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Pediatric Diabetes Consortium Type 1 Diabetes (T1D) New Onset (NeOn) Study: Factors Associated with HbA1c Levels One Year after Diagnosis

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Abstract

Objective—To identify determinants of HbA1c levels one year after the diagnosis of type 1 diabetes (T1D) in participants in the Pediatric Diabetes Consortium (PDC) T1D New Onset (NeOn) Study.

Research Design and Methods—Diabetes-specific as well as socioeconomic factors during the first year following diagnosis were analyzed in 857 participants (mean age 9.1 years, 51% female, 66% non-Hispanic White) not participating in an intervention study who had an HbA1c value at 12 months.

Results—Mean ± SD HbA1c at one year was 62 ± 16 mmol/mol (7.8% ± 1.5). In univariate and multivariate analyses, clinical center, non-Hispanic White race, private health insurance, living with both parents, higher frequency of self-monitoring of blood glucose (SMBG), and lower insulin requirements were associated with lower HbA1c concentrations at one year ($p<0.01$). No association was found with gender, age, Tanner stage, BMI, DKA at onset, number of positive autoantibodies or HbA1c at onset, or number of visits to diabetes physician during the first year.

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Conclusions—White race, higher socioeconomic status, two-parent household, more frequent SMBG and low insulin requirements are associated with lower HbA1c concentration one year after the onset of T1D in children.

Keywords

Diabetes Mellitus; Type 1; Hemoglobin A1c

Introduction

Glucose control, as reflected by HbA1c, is one of the strongest predictors of chronic complications in adults (1) and children (2) with type 1 diabetes (T1D) including proliferative retinopathy (3), diabetic nephropathy (4), cardiovascular disease and age-adjusted mortality (5, 6) rates. Poor glucose control during the first few years of T1D in children predicts development of microalbuminuria (7) and retinopathy (8) in adulthood.

Although there are reports on factors associated with HbA1c in patients with established T1D (9–19), there are no published studies on the association of diabetes-specific and socioeconomic factors with HbA1c during the early stages of the disease in a representative sample of children who receive their care at pediatric diabetes centers in the United States.

The Pediatric Diabetes Consortium (PDC) T1D New Onset (NeOn) study aims to improve the care of children with diabetes through sharing of best practices (20). This large and geographically and ethnically diverse cohort of youth with T1D offered us the unique opportunity to study possible factors that may be associated with metabolic control one year after the diagnosis of T1D.

Study Design and Methods

Patients

The PDC T1D NeOn Study enrolled 1,052 patients with T1D between July 2009 and April 2011. The protocol was approved by the Institutional Review Board (IRB) at each of the 7 participating centers. Informed consent was obtained from participants 18 years of age and older and from parents of those less than 18 years of age. Assent was obtained from participants <18 years of age as required by local IRB regulations. To be eligible for enrollment in the study, patients had to be <19 years of age and managed at one of the 7 participating PDC centers within 3 months of diagnosis. A detailed description of PDC and the design of the study have been published previously (20). The analyses reported herein included data from 857 participants; 163 were excluded from this analysis due to no HbA1c measurement available at one year (319–455 days from diagnosis), and 32 were excluded due to participation in an intervention study.

Data Collection

Demographic, socioeconomic status (SES) and clinical characteristic data were collected from medical records and from interviews with the patient and/or parent. Follow-up visits

were completed per usual care and all visits during the first year post-diagnosis were entered in standardized electronic case report forms for the study.

Body mass index (BMI) at diagnosis was computed from the closest height and weight within ± 14 days of diagnosis. BMI percentiles and Z scores adjusted for age and gender were calculated using 2000 CDC population growth chart data (21). If height and/or weight was missing within ± 14 days of diagnosis or if the participant was <2 years of age at the time of diagnosis, BMI percentile was not calculated.

Diabetes ketoacidosis (DKA) was defined according to DCCT criteria of pH <7.3 or HCO3 <15mEq/L and treatment in a healthcare facility (22). Tests for diabetes autoantibodies were included any time prior to diagnosis or within 91 days after diagnosis for insulinoma-associated autoantibody (IA-2) or glutamic acid decarboxylase (GADA) and within 14 days after diagnosis for insulin autoantibody (IAA).

