

Role of dietary factors in ethnic differences in early risk of cardiovascular disease and type 2 diabetes¹⁻³

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ABSTRACT

Background: The disparity in the prevalence of cardiovascular disease and type 2 diabetes between African Americans and whites has been well established, and ethnic differences in several risk factors for these diseases are evident in childhood.

Objective: The current study explored whether dietary factors explain ethnic differences in serum lipids and insulin profiles in children, independent of body composition and social class background.

Design: The sample included 95 African American and white children (mean age: 10.0 y). Macronutrient and food group intakes were derived from three 24-h recalls. Cardiovascular disease and type 2 diabetes risk were determined on the basis of total cholesterol, triacylglycerol, insulin sensitivity (S_i), and acute insulin response (AIR). Data were analyzed by using *t* tests, analysis of covariance, and multiple regression.

Results: African American children had lower triacylglycerol ($P < 0.01$), lower S_i ($P < 0.001$), and higher AIR ($P < 0.001$) than whites. Intake of fruit and vegetables was significantly higher, and dairy intake lower, in African American than in white children after adjustment for social class and total energy intake. Several direct relations were observed between diet and insulin action: carbohydrate and fruit intakes were positively associated with S_i ($P = 0.02$), and vegetable intake was negatively associated with AIR ($P = 0.01$). However, neither macronutrient nor food group intake accounted for the ethnic differences in triacylglycerol and AIR.

Conclusions: The African American children in our sample showed a greater disease risk than did the white children, even after body composition, social class background, and dietary patterns were adjusted for. *Am J Clin Nutr* 2000;71:725–32.

KEY WORDS Ethnicity, children, dietary patterns, food groups, insulin, blood lipids, diabetes, white, Caucasian, African American, black, fruit and vegetable intake

INTRODUCTION

Ethnic differences in the risk of cardiovascular disease (CVD) and type 2 diabetes have consistently been identified, with the most studies comparing the risk between African Americans and whites. The risk of African American adults developing type 2 diabetes is twice that of whites (1). An ethnic disparity in the prevalence of CVD and mortality attributed to CVD has also been identified (2, 3),

with studies in women indicating a 22% higher age-adjusted mortality rate for CVD in African Americans (4). Ethnic differences in risk factors associated with the development of CVD and type 2 diabetes appear to be evident even in childhood.

Certain variations in serum lipids (particularly total cholesterol, HDL cholesterol, and triacylglycerol) are often considered risk factors for CVD (5, 6). On the basis of such indicators, the risk of developing CVD is higher in African American than in white children, as shown in both the third National Health and Nutrition Examination Survey (7) and the Bogalusa Heart Study (8). Data from both studies indicated higher total cholesterol in African American children, although lower triacylglycerol concentrations (a favorable profile) were evident in black adolescents (7).

The development of type 2 diabetes may be preceded by reduced insulin sensitivity, enhanced insulin secretion, and impaired glucose tolerance (9). Such indicators appear markedly different between African American and white children. Insulin sensitivity has been reported to be nearly 50% lower (10), and insulin secretion higher (11, 12), in African American children than in whites, particularly girls (13).

Ethnic differences in the risk of developing CVD and type 2 diabetes are difficult to explain and do not appear to result from differences in body fat or fat distribution (10, 14). As suggested by previous research, dietary factors may play a role in both lipid and insulin profiles, although these patterns may be mediated by body fat content (14, 15). Total fat (and saturated fat) intake has been shown to adversely affect total cholesterol concentrations in children (16, 17), adolescents (18), and young adults (19). Total fat intake is also associated with reduced insulin sensitivity in adults (20–22). Although not examined previously, dietary components other than macronutrients may be relevant to disease risk. Because

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they contain unique combinations of dietary compounds and nutrients (23, 24), individual foods and food groups may play a role in the development of CVD and type 2 diabetes.

The potential role of diet in early disease risk and the identification of ethnic differences in both macronutrient composition (14) and food group intake (25) by previous researchers suggest that the unfavorable lipid and insulin profiles observed in African American children may be because of differences in dietary patterns. The current study explores the role of dietary factors (including both macronutrient composition and food group intake) in ethnic differences in early disease risk among African American and white children. We hypothesize that dietary patterns account for ethnic differences in lipid and insulin measures, independent of body composition and social class background.

SUBJECTS AND METHODS

Sample

The data used in this study were derived from an ongoing, longitudinal study of childhood obesity conducted in Birmingham, AL. The children who participated in the study were recruited through advertisements, through flyers, and by word of mouth. Most children began participating at ≈ 7 y of age and were in year 2 or 3 of their participation during our study; they were aged from 6.5 to 13.0 y (mean age: 10.0 y). The participants were screened for any serious medical condition since birth (such as type 1 diabetes, Down syndrome, Cushing syndrome, and hypothyroidism). The protocol included a yearly overnight visit to the General Clinical Research Center (GCRC). Data were collected on anthropometry, sociodemographic background, dietary intake, and physical activity. In addition, after an overnight fast, data on resting metabolic rate were obtained, a blood sample collected, and an insulin sensitivity test conducted. Two weeks later, the children underwent further testing at the Department of Nutrition Science, at which time data on dietary intake, aerobic fitness, and body composition were gathered (with the children arriving in the fasted state). The study was approved by the University of Alabama at Birmingham institutional review board for human use, and informed consent was obtained from all subjects and their parents before testing.

Approximately 120 healthy white and African American children, representing a wide range of body sizes, participated in the study. However, because lipid and insulin profiles are strongly associated with stage of pubertal development, the sample used in the current analyses included children assessed at pubertal development stage 1 or 2 only (determined by using the criteria of Tanner and assessed by physical examination by a pediatrician; 26, 27). This resulted in a sample size of 95 (54 whites and 41 African Americans). The analyses in the current study were cross-sectional, with all measurements derived from data collected during the 1997–1998 school year.

Dietary variables

All dietary intake variables were based on three 24-h dietary recalls collected during a 2-wk period, 2 of which were obtained on weekdays and 1 that was obtained on a weekend. The first 24-h recall was conducted at the child's GCRC visit, the second over the telephone, and the third at the Department of Nutrition Sciences. The dietary recalls were conducted by using the "multiple pass" method, which has been validated against the doubly labeled water method

and described previously in detail by Johnson et al (28). This method requires that a trained interviewer gather food consumption information from the subjects in 3 passes: a quick list, a detailed description, and a review. All dietary recalls were conducted in the presence of one of the child's parents (usually the mother). The validity of the 24-h recall method in assessing energy intake in our sample was recently established in a subsample of the children ($n = 17$ whites and 13 African Americans) who were dosed with doubly labeled water (29). Comparisons of the mean energy intake and energy expenditure (assessed by doubly labeled water) showed nearly identical group-level estimates, with the difference in the 2 measures being only 0.04 MJ/d. Individual-level validity appeared weaker, with the correlation between energy intake measured by the 24-h recalls and energy expenditure being only 0.32 ($P = 0.08$) (29).

The dietary recalls were coded and entered into the Food Intake Analysis System (FIAS; Health Science Center, University of Texas, Houston) by a trained interviewer. The FIAS program is based on the US Department of Agriculture Nationwide Food Consumption Survey, Continuing Survey of Food Intakes by Individuals (CSFII), and allows for the addition of new foods, the modification of recipes, and the selection of default portion sizes representing commonly consumed portions of individual foods. The dietary variables generated by FIAS that were used in the analyses were total energy (MJ/d), protein (g/d), carbohydrate (g/d), and total fat intake (g/d). These measures reflected the subject's average intake over the 3 recalls.

In addition to macronutrient intake, the subject's average intake of food group servings was determined by using the USDA Pyramid Serving Database (25, 30, 31). The methodology used to generate this database operationalized food groups and serving sizes specified in the USDA food guide pyramid and consisted of 3 basic steps: 1) translating gram weight information for foods included in the CSFII database into serving sizes compatible with pyramid guidelines, 2) disaggregating combination foods into individual ingredients, and 3) assigning all foods or ingredients to a food group. These procedures result in an estimate of the average daily servings from grain, vegetable, fruit, dairy, and meat groups, as well as the average energy consumed daily as added sugar and discretionary fat (which constitute the pyramid tip). The separation of added sugar and discretionary fat (defined as fat and sugar either added in preparation or in excess of that which would have been consumed if only the lowest fat or sugar food choices were chosen from a particular food group) was a major advantage of this methodology because the effects of consuming the "healthiest" foods from each food group could be determined, as well as the consequences of components of the pyramid tip. Thus, by using this methodology, all foods and beverages consumed by the subjects over the 3-d period were categorized, and each subject's average daily servings of the 5 major food groups and intake of added sugar and discretionary fat were derived. Because intakes of the dairy, fruit, and vegetable groups were not normally distributed among the sample, these 3 variables were log transformed for use in the multivariate analyses.

Lipid profile

To assess total cholesterol and triacylglycerol, blood samples were collected at the GCRC after the overnight fast (5 mL venous blood). Serum was separated and stored in cryotubes at -85°C . Lipids were analyzed by using Johnson and Johnson Ektachem DT slides and reagents and a Johnson and Johnson Ektachem DT60 II Analyzer (Rochester, NY). The Ektachem analyzer was



calibrated by Johnson and Johnson Clinical Diagnostics against the National Cholesterol Education Program guidelines. The values for total cholesterol (mmol/L) and triacylglycerol (mmol/L) were log transformed for the statistical analyses because of uneven distributions.

Insulin measures

Insulin sensitivity and acute insulin response were measured by tolbutamide-modified, frequently sampled intravenous-glucose-tolerance tests (32–34). The intravenous-glucose-tolerance test is advantageous because it is better tolerated in children than other, more invasive methods, and is relatively easy to perform, requiring the same amount of time as an oral test yet providing more specific information regarding insulin secretion and function. Because it takes into account changing concentrations of both glucose and insulin over time, the intravenous-glucose-tolerance test provides an index of insulin sensitivity that correlates well with that determined by the euglycemic clamp (35), which is more technically difficult to perform and less suitable for use with children (36). The tests were initialized at 0600 on the morning after the child's GCRC admission. A topical anesthetic (Emla cream; Astra Pharmaceutical Products, Inc, Westborough, MA) was applied to the antecubital space of both of the child's arms. At ≈0700, flexible intravenous catheters were introduced into a forearm vein, and 3 blood samples (2.0 mL) were taken for determination of basal glucose and insulin. At time 0, glucose (25% dextrose; 11.4 g/m²) was administered intravenously. Blood samples (2.0 mL) were then collected at the following times (min) relative to glucose injection: 2, 3, 4, 5, 6, 8, 10, 14, 19, 22, 25, 30, 40, 50, 70, 100, 140, and 180. Tolbutamide (125 mg/m²; Upjohn, Kalamazoo, MI) was injected intravenously at 20 min.

Sera were analyzed for glucose and insulin, and values were entered into the MINMOD computer program (version 3.0; 33) for determination of insulin sensitivity and acute insulin response (the insulin area under the curve above basal for the first 10 min). Insulin was assayed in duplicate 200:1 aliquots with Diagnostic Products Corporation (Los Angeles) Coat-A-Count kits. According to the supplier, cross-reactivity of this assay with proinsulin is ≈40% at midcurve; C-peptide is not detected. In our laboratory, this assay has a sensitivity of 11.4 pmol/L (1.9 IU/mL), a mean intraassay covariance of 5%, and a mean interassay covariance of 6%. Commercial quality-control sera of low, medium, and high insulin concentration (Lyphochek; Bio-Rad, Anaheim, CA) are included in every assay to monitor variation over time. The insulin sensitivity and acute insulin response variables were highly skewed and were therefore log transformed for use in statistical analyses.

Control variables

To adjust for physiologic covariates of serum lipid and insulin profiles, relevant control variables were included in the analysis for each outcome variable. For triacylglycerol, intraabdominal adipose tissue (IAAT) was included as a control variable. We showed previously that IAAT is a significant predictor of triacylglycerol concentration, independent of total fat mass (10). IAAT was measured by computed-tomography scanning (HiLight/AdvantageScanner; General Electric, Milwaukee) of a single slice of the abdomen (for a detailed description, *see* reference 37). Because, in our sample, total body fat was shown to be related to insulin sensitivity independent of fat distribution (10), total fat mass was selected as the most relevant control variable

for insulin sensitivity. Total-body fat mass was measured by dual-energy X-ray absorptiometry (DXA) with a DPX-L densitometer and pediatric software (previously validated in reference 38; Lunar, Madison, WI). Because insulin sensitivity is an important determinant of β -cell function (39), we included this variable as a covariate for acute insulin response. IAAT and fat mass were log transformed to correct nonnormal distributions.

In addition to relevant measures of body composition, social class background was included as a control variable to determine the influence of social class on risk of CVD and type 2 diabetes. The Hollingshead 4-factor index of social class (40), which combines the educational attainment and occupational prestige for the number of working parents in the child's family, was included as a control variable for all outcomes. The derived social class score ranges from 8 to 66, with higher values indicating a higher social class background.

Statistical analysis

The data were analyzed by using SPSS/PC+ software (version 8.0; SPSS Inc, Chicago). First, to determine ethnic differences in relevant background characteristics and lipid profile and insulin sensitivity measures, *t* tests were conducted for interval or ratio variables and chi-square tests were performed for categorical variables (Tanner stage). Second, ethnic differences in dietary patterns (including both macronutrient and food group intakes) were determined by using analysis of variance. Then, to avoid the confounding influence of total energy intake and social class background on estimates of macronutrient intake and food group servings, mean intakes were adjusted for these variables by using analysis of covariance. Finally, the influence of dietary patterns on ethnic differences in lipid and insulin measures was determined by using multiple regression analyses. For each outcome, the analyses were conducted in several stages. First, the bivariate relation between ethnicity and the outcome was determined. Ethnicity was dummy coded (0 = whites, 1 = African Americans). Second, relevant control variables were entered into the equation in successive steps: for total cholesterol, social class was the only control variable; for triacylglycerol, IAAT and social class were included; for insulin sensitivity, fat mass and social class were included as control variables; and for acute insulin response, insulin sensitivity and social class were included in the model. Third, the macronutrient variables were entered into the equations. Finally, food groups (and added fat and discretionary sugar) were included in the model. This sequential procedure enabled the magnitude of ethnicity as a predictor of disease outcomes to be compared at each stage of the analyses. Our hypothesis was that the addition of dietary variables would significantly reduce the influence of ethnicity on each risk factor. The analytic design also allowed the change in explained variation after each equation to be observed, and individual associations between social class, macronutrients, and food groups and disease risk indicators to be identified.

RESULTS

Bivariate analyses

Background characteristics of the sample ($n = 95$) are presented in **Table 1**. No ethnic differences in age, stage of pubertal development, or body composition were evident. However, social class background differed significantly by ethnicity, with



TABLE 1
Background characteristics of the sample of children, by ethnicity

	Whites (n = 54)	African Americans (n = 41)
Age (y)	9.8 ± 1.5 ¹	9.3 ± 1.5
Height (cm)	139.5 ± 10.7	139.9 ± 10.6
Weight (kg)	39.2 ± 12.8	42.7 ± 15.4
BMI (kg/m ²)	19.8 ± 4.2	21.3 ± 5.5
Fat mass (kg)	11.3 ± 8.2	12.9 ± 9.6
Fat-free mass (kg)	25.4 ± 5.4	26.9 ± 5.9
Percentage body fat (%)	27.0 ± 10.8	27.8 ± 11.6
Social class ²	53.1 ± 7.9	32.6 ± 13.4 ³
Tanner stage		
1	38 (70) ⁴	22 (63)
2	16 (30)	19 (46)

¹ $\bar{x} \pm SD$.

²Hollingshead index; score ranges from 8 to 66 (lowest to highest social class background) (40).

³Significantly different from whites, $P < 0.001$.

⁴n; percentage in parentheses.

white children having a significantly higher social class background than the African American children in the sample. Although not shown in the table, no sex differences in these characteristics were significant; therefore, subsequent analyses were conducted with boys and girls combined.

Ethnic differences in the 4 outcome variables examined in this study are shown in **Table 2**. Total cholesterol tended to be higher and triacylglycerol concentrations were significantly lower for African American than for white children. African American children had a significantly lower insulin sensitivity (by $\approx 40\%$) and a 2-fold greater acute insulin response than the white children.

Dietary information, including both macronutrient intake and food group consumption, is presented in **Table 3**. The means are presented both unadjusted and adjusted for total energy intake and social class background. Several ethnic differences were evident when the unadjusted values were examined. African American children consumed fewer grams carbohydrate per day, less energy as added sugar per day, fewer servings of dairy products per day, and more servings of fruit and meat than did white children. However, when total energy intake and social class were adjusted for, few ethnic differences remained. The adjusted means only differed significantly for dairy (with African American children consuming 40% less dairy than white children), fruit (with African American children consuming twice as many daily servings of fruit than whites), and vegetables (with African American children consuming 25% more vegetable servings than whites).

Multivariate analyses

The final series of analyses examined the role of macronutrients and food group intake in ethnic differences in early disease risk indicators. The results of the regression analyses for total cholesterol are presented in **Table 4**. The trend toward higher cholesterol for African American children decreased notably when social class was included in the model. Although there was a trend toward a positive relation between protein intake and total cholesterol, no significant relations between macronutrients and cholesterol were evident. The model containing macronutrient intake resulted in the smallest ethnic difference in total cholesterol, with no further reductions observed when food group intake measures were included.

The significantly lower triacylglycerol concentration of the African American children was evident in all multivariate models, as indicated in **Table 5**. The favorable triacylglycerol profile of African Americans was slightly reduced when visceral fat, social class, and macronutrient intake were controlled for, but increased slightly when food group consumption was entered into the model. The ethnic difference was smallest when only visceral fat was included (slope for ethnicity in model 2 = -0.108) and largest when food group intake was accounted for (slope for ethnicity in model 5 = -0.134). In fact, when food group intake was accounted for, the bivariate ethnic difference in triacylglycerol increased by 11%. None of the dietary variables were independently related to triacylglycerol (although the relation between triacylglycerol and grain intake approached significance), and the ethnic difference remained significant in all models.

The results of the regression analyses for insulin sensitivity are shown in **Table 6**. The ethnic difference in insulin sensitivity was highly significant in all 5 models. Inclusion of total fat mass in the model slightly reduced the magnitude of the ethnic effect. Social class was not significantly related to insulin sensitivity in any model and its inclusion did not reduce the ethnic difference in insulin sensitivity (model 3). Although dietary factors did not account for ethnic differences in insulin sensitivity, several individual relations were evident. Carbohydrate (relative to protein and dietary fat) and fruit intake were positively associated with insulin sensitivity. The models containing macronutrient and food group intake explained 61% and 62% of the variation in insulin sensitivity, respectively. The model resulting in the smallest ethnic difference in insulin sensitivity contained only fat mass (model 2), and the model resulting in the largest ethnic difference in insulin sensitivity included food group intake. This model increased the bivariate ethnic difference in insulin sensitivity by 18%.

Acute insulin response was also significantly associated with ethnicity in all models (**Table 7**). However, the magnitude of the ethnic effect decreased by nearly 30% when insulin sensitivity was added (model 2), 52% when both social class and insulin sensitivity were added (model 3), 48% when the macronutrients were added (model 4), and 40% when food group intake was added (model 5). The model including insulin sensitivity and social class resulted in the smallest ethnic difference in insulin secretion ($\beta = 0.193$). Although not significant, a trend toward lower insulin secretion among children from higher social class backgrounds was evident ($P = 0.06$). Neither macronutrient nor food group intake contributed to a further reduction in the effect of ethnicity on the acute insulin response. None of the macronutrients were independently associated with the acute insulin response (model 4). Higher intake of vegetables was significantly (negatively) associated

TABLE 2
Ethnic differences in blood lipids and insulin sensitivity measures¹

	Whites (n = 54)	African Americans (n = 41)
Total cholesterol (mmol/L)	3.82 ± 1.14	4.07 ± 1.23 ²
Triacylglycerol (mmol/L)	0.71 ± 0.02	0.54 ± 0.02 ³
Insulin sensitivity ($\times 10^{-4} \text{ L} \cdot \text{min}^{-1} \cdot \text{pmol}^{-1}$)	34.05 ± 1.97	20.40 ± 1.98 ⁴
Acute insulin response (pmol/L)	3501.06 ± 1.72	8887.92 ± 1.95 ⁴

¹ $\bar{x} \pm SD$.

²Nearly significantly different from whites, $P = 0.07$.

^{3,4}Significantly different from whites: ³ $P < 0.01$, ⁴ $P < 0.001$.

TABLE 3
Ethnic differences in dietary patterns¹

	Whites (n = 54)	African Americans (n = 41)
Macronutrient composition		
Energy (MJ/d)	7.9 ± 2.0	7.3 ± 1.7
Protein (g/d)		
Unadjusted	64.6 ± 2.7	62.6 ± 2.8
Adjusted	64.1 ± 2.2	63.3 ± 2.6
Carbohydrate (g/d)		
Unadjusted	254.7 ± 8.9	215.9 ± 9.1 ²
Adjusted	240.9 ± 4.7	233.8 ± 5.5
Total dietary fat (g/d)		
Unadjusted	70.7 ± 3.2	72.4 ± 3.0
Adjusted	69.8 ± 1.5	73.5 ± 1.7
Food group composition		
Added sugar (MJ/d)		
Unadjusted	1.6 ± 0.1	1.3 ± 0.1 ³
Adjusted	1.5 ± 0.1	1.5 ± 0.1
Discretionary fat (MJ/d)		
Unadjusted	2.1 ± 0.1	2.0 ± 0.1
Adjusted	2.0 ± 0.1	2.1 ± 0.1
Dairy group (servings/d)		
Unadjusted	1.4 ± 0.1	0.8 ± 0.1 ⁴
Adjusted	1.5 ± 0.1	0.9 ± 0.1 ⁴
Fruit group (servings/d)		
Unadjusted	0.7 ± 0.1	1.0 ± 0.1 ⁵
Adjusted	0.6 ± 0.1	1.2 ± 0.1 ²
Grain group (servings/d)		
Unadjusted	7.0 ± 0.3	6.3 ± 0.4
Adjusted	6.8 ± 0.3	6.6 ± 0.4
Meat group (servings/d)		
Unadjusted	3.6 ± 0.3	4.7 ± 0.3 ³
Adjusted	3.7 ± 0.3	4.5 ± 0.3
Vegetable group (servings/d)		
Unadjusted	2.3 ± 0.2	2.6 ± 0.2
Adjusted	2.1 ± 0.2	2.8 ± 0.2 ⁵

¹ $\bar{x} \pm SE$. Adjusted values are adjusted for total energy intake and social class.

²⁻⁵Significantly different from whites: ² $P < 0.01$, ³ $P = 0.01$, ⁴ $P < 0.001$, ⁵ $P = 0.03$.

with insulin secretion, and the effects of several other food groups on insulin secretion approached statistical significance. The final model explained 72% of the variation in insulin secretion.

DISCUSSION

The results of this study suggest that African American children do not have a more unfavorable serum lipid profile than do whites. Although total cholesterol tended to be higher in African American children, this trend was not evident in the multivariate analyses. In addition, triacylglycerol concentrations were significantly lower (a favorable profile) for African Americans in all models. Similar to previous research (10, 11, 13), the current study identified lower insulin sensitivity in African American children. Insulin sensitivity was 40% lower and insulin secretion was 2-fold higher in African American than in white children. These differences were highly significant and were evident in all multivariate models. The pattern of activity observed in African American children is consistent with previous findings (10). Although this observation is paradoxical, the explanation may be the lower amount of visceral fat observed

in African American children and adults (41, 42). The ethnic difference in insulin sensitivity is independent of body composition and fat distribution, whereas lower visceral fat in African Americans confers lower triacylglycerol concentrations (10).

Although it was hypothesized that diet would account for ethnic differences in serum lipid and insulin profiles, macronutrient and food group intakes did not reduce the effect of ethnicity. For the serum lipid outcomes, dietary variables did not decrease the significance of ethnicity as a predictor. Indeed, inclusion of food group intake increased the ethnic difference in triacylglycerol. The relatively small influence of diet on serum lipids is not inconsistent with some previous studies. Several researchers (5) have described the relation between macronutrients and lipid concentrations as being "weak and inconsistent," with numerous studies finding no association, particularly when body fat is controlled for (14, 43). Previous studies from our laboratory suggested that body fat may be more influential than dietary fat in explaining lipid concentrations (particularly triacylglycerol) in prepubertal children (14). However, a recent meta-analysis of the National Cholesterol Education Program's dietary intervention programs showed beneficial effects of diets low in fat (and saturated fatty acids) on plasma lipid profiles (44). Of relevance to the current study, total cholesterol decreased by 10–13%, depending on the intensity of the dietary intervention, and triacylglycerol concentrations decreased by 8%—all statistically significant decreases (44). Lipid profile was not influenced by either macronutrient or food group intake in the current study. The latter factor has not been considered in any previous studies that we are aware of, although fruit and vegetable intake has been shown to produce protective effects against CVD in adults (45–47). Future studies exploring the relation between components of the food guide

TABLE 4
Regression of total cholesterol (mmol/L – log) on ethnicity and dietary patterns¹

	Parameter estimate	P ²	R ² for model
Model 1			
Ethnicity (African American = 1)	0.028	0.07	0.04
Model 2			
Ethnicity (African American = 1)	0.006	0.76	
Social class	–0.001	0.14	0.06
Model 3			
Ethnicity (African American = 1)	0.004	0.83	
Social class	–0.001	0.22	
Protein (g/d)	0.001	0.06	
Carbohydrate (g/d)	–0.000	0.10	
Total dietary fat (g/d)	–0.000	0.37	0.14 ³
Model 4			
Ethnicity (African American = 1)	0.013	0.58	
Social class	–0.000	0.38	
Added sugar (MJ/d)	–0.018	0.16	
Discretionary fat (MJ/d)	–0.010	0.58	
Dairy (servings/d – log)	0.015	0.52	
Fruit (servings/d – log)	–0.019	0.22	
Grain (servings/d)	–0.002	0.61	
Meat (servings/d)	0.004	0.37	
Vegetable (servings/d – log)	–0.020	0.60	0.17

¹n = 52 whites, 41 African Americans.

²Significance of the parameter estimate in the multiple regression model.

³P for the model < 0.05.

TABLE 5
Regression of triacylglycerol (mmol/L – log) on ethnicity and dietary patterns¹

	Parameter estimate	P ²	R ² for model
Model 1			
Ethnicity (African American = 1)	-0.119	0.00	0.10 ³
Model 2			
Ethnicity (African American = 1)	-0.108	0.01	
Visceral fat (cm ² – log)	0.186	0.02	0.15 ⁴
Model 3			
Ethnicity (African American = 1)	-0.111	0.05	
Visceral fat (cm ² – log)	0.186	0.02	
Social class	-0.000	0.94	0.15 ⁵
Model 4			
Ethnicity (African American = 1)	-0.117	0.04	
Visceral fat (cm ² – log)	0.203	0.02	
Social class	-0.000	0.86	
Protein (g/d)	-0.000	0.79	
Carbohydrate (g/d)	-0.000	0.92	
Total dietary fat (g/d)	-0.001	0.64	0.16 ⁵
Model 5			
Ethnicity (African American = 1)	-0.134	0.04	
Visceral fat (cm ² – log)	0.199	0.02	
Social class	-0.000	0.95	
Added sugar (MJ/d)	0.026	0.45	
Discretionary fat (MJ/d)	0.011	0.83	
Dairy (servings/d – log)	-0.080	0.20	
Fruit (servings/d – log)	-0.008	0.85	
Grain (servings/d)	-0.021	0.07	
Meat (servings/d)	0.005	0.68	
Vegetable (servings/d – log)	-0.048	0.64	0.21 ⁵

¹n = 52 whites, 41 African Americans.

²Significance of the parameter estimate in the multiple regression model.

³P for the model < 0.01.

⁴P for the model < 0.001.

⁵P for the model < 0.05.

pyramid and serum lipids are needed to determine whether compliance with dietary guidelines results in cardiovascular health.

Dietary patterns appeared to have a greater influence on insulin action than on lipid concentration. Although macronutrient intake did not reduce the ethnic difference in insulin sensitivity, and food group intake actually increased the effect of ethnicity, several dietary variables were directly associated with this outcome. Carbohydrate consumption (relative to protein and dietary fat intake) and fruit intake (relative to other pyramid components) were positively associated with insulin sensitivity, and vegetable intake independently predicted insulin secretion. The positive effect of carbohydrate intake on insulin sensitivity observed in our cross-sectional analyses appears to be supported by previous intervention studies. Several controlled dietary studies involving high-carbohydrate, low-fat diets have shown improvements in insulin sensitivity (48, 49), although the beneficial effects could have been attributed to either dietary component. The positive associations we observed between fruit and vegetable intake and insulin action are difficult to interpret because no other studies that we are aware of have studied the relation between consumption of particular food groups and insulin measures. It is clear, however, that fruit and vegetables contain complex organic compounds that may influence health outcomes and require further investigation (50).

Although the individual associations between dietary variables and insulin action observed in this study are of interest and contribute to our understanding of the relation between diet and health, the major conclusion to be derived from this study is that dietary patterns did not explain the ethnic differences in early disease risk indicators. There are several potential explanations for this finding. First, after adjustment for relevant confounders, the dietary patterns of the African American and white children were extremely similar and the differences that were evident were more favorable for African American children. For example, African American children consumed more daily servings of fruit and vegetables than did whites. The greater consumption of vegetables (and lower dairy intake) by the African American children in our sample was also reported in large, nationally representative samples of children (25). The fact that this intake pattern is perhaps more desirable regarding early disease risk indicators may preclude the reduction of ethnic differences in such outcomes. The more favorable dietary pattern among African Americans suggests that once dietary factors are controlled for, the ethnic difference in early disease risk would actually increase. Indeed, this proposition is supported by the increase in ethnic differences in triacylglycerol and insulin sensitivity observed in the current analyses once food group intake was controlled for. However, it is important to point out that the diets consumed by most of the children in our sample (both African American and white) were

TABLE 6
Regression of insulin sensitivity [$\times 10^{-4}$ L · min⁻¹ · pmol⁻¹) – log] on ethnicity and dietary patterns¹

	Parameter estimate	P ²	R ² for model
Model 1			
Ethnicity (African American = 1)	-0.223	0.00	0.12 ³
Model 2			
Ethnicity (African American = 1)	-0.198	0.00	
Total fat mass (kg – log)	-0.691	0.00	0.57 ³
Model 3			
Ethnicity (African American = 1)	-0.248	0.00	
Total fat mass (kg – log)	-0.693	0.00	
Social class	-0.002	0.27	0.58 ³
Model 4			
Ethnicity (African American = 1)	-0.223	0.00	
Total fat mass (kg – log)	-0.710	0.00	
Social class	-0.004	0.10	
Protein (g/d)	0.001	0.45	
Carbohydrate (g/d)	0.001	0.02	
Total dietary fat (g/d)	-0.003	0.12	0.61 ³
Model 5			
Ethnicity (African American = 1)	-0.270	0.00	
Total fat mass (kg – log)	-0.693	0.00	
Social class	-0.003	0.16	
Added sugar (MJ/d)	0.020	0.62	
Discretionary fat (MJ/d)	-0.056	0.32	
Dairy (servings/d – log)	0.067	0.42	
Fruit (servings/day – log)	0.111	0.02	
Grain (servings/d)	0.011	0.37	
Meat (servings/d)	0.005	0.74	
Vegetable (servings/d – log)	0.023	0.85	0.62 ³

¹n = 50 whites, 36 African Americans.

²Significance of the parameter estimate in the multiple regression model.

³P for the model < 0.001.



TABLE 7

Regression of acute insulin response (pmol/L – log) on ethnicity and dietary patterns¹


	Parameter estimate	P ²	R ² for model	
Model 1				
Ethnicity (African American = 1)	0.405	0.00	0.38 ³	
Model 2				
Ethnicity (African American = 1)	0.278	0.00	0.64 ³	
Insulin sensitivity [(×10 ⁻⁴ L·min ⁻¹ ·pmol ⁻¹) – log]	-0.570	0.00		
Model 3				
Ethnicity (African American = 1)	0.193	0.00	0.65 ³	
Insulin sensitivity [(×10 ⁻⁴ L·min ⁻¹ ·pmol ⁻¹) – log]	-0.580	0.00		
Social class	-0.004	0.06		
Model 4				
Ethnicity (African American = 1)	0.213	0.00	0.67 ³	
Insulin sensitivity [(×10 ⁻⁴ L·min ⁻¹ ·pmol ⁻¹) – log]	-0.587	0.00		
Social class	-0.004	0.06		
Protein (g/d)	0.000	0.76		
Carbohydrate (g/d)	0.000	0.28		
Total dietary fat (g/d)	0.000	0.81		
Model 5				
Ethnicity (African American = 1)	0.241	0.00		0.72 ³
Insulin sensitivity [(×10 ⁻⁴ L·min ⁻¹ ·pmol ⁻¹) – log]	-0.576	0.00		
Social class	-0.004	0.09		
Added sugar (MJ/d)	-0.054	0.12		
Discretionary fat (MJ/d)	0.082	0.10		
Dairy (servings/d – log)	0.115	0.06		
Fruit (servings/d – log)	0.040	0.36		
Grain (servings/d)	0.021	0.07		
Meat (servings/d)	-0.005	0.70		
Vegetable (servings/d – log)	-0.275	0.01		

¹n = 50 whites, 36 African Americans.²Significance of the parameter estimate in the multiple regression model.³P for the model < 0.001.

generally of poor quality according to national recommendations. A comparison of our sample's food intakes with pyramid guidelines showed that consumption of all major food groups was well below guidelines, and nearly half of the children's energy intake (46%) was derived from the pyramid tip.

Other caveats of the current study are important to consider. Our analyses were cross-sectional, preventing causal inferences of the relation between diet and risk of CVD or type 2 diabetes. Longitudinal and intervention studies are needed to determine the magnitude of ethnic differences in insulin action throughout pubertal development and adulthood, and the ability of dietary patterns to influence serum lipid and insulin profiles. The current study is also limited by the use of self-reports of dietary intake, which were dependent on memory skills. However, we took several steps to improve the accuracy of the recalls (ie, conducting recalls in the presence of a parent and doing multiple passes), and our group-level estimates of dietary intake had extremely high validity. Finally, the current study relied on a relatively small sample size (n = 95) of children in the earliest stages of puberty. Although we deliberately included only pre- or early pubertal children to avoid the confounding influence of sex hormones on serum lipid and insulin profiles, it is possible that analyses of

larger samples with a wider age range might yield more generalizable results. The weaknesses caused by the small sample size in the current study are likely to have been offset by the use of strong measures of body composition (DXA and computed tomography) and insulin action (frequently sampled, intravenous-glucose-tolerance test).

In summary, our study showed an ethnic difference in disease risk that was not substantially reduced by dietary patterns. African American children still had significantly lower insulin sensitivity and a greater acute insulin response than did whites after adjustment for social class, body composition, macronutrient intake, and food group consumption. Although dietary factors did not appear to account for a major portion of the ethnic difference in disease risk, the results of the current study contribute to existing knowledge regarding the role of diet in metabolic disorders. Future research exploring the health effects of food group consumption and other indicators of dietary quality is needed. 

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