Risk for Metabolic Control Problems in Minority Youth With Diabetes

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OBJECTIVE — We examined and quantified the degree of risk for poor glycemic control and hospitalizations for diabetic ketoacidosis (DKA) among black, Hispanic, and white children and adolescents with diabetes.

RESEARCH DESIGN AND METHODS — We examined ethnic differences in metabolic control among 68 black, 145 Hispanic, and 44 white children and adolescents with type 1 diabetes (mean age 12.9 [range 1–21] years), who were primarily of low socioeconomic status. Clinical and demographic data were obtained by medical chart review. Glycohemoglobins were standardized and compared across ethnic groups. Odds ratios among the ethnic groups for poor glycemic control and hospitalizations for DKA were also calculated.

RESULTS — The ethnic groups were not different with respect to age, BMI, insulin dose, or hospitalizations for DKA, but black children were older at the time of diagnosis than Hispanics ($P < 0.05$) and were less likely to have private health insurance than white and Hispanic children ($P < 0.001$). Black youths had higher glycohemoglobin levels than white and Hispanic youths ($P < 0.001$ after controlling for age at diagnosis). Black youths were also at greatest risk for poor glycemic control (OR = 3.9, relative to whites; OR = 2.5, relative to Hispanics).

CONCLUSIONS — These results underscore and quantify the increased risk for glycemic control problems of lower-income, black children with diabetes. In the absence of effective intervention, these youths are likely to be overrepresented in the health care system as a result of increased health complications related to diabetes.

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Research is beginning to demonstrate that black children with type 1 diabetes have more problems with metabolic control than white children with diabetes (1–3). Metabolic control problems, especially chronic hyperglycemia and recurrent diabetic ketoacidosis (DKA), are likely to lead to increased health complications (4) and reduced quality of life. For example, a study of >200 children and adolescents (2) found that black youths, in contrast to white youths, had poorer glycemic control after adjusting for insulin dosage (higher among blacks), diabetes duration, and socioeconomic status (SES; lower for blacks). Blacks also had significantly more hospitalizations for DKA. Thus, the increased morbidity documented in adult blacks with diabetes (5–8) may begin as early as childhood or adolescence.

Although the above patterns are being documented in black youth, less research is available regarding the degree of risk among youths from other minority groups, particularly Hispanic youths. Studies of adult Mexican Americans with type 2 diabetes have shown more severe hyperglycemia and higher rates of diabetic retinopathy, proteinuria, and end-stage renal disease than whites (9–11), even after accounting for the lower SES of the Mexican-American groups (12). Very little information is available concerning metabolic control of Hispanic youths with type 1 diabetes; however, one study found similar levels of glycosylated hemoglobin A_1 and insulin dose in Hispanic compared with non-Hispanic white youths in Colorado (13).

Identifying and quantifying risk factors for metabolic control problems in minority youths becomes increasingly important given that racial and ethnic minorities are a rapidly growing segment of the U.S. population. Minority individuals under the age of 19 years are expected to represent an estimated 33% of the pediatric population by 2000 (14). Projections indicate that by 2000, Hispanics will be the largest group of minority children and youth in the U.S. (15). Furthermore, minority youths, especially those of lower SES, encounter more health problems and impact disproportionately on the health care system (16,17). This increased morbidity has been linked to poor access to routine care and the resulting increased use of emergency care (18), but relevant biological, behavioral, and psychosocial factors underlying these phenomena need to be identified.

Epidemiological evidence indicates that minority Hispanic and black children have a lower incidence of type 1 diabetes than do non-Hispanic white children (19). For example, the age-adjusted incidence among white children and youths ranges from ~13 to 20/100,000 per year, whereas for blacks it is ~3 to 11/100,000 per year (19–23). Fewer studies have addressed the incidence of type 1 diabetes in Hispanic children, but available results indicate incidence ranges from ~4 to 15/100,000 per year (22,24–25), with relatively higher incidence among children who have a geographic origin of Puerto Rico as compared with Mexico.