Statistical Analysis

Least squares regression was used to assess the association of baseline (demographic, SES, and clinical) and one-year (total daily insulin dose, SMBG frequency, and number of visits to diabetes management providers [excluding visits during the first 3 weeks following diagnosis, which correspond to initial education in centers where patients are not routinely admitted at diagnosis]) factors with HbA1c at one year from T1D diagnosis. Initial models were constructed for each baseline factor individually. A multivariate model of baseline factors was then constructed using stepwise selection with p-values <0.10 required to be included in the model. Due to multiple comparisons, only factors with p-values <0.01 were considered statistically significant although factors with p-values <0.10 were included in the model to adjust for potential confounding. Interaction terms were tested for all variables included in the final multivariate model with a p-value <0.01 required to be included. A second multivariate model added the one-year factors to the significant baseline factors. Continuous variables were tested for linearity by adding a quadratic term to the model. If significant non-linearity was detected the variable was divided into categories and analyzed as discrete variables. Missing covariates were treated as a separate category for discrete variables and a missing value indicator was added to the model for continuous variables. All reported p-values are two-sided. All analyses were conducted using SAS version 9.3 (SAS Institute, Cary, NC).

RESULTS

At the time of diagnosis, the mean ± SD age of the 857 participants was 9.1 ± 4.1 years, 51% were female, 66% were Non-Hispanic White, and 68% had private health insurance. Mean ± SD HbA1c was 102 mmol/mol ± 25 (11.4% ± 2.3), 19% of participants were overweight or obese (BMI ≥85th percentile for age and gender) and 33% had DKA. Of the 510 participants who were tested for all three autoantibodies, 95% were positive for at least one (Table 1).

One year after diagnosis of T1D, mean ± SD HbA1c was 62 mmol/mol ± 16 (7.8% ± 1.5) with a median insulin dose of 0.6 units/kg/day (interquartile range [IQR] 0.5 to 0.8). The

median number of self-reported SMBG tests per day was 5 (IQR 4 to 6) and the median number of visits to diabetes providers during the first year of T1D was 4 (IQR 4 to 5). In cases where a blood glucose meter download was available (n=469), the median (and IQR) number of SMBG tests per day was similar to self-report. Insulin pump therapy was being used by 34% of participants at one year and 59% of all participants were taking three or more injections of insulin per day (Table 2).

Several baseline (i.e., at diagnosis) characteristics were significantly associated with lower HbA1c at one year in univariate analyses: clinical site, white race, private health insurance, higher household income, higher parent education level, living with both parents, and absence of DKA at diagnosis (p-value <0.01 for each, Table 3). In the multivariate analysis of the baseline characteristics, clinical site, race, health insurance status and family structure remained significant. The effects of family income and parent education were confounded with the other SES factors so that possible independent effects of these factors cannot be confirmed nor ruled out. In the multivariate analysis, there was no detectable association with gender, age, Tanner stage, BMI, number of positive anti-islet autoantibodies, DKA at onset or HbA1c at onset. Positivity for each of the autoantibodies, i.e., GADA, IAA or IA-2, was not associated with one-year HbA1c (p-value >0.10; data not shown).

In the multivariate model for one-year factors, adjusting for the significant baseline factors indicated above, lower one-year HbA1c was associated with higher frequency of SMBG testing and a lower total daily insulin dose per kg of body weight (both p-value <0.001; Table 4, Figure 1).

DISCUSSION

This study describes a large cohort of racially diverse children from seven pediatric diabetes treatment centers across the United States and reports characteristics associated with glycemic control one year following diagnosis of T1D. We observed that race/ethnicity, socioeconomic factors, frequency of SMBG and lower dose of insulin were independently associated with HbA1c one year after diagnosis of T1D in children.

There have been few previous studies analyzing factors associated with HbA1c at the early stages of T1D in children. In a study of 275 European children, Mortensen et al. identified ethnic minority, higher HbA1c at diagnosis and positivity for GAD65 autoantibodies as predictors of higher HbA1c one year after diagnosis (23). Our study found that the association between race/ethnicity and one-year HbA1c was independent of SES, frequency of SMBG, pump use and insulin dose. Higher levels of HbA1c have been previously reported in African-American adults with and without diabetes (24) and minority youth with diabetes (9) compared with their white counterparts. Whether this difference reflects higher glycemia, or higher rates of glycosylation, is currently unknown (25, 26). It is also possible that the distribution of socioeconomic factors is narrower for minorities thus not allowing studies to completely rule out confounding. It is concerning, however, that African-Americans also have higher mortality rates and risk of diabetic complications than whites (27).

