Characteristics of Young Offspring of Type 2 Diabetic Parents in a Biracial (black-white) Community-Based Sample: The Bogalusa Heart Study

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The impact of race (black-white) and family history of type 2 diabetes mellitus on metabolic characteristics in early life was examined in a community-based sample from Bogalusa, LA. Study subjects included offspring of type 2 diabetics (n = 53, 47% black) and nondiabetics (n = 52, 40% black), with the mean age of each group ranging from 14.2 to 15.6 years. Offspring were given a 1-hour oral glucose tolerance test. Measures of body fatness such as body weight, body-mass index (BMI; weight/height^2), and triceps and subscapular thicknesses were significantly higher only in white offspring of diabetics versus nondiabetics; measures of abdominal fat (waist circumference and waist-to-hip ratio) were significantly higher among offspring of diabetics of both races. Among the measures of glucose homeostasis, basal glucose, insulin, insulin–to–C-peptide ratio (a measure of hepatic insulin extraction), insulin resistance index (derived from basal glucose and insulin levels), and glucose response after glucose challenge were higher in the offspring of diabetics of both races. The differences in insulin–to–C-peptide ratio and glucose response remained significant after adjusting for BMI; further, these two variables were independently associated with parental diabetes in both races. Waist-to-hip ratio, glucose response, C-peptide response (a measure of insulin secretion) were lower, and basal insulin–to–C-peptide ratio and postglucose suppression of free fatty acids greater in blacks versus whites, regardless of status of parental diabetes. Black-white differences in postglucose suppression of free fatty acids disappeared after adjusting for BMI. Thus, blacks and whites with parental type 2 diabetes show multiple abnormalities in parameters governing glucose homeostasis early in life, and some of these traits differ between the races, regardless of status of parental diabetes.

TYPE 2 DIABETES MELLITUS is a major contributor to adult coronary heart disease morbidity and mortality and often accompanies hypertensive cardiovascular renal disease.[1] The prevalence of type 2 diabetes varies depending on race and ethnicity.[2] In the United States, blacks have a twofold to threefold greater prevalence of the disease than whites.[3] Although type 2 diabetes has a strong genetic and familial component,[4-6] the molecular genetics of this complex syndrome are just being elucidated.[7] Further, identification of underlying defects that are antecedent of its clinical expression remains elusive. Studies of asymptomatic first-degree relatives of diabetics[8-14] and ethnically distinct populations with high prevalence of the disease[15-17] are being used to identify underlying markers of type 2 diabetes. Studies in this regard have focused mainly on adults, because type 2 diabetes is essentially an adult-onset disorder. The few studies that have addressed this issue in children have involved genetically distinct, extraordinarily high-risk ethnic groups,[18,19] the results of which may not be applicable to other racial and ethnic groups. Recent studies, including our own, clearly demonstrated black-white differences in glucose and insulin metabolism in young nondiabetic subjects.[20-22] However, whether the antecedent factors governing the evolution of the disease process early in life are different between the two races remain uncertain.

The Bogalusa Heart Study is a long-term, community-based investigation of cardiovascular disease risk factors in a biracial (black-white) population of children and young adults.[23,24] Information on parental history of diabetes among the study population provided the opportunity to study metabolic abnormalities in the young offspring. Our initial observations in young white offspring of diabetics suggest that subtle abnormalities related to type 2 diabetes are already apparent in childhood.[25] The present study was initiated to compare the characteristics of young white and black offspring of type 2 diabetic parents.

MATERIALS AND METHODS

Parents with a history of diabetes were identified by a questionnaire administered during a cross-sectional survey of school children from the community of Bogalusa, LA. Fifty-three offspring (53% white, 48% female) from 30 families with parental history of type 2 diabetes (onset after the age of 30) were available for the study as cases. The nature and onset of parental diabetes was verified by a physician through medical records and interviews to exclude possible type 1 diabetics. None of the case offspring had two diabetic parents; in 57% of black cases and 60% of white cases, it was the mother who was diabetic. Matched by the age of the parents, 52 offspring (60% white, 50% female) from 35 families with no history of diabetes in parents, grandparents, uncles, or aunts were selected as controls. Table 1 provides age, race, and sex distribution of the subjects under study. Although the age range of case and control offspring ranged from 7 to 25 years, with an average of 15.0 years, 81% of the offspring were under the age of 18.

Measurements of weight, height, thickness of triceps and subscapular skinfolds, waist and hip circumferences, and replicate measurements of blood pressure were obtained adhering to rigid protocols previously used in the Bogalusa Heart Study.[24]

After an overnight fast (12 hours), a heparin lock with a butterfly needle was inserted into subjects' antecubital vein to perform an abbreviated oral glucose tolerance test. Venous blood was drawn immediately before and at 15, 30, and 60 minutes after oral administration of 1.75 g glucose/kg body weight. A maximum dose of 75 g (225 mL) was used.

Plasma glucose was determined by a glucose oxidase method using Abbott quick start reagent (Abbott Laboratories, North Chicago, IL); plasma immunoreactive insulin (which cross-reacts 41% with proinsu-

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Table 1. Age, Race, and Sex Distributions of Young Offspring of Diabetic and Nondiabetic Parents: The Bogalusa Heart Study

Parental Diabetes	Age (yr)							
	7-12		13-17		18-25		Total	
	White	Black	White	Black	White	Black	White	Black
Yes								
Male	5	0	3	10	3	1	11	11
Female	4	4	8	6	5	4	17	14
No								
Male	8	2	7	6	1	2	16	10
Female	4	5	8	5	3	1	15	11
Total	21	11	26	27	12	8	59	46

lin) by a radioimmunoassay (RIA) procedure, using the Phadebas insulin kit (Pharmacia Diagnostics, Piscataway, NJ); specific insulin (which does not cross-react with proinsulin) by a specific double-antibody RIA procedure (Linco, St Louis, MO); proinsulin (intact and split product des 31,32) by a nonequilibrium RIA method (Linco); plasma C-peptide by a RIA kit (Incstar, Stillwater, MN); plasma glucagon by a RIA kit (ICN Biomedical, Costa Mesa, CA); plasma free fatty acids by a colormetric procedure (Wako Chemicals USA, Richmond, VA); and blood glycated hemoglobin by an affinity chromatography method using Glyc-Affin GHb (Isolab, Akron, OH). Serum total cholesterol and triglycerides were measured by enzymatic procedures in an Abbott VP analyzer (Abbott Laboratories); lipoprotein cholesterols were measured by a combination of heparin-calcium precipitation and agar-agarose gel electrophoresis.[26]

The incremental areas under the curve (ie, above the basal concentrations during a 1-hour period) after glucose challenge were calculated by the trapezoidal rule: 0.25 h (0.5 · fasting value + 15-min value + 0.5 · 30-min value) + 0.5 (0.5 · 30-min value + 0.5 · 60-min value) − fasting value. Insulin resistance index and β-cell function index were calculated according to the homeostasis model assessment formulae[27]: insulin resistance index = fasting insulin (μU/mL) × fasting glucose (mmol/L) ÷ 22.5 and β-cell function index = 20 × fasting insulin (μU/mL) ÷ fasting glucose (mmol/L) − 3.5. This model is considered useful to assess insulin resistance and β-cell function in epidemiological studies.[28]

A multiway ANCOVA was performed for each dependent variable. The independent variables used in the model were age, sex, race, parental history of diabetes, and race by parental history interaction. The same analysis was repeated including body-mass index (BMI; weight in kilograms divided by height in meter squared) as another independent variable to adjust for the influence of obesity. Step-wise logistic regression was performed to determine what characteristics are independently associated with parental history. Age, sex, and BMI were forced into the model. The Statistical Analysis System (SAS) was used to perform the various analyses.[29]

RESULTS

Selected Clinical Characteristics

Information on age, anthropometric, blood pressure, and lipoprotein variables in young offspring of diabetics versus nondiabetics by race are presented in Table 2. Mean age, which ranged from 14.2 years to 15.6 years, was similar between groups. Body weight and measures of body fatness, such as BMI, triceps skinfold thickness, and subscapular skinfold thickness, were significantly higher in offspring of diabetics than in offspring of nondiabetics only among whites (P = .04 to .01); both waist circumference and waist-to-hip ratio, measures of abdominal fat, were significantly higher among offspring of diabetics of both races. In addition, the waist-to-hip ratio was significantly greater in white offspring than in black offspring, regardless of parental history of diabetes.