Thus, although incidence is somewhat lower among minority Hispanic and black

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Abbreviations: ANCOVA, analysis of covariance; ANOVA, analysis of variance; CMS, Children's Medical Service; DCCT, Diabetes Control and Complications Trial; DKA, diabetic ketoacidosis; SES, socioeconomic status.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

children in the U.S., in light of the fact that minority populations are rapidly growing (14) and that the incidence of type 1 diabetes is increasing (20,26), poor metabolic control in minority youths is a significant health care issue. It therefore becomes imperative to understand the risk factors that influence the health outcomes of minority youths with diabetes and their implications for health care providers. The first steps in this process are documenting and quantifying the degree of risk among these populations. Quantifying risk is also important for health care administration and planning for resource allocation. Therefore, the purpose of the current study was to quantify the degree of risk for poor metabolic control among black, Hispanic, and white children and adolescents with diabetes.

RESEARCH DESIGN AND METHODS

— Medical charts of all children and adolescents (ages 1–21, mean age = 12.9 years; 120 boys and 137 girls) with a primary diagnosis of type 1 diabetes were reviewed over a 1-year period (1992) in a university medical school (n = 167) and a children's hospital (n = 90) diabetes clinic. There were a few cases in the two centers of children who had a primary diagnosis of type 2 diabetes during the year of the study. These cases were not included in the data analyses that follow.

Relevant data were collected from medical charts, including glycohemoglobin, number of hospitalizations for DKA in the previous year (excluding diagnosis), age of diagnosis, current age, insulin dose, height, weight, pubertal status (by Tanner stage), and type of health insurance. BMI was calculated from height and weight. Type of health insurance was classified as private commercial (including health maintenance organizations), Medicaid, or Children's Medical Service (CMS; an insurance program funded by the state of Florida for children with chronic health problems). This classification was used as a marker for SES given that CMS was based on the presence of a chronic health condition and the same income eligibility requirements as Medicaid (annual family income, adjusted for family size, <150% of the federal poverty level).

Glycemic control was measured by glycohemoglobin (GHb). At least one GHb was recorded in the charts of all 257 patients. In cases where more than one GHb was available, we recorded the value taken latest in the calendar year; other clinical data were retrieved from the chart based on the date of this GHb and were utilized in the primary analysis of glycemic control. For cases with more than one GHb, we recorded the value that next preceded the first value; this second GHb was available for 203 patients and was utilized in a secondary analysis of glycemic control. Because six laboratories were used to conduct the assays, GHb was standardized by determining the percentage of the upper limit of the nondiabetic normal ranges for each assay: two assays were used by one site (upper limits of 5.8 and 8.0), and four assays were used by the other site (upper limits of 6.1, 6.8, 8.0, and 8.2). All black children had previous hemoglobin electrophoresis to confirm the validity of this method for the determination of GHb.

For the purposes of the present study, ethnicity was defined as listed in the medical chart, and the categories were then independently confirmed by the nurse educator who was most familiar with the patient and family. The Hispanic population (n = 145) of this clinic was primarily of Cuban descent, with Central American countries also being represented. The black population (n = 68) was predominately African American (n = 54), but the Caribbean islands were also represented (n = 14). There were no differences ($P > 0.05$) in age, age at diagnosis, insulin dose, BMI, or GHb between African-American and Caribbean black children, so these children were combined for the analyses of ethnic effects. It is important to note that although we recognize the diversity within these ethnic groupings, these broad categories were necessary for description and quantification of risks in this study.

There were some differences in patient characteristics between the two sites. Patients from the children's hospital clinic were younger in age (10.3 vs. 14.4 years, $P < 0.001$); had a younger age at diagnosis of diabetes (6.9 vs. 9.6 years, $P < 0.001$); had a lower BMI (19.8 vs. 23.3, $P < 0.001$); had more private (including health maintenance organization) health insurance (62 vs. 21%, $P < 0.001$); were less likely to be black (11 vs. 34%, $P < 0.001$); and had lower GHb levels (160 vs. 174%, $P < 0.02$). These differences indicate variations in the referral patterns for the two settings. However, there were no differences in the treatment philosophy, as indicated by the insulin dose prescribed (0.91 vs. 0.85 U/kg) and the modal treatment regimen, as described below.

Treatment was provided in both settings by interdisciplinary teams led by pediatric endocrinologists, including nurses, dietitians, and referral to pediatric psychologists as clinically indicated. During the time period sampled, the modal treatment regimen prescribed included two daily injections of insulin (mix of regular and intermediate-acting), three or four blood glucose tests per day, an individualized meal plan provided by a registered dietitian (using the American Diabetes Association guidelines, with daily caloric goals dependent on children's age and physical activity levels), and outpatient visits scheduled at least four times per year (or more frequently as clinically indicated). In some cases, a third insulin injection (of regular insulin) was prescribed, particularly for those times when children had very high blood glucose. At the university-based center, the treatment goal for the blood glucose range of children <5 years of age was 80–200 mg/dl; and for children 5 and older, it was 80–150 mg/dl. At the children's hospital clinic, the treatment goal for the blood glucose ranges were similar: for children <5 years of age, the target range was 70–200 mg/dl; and for children 5 and older, it was 70–180 mg/dl. Efforts were made to obtain a GHb at each clinic visit, but GHbs could not be obtained at each clinic visit for every patient during the time period sampled for this study.

To increase the sample size and demographic variability of the study sample, the two sites were combined for the purpose of data analyses. Statistical analyses included one-way analyses of variance and covariance (ANOVAs and ANCOVAs) with Tukey-HSD Range post hoc analyses, χ^2 tests, odds ratios, Pearson product-moment correlations, and independent t tests. For categorical tests of glycemic control, 164% of the upper limit of normal was the cut-point used for classification of good–fair versus poor control. This decision was made based on the fact that 164% was the sample median and corresponded to a GHb of 10% for an assay with an upper limit of normal of 6.1%.

RESULTS

— Patient characteristics are presented in Table 1. There were no differences in current age, insulin dose, BMI, sex, or Tanner stage among the ethnic groups. However, a significant difference in ethnic group was obtained for mean age of diagnosis [$F(2,248) = 7.85$, $P < 0.001$], indicating that blacks were older than Hispanics at diagnosis of diabetes (10.5 vs. 7.9 years, $P < 0.05$). To examine possible effects of

Table 1—*Patient characteristics*

	White	Hispanic	Black
n	44	145	68
Age (years)	12.9 ± 4.8	12.5 ± 5.0	14.2 ± 4.4
Age at diagnosis (years)	8.9 ± 4.2*	7.9 ± 4.8*	10.5 ± 3.9*
Insulin dose (U/kg)	0.84 ± 0.3	0.90 ± 0.3	0.87 ± 0.3
BMI	21.1 ± 4.3	21.7 ± 8.1	23.6 ± 6.6
Tanner stage	3.8 ± 1.4	3.5 ± 1.5	4.0 ± 1.4
GHb	154.1 ± 39.9*	166.1 ± 46.0*	187.3 ± 52.7*
Previous GHb	143.8 ± 34.7†	164.1 ± 44.1†	169.0 ± 50.7†
Number of DKA events (%)	8 (18.2)	22 (15.2)	16 (23.5)
Number of female subjects (%)	28 (64)	75 (52)	34 (50)
Number of subjects with private insurance (%)	29 (66)	52 (36)	9 (13)

Data are means ± SD or *n* (%). GHb is percentage of upper limit of normal range; *n* = 36 white, 115 Hispanic, and 52 black subjects for analysis of previous GHb; *n* = 26 white, 87 Hispanic, and 43 black subjects for analysis of Tanner stage. *$P < 0.001$; †$P < 0.02$.

sex related to this finding, an ethnicity-by-sex ANOVA was conducted and did not reveal a main or interactive effect involving sex. Boys and girls did not differ in mean age (13.1 vs. 12.8 years), age at diagnosis (9.1 vs. 8.4 years), Tanner stage (3.7 vs. 3.6), insulin dose (0.86 vs. 0.89 U/kg), BMI (21.7 vs. 22.4), GHb (166.9 vs. 171.6%), or previous GHb (158.0 vs. 164.4%).

Regarding health insurance, 52% of children had state-provided health insurance (CMS), 14% had Medicaid (without CMS), and 34% had some type of private insurance. In addition, although this clinic population was primarily of lower SES, fewer whites (23%) had state-provided health insurance than Hispanics (48%) or blacks (77%) [$\chi^2(2) = 32.2$, $P < 0.001$]. More whites (66%) had private insurance than Hispanics (36%) or blacks (13%) [$\chi^2(2) = 32.7$, $P < 001$].

Mean GHb for the entire sample was 169.7% of the upper limit of normal. Preliminary analyses indicated that GHb was significantly correlated with age ($r = 0.21$, $P < 0.001$), age at diagnosis ($r = 0.20$, $P < 0.001$), and insulin dose ($r = 0.26$, $P < 0.001$), but not with BMI. A marginal association was observed between GHb and Tanner stage ($r = 0.15$, $P < 0.06$) for the 156 children having Tanner scores recorded in the medical chart (at the time the primary GHb was taken).

A primary purpose of the present study was to determine if any significant differences existed among the ethnic groups with respect to glycemic control as measured by standardized GHb. ANOVA of GHb by ethnic group (black, Hispanic, and white) revealed a significant ethnic group difference [$F(2,254) = 7.61$, $P < 0.0001$]. The post-hoc test indicated that black youths (M = 187.3%) had higher ($P < 0.05$) GHb levels than both Hispanic (M = 166.1%) and white (M = 154.1%) youths. Because the ethnic groups differed with regard to age at diagnosis, the GHb analysis was reexamined with age included as a covariate. This analysis revealed significant effects for both the covariate [$F(1,247) = 6.93$, $P < 0.009$] and the ethnic group [$F(2,247) = 5.15$, $P < 0.006$], with the same pattern of results. ANOVA of the previously recorded GHb ($n = 203$) also showed an ethnicity effect [$F(2,200) = 3.79$, $P < 0.02$], but the post-hoc test revealed higher ($P < 0.05$) GHb levels in both black and Hispanic children compared with white children (169.0 and 164.1 vs. 143.8%).

To examine possible interactive effects of ethnicity with sex, additional ANCOVAs on GHb and previous GHb were conducted, with age at diagnosis as the covariate. Significant effects were again obtained for the covariate ($P < 0.001$) and main effect of ethnicity [$F(2,244) = 5.54$, $P < 0.004$] in the analysis of GHb, but was qualified by a significant sex-by-ethnicity interaction [$F(2, 244) = 3.73$, $P < 0.025$]. Inspection of the mean GHb levels for the groups showed similar glycemic control in white boys and girls (155.2 vs. 153.5%) but higher GHb in Hispanic girls than Hispanic boys (174.9 vs. 156.7%) and higher GHb in black boys than black girls (194.7 vs. 179.8%). However, the ANCOVA on previous GHb indicated only a main effect for ethnicity [$F(2,190) = 3.36$, $P < 0.04$].

Because the ethnic groups differed with regard to insurance status, an ANOVA was also conducted on GHb with ethnicity and insurance status (private versus other insurance) as the independent variables. The results of this analysis revealed main effects for both insurance status [$F(1,251) = 5.32$, $P < 0.02$] and ethnicity [$F(2,251) = 4.28$, $P < 0.015$], but no interaction between insurance status and ethnicity. Children with private insurance had lower GHb than those with state-funded insurance or Medicaid (155.8 vs. 177.1%).

To estimate the degree of risk for poor glycemic control, subjects were dichotomized by GHb level into good–fair (<164% of the upper limit of normal) vs. poor glycemic control subgroups (≥164%). A significant effect for ethnic groups was found [$\chi^2(2) = 13.7$, $P < 0.001$], with 69.1% of blacks, 46.9% of Hispanics, and 36.4% of whites in poor glycemic control. Odds ratios were calculated, and they indicated that blacks were 3.9 times more likely than whites (95% CI 1.8–8.6) and 2.5 times more likely than Hispanics (95% CI 1.4–4.6) to be classified as being in poor glycemic control. Hispanics were not at increased risk relative to whites.

We were also interested in hospitalizations for DKA as an important marker of health status, with substantial associated medical and psychological costs. Analysis of the entire sample failed to show an effect for ethnicity, although 46 children (18% of the sample) had been hospitalized in the previous year for DKA, including 18.2% of white, 15.2% of Hispanic, and 23.5% of black children. Likewise, the odds ratio analyses did not show a significantly increased risk of DKA hospitalizations for black children. However, χ^2 analysis of children from the university-based clinic site revealed significant differences among the ethnic groups [$\chi^2(2) = 5.83$, $P < 0.05$], with 8.7% of white, 9.1% of Hispanic, and 22.4% of black youths having DKA hospitalizations in the previous year.

CONCLUSIONS — In our sample of mostly lower-income, minority youths with diabetes, glycemic control was insufficient compared with recommended levels. The mean levels of GHb were higher than those achieved by youths during the Diabetes Control and Complications Trial (DCCT) (4), reflecting both that intensive insulin regimens were not as commonly prescribed during the time period of the study and that the sample was demographically dif-

ferent from the DCCT sample. The findings of the current study indicated that black youths were in particularly poor glycemic control; they were estimated to have a nearly 4 times greater risk for poor glycemic control than white youths and a 2.5 times greater risk than Hispanic youths. Hispanic youths were not at increased risk relative to whites in the primary analyses and were intermediate between white and black youths. To our knowledge, this report is the first to quantify this relationship relative to Hispanic youths. The findings of generally similar glycemic control in Hispanic relative to white youths is consistent with a previous report (13). Although the analysis of the secondary GHb measure did show significantly increased GHb in both black and Hispanic children, this finding should be interpreted cautiously in light of the smaller subject sample for that analysis.

To increase the size and demographic variability of the study sample, we included a second clinical site that utilized several laboratories for the GHb assay. Although use of one assay would have been preferable, results from different assays were standardized in our study by computing the percentage of the upper limit of normal for each assay. Replication of these findings with one assay would be helpful, but our obtained findings of worse glycemic control in black youths are consistent with previous published reports (1–3).

Patient age, insulin dose, BMI, and Tanner stage did not help to explain the observed differences in GHb between black and white youths. Furthermore, private health insurance status, significantly less frequent among black youths, also did not explain the differences in glycemic control. Particularly poor glycemic control was observed in black boys, a patient subgroup at highest risk for metabolic control problems. This was especially evident in the analysis of the university-based site, which had significantly more black youths than white or Hispanic youths hospitalized for DKA in the preceding year.

Health care system factors may be important to consider in understanding ethnic differences in metabolic control. This is important in the current study because two sites were combined for data analyses, and one of the sites had relatively more black children and adolescents than the other. However, the treatment given patients at both sites was provided by pediatric endocrinologists assisted by an interdisciplinary team of nurses, dietitians, and psychologists. The health care teams at both sites included minority individuals, both black and Hispanic. The modal treatment regimens prescribed, including the insulin doses, were similar. Therefore, it is unlikely that the obtained findings were an artifact of differences in health care system variables, although one such variable, frequency of outpatient visits attended, was not recorded as part of the current study. A previous report on a different patient sample demonstrated that racial differences in metabolic control could not be attributed to frequency of outpatient visits attended (2). However, in the current study, because we did not have data on total contact with the health care team during the year, we cannot rule out the possibility that differences in this variable could account for the observed racial disparity in metabolic control.

Why, then, did this cohort of patients have such high GHb levels, and what could account for the worse glycemic control of black patients? Because this study did not set out to answer this question, we can only speculate as to possible explanations for the observed findings. Besides the impact of sociodemographic risk factors such as poverty and single-parent families, the most likely possibilities are regimen nonadherence and psychosocial variables, such as stress, inadequate health beliefs, and family dysfunction. Recent reviews (27,28) indicate that a number of studies have found relationships between these behavioral and psychosocial factors and metabolic control in youths with diabetes; however, it is important to note that most of the subjects in these studies were white children from two-parent, middle-income families.

Few studies have examined these behavioral and psychosocial variables in minority children from lower-income groups. One retrospective clinical report found that children with repeated hospitalizations for DKA were likely to be black and from lower-income, single-parent families (29). Another study indicated that poor glycemic control of black youths could in part be explained by their lower levels of regimen adherence (30). We have recently shown that poor metabolic control in lower-income black youths was associated with lower levels of regimen adherence, more regimen barriers, greater family stress, and diffusion of responsibilities and less family support for diabetes care (31).

It is also possible that biological factors may be implicated. The mean age of diagnosis of black youths was almost 2 years older than that of Hispanic children and also older than that of white children. We are not aware of this difference being documented in other studies. It may suggest different pathophysiological mechanisms underlying disease onset as well as glycemic control. Other racial differences have been previously documented, such as age of puberty, which is typically lower among black children (32). Research has also shown that some black youths, rather than having type 1 diabetes, have a different form of diabetes more like type 2 diabetes (33). Recent findings from Arkansas (34) and Ohio (35) indicate that an increasing number of obese black youths have type 2 diabetes.

It is possible that some black youths in our sample actually had type 2 diabetes and were incorrectly classified as type 1, which could account for their later age at diagnosis. Furthermore, because of other factors, such as differences in health insurance or access to health care, they may not have come into contact with the health care system until later in their disease course. The current study's findings that black patients were not on average obese, that all required daily insulin, and that they were diagnosed on average 4 years earlier than those determined to have type 2 diabetes in the Arkansas study (34) suggest, but do not prove, that the majority of these patients actually did have type 1 diabetes. Even with some misclassification, it is doubtful that this could amount to more than a small percentage of the minority sample, and thus it is unlikely to have an appreciable impact on the study findings. Further studies of this phenomenon, including measures of C-peptide and islet-cell antibodies, are needed.

In our study, the majority of youths were of lower income status, as defined by their having state-funded insurance or Medicaid. It is important to point out, however, that health insurance of children may not necessarily be an accurate index of family SES. It could be that SES, which is often confounded with ethnicity, is the more important risk factor for metabolic control problems than ethnicity. Both ethnicity and SES may serve as markers or proxies for a variety of underlying independent and interactive risk factors (36). For example, family structure—such as living in single-parent or step-parent versus two-parent households—may be an important moderating variable (2,37). A previous report observed that diabetic children and adolescents who were black, from single-parent

families, and/or without health insurance were more frequently readmitted to the hospital for DKA (29). Similarly, a recent study found that poor glycemic control of black youths was partially explained by their more frequently living in single-parent households and by their lower levels of regimen adherence (30). Although further studies should utilize more precise measurement of SES than health insurance, it is important to note that a previous study found that only 3% of the variance in GHb could be explained by SES (2). The mechanisms by which such demographic, psychosocial, and behavioral factors influence health outcomes need to be further investigated in minority youths with diabetes.

The role of ethnicity in affecting health outcomes is complex, especially because of the tremendous variability within ethnic groups. In the current study, ethnicity was broadly categorized into three primary groups, but it is clear that within any one group there are differences in such factors as SES and geographic origins. The validity of such broad categorization may therefore be somewhat limited, particularly when ethnic classification is made based on medical chart review, as in our study. However, confirmation of the ethnic grouping was made by a member of the health care team who knew the families well over a period of time. Future studies in this area should utilize self-defined ethnic status as well as geographic origins. This is particularly important to consider in light of the international variance in the incidence of type 1 diabetes (19,26).

These results provide further support that inner-city, lower-income, black children and adolescents are at significantly increased risk for glycemic control problems and quantifies the degree of risk. In the absence of effective intervention, these youths are likely to be overrepresented in the health care system as a result of increased diabetes-related complications. Given that poor metabolic control is evident in minority youths, early intervention and more comprehensive services (medical, psychosocial, educational, and behavioral) for this patient population are needed. Further studies should identify biological, behavioral, psychosocial, and cultural factors that result in this increased risk and could therefore be the target of interventions.

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