Several socioeconomic factors were significant in univariate analysis, but because of colinearity, were not included in the multivariate model. Health insurance, the SES factor that was included in the multivariate analysis, was independently associated with HbA1c at one year. Our analysis does not allow us to completely rule out a possible independent effect of family income, structure or education. Socioeconomic factors were not analyzed in Mortensen's study (23) but analyses in established, long-duration T1D have demonstrated a strong impact of health insurance type (28, 29), family structure (12–14, 18), education (18), family income (19, 30) and other social circumstances (e.g. immigration) (17, 31, 32) on glucose control. Although health insurance type may impact the choice of insulin delivery modality (28), in our analysis both factors were analyzed as independent variables associated with HbA1c.

Higher frequency of SMBG was associated with lower one-year HbA1c after adjustment for the other variables in the model. The association between frequency of SMBG and glucose control has been reported in patients with established T1D in several (15, 16, 18, 19, 33–35) but not all (14, 36) studies. The disagreement among studies might be explained by the strong and complex relationships between SMBG and other factors, such as use of insulin pumps or family structure and support (14), which also affect HbA1c. Our and others' studies (18) suggest that frequency of SMBG is associated with HbA1c independently of those factors, and thus frequent BG monitoring should be encouraged.

We found that lower dose of insulin was independently associated with HbA1c at one year. In patients who retain residual endogenous insulin secretion, the dose of exogenous insulin adjusted for body weight is a measure of both beta-cell function and insulin resistance. In this fashion, insulin dose has been used to define the partial remission period (37, 38). The long known association between greater beta-cell function and lower HbA1c (39) provides the rationale for trials that aim to preserve beta-cell function and prolong the partial remission period in an effort to prevent complications of diabetes.

Clinical center was independently associated with HbA1c concentrations after adjustment for other variables. Differences in the therapeutic approaches by different centers may explain this finding, highlighting the importance of networks that promote sharing clinical practices among centers.

In our study, age was not associated with HbA1c. This is contrary to studies in established T1D that have found that age, and in particular late adolescence, is associated with higher HbA1c (11, 14, 16, 31). A likely explanation for this discrepancy is that we studied children and adolescents only one year after onset and therefore before the deterioration in compliance with diabetes tasks and glucose control that are typical during adolescence (40, 41). Alternatively, the challenges of managing diabetes in teenagers may have been offset by the accelerated rate of autoimmune destruction of beta-cells in younger patients, compared with adolescents. Mortensen et al. (23) also did not find an association with age.

Several studies have demonstrated that the influence of glucose control on risk of complications persists for many years (5). HbA1c in the first five years of childhood-onset T1D predicts microalbuminuria (7) and retinopathy (8) on follow-up. HbA1c at onset of

diabetes correlates with glucose control in subsequent years (36, 42) and predicted microalbuminuria 11 years later (6). Our study sought to understand the factors associated with metabolic control one year after T1D diagnosis in children. By addressing characteristics that are associated with HbA1c at the early stages of disease, long-term chronic complications could be prevented in patients with childhood-onset T1D.

Although the list of predictors that our study was able to assess was comprehensive, one limitation is that we did not explore psychological factors. Previous literature indicates that diabetes-specific conflict (15, 34), self-care behavior, eating disturbances, depression (11), and family behavior factors (12, 43) influence glucose control in established T1D likely through their effect on adherence to treatment (40). It is likely that these factors also affect HbA1c at the early stages of disease. Missing data for some of the factors studied at one year of onset is another limitation of the study.

In summary, we studied a large sample of children with T1D and analyzed a comprehensive series of variables at diagnosis and during the first year of T1D. We found that sociodemographic factors, SMBG frequency and low insulin requirements are associated with HbA1c at one year of childhood-onset T1D. Further investigation is needed to identify potentially modifiable factors that mediate these associations.

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Abbreviations

HbA1c	Hemoglobin A1c
T1D	type 1 diabetes
PDC	Pediatric Diabetes Consortium
NeOn	New Onset Study
SMBG	self-monitoring of blood glucose
BMI	body mass index
DKA	diabetic ketoacidosis
IRB	Institutional Review Board
SES	socioeconomic status
CDC	Centers for Disease Control and Prevention
DCCT	The Diabetes Control and Complications Trial
IA-2	insulinoma-associated autoantibody
GADA	glutamic acid decarboxylase

IAA	insulin autoantibody
SD	standard deviation
BG	blood glucose

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References

1. The Diabetes Control and Complications Trial Research Group. The Effect of Intensive Treatment of Diabetes on the Development and Progression of Long-Term Complications in Insulin-Dependent Diabetes Mellitus. N Engl J Med. 1993; 329:977–986. [PubMed: 8366922]
2. Amin R, Widmer B, Prevost AT, et al. Risk of microalbuminuria and progression to macroalbuminuria in a cohort with childhood onset type 1 diabetes: prospective observational study. BMJ. 2008; 336:367–701.
3. Skrivarhaug T, Fosmark DS, Stene LC, et al. Low cumulative incidence of proliferative retinopathy in childhood-onset type 1 diabetes: a 24-year follow-up study. Diabetologia. 2006; 49:2281–2290. [PubMed: 16955208]
4. Skrivarhaug T, Bangstad H-J, Stene LC, Sandvik L, Hanssen KF, Joner G. Low risk of overt nephropathy after 24 yr of childhood-onset type 1 diabetes mellitus (T1DM) in Norway. Pediatr Diabetes. 2006; 7:239–246. [PubMed: 17054444]
5. Stadler M, Auinger M, Anderwald C, et al. Long-Term Mortality and Incidence of Renal Dialysis and Transplantation in Type 1 Diabetes Mellitus. J Clin Endocrinol Metab. 2006; 91:3814–3820. [PubMed: 16882744]
6. Giordano C, Amato MC, Ciresi A, et al. Predictors of microvascular complications in type 1 diabetic patients at onset: The role of metabolic memory. Eur J Intern Med. 2011; 22:266–274. [PubMed: 21570646]

7. Rudberg S, Ullman E, Dahlquist G. Relationship between early metabolic control and the development of microalbuminuria--a longitudinal study in children with type 1 (insulin-dependent) diabetes mellitus. Diabetologia. 1993; 36:1309–1314. [PubMed: 8307261]
8. Svensson M, Eriksson JW, Dahlquist G. Early Glycemic Control, Age at Onset, and Development of Microvascular Complications in Childhood-Onset Type 1 Diabetes: A population-based study in northern Sweden. Diabetes Care. 2004; 27:955–962. [PubMed: 15047655]
9. Petitti DB, Klingensmith GJ, Bell RA, et al. Glycemic Control in Youth with Diabetes: The SEARCH for Diabetes in Youth Study. J Pediatr. 2009; 155:668–672. e1–3. [PubMed: 19643434]
10. Paris CA, Imperatore G, Klingensmith G, et al. Predictors of Insulin Regimens and Impact on Outcomes in Youth with Type 1 Diabetes: The SEARCH for Diabetes in Youth Study. J Pediatr. 2009; 155:183–189. e1. [PubMed: 19394043]
11. Helgeson VS, Siminerio L, Escobar O, Becker D. Predictors of Metabolic Control among Adolescents with Diabetes: A 4-Year Longitudinal Study. J Pediatr Psychol. 2009; 34:254–270. [PubMed: 18667479]
12. Meunier J, Dorchy H, Luminet O. Does family cohesiveness and parental alexithymia predict glycaemic control in children and adolescents with diabetes? Diabet Metab. 2008; 34:473–481.
13. Swift EE, Chen R, Hershberger A, Holmes CS. Demographic risk factors, mediators, and moderators in youths' diabetes metabolic control. Ann Behav Med. 2006; 32:39–49. [PubMed: 16827628]
14. Urbach SL, LaFranchi S, Lambert L, Lapidus JA, Daneman D, Becker TM. Predictors of glucose control in children and adolescents with type 1 diabetes mellitus. Pediatr Diabetes. 2005; 6:69–74. [PubMed: 15963032]
15. Moreland EC, Tovar A, Zuehlke JB, Butler DA, Milaszewski K, Laffell LM. The impact of physiological, therapeutic and psychosocial variables on glycemic control in youth with type 1 diabetes mellitus. J Pediatr Endocrinol Metab. 2004; 17:1533–1544. [PubMed: 15570991]
16. Haller MJ, Stalvey MS, Silverstein JH. Predictors of Control of Diabetes: Monitoring May be the Key. J Pediatr. 2004; 144:660–661. [PubMed: 15127007]
17. Scottish Study Group for the Care of the Young Diabetic. Factors Influencing Glycemic Control in Young People With Type 1 Diabetes in Scotland: A population-based study (DIABAUD2). Diabetes Care. 2001; 24:239–244. [PubMed: 11213872]
18. Levine BS, Anderson BJ, Butler DA, Antisdel JE, Brackett J, Laffel LM. Predictors of glycemic control and short-term adverse outcomes in youth with type 1 diabetes. J Pediatr. 2001; 139:197–203. [PubMed: 11487743]
19. Rosilio M, Cotton JB, Wieliczko MC, et al. Factors associated with glycemic control. A cross-sectional nationwide study in 2,579 French children with type 1 diabetes The French Pediatric Diabetes Group. Diabetes Care. 1998; 21:1146–1153. [PubMed: 9653610]
20. Pediatric Diabetes Consortium. The Pediatric Diabetes Consortium: Improving Care of Children with Type 1 Diabetes Through Collaborative Research. Diabetes Technol Ther. 2010; 12:685–688. [PubMed: 20687862]
21. Kuczmarski R, Ogden C, Grummer-Strawn L, et al. CDC growth charts: United States. Adv Data. 2000; 314:1–27. [PubMed: 11183293]
22. The Diabetes Control and Complications Trial Research Group. The Diabetes Control and Complications Trial (DCCT) Design and methodologic considerations for the feasibility phase. Diabetes. 1986; 35:530–545. [PubMed: 2869996]
23. Mortensen HB, Swift PG, Holl RW, et al. Multinational study in children and adolescents with newly diagnosed type 1 diabetes: association of age, ketoacidosis, HLA status, and autoantibodies on residual beta-cell function and glycemic control 12 months after diagnosis. Pediatr Diabetes. 2010; 11:218–226. [PubMed: 19708904]
24. Ziemer DC, Kolm P, Weintraub WS, et al. Glucose-Independent, Black-White Differences in Hemoglobin A1c Levels A Cross-sectional Analysis of 2 Studies. Ann Intern Med. 2010; 152:770–777. [PubMed: 20547905]
25. Selvin E, Steffes MW, Ballantyne CM, Hoogeveen RC, Coresh J, Brancati FL. Racial Differences in Glycemic Markers: A Cross-sectional Analysis of Community-Based Data. Ann Intern Med. 2011; 154:303–309. [PubMed: 21357907]

26. Kamps JL, Hempe JM, Chalew SA. Racial Disparity in A1C Independent of Mean Blood Glucose in Children With Type 1 Diabetes. Diabetes Care. 2010; 33:1025–1027. [PubMed: 20185743]
27. Secrest AM, Becker DJ, Kelsey SF, LaPorte RE, Orchard TJ. Cause-Specific Mortality Trends in a Large Population-Based Cohort With Long-Standing Childhood-Onset Type 1 Diabetes. Diabetes. 2010; 59:3216–3222. [PubMed: 20739685]
28. Wintergerst KA, Hinkle KM, Barnes CN, Omoruyi AO, Foster MB. The impact of health insurance coverage on pediatric diabetes management. Diabetes Res Clin Pract. 2010; 90:40–44. [PubMed: 20630611]
29. Beck JK, Lewis TV, Logan KJ, Harrison DL, Gardner AW, Copeland KC. Intensive vs conventional insulin management initiated at diagnosis in children with diabetes: Should payer source influence the choice of therapy? Pediatr Diabetes. 2009; 10:368–373. [PubMed: 19522746]
30. Gallegos-Macias AR, Macias SR, Kaufman E, Skipper B, Kalishman N. Relationship between glycemic control, ethnicity and socioeconomic status in Hispanic and white non-Hispanic youths with type 1 diabetes mellitus. Pediatr Diabetes. 2003; 4:19–23. [PubMed: 14655519]
31. Wilf-Miron R, Peled R, Yaari E, et al. Disparities in diabetes care: role of the patient's socio-demographic characteristics. BMC Public Health. 2010; 10:729. [PubMed: 21108780]
32. Rosenbauer J, Dost A, Karges B, et al. Improved Metabolic Control in Children and Adolescents With Type 1 Diabetes: A trend analysis using prospective multicenter data from Germany and Austria. Diabetes Care. 2012; 35:80–86. [PubMed: 22074726]
33. Ziegler R, Heidtmann B, Hilgard D, et al. Frequency of SMBG correlates with HbA1c and acute complications in children and adolescents with type 1 diabetes. Pediatr Diabetes. 2011; 12:11–17. [PubMed: 20337978]
34. Hilliard ME, Guilfoyle SM, Dolan LM, Hood KK. Prediction of Adolescents' