LDL and smaller VLDL particle sizes than whites (Supplementary Table A1, available in an online appendix at http://care.diabetesjournals.org/cgi/content/full/dc09-0380). After correcting for sex and VAT, race differences remained in VLDL particle size and total, large, and medium VLDL and total HDL concentrations (Table A1).

Table 2 depicts race data grouped by sex. Black male children had smaller VLDL particle size and black female children had larger HDL size than their white counterparts. After adjustment for VAT, differences in VLDL particle size remained in male children \((P = 0.028)\) and for HDL particle size persisted in female children \((P = 0.084)\). Black male and female children had lower concentrations of total, large, and medium VLDL, and black male children had lower concentrations of total, small, and very small LDL than their white counterparts. After adjustment for VAT, differences in VLDL remained in both sexes but differences in HDL disappeared in male children.

**Insulin sensitivity, lipoprotein particle size, and concentrations**

Figure 1 depicts lipoprotein particle size by in vivo insulin sensitivity quartiles. Irrespective of sex, black and white children in the lowest quartile of insulin sensitivity had smaller HDL (Fig. 1A) and LDL (Fig. 1B) size than children in the upper quartiles. White male children in the lowest two quartiles of insulin sensitivity had larger VLDL particle size (Fig. 1C) than their counterparts in the upper quartiles.

Figures 2 and 3 depict lipoprotein particle concentrations by in vivo insulin sensitivity quartiles. In white male children and in both sexes for black children, those in the lowest quartile of insulin sensitivity had lower concentrations of large and higher concentrations of small HDL than children in the top quartile (Fig. 2A and B). Similarly, small dense LDL and very small LDL concentrations were higher in the lowest than in the uppermost quartile of insulin sensitivity in both races (Fig. 3A and B), irrespective of sex. Large VLDL and chylomicron concentrations were significantly higher in the most insulin-resistant quartile of black male children \((P < 0.05)\) and in the bottom two quartiles of white children \((P < 0.05)\) compared with the most insulin-sensitive children in each group (Fig. 3C).

After correction for pubertal development across insulin sensitivity quartiles, the significance for large HDL

---

### Table 2—Lipoprotein subclass concentration, particle size, and plasma lipids in black versus white children

|                  | Male |         | Female | Nominal P |         |
|------------------|------|---------|--------|-----------|---------|
|                  | Blacks | Whites | Blacks | Whites |         |
|                  | 49    | 52      | 68     | 57       |         |
| Total VLDL and chylomicrons (nmol/l) |        |        |        |          |         |
| Total VLDL and chylomicrons | 41.7 ± 2.9 | 54.8 ± 2.9 | 40.3 ± 2.2 | 53.5 ± 2.5 | 0.002     |
| Large VLDL and chylomicrons | 1.7 ± 0.4 | 4.1 ± 0.7 | 1.3 ± 0.3 | 2.7 ± 0.4 | <0.001   |
| Medium VLDL | 11.4 ± 1.5 | 19.4 ± 1.9 | 10.8 ± 1.1 | 18.3 ± 1.3 | 0.005     |
| Small VLDL | 28.6 ± 1.7 | 31.4 ± 2.1 | 28.2 ± 1.6 | 32.4 ± 1.6 | 0.007     |
| Total LDL (nmol/l) |        |        |        |          |         |
| Total LDL | 833.0 ± 40.4 | 993.8 ± 53.3 | 828.6 ± 35.6 | 887.7 ± 43.9 | 0.019     |
| Large LDL | 316.1 ± 20.0 | 302.6 ± 22.2 | 321.3 ± 15.3 | 296.4 ± 16.4 | NS       |
| Small LDL | 481.6 ± 41.3 | 649.8 ± 55.3 | 474.5 ± 36.1 | 553.7 ± 48.3 | 0.017     |
| Medium small LDL | 104.9 ± 8.4 | 133.8 ± 12.4 | 100.2 ± 7.1 | 116.4 ± 9.4 | NS       |
| Very small LDL | 376.8 ± 33.2 | 515.9 ± 43.2 | 374.3 ± 29.3 | 437.3 ± 39.1 | 0.012     |
| Total HDL (µmol/l) |        |        |        |          |         |
| Total HDL | 27.1 ± 0.7 | 25.8 ± 0.7 | 25.1 ± 0.5 | 24.7 ± 0.5 | NS       |
| Large HDL | 6.2 ± 0.5 | 5.5 ± 0.5 | 6.2 ± 0.3 | 5.4 ± 0.4 | NS       |
| Medium HDL | 4.8 ± 0.5 | 4.5 ± 0.7 | 3.5 ± 0.4 | 3.7 ± 0.3 | NS       |
| Small HDL | 16.1 ± 0.6 | 15.8 ± 0.7 | 15.4 ± 0.6 | 15.6 ± 0.5 | NS       |
| IDL (nmol/l) | 35.3 ± 4.7 | 41.4 ± 7.4 | 32.7 ± 4.1 | 37.4 ± 4.8 | NS       |
| Lipoprotein particle size (nm) |        |        |        |          |         |
| VLDL | 50.8 ± 1.2 | 56.4 ± 1.5 | 50.7 ± 1.3 | 52.8 ± 1.0 | 0.004     |
| LDL | 21.2 ± 0.1 | 20.9 ± 0.1 | 21.2 ± 0.1 | 21.2 ± 0.1 | NS       |
| HDL | 9.1 ± 0.1 | 9.0 ± 0.1 | 9.1 ± 0.1 | 8.9 ± 0.1 | 0.007     |
| Plasma lipids (mmol/l) |        |        |        |          |         |
| Total cholesterol | 4.09 ± 0.12 | 4.49 ± 0.13 | 3.93 ± 0.09 | 4.12 ± 0.09 | 0.031     |
| LDL cholesterol | 2.44 ± 0.10 | 2.66 ± 0.12 | 2.36 ± 0.09 | 2.45 ± 0.08 | NS       |
| HDL cholesterol | 1.20 ± 0.05 | 1.16 ± 0.04 | 1.18 ± 0.03 | 1.11 ± 0.03 | NS       |
| Total triglycerides | 1.00 ± 0.08 | 1.50 ± 0.14 | 0.94 ± 0.07 | 1.26 ± 0.08 | 0.003     |
| VLDL triglycerides | 0.20 ± 0.02 | 0.30 ± 0.03 | 0.19 ± 0.01 | 0.25 ± 0.02 | 0.003     |

Data are means ± SEM. All comparisons were made using independent t tests. Nominal P values indicate that significance values are unadjusted for multiple comparisons. IDL, intermediate-density lipoprotein.
concentrations in black male children changed from $P = 0.022$ to $P = 0.111$; in black female children, the significance for small HDL changed from $P = 0.037$ to $P = 0.109$ and, in white male children, it changed from $P = 0.027$ to $P = 0.078$. Excluding black or white girls with PCOS from their respective datasets did not change significance values across quartiles.

**Contribution of insulin sensitivity and visceral adiposity to lipoprotein particle size**

In multiple regression analyses with lipoprotein particle size as the dependent variable and race, sex, age, insulin sensitivity, and VAT as the independent variables, VAT and insulin sensitivity independently and together explained 26% of the variance ($P < 0.001$) in LDL size (VAT, partial $r = −0.293, P < 0.001$; insulin sensitivity, partial $r = 0.194, P = 0.008$) and 41% of the variance ($P < 0.001$) in HDL size (VAT, partial $r = −0.368, P < 0.001$; insulin sensitivity, partial $r = 0.301, P < 0.001$), whereas VAT and race explained 12% of the variance ($P < 0.001$) in VLDL size (VAT, partial $r = −0.266, P < 0.001$; race, partial $r = 0.199, P = 0.007$).

---

**Figure 1**—HDL (A), LDL (B), and VLDL (C) particle size by quartiles of insulin sensitivity in black male ($n = 43$; □) and female ($n = 57$; □) and white male ($n = 43$; □) and female ($n = 49$; □) children. Differences within each group were compared using one-way ANOVA with post hoc Tukey correction. a, significant difference versus 4; b, significant difference versus 3; c, significant difference versus 2; $P < 0.05$. Range of insulin sensitivity (in micromoles per kilogram per minute per picomole per liter) for black children: quartile 1, 0.45–1.95; quartile 2, 2.16–4.37; quartile 3, 4.40–9.20; quartile 4, 9.21–18.39; for white children: quartile 1, 0.68–1.75; quartile 2, 1.78–3.76; quartile 3, 3.80–9.21; quartile 4, 9.28–25.32.
CONCLUSIONS — The present study advances previous observations of favorable lipoprotein profiles in black compared with white children (3,6,8,14,15) and demonstrates that this finding is partly explained by lower visceral adiposity in blacks. Moreover, we show that in both racial groups, the most insulin-resistant youths have an atherogenic lipoprotein profile of small dense LDL, small HDL, and large VLDL, akin to the atherogenic pattern in adults with coronary artery disease (10,11). In consideration of the fact that atherosclerosis starts in childhood (20), such a lipoprotein pattern may have serious health consequences.

Our findings from the whole group (Table A1, available in an online appendix) are consistent with those of the Bogalusa Heart Study (6,8), in which black children had HDL and LDL particles on average 0.3 and 0.2 nm larger (6,8) and VLDL particles 3.6 nm smaller than those of their white peers (6). These values are similar to the mean differences we observed: 0.2 nm larger, 0.2 nm larger, and 3.8 nm smaller for HDL, LDL, and VLDL in blacks, respectively. Several studies demonstrated favorable lipid profiles in black compared with white children (6,8,14,15) despite insulin resistance (12,13) with similar observations in adults (21). Importantly, for similar overall adiposity, blacks have lower visceral adiposity than whites (3,15,22), an observation repeated in the current study. Controlling for VAT abolished black-white differences in LDL and HDL particle size and concentration in the present study. However, visceral adiposity did not account for the race-related differences in VLDL particle size and concentration, which remained significant after adjustment for VAT. Two potential explanations for this are 1) the lower concentrations of triglycerides in black children because lipoprotein size is related to concentration (6) and 2) increased lipoprotein triglyceride clearance (21), as postheparin lipoprotein lipase activity is reported to be higher and hepatic lipase activity lower in black adults (21,23).

The present study suggests that, besides race, sex-specific analyses are important. Black male children had smaller VLDL particles and black female children had larger HDL particles than their white counterparts. For particle concentration, race differences existed in the larger VLDL in children of both sexes and for male children only in the small dense LDL. One note of caution is that we report nominal significance values for race comparisons on lipoprotein variables in both the group as a whole (Table A1) and for sex-specific analyses (Table 2). The use of multiple t tests may have increased the chance of finding a difference in our data. Nevertheless, significant race differences in some findings remain even if adjusted for multiple comparisons, particularly for VLDL concentrations in both sexes and LDL concentrations in male children.

CONCLUSIONS — The present study advances previous observations of favorable lipoprotein profiles in black compared with white children (3,6,8,14,15) and demonstrates that this finding is partly explained by lower visceral adiposity in blacks. Moreover, we show that in both racial groups, the most insulin-resistant youths have an atherogenic lipoprotein profile of small dense LDL, small HDL, and large VLDL, akin to the atherogenic pattern in adults with coronary artery disease (10,11). In consideration of the fact that atherosclerosis starts in childhood (20), such a lipoprotein pattern may have serious health consequences.

Our findings from the whole group (Table A1, available in an online appendix) are consistent with those of the Bogalusa Heart Study (6,8), in which black children had HDL and LDL particles on average 0.3 and 0.2 nm larger (6,8) and VLDL particles 3.6 nm smaller than those of their white peers (6). These values are similar to the mean differences we observed: 0.2 nm larger, 0.2 nm larger, and 3.8 nm smaller for HDL, LDL, and VLDL in blacks, respectively. Several studies demonstrated favorable lipid profiles in black compared with white children (6,8,14,15) despite insulin resistance (12,13) with similar observations in adults (21). Importantly, for similar overall adiposity, blacks have lower visceral adiposity than whites (3,15,22), an observation repeated in the current study. Controlling for VAT abolished black-white differences in LDL and HDL particle size and concentration in the present study. However, visceral adiposity did not account for the race-related differences in VLDL particle size and concentration, which remained significant after adjustment for VAT. Two potential explanations for this are 1) the lower concentrations of triglycerides in black children because lipoprotein size is related to concentration (6) and 2) increased lipoprotein triglyceride clearance (21), as postheparin lipoprotein lipase activity is reported to be higher and hepatic lipase activity lower in black adults (21,23).

The present study suggests that, besides race, sex-specific analyses are important. Black male children had smaller VLDL particles and black female children had larger HDL particles than their white counterparts. For particle concentration, race differences existed in the larger VLDL in children of both sexes and for male children only in the small dense LDL. One note of caution is that we report nominal significance values for race comparisons on lipoprotein variables in both the group as a whole (Table A1) and for sex-specific analyses (Table 2). The use of multiple t tests may have increased the chance of finding a difference in our data. Nevertheless, significant race differences in some findings remain even if adjusted for multiple comparisons, particularly for VLDL concentrations in both sexes and LDL concentrations in male children.

CONCLUSIONS — The present study advances previous observations of favorable lipoprotein profiles in black compared with white children (3,6,8,14,15) and demonstrates that this finding is partly explained by lower visceral adiposity in blacks. Moreover, we show that in both racial groups, the most insulin-resistant youths have an atherogenic lipoprotein profile of small dense LDL, small HDL, and large VLDL, akin to the atherogenic pattern in adults with coronary artery disease (10,11). In consideration of the fact that atherosclerosis starts in childhood (20), such a lipoprotein pattern may have serious health consequences.

Our findings from the whole group (Table A1, available in an online appendix) are consistent with those of the Bogalusa Heart Study (6,8), in which black children had HDL and LDL particles on average 0.3 and 0.2 nm larger (6,8) and VLDL particles 3.6 nm smaller than those of their white peers (6). These values are similar to the mean differences we observed: 0.2 nm larger, 0.2 nm larger, and 3.8 nm smaller for HDL, LDL, and VLDL in blacks, respectively. Several studies demonstrated favorable lipid profiles in black compared with white children (6,8,14,15) despite insulin resistance (12,13) with similar observations in adults (21). Importantly, for similar overall adiposity, blacks have lower visceral adiposity than whites (3,15,22), an observation repeated in the current study. Controlling for VAT abolished black-white differences in LDL and HDL particle size and concentration in the present study. However, visceral adiposity did not account for the race-related differences in VLDL particle size and concentration, which remained significant after adjustment for VAT. Two potential explanations for this are 1) the lower concentrations of triglycerides in black children because lipoprotein size is related to concentration (6) and 2) increased lipoprotein triglyceride clearance (21), as postheparin lipoprotein lipase activity is reported to be higher and hepatic lipase activity lower in black adults (21,23).

The present study suggests that, besides race, sex-specific analyses are important. Black male children had smaller VLDL particles and black female children had larger HDL particles than their white counterparts. For particle concentration, race differences existed in the larger VLDL in children of both sexes and for male children only in the small dense LDL. One note of caution is that we report nominal significance values for race comparisons on lipoprotein variables in both the group as a whole (Table A1) and for sex-specific analyses (Table 2). The use of multiple t tests may have increased the chance of finding a difference in our data. Nevertheless, significant race differences in some findings remain even if adjusted for multiple comparisons, particularly for VLDL concentrations in both sexes and LDL concentrations in male children.
prevalence of small dense LDL was 10% in children with insulin resistance syndrome (IRS) in contrast with 1% in those without IRS (7). However, IRS was defined based on fasting insulin. Small dense LDL particles are predictive of coronary heart disease (5,24). Conversely, large HDL particles have an inverse relationship with coronary heart disease, whereas small HDL has a positive association (25). Our data demonstrate that in both races, insulin resistance was associated with small HDL particle size, increased small HDL concentration, and low large HDL concentration. Finally, VLDL particles differ in atherogeneity, with some investigations suggesting that large particles are most strongly related to arterial disease and obesity (25). Our data demonstrate that the more insulin-resistant white and black children had higher concentrations of large VLDL particles and bigger VLDL particle size. Last, in multiple regression analyses, both VAT and insulin sensitivity were significant determinants of LDL and HDL particle size, whereas VAT and race were significant for VLDL particle size.

In summary, our data confirm prior observations of favorable lipoprotein profiles in black youth compared with white youth and advance them by showing the role of the lower visceral adiposity in blacks. Sex affects the extent of these differences. Moreover, for both blacks and whites, the most insulin-resistant youth exhibit small dense LDL, small HDL, and large VLDL profiles similar to the atherogeneic.
genic lipoprotein pattern in adults with coronary artery disease (10,11). Such data underscore the need to initiate therapeutic interventions early in childhood to lessen abdominal obesity and insulin resistance and improve the associated adverse alterations in lipoprotein profile, irrespective of race or sex, and reduce the potential risk of atherosclerotic cardiovascular changes.

Acknowledgments—This work was supported by the National Institutes of Health (Grants R01-HD-27503 to S.A.A., K24-HD-01357 to S.A.A., and UL1-RR-024153 CTSA [previously M01-RR-00084]).

No potential conflicts of interest relevant to this article were reported.

Parts of this study were presented in abstract form at the 69th Scientific Sessions of the American Diabetes Association, New Orleans, Louisiana, 5–9 June 2009.

We express our gratitude to the children and their parents who volunteered to participate in this study. We are grateful to the nursing staff of the Pediatric Clinical and Translational Research Center for their outstanding care of the participants and meticulous attention to the research and to Drs. Neslihan Gungor and Fida Bacha and all past endocrine fellows for their assistance with some of the clamp experiments.

References

1. Howard BV, Mayer-Davis EJ, Golf D, Zac-caro DJ, Laws A, Robbins DC, Saad MF, Selby J, Hamman RF, Krauss RM, Haffner SM. Relationships between insulin resistance and lipoproteins in non-diabetic African Americans, Hispanics, and non-Hispanic whites: the Insulin Resistance Atherosclerosis Study. Metabolism 1998; 47:1174–1179

2. Albers JJ, Marcovina SM, Imperatore G, Snively BM, Stafford J, Fujimoto WY, Mayer-Davis EJ, Petitt DB, Pihoker C, Dolan L, Dabelea DM. Prevalence and determinants of elevated apolipoprotein B100 in youth with type 1 and type 2 diabetes. J Clin Endocrinol Metab 2008;93:735–742

3. Lee S, Gungor N, Bacha F, Arslanian S. Insulin resistance: link to the components of the metabolic syndrome and biomarkers of endothelial dysfunction in youth. Diabetes Care 2007;30:2091–2097

4. Pérez-Méndez O, Torres-Tamayo M, Posadas-Romero C, Vidarte Garccés V, Carreón-Torres E, Mendoza-Pérez E, Medina Urrutia A, Huesca-Gómez C, Zamora-González J, Aguilar-Herrera B. Abnormal HDL subclass distribution in overweight children with insulin resistance or type 2 diabetes mellitus. Clin Chim Acta 2007;376:17–22

5. Packard CJ. LDL subfractions and atherogenicity: an hypothesis from the University of Glasgow. Curr Med Res Opin 1996; 13:379–390

6. Freedman DS, Bowman BA, Otvos JD, Srinivasan SR, Berenson GS. Levels and correlates of LDL and VLDL particle sizes among children: the Bogalusa Heart Study. Atherosclerosis 2000;152:441–449

7. Stan S, Levy E, Devlin EE, Hanley JA, LaMarche B, O’Loughlin J, Paradis G, Lambert M. Distribution of LDL particle size in a population-based sample of children and adolescents and relationship with other cardiovascular risk factors. Clin Chem 2005;51:1192–1200

8. Freedman DS, Bowman BA, Srinivasan SR, Berenson GS, Otvos JD. Distribution and correlates of high-density lipoprotein subclasses among children and adolescents. Metabolism 2001;50:370–376

9. Kang H-S, Gutin B, Barbeau P, Litaker MS, Allison J, Le NA. Low-density lipoprotein particle size, central obesity, cardiovascular fitness, and insulin resistance syndrome markers in obese youth. Int J Obes 2002;26:1030–1035

10. Austin MA, King MC, Vranizan KM, Krauss RM. Atherogenic lipoprotein phenotype: a proposed genetic marker for coronary heart disease risk. Circulation 1990;82:495–506

11. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation 2002;106:3143–3421

12. Arslanian SA, Saad R, Levy W, Danadian K, Janosky J. Hyperinsulinemia in African-American children: decreased insulin clearance and increased insulin secretion and its relationship to insulin sensitivity. Diabetes 2002;51:3014–3019

13. Gower BA, Granger WM, Franklin F, Shechwuk RM, Goran MI. Contribution of insulin secretion and clearance to glucose-induced insulin concentration in African-American and Caucasian children. J Clin Endocrinol Metab 2002;87:2218–2224

14. Herd SL, Gower BA, Dashni N, Goran MI. Body fat, fat distribution and serum lipids, lipoproteins and apolipoproteins in African-American and Caucasian-American prepubertal children. Int J Obes 2001;25:198–204

15. Bacha F, Saad R, Gungor N, Janosky J, Arslanian SA. Obesity, regional fat distribution, and syndrome X in obese black versus white adolescents: race differential in diabeticogenic and atherogenic risk factors. J Clin Endocrinol Metab 2003;88:2534–2540

16. Sumner AE, Finley KB, Genovese DJ, Criqui MH, Boston RC. Fasting triglyceride and the triglyceride-HDL cholesterol ratio are not markers of insulin resistance in African Americans. Arch Intern Med 2005;165:1395–1400

17. Lee S, Bacha F, Gungor N, Arslanian SA. Waist circumference is an independent predictor of insulin resistance in black and white youths. J Pediatr 2006;148:188–194

18. Matthews KA, Kuller LH, Wing RR, Meil-lan EN, Plantinga P. Prior to use of estrogen replacement therapy are users healthier than nonusers. Am J Epidemiol 1996;143:971–978

19. Otvos JD, Jeyarajah EJ, Bennett DW, Krauss RM. Development of a proton magnetic resonance spectroscopic method for determining plasma lipoprotein concentrations and subspecies distributions from a single, rapid measurement. Clin Chem 1992;38:1632–1638

20. Strong JP, Malcom GT, McMahan CA, Tracy RE, Newman WP 3rd, Herderick EE, Cornhill JF. Prevalence and extent of atherosclerosis in adolescents and young adults: implications for prevention from the Pathobiological Determinants of Atherosclerosis in Youth Study. JAMA 1999;281:727–733

21. Sumner AE, Vega GL, Genovese DJ, Finley KB, Bergman RN, Boston RC. Normal triglyceride levels despite insulin resistance in African Americans: role of lipoprotein lipase. Metabolism 2005;54:902–909

22. Lee S, Kuk JL, Hannon TS, Arslanian SA. Race and gender differences in the relationships between anthropometrics and abdominal fat in youth. Obesity 2008;16:1066–1071

23. Friday KE, Srinivasan SR, Elkasabany A, Dong C, Wattigney WA, Dalleres E Jr, Bergeron GS. Black-white differences in postprandial triglyceride response and postheparin lipoprotein lipase and hepatic triglyceride lipase among young men. Metabolism 1999;48:749–754

24. Stamper MJ, Krauss RM, Ma J, Blanche PJ, Holl LG, Sacks FM, Hennekens CH. A prospective study of triglyceride level, low-density lipoprotein particle diameter, and risk of myocardial infarction. JAMA 1996;276:882–888

25. Freedman DS, Otvos JD, Jeyarajah EJ, Barbiorjak JJ, Anderson AJ, Walker J. The measurement of lipoprotein subclasses with proton nuclear magnetic resonance spectroscopy: associations with the extent of documented coronary artery disease. Arterioscler Thromb Vasc Biol 1998;18:1046–1053