Patterns of Glucose and Lipid Abnormalities in Black NIDDM Subjects

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Objective: We had previously shown two variants among black non-insulin-dependent diabetic (NIDDM) subjects in a normoglycemic remission: one with insulin resistance and the other with normal insulin sensitivity. This study examined whether these two variants exist in the ordinary hyperglycemic black NIDDM population. Research Design and Methods: Fifty-two black NIDDM subjects were assessed for insulin-stimulated glucose disposal (euglycemic clamp), glycemic control (fasting plasma glucose and HbA_{1c}), and fasting lipid profiles. Results: The distribution of glucose disposal in 30 black NIDDM subjects (body mass index; BMI <30 kg/m²) was bimodal, which indicated two populations. Eighteen of 30 subjects (BMI 26.4 ± 0.5 kg/m²) had insulin resistance (glucose disposal 3.21 ± 0.24 mg · kg^{-1} · min^{-1}). Twelve of 30 subjects (BMI 24.83 ± 1.1 kg/m²) had normal insulin sensitivity (glucose disposal 7.19 ± 0.46 mg · kg^{-1} · min^{-1}). Twenty-one of the remaining 22 subjects (BMI 33.4 ± 0.7 kg/m²) were insulin resistant (glucose disposal 2.88 ± 0.21 mg · kg^{-1} · min^{-1}). Fasting serum triglyceride levels were lowest in the insulin-sensitive population (0.91 ± 0.07 mM) and different from the insulin-resistant population, BMI <30 and >30 kg/m², (1.20 ± 0.10 mM, $P < 0.05$ and 1.42 ± 0.17 mM, $P < 0.025$, respectively). Fasting serum low-density lipoprotein cholesterol levels were not significantly different among the groups, although it did increase with insulin resistance and increasing obesity. Total serum cholesterol levels and glycemic control were similar for all three groups. Serum high-density lipoprotein cholesterol levels were higher in women compared with men. Conclusions: In the hyperglycemic black NIDDM population, two variants exist: one with insulin resistance and one with normal insulin sensitivity. This insulin-sensitive variant represents 40% of subjects with a BMI <30 kg/m². Moreover, the insulin-sensitive group has a lower risk profile for cardiovascular disease. *Diabetes Care* 14:1036–42, 1991

In a series of investigations undertaken to define the pathogenesis of non-insulin-dependent diabetes mellitus (NIDDM), we studied a unique population of black NIDDM subjects who were in near-normoglycemic remission and off all pharmacological therapy (1,2). For that population, we found that NIDDM consisted of two different variants: one with primary peripheral insulin resistance and one with normal peripheral insulin sensitivity. Each of these variants had its own specific metabolic profile (3). The subjects with insulin resistance had decreased insulin-mediated glucose disposal, hyperinsulinemia, increased obesity, high serum cholesterol and low-density lipoprotein (LDL) cholesterol, high serum triglycerides, and normal serum high-density lipoprotein (HDL) cholesterol. The patients with normal insulin sensitivity had normal insulin-mediated glucose disposal, normal fasting insulin levels, decreased glucose-stimulated insulin secretion, mild obesity, normal serum cholesterol and LDL cholesterol, low serum triglycerides, and normal serum HDL cholesterol. These different metabolic profiles suggest not only that the underlying disease is different, but that the risk of macrovascular disease complications will be high in one of the variants and low in the other.

An important question is whether the two variants described exist only in this small unique population of

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black NIDDM subjects who can achieve near-normoglycemic remission or are characteristic of the black NIDDM population in general. This study describes the metabolic profiles of a random sample of 52 black NIDDM subjects representative of the general NIDDM population.

RESEARCH DESIGN AND METHODS

Fifty-two black subjects with NIDDM were randomly selected from our diabetes clinic population. The population ranged in age from 28 to 65 yr and consisted of 31 women and 21 men. Their ethnic origins were equally distributed among American-born and Caribbean-born blacks. Body mass index (BMI, kg/m^2) ranged from 20.0 to 42.1. NIDDM had been diagnosed based on clinical course and response to therapy. None of the subjects had experienced a remission from their hyperglycemic state. Thirteen of the subjects were being treated with insulin, 3 with diet alone, and the remainder were treated with oral sulfonylurea agents. All subjects were instructed in an appropriate American Diabetes Association diet, and compliance with the diet had not varied during the several months preceding the study, because body weight had not changed significantly during that time. No subject had a history of significant hepatic, renal, or endocrine disease. Studies were performed at the Clinical Research Center at State University of New York—Health Science Center at Brooklyn after written informed consent was obtained.

Insulin sensitivity and hepatic glucose production were determined by a modification of the euglycemic insulin-clamp technique after an overnight fast (4). Diabetes medication was withheld on the morning of the study. A catheter was placed in the antecubital vein antegrade for the administration of infusates. A catheter was placed retrograde in the hand and the hand was kept in a warming box at 68°C to provide arterialized venous blood for sampling. [3-^{3}H]D-glucose was infused in a primed continuous manner for 180–210 min before the start of the insulin infusion. Two hundred thirty-six nCi/kg [3-^{3}H]D-glucose was injected as a bolus, followed by a continuous infusion at 2.36 $nCi \cdot kg^{-1} \cdot min^{-1}$. After the equilibration period, regular human insulin was infused initially as a priming dose to acutely raise plasma insulin to the desired level (5), and then was infused at a rate of 1 $mU \cdot kg^{-1} \cdot min^{-1}$ for 120 min. Plasma glucose was measured every 5 min and was allowed to fall to 5.5 mM during the 1st h of the insulin infusion.

Samples were obtained every 15 min for hormones and specific activity of [3-^{3}H]D-glucose during the insulin infusions. Urine was assayed for glucose losses throughout the study, and basal and insulin-stimulated glucose disposal rates were corrected for these losses.

To verify that the decrease in plasma glucose to 5.5 mM at the start of the study does not significantly alter the response to the subsequent continuous 1-$mU \cdot kg^{-1} \cdot min^{-1}$ insulin infusion, we compared the effects of the acute decrease in plasma glucose to a decrease in plasma glucose with an overnight insulin infusion. Four insulin-resistant NIDDM subjects with moderate glycemic control on glipizide had replicate 1-$mU \cdot kg^{-1} \cdot min^{-1}$ insulin clamps separated by several days to several weeks. For one insulin-clamp study, the ambient plasma glucose level was allowed to decrease to 5.5 mM at the start of the study, as described above in our study subjects. For the other insulin-clamp study, the ambient plasma glucose level was lowered to 5.5 mM by a 10-h overnight monitored insulin infusion. The glucose disposal rate during the 2nd h of the 1-$mU \cdot kg^{-1} \cdot min^{-1}$ insulin infusion during the euglycemic clamp was not significantly different between the two techniques to lower the ambient glucose level to euglycemia (3.75 ± 0.39 vs. 3.22 ± 0.27 $mg \cdot kg^{-1} \cdot min^{-1}$, acute vs. overnight, respectively).

The rate of glucose appearance and disappearance was measured in the basal state and during the last 60 min of the insulin-infusion period. Before basal measurements were taken [3-^{3}H]D-glucose was infused for at least 180–210 min. Because plasma counts per minute were stable in multiple samples taken every 10 min for the 40 min before the start of the insulin infusion, it was assumed that we had reached steady state. Thus, hepatic glucose production in the basal state was calculated on the basis of steady-state kinetics. Steele's equations were used to calculate the rates of appearance and disappearance of glucose during the insulin-infusion period (6). Hepatic glucose production is equal to the total rate of appearance of glucose minus the rate of exogenously administered glucose.

Insulin-stimulated glucose disposal (M) was defined as the sum of the hepatic glucose production and the rate of exogenous glucose infusion. Urinary glucose losses were calculated for each insulin-infusion period and subtracted as appropriate.

Plasma glucose was measured by the glucose oxidase method with a Beckman Glucose Analyzer (Fullerton, CA). Specific activity for [3-^{3}H]D-glucose was determined on plasma samples deproteinized with barium hydroxide and zinc sulfate. Plasma insulin levels were assayed with a standard double-antibody radioimmunoassay (7). Serum lipids were measured after a 12-h fast. Total cholesterol and triglyceride were measured enzymatically with the Kodak Ektachem 400 system (Rochester, NY; 8,9). HDL cholesterol was measured enzymatically after the removal of LDL and very-low-density lipoproteins with a dextran magnesium precipitation (10). The interassay coefficient of variation for total cholesterol and triglyceride was 2.6 and 3.0, whereas that for HDL cholesterol was 4.8%. Control sera were provided by the College of American Pathology. All lipid assays met the Centers for Disease Control and College of American Pathology Standardization programs. LDL cholesterol was estimated according to Friedewald et al. (11), i.e., LDL cholesterol equals total cholesterol minus (HDL cholesterol + 0.2 × triglycer-

ide). HbA_{1c} was performed by high-performance liquid chromatography in most subjects or affinity chromatography in subjects with hemoglobin variants. The data were presented as a ratio of normal because the normal range was different for both methods.

Statistical analyses. Analyses were performed with the Student's *t* test, one-way analysis of variance (ANOVA), and Scheppe's multiple range test (12). Bimodality of insulin action was determined according to Haldane with the *Z* distribution (13). Data are presented as means ± SE or as means ± SD, as described.

RESULTS

Insulin-stimulated glucose disposal (M), which is a sensitive measure of insulin action, was determined during a 1 mU · kg^{-1} · min^{-1} insulin infusion after the plasma glucose level was allowed to decrease and then be maintained at 5.5 mM (see METHODS). Figure 1 presents the data obtained in the 52 subjects. The population was grouped according to a BMI of <30 or >30 kg/m^2. We have previously shown in the insulin-sensitive near-normoglycemic black NIDDM population that insulin sensitivity has an inverse relationship with BMI. For a BMI >30 kg/m^2, insulin resistance that is secondary to obesity becomes manifest (14) and cannot be distinguished from primary insulin resistance, which is independent of BMI. The plot of frequency versus insulin-mediated glucose disposal for black NIDDM subjects with a BMI <30 kg/m^2 shows a bimodal distribution (Fig. 1*A*, $P < 0.0014$; 13). One group distributes between an M value of 1.5–4.5 mg · kg^{-1} · min^{-1}, which is below the mean −2SD of our control group (5.57 mg · kg^{-1} · min^{-1}), whereas the other distributes between 5.5 and 11.5 mg · kg^{-1} · min^{-1}. Forty percent of black NIDDM subjects with a BMI <30 kg/m^2 had an insulin-mediated glucose disposal rate >5.5 mg · kg^{-1} · min^{-1}. There was no difference in insulin sensitivity based on sex in this population (BMI <30 kg/m^2), because 43% of the women and 38% of the men had an insulin-stimulated glucose disposal rate that was considered to be within the normal range (Fig. 2).

Only 1 of 22 (4.5%) black NIDDM subjects with a BMI >30 kg/m^2 had an insulin-mediated glucose disposal rate that was >5.5 mg · kg^{-1} · min^{-1}. Nine healthy nondiabetic subjects constituted our control population for insulin-mediated glucose disposal. The mean ± SD of their data were age 42. 7 ± 8.6 yr, fasting plasma glucose 5.49 ± 0.17 mM, BMI 25.2 ± 2.9 kg/m^2, and 1-mU · kg^{-1} · min^{-1} insulin–mediated glucose disposal 7.59 ± 1.01 mg · kg^{-1} · min^{-1}.

Figure 2 shows the relationship of insulin sensitivity and insulin resistance to obesity in black male and female NIDDM subjects. For subjects with a BMI <30 kg/m^2, normal insulin sensitivity is almost as frequent

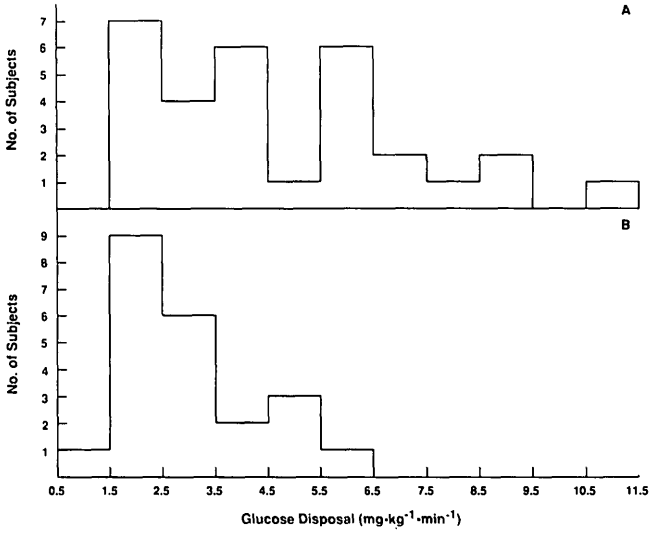

FIG. 1. Distribution of glucose disposal (1 mU · kg^{-1} · min^{-1} insulin clamp) in black non-insulin-dependent diabetic subjects. *A*: distribution of glucose disposal for subjects with body mass index (BMI) <30 kg/m^2. Note that there is bimodal distribution ($P < 0.0014$) showing 1 group with insulin resistance distributing between 1.5 and 4.5 mg · kg^{-1} · min^{-1} and 1 group with normal insulin sensitivity distributing between 5.5 and 11.5 mg · kg^{-1} · min^{-1}. *B*: distribution of glucose disposal for subjects with BMI >30 kg/m^2. Only 1 subject has glucose disposal in normal range.

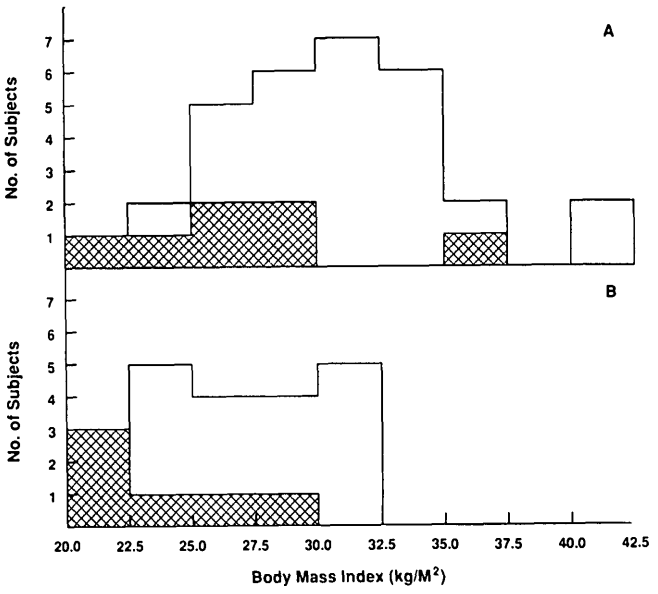

FIG. 2. Insulin action and body mass index (BMI) as function of sex. Frequency of insulin sensitivity and resistance at varying BMI in women (*A*, *n* = 31) and men (*B*, *n* = 21). *Hatched area*, insulin-sensitive subjects; *open area*, insulin-resistant subjects.

TABLE 1
Characteristics of insulin-sensitive and insulin-resistant black non-insulin-dependent diabetic subjects

	Insulin sensitive	Insulin resistant (BMI <30 kg/m²)	Insulin resistant (BMI >30 kg/m²)
	13	18	21
Age (yr)	49.3 ± 2.4	44.3 ± 2.4	49.4 ± 2.2
BMI (kg/m²)	25.6 ± 1.3	26.4 ± 0.5	33.4 ± 0.7*
Insulin-stimulated glucose disposal (mg · kg⁻¹ · min⁻¹)	7.06 ± 0.46	3.21 ± 0.24*	2.88 ± 0.21*
Triglycerides	0.91 ± 0.07	1.20 ± 0.10†	1.42 ± 0.17‡
Cholesterol (mM)	5.16 ± 0.39	5.15 ± 0.29	6.08 ± 0.38
HDL cholesterol (mM)	1.34 ± 0.08	1.15 ± 0.05†	1.34 ± 0.11
LDL cholesterol (mM)	3.40 ± 0.38	3.50 ± 0.31	3.98 ± 0.34
Fasting plasma glucose (mM)	10.6 ± 1.0	8.2 ± 0.5†	9.7 ± 0.8
HbA_{1c} (ratio)§	1.53 ± 0.10	1.46 ± 0.09	1.64 ± 0.14

BMI, body mass index; HDL, high-density lipoprotein; LDL, low-density lipoprotein.
*$P < 0.001$ vs. insulin-sensitive group.
†$P < 0.05$ vs. insulin-sensitive group.
‡$P < 0.025$ vs. insulin-sensitive group.
§Subject's HbA_{1c} divided by normal HbA_{1c}.

as insulin resistance. Table 1 compares the characteristics of the insulin-sensitive group to the minimally obese insulin-resistant group with a BMI <30 kg/m² and to the significantly obese group with a BMI >30 kg/m². The groups did not differ in age. The insulin-sensitive and the insulin-resistant groups with a BMI <30 kg/m² had comparable obesity so that the difference in insulin sensitivity could not be attributed to obesity. The significantly obese insulin-resistant group had no more insulin resistance than the mildly obese insulin-resistant group.

Fasting plasma glucose was mildly elevated in all three groups. Fasting plasma glucose for the insulin-resistant group with a BMI <30 kg/m² was different from the insulin-sensitive group ($P < 0.05$ by Student's t test but NS by ANOVA and Scheppe's multiple range test; Table 1). The HbA_{1c} levels were similar for all groups, suggesting that their glycemic control was equivalent. Basal hepatic glucose production rates were similar for all groups (data not given). Diabetes treatment regimens were similar for all groups with two-thirds of subjects in all groups treated with sulfonylureas and the remainder treated with diet, insulin, or combination insulin and sulfonylurea therapy.

Fasting serum triglyceride levels were lowest in the insulin-sensitive group (0.91 ± 0.07 mM) and different from the insulin-resistant group with a BMI <30 kg/m² (1.20 ± 0.10 mM, $P < 0.05$) and from the insulin-resistant group with a BMI >30 kg/m² (1.42 ± 0.17 mM, $P < 0.025$; Table 1). One-way ANOVA of triglyceride among the groups was significant at $P < 0.03$. No one in the insulin-sensitive group had a fasting triglyceride level >1.41 mM. However, 33.3% of the insulin-resistant group with a BMI <30 kg/m² and 47.4% of the insulin-resistant group with a BMI >30 kg/m² had a fasting triglyceride level >1.41 mM (Fig. 3). Fasting serum cholesterol levels were similar in all three groups (Table 1).

Fasting serum LDL cholesterol levels were not different in the three groups (insulin sensitive 3.40 ± 0.38 mM, insulin resistant with BMI <30 kg/m² 3.50 ± 0.31 mM, insulin resistant with BMI >30 kg/m² 3.98 ± 0.34 mM), although there was an increase in LDL cholesterol with increasing BMI and insulin resistance (Fig. 4).

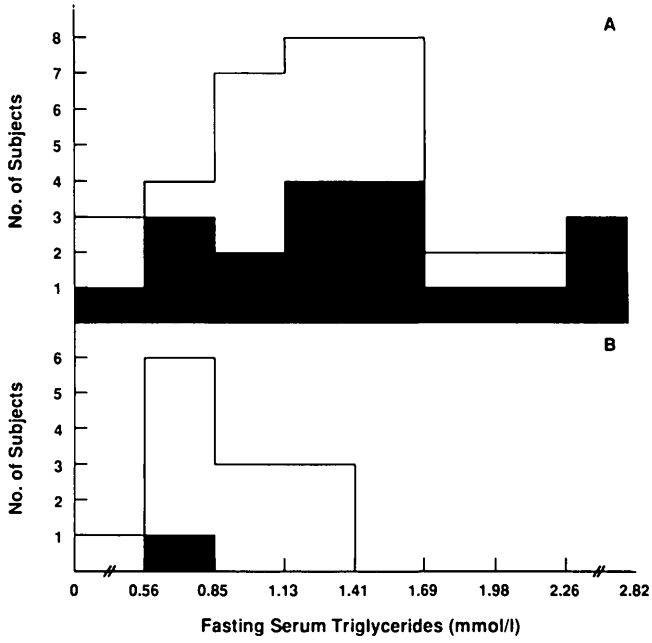

FIG. 3. Distribution of fasting serum triglyceride levels in black non-insulin-dependent diabetic subjects. Frequency of triglyceride levels in insulin-resistant group (*A*) and insulin-sensitive group (*B*). *Solid area*, subjects with body mass index (BMI) >30 kg/m²; *open area*, subjects with BMI <30 kg/m².

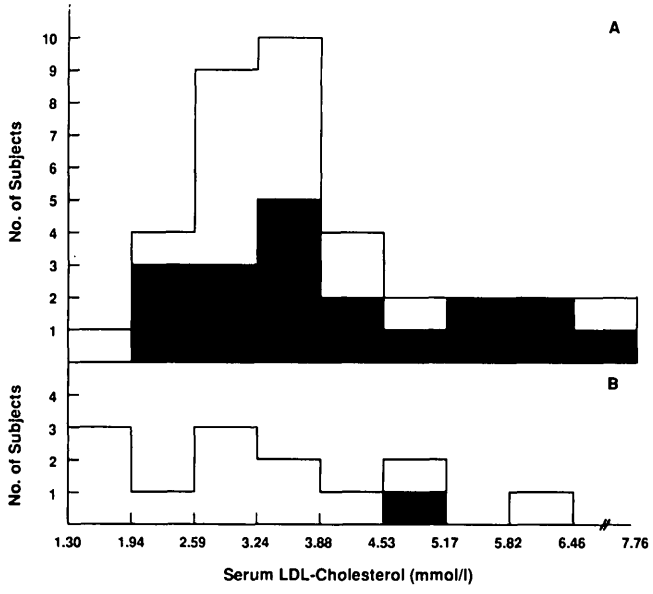

FIG. 4. Distribution of serum low-density lipoprotein (LDL) cholesterol levels in black non-insulin-dependent diabetic subjects. Frequency of LDL cholesterol for insulin-resistant subjects (*A*) and insulin-sensitive subjects (*B*). *Solid area*, subjects with body mass index (BMI) >30 kg/m²; *open area*, subjects with BMI <30 kg/m².

Fasting serum HDL cholesterol levels were lower in the insulin-resistant group with a BMI <30 kg/m² (1.15 mM, $P < 0.05$) compared with the insulin-sensitive group. They were the same for both the insulin-sensitive and insulin-resistant groups with a BMI >30 kg/m² (1.34 ± 0.08 and 1.34 ± 0.11 mM, respectively). This difference was not sustained when HDL cholesterol was analyzed with ANOVA and Scheppe's multiple range test (NS).

The obese insulin-resistant group consisted of a significant preponderance of women. The levels of serum HDL cholesterol in our black NIDDM women were strikingly different from that in our black NIDDM men (Fig. 5). Forty percent of our black NIDDM men had serum HDL cholesterol levels <1.03 mM and only 20% had levels >1.29 mM. In contrast, only 3.5% of our black NIDDM women had serum cholesterol levels <1.03 mM and 55% had values that exceeded 1.29 mM.

CONCLUSIONS

During a detailed study of glucose and lipid metabolism in hyperglycemic black subjects with NIDDM, we measured peripheral insulin sensitivity and baseline lipid profiles. Thirteen of 52 subjects (25%) had normal insulin-stimulated glucose disposal. When matched for BMI of <30 kg/m², 12 of 30 (40%) of our NIDDM subjects were insulin sensitive. Our insulin-resistant NIDDM subjects with a BMI <30 kg/m² had an insulin-stimulated glucose disposal rate that was no different from their obese (BMI >30 kg/m²) counterparts. Although we lowered the plasma glucose level of our subjects to 5.5 mM at the beginning of the insulin clamp, our validation studies of this method showed that peripheral sensitivity to insulin was unchanged whether plasma glucose was lowered slowly overnight or as we had done.

Furthermore, in both insulin-resistant groups, the fasting serum triglyceride levels were significantly higher than those in the insulin-sensitive group.

Although the fasting plasma glucose levels were slightly lower in the insulin-resistant group with a BMI <30 kg/m², the HbA_{1c} levels for all three groups were similar. The groups were similar in age and serum cholesterol and LDL cholesterol levels. HDL cholesterol levels were signficantly lower in the insulin-resistant group with a BMI <30 kg/m². Black women had higher HDL cholesterol levels compared with black men.

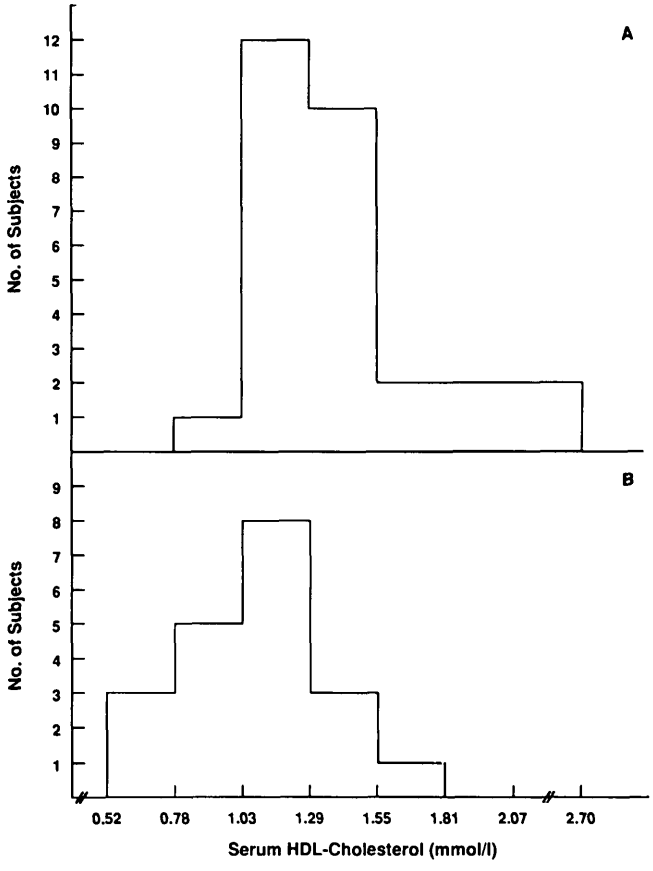

FIG. 5. Serum high-density lipoprotein (HDL) cholesterol levels as function of sex. *A*: distribution of serum HDL cholesterol levels in black non-insulin-dependent diabetic (NIDDM) women ($n = 29$). *B*: distribution in black NIDDM men ($n = 20$).

The emergence of two groups of NIDDM subjects determined by peripheral insulin sensitivity complements the data reported from our laboratory by Banerji and Lebovitz (1,2). They showed that 7 of 16 NIDDM subjects who had been in near-normoglycemic remission while receiving no antidiabetic therapy for several months had normal peripheral and hepatic sensitivity to insulin action. They proposed that NIDDM consists of at least two discrete variants: one insulin sensitive and the other insulin resistant. In assessing why they observed a large insulin-sensitive NIDDM population in their study subjects, whereas previous studies of subjects with NIDDM did not, they hypothesized that the insulin-sensitive form of the disease is a variant of NIDDM in blacks (all 7 of their insulin-sensitive subjects were black). Other investigators have also suggested NIDDM may be more heterogeneous in blacks compared with whites (15). Our data show that an insulin-sensitive form of NIDDM is a common entity in the black NIDDM population accounting for a minimum of 25% of the population. It appears that this is the same insulin-sensitive variant described by Banerji and Lebovitz (1,2) in near-normoglycemic remission NIDDM subjects. This study clearly demonstrates this sensitive variant in a separate randomly selected hyperglycemic NIDDM group. Thus, it is likely that this represents a different disorder than the classically described insulin-resistant NIDDM with its increased cardiovascular disease risk factors and increased prevalence of macrovascular disease.

It is unclear why insulin-sensitive NIDDM has not been described in other populations. Several investigators have commented that an occasional subject with hyperglycemia and NIDDM is insulin sensitive, but the race of the individual is not given. Ginsberg and Rayfield (16) studied the effects of insulin therapy on insulin resistance in NIDDM and noted that in baseline studies 2 of 15 subjects had normal insulin sensitivity, as measured by steady-state plasma glucose techniques. Reaven et al. (17) had 1 of 10 NIDDM subjects with normal insulin sensitivity during an insulin clamp. An analysis of the data of Campbell et al. (18) shows that several of their NIDDM subjects had an ED_{50} of insulin-stimulated glucose uptake that overlapped with the nondiabetic population. The significance of normal insulin sensitivity in the occasional hyperglycemic subject with NIDDM has not been explained previously.

There has been discussion in the literature whether insulin resistance is more impacted by diabetes or the obesity that often accompanies diabetes. Investigations have shown that nondiabetic obese subjects and lean diabetic subjects are insulin resistant (19,20). Several studies suggest that obesity impacts only minimally on insulin resistance in NIDDM (21,22). In any event, we compared the insulin-stimulated glucose disposal in diabetic groups matched for BMI (insulin sensitive vs. insulin resistant with a BMI <30 kg/m^2) and this eliminates any impact that obesity might have on the differences in insulin sensitivity, which we observed in our black NIDDM subjects.

Our laboratory observed that in near-normoglycemic black NIDDM subjects in remission fasting serum triglyceride and LDL cholesterol levels show a significant negative correlation with insulin-stimulated glucose disposal and a positive correlation with fasting plasma insulin levels. Banerji and Lebovitz (3) postulated that the insulin-sensitive euglycemic black NIDDM subject has a lower risk factor profile for the development of macrovascular disease. In our hyperglycemic NIDDM subjects, the insulin-sensitive group had lower fasting serum triglyceride levels, suggesting that this group may be at a lower risk for macrovascular disease than their insulin-resistant counterparts. Although there was no statistical difference between the LDL cholesterol levels in the three groups we studied, there was a trend of increasing LDL cholesterol levels with insulin resistance and increasing BMI. The high HDL cholesterol levels in black NIDDM women, regardless of insulin sensitivity or BMI, may have been a reflection of racial differences in lipid metabolism, because black Americans and black Africans have been reported to have higher HDL cholesterol levels than whites (23,24). These differences in lipids and lipoproteins in black NIDDM subjects may partially explain the lower rates of existing coronary artery disease in newly diagnosed black NIDDM subjects compared with newly diagnosed white NIDDM subjects (25).

Because some of our subjects were on sulfonylureas at the time of study, it is appropriate to question whether normal peripheral sensitivity to insulin in some of our subjects could be explained by an effect of this therapy. If sulfonylurea therapy does improve insulin resistance in NIDDM subjects, it rarely restores insulin sensitivity to normal (26,27). Recently, investigators have used sulfonylurea agents in combination with insulin to elucidate the extra pancreatic effects of these agents independent of glycemic control. In studies by Simonson et al. (28) and Gutniak et al. (29), insulin sensitivity in NIDDM subjects did not change, as measured by the euglycemic insulin clamp, despite the addition of sulfonylurea agents to the insulin regimen and improvement in glycemic control. Thus, they concluded that sulfonylurea agents did not have a major impact on peripheral insulin sensitivity, as measured by the clamp method. Moreover, the same percentage of subjects in each of our three groups was on sulfonylurea therapy, thus implying that the drug would had to have had a selective action on some subjects and not others.

The data presented herein indicate that in our population of hyperglycemic black NIDDM subjects, peripheral insulin resistance is not always a major factor in the pathogenesis of this disorder. Regardless of whether our population is two distinct subgroups or is one continuous group is less important than recognizing that in 40% of the subjects with a BMI <30 kg/m^2 insulin sensitivity is normal. In this group, the primary metabolic defect is most likely the site of the β-cell with insulin deficiency alone leading to hyperglycemia. Moreover, this group has lower fasting serum triglyceride levels and perhaps a tendency for lower fasting serum LDL cholesterol levels. In contrast, for the insulin resistant groups, periph-

eral insulin resistance is a major factor leading to hyperglycemia. Obesity, as measured by a BMI >30 kg/m^2, has little impact on insulin-stimulated glucose disposal. Furthermore, the lipid profile of these insulin-resistant black NIDDM subjects infers that macrovascular complications may be more prevalent in them compared with their insulin-sensitive black NIDDM counterparts.

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