Diastolic blood pressure was lower in black offspring of diabetics versus nondiabetics (P = .04); however, this difference disappeared after adjusting for BMI and waist. Levels of very–low-density lipoprotein (VLDL) cholesterol were lower

Table 2. Anthropometric, Blood Pressure, and Lipoprotein Variables in Young Offspring of Diabetic Versus Nondiabetic Parents: The Bogalusa Heart Study

Variable	Parental Diabetes				Without Adjusting for BMI and Waist			Adjusting for BMI and Waist		
	White		Black							
	Yes (n = 28)	No (n = 31)	Yes (n = 25)	No (n = 21)	Parental Diabetes	Race	Interaction	Parental Diabetes	Race	Interaction
Age (yr)	15.2 ± 0.9	14.2 ± 0.7	15.6 ± 0.7	15.3 ± 0.6						
Weight (kg)	66.0 ± 4.6	52.8 ± 2.9	64.9 ± 4.3	67.5 ± 5.9	—	—*	.049			
BMI (kg/m²)	25.7 ± 1.4	21.1 ± 0.8	23.4 ± 1.2	24.6 ± 1.7	—	—	.025			
Triceps skinfold (mm)	22.2 ± 1.9	16.3 ± 1.2	15.7 ± 1.9	19.2 ± 2.7	—	—	.019			
Subscapular skinfold (mm)	20.7 ± 2.5	12.9 ± 1.6	14.7 ± 1.8	15.4 ± 2.1	—	—	.048			
Waist (cm)	83.7 ± 3.4	73.2 ± 2.2	76.9 ± 2.4	76.0 ± 3.3	.037	—	—			
Waist/hip ratio	0.84 ± 0.01	0.82 ± 0.01	0.81 ± 0.01	0.77 ± 0.01	.010	.003	—			
Systolic blood pressure (mm Hg)	111.5 ± 2.0	106.0 ± 1.4	108.5 ± 1.9	108.7 ± 2.3	—	—	—	—	—	—
Diastolic blood pressure (mm Hg)	66.4 ± 1.6	63.6 ± 1.6	64.4 ± 1.6	68.2 ± 1.3	—	—	.025	—	—	—
Cholesterol (mg/dL)										
Total	174.0 ± 6.1	166.5 ± 5.6	159.5 ± 5.2	171.0 ± 6.5	—	—	—	—	—	—
VLDL cholesterol	18.5 ± 2.0	15.2 ± 1.6	7.8 ± 0.9	16.7 ± 2.0	—	.007	.001	−.009	.032	.001
LDL cholesterol	109.7 ± 5.4	102.1 ± 1.6	92.1 ± 2.6	105.1 ± 6.0	—	—	.042	—	—	.035
HDL cholesterol	45.9 ± 2.1	49.3 ± 1.9	59.6 ± 4.4	49.2 ± 2.4	—	.014	.012	—	.04	.028
Triglycerides (mg/dL)	133.6 ± 16.7	99.4 ± 7.6	66.9 ± 5.0	89.7 ± 9.9	—	.001	.014	—	.001	.014

NOTE. Comparisons adjusted for age and sex. Values are mean ± SE.
*Not significant.

($P = .001$) and high-density lipoprotein (HDL) cholesterol higher ($P = .02$) in black offspring of diabetics versus nondiabetics, with or without adjusting for BMI and waist. White offspring of diabetics compared with control offspring had higher serum triglyceride levels ($P = .03$); however, this difference disappeared after adjusting for BMI and waist. In addition, the well-known race differential in VLDL cholesterol (whites > blacks), triglycerides (whites > blacks) and HDL cholesterol (blacks > whites) were noted, regardless of parental history of diabetes.

Measures of Glucose Homeostasis

Basal. As seen in Table 3, fasting glucose levels were slightly higher in offspring of diabetic parents of both races, but this difference was not significant when adjusted for BMI and waist. Similarly, fasting immunoreactive insulin levels were higher in case offspring of both races, but the difference was not significant when corrected for BMI and waist. Fasting specific insulin and C-peptide levels also tended to be higher in offspring of diabetics of both races, but not at a significant level. However, the immunoreactive insulin–to–C-peptide ratio was significantly higher in offspring of diabetics of both races. The insulin–to–C-peptide ratio showed a significant race differential (blacks > whites), regardless of status of BMI and waist or parental disease. In addition, BMI- and waist-adjusted fasting specific insulin levels were significantly higher in blacks versus whites regardless of status of parental disease. Offspring of diabetics of both races had an increased insulin resistance index assessed by homeostasis model compared with their control counterparts, but this difference disappeared after adjusting for BMI and waist. β-cell function index assessed by homeostasis model showed no significant difference in offspring of diabetics of both races versus their counterparts.

Responses to oral glucose load. Postglucose plasma responses (incremental area above the fasting level) of parameters of glucose homeostasis are given in Table 4. In addition, race- and sex-specific mean levels of glucose (Fig 1), immunoreactive insulin (Fig 2), and C-peptide (Fig 3) during the glucose tolerance test by parental history of diabetes are shown. Among the variables listed in Table 4, glucose response differed significantly between the offspring of both races with and without parental diabetes; the values remained higher in offspring of diabetics after adjusting for BMI and waist.

White offspring of diabetics versus nondiabetics tended to show higher insulin and C-peptide responses; this trend was just the opposite in black offspring. This divergent trend was mainly due to 30-minute postglucose values noted among black males and white females (Figs 2 and 3). However, this divergent trend was no longer significant after adjusting for BMI and waist.

There was a significant racial difference in glucose and C-peptide responses, with white offspring showing higher responses than black offspring, irrespective of status of BMI and waist or parental diabetes. Postglucose suppression of free fatty acids was less in white offspring of both diabetics and nondiabetics compared with their black counterparts; however, this difference disappeared after adjusting for BMI and waist.

Early-phase insulin and glucose responses were examined in terms of plasma insulin and glucose levels at 30 minutes after the glucose challenge. Thirty-minute glucose levels, rather than 30-minute insulin levels, showed a distinct pattern of distributions only among white offspring (mean ± SE: 152.4 ± 7.2 mg/dL for offspring of diabetics *v* 129.1 ± 3.4 mg/dL for offspring of nondiabetics, $P < .001$). This pattern was not found among black offspring (123.1 ± 4.0 mg/dL *v* 122.8 ± 4.2 mg/dL, $P > .05$). With respect to 30-minute insulin, the values were 118.6 ± 16.2 μU/mL for white offspring of diabetics versus 79.2 ± 12.1 μU/mL for nondiabetics ($P > .05$) and 101.6 ± 13.3 μU/mL for black offspring of diabetics versus 135.7 ± 17.3 μU/mL for nondiabetics ($P > .05$).

Table 3. Fasting Levels of Measures of Carbohydrate Tolerance in Young Offspring of Diabetic Versus Nondiabetic Parents: The Bogalusa Heart Study

	Parental Diabetes				Without Adjusting for BMI and Waist			Adjusting for BMI and Waist		
	White		Black							
Variable	Yes (n = 28)	No (n = 31)	Yes (n = 25)	No (n = 21)	Parental Diabetes	Race	Interaction	Parental Diabetes	Race	Interaction
Glucose (mg/dL)	85.6 ± 1.2	82.2 ± 1.0	83.8 ± 1.8	82.7 ± 1.8	.060	—†	—	—	—	—
Insulin (μU/mL)*	19.6 ± 2.8	12.4 ± 1.4	17.5 ± 2.6	15.1 ± 2.2	.039	—	—	—	—	—
Specific insulin (pmol/L)	124.7 ± 14.5	89.8 ± 11.4	134.5 ± 21.8	119.1 ± 22.3	—	—	—	—	.031	—
Proinsulin (pmol/L)	11.9 ± 2.6	13.4 ± 1.8	10.6 ± 1.7	12.4 ± 2.2	—	—	—	—	—	—
C-peptide (ng/mL)	2.1 ± 0.2	1.7 ± 0.2	1.9 ± 0.3	1.6 ± 0.2	—	—	—	—	—	—
Insulin/C-peptide ratio	9.0 ± 0.6	6.9 ± 0.3	10.9 ± 1.3	9.2 ± 0.7	.011	.005	—	.054	.001	—
Glucagon (pg/mL)	245.8 ± 16.0	208.6 ± 12.1	222.2 ± 12.1	222.4 ± 20.2	—	—	—	—	—	—
Free fatty acids (μmol/L)	511.9 ± 40.1	553.4 ± 31.9	529.7 ± 39.0	539.5 ± 44.8	—	—	—	—	—	—
Glycated hemoglobin (%)	5.6 ± 0.1	5.5 ± 0.1	5.6 ± 0.1	5.5 ± 0.1	—	—	—	—	—	—
Insulin resistance index	4.2 ± 0.6	2.6 ± 0.3	3.8 ± 0.6	3.2 ± 0.6	.036	—	—	—	—	—
β-cell function index	323.4 ± 49.6	234.8 ± 28.4	303.1 ± 38.7	278.3 ± 31.8	—	—	—	—	—	—

NOTE. Comparisons adjusted for age and sex. Values are mean ± SE.

*Immunoreactive insulin.

†Not significant.

Table 4. Incremental Response to Oral Glucose Load of Measures of Carbohydrate Tolerance in Young Offspring of Diabetic Versus Nondiabetic Parents: The Bogalusa Heart Study

	Parental Diabetes				Without Adjusting for BMI and Waist			Adjusting for BMI and Waist		
	White		Black							
Incremental Response*	Yes (n = 28)	No (n = 31)	Yes (n = 25)	No (n = 21)	Parental Diabetes	Race	Interaction	Parental Diabetes	Race	Interaction
Glucose (mg/dL · h)	46.8 ± 5.1	34.1 ± 2.5	27.8 ± 3.2	26.0 ± 2.4	.032	.000	—‡	.075	.001	—
Insulin (μU/mL · h)†	74.2 ± 10.4	54.5 ± 9.3	58.6 ± 7.5	78.2 ± 9.1	—	—	.048	—	—	—
Specific insulin (pmol/L · h)	575.1 ± 92.3	367.9 ± 76.8	382.4 ± 63.6	652.8 ± 155.6	—	—	.024	—	—	—
Proinsulin (pmol/L · h)	21.8 ± 3.3	22.9 ± 3.5	17.1 ± 2.6	20.6 ± 2.6	—	—	—	—	—	—
C-peptide (ng/mL · h)	3.6 ± 0.4	2.8 ± 0.4	1.9 ± 0.3	3.0 ± 0.4	—	.044	.028	—	.053	—
Glucagon (pg/mL · h)	25.3 ± 9.4	17.3 ± 7.9	3.4 ± 5.5	20.6 ± 7.4	—	—	—	—	—	—
Free fatty acids (μmol/L · h)	−138.0 ± 29.5	−109.0 ± 22.9	−148.0 ± 30.6	−201.0 ± 27.6	—	.060	—	—	—	—

NOTE. Comparisons adjusted for age and sex. Values are mean ± SE.
*Area under the curve–fasting value.
†Immunoreactive insulin.
‡Not significant.

Predictor Variables

The independent correlates in young offspring associated with parental diabetes are shown in Table 5. Insulin–to–C-peptide ratio and glucose response in young offspring were independently associated with parental diabetes. For example, compared with offspring whose glucose response was at the 25th percentile (22.1 mg/dL · h), offspring whose response was at the 75th percentile (45.6 mg/dL · h) were 2.01 times more likely to have diabetic parents.

DISCUSSION

The present study of healthy black and white subjects, conducted in an epidemiologic setting, shows that abnormalities in carbohydrate metabolism occur early in life in those with parental history of type 2 diabetes. Many previous studies in adults demonstrated that hyperinsulinemia and insulin resistance predate the development of disease.[13-15,30-34] Further, studies in ethnic groups with an unusually high prevalence of type 2 diabetes have shown childhood hyperinsulinemia to be a predictor of subsequent disease.[18,19] In this study, increased basal immunoreactive insulin–to–C-peptide ratio and postglucose glucose response were the two independent metabolic characteristics that distinguished both black and white young offspring of diabetics from those without parental diabetes. This trend was independent of other confounding variables, including body fatness measures. To our knowledge, no comparable black versus white data are available in this age group.

Since clinical methods to assay insulin sensitivity and secretion are difficult to apply in the epidemiologic setting, we used simple measures of glucose homeostasis for this purpose. Plasma C-peptide has been used as a semiquantitative measure of the pancreatic β-cell function.[35,36] It has been suggested that the insulin–to–C-peptide ratio in plasma reflects changes in hepatic insulin extraction.[37,38] The increased insulin–to–C-peptide ratio with no changes in C-peptide levels, independent of body fatness and other covariates, among white and black offspring of diabetics (*v* nondiabetics) in the present study suggests hepatic resistance and its attendent reduced insulin clearance by the liver in those at high risk to develop type 2 diabetes. In addition, the offspring of diabetics of both races had increased basal insulin and insulin resistance index associated with increased body fatness in the absence of significant

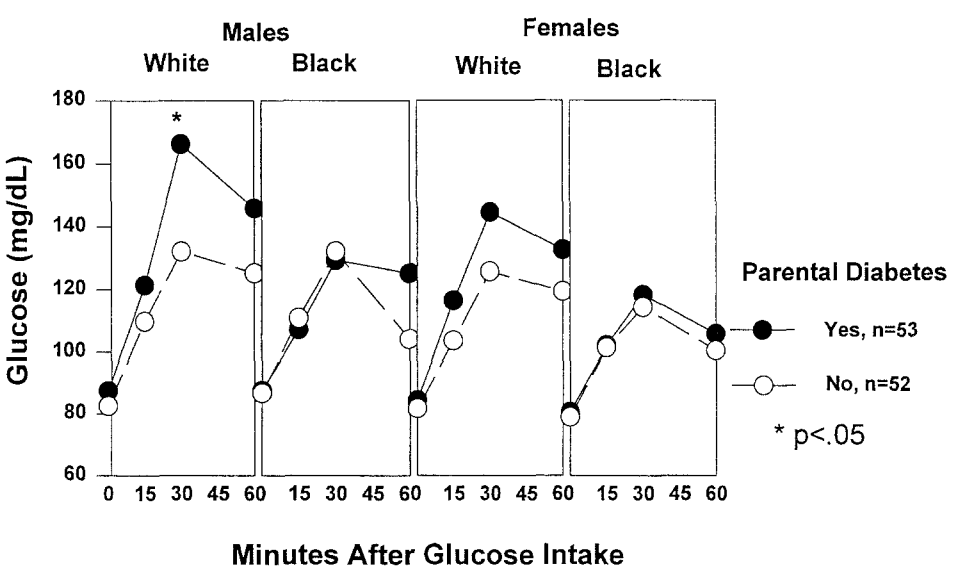

Fig 1. Mean glucose levels during a glucose tolerance test in young offspring of diabetic and nondiabetic parents by race and sex.

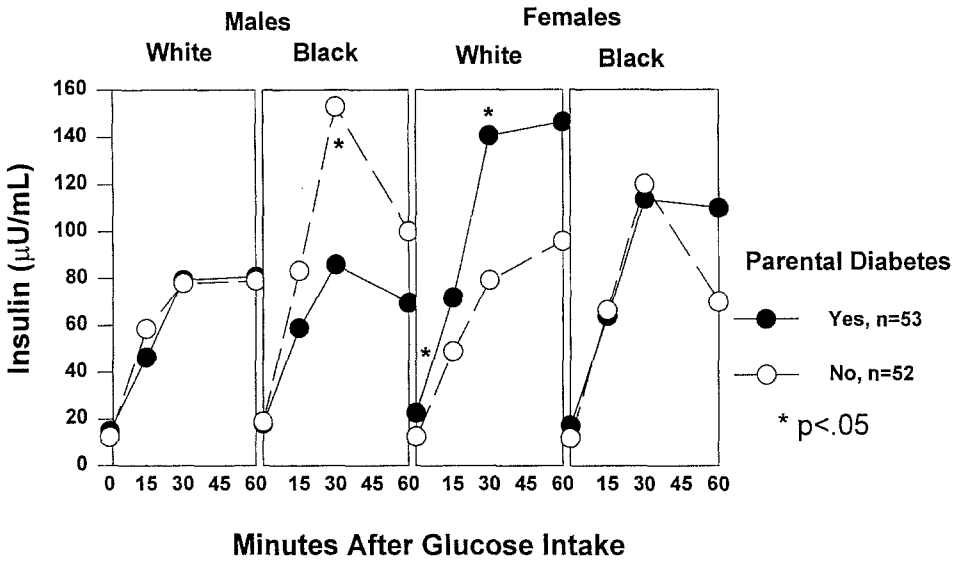

Fig 2. Mean immunoreactive insulin levels during a glucose tolerance test in young offspring of diabetic and nondiabetic parents by race and sex.

increase in postglucose insulin or C-peptide response. A decrease in insulin action may antedate the development of significant hyperinsulinemia, because insulin resistance without concomitant hyperinsulinemia was found in adult first-degree relatives of type 2 diabetics.[12]

It has been reported that plasma proinsulin, a marker of islet-cell distress or compromised insulin secretion, is increased in type 2 diabetics,[39,40] prediabetics,[41,42] subjects with impaired glucose tolerance,[43,44] and nondiabetic offspring of diabetic parents.[45] However, the present study, along with other earlier studies,[40,46] found no such trend among nondiabetic subjects with a family history of diabetes. It is likely that abnormal proinsulin processing may not occur in early life among those at high risk to develop type 2 diabetes.

In addition to impaired insulin sensitivity, a defect in insulin independent glucose utilization (glucose effectiveness) is considered an important characteristic of diabetes susceptibility.[13,14] Our results suggest slow glucose removal in the young offspring of diabetics of both races, especially in white children. This trend was clearly apparent in the early phase (30 minutes) of postglucose glucose response among the white offspring of diabetics. It is of interest that 38% of white offspring of diabetics had 30-minute glucose levels greater than 162 mg/dL, whereas only one white offspring of nondiabetic exceeded this value.

The present study also demonstrates black-white divergences in certain parameters associated with glucose homeostasis, regardless of parental history of type 2 diabetes. The postglucose glucose response, basal hepatic insulin extraction (insulin–to–C-peptide ratio), and insulin secretion (C-peptide response to glucose load) were all relatively lower in black offspring than in white offspring. Although the abdominal fat (waist and waist-to-hip ratio) was lower in black offspring, it should be noted that black-white differences in glucose response, insulin secretion, and hepatic insulin extraction were independent of body fatness measures. In addition, insulin-mediated postglucose suppression of free fatty acids was relatively greater in blacks than in whites. This may be related to the observed racial

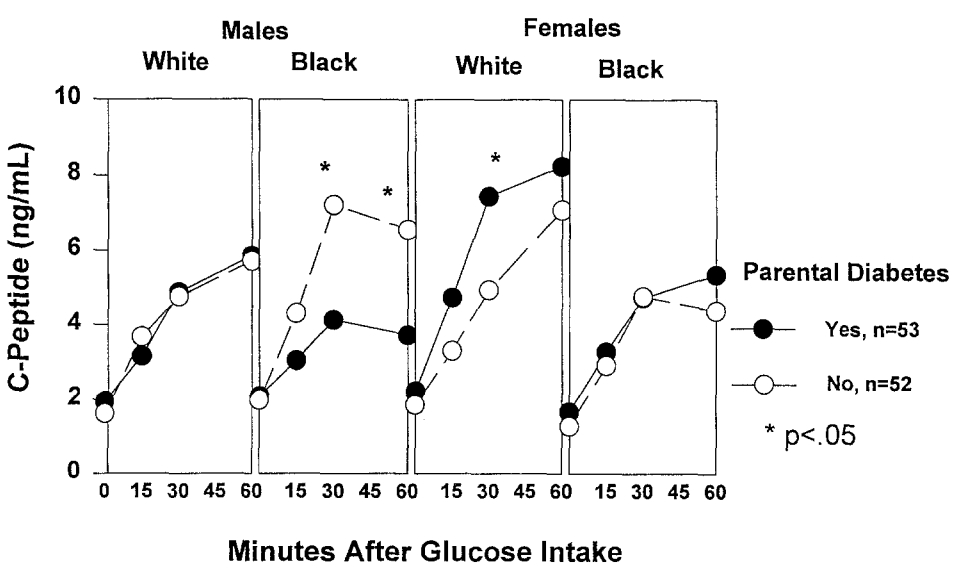

Fig 3. Mean C-peptide levels during a glucose tolerance test in young offspring of diabetic and nondiabetic parents by race and sex.

Table 5. Predictor Variables of Young Offspring Associated With Parental Diabetes: The Bogalusa Heart Study

Independent Variables*	Odds Ratio (95% Confidence Interval)	P Value
Insulin/C-peptide ratio (10.6 *v* 6.4)	1.92 (1.09-3.37)	.023
Incremental glucose response (45.6 mg/dL · h *v* 22.1 mg/dL · h)	2.01 (1.04-3.87)	.035

NOTE. Included variables that were significant in the univariate analysis: BMI, waist, waist/hip ratio, fasting glucose, fasting insulin, insulin resistance index, insulin/C-peptide ratio, and incremental glucose response.

*Comparing high (75th percentile) *v* low (25th percentile) values, adjusting for age and sex.

difference in abdominal fat, which is known to have a low responsiveness to the antilipolytic effect of insulin.[47-49] Greater suppression of free fatty acids may relate to the observed lower serum triglycerides and VLDL cholesterol in black offspring.

Earlier studies, including our own, noted black-white differences in hepatic insulin extraction,[20,22,50] glucose response,[21,51,52] and abdominal fat.[52-55] Decreased hepatic insulin extraction in blacks has been implicated in their higher prevalence of type 2 diabetes,[20,50] although no association between hepatic insulin extraction and insulin sensitivity was found either in whites or blacks.[20] The present finding that insulin secretion was lower in blacks compared with whites differs from earlier studies, which found insulin secretion to be either similar in blacks and whites[50] or higher in blacks.[51,56] Also, unlike earlier studies,[20,51,56] this study found no black-white difference in insulin resistance. The reasons for these discrepancies are not clear, except that our study subjects are very young. Importantly, the observed black-white differences in certain parameters associated with glucose homeostasis likely has a bearing on the disparities in the prevalent rates of the disease.

In summary, our study demonstrates that it is possible to identify metabolic abnormalities in early life among those at high risk to develop type 2 diabetes, and that black-white differences in factors governing glucose homeostasis occur in youth, regardless of parental history of disease. Since type 2 diabetes is a heterogeneous disorder, long-term follow-up studies are needed to ascertain the relationships between the observed correlates of parental disease and development of diabetes later in life in the two racial groups. Nonetheless, recent studies show an increasing incidence of type 2 diabetes among adolescents, accompanying the upward secular trend in obesity in American youth.[57-60] An increased insulin resistance associated with body fatness in the young offspring of diabetics, as seen in the present study, has practical implications for prevention or delay the onset of disease through control of obesity and increased physical activity early in life.

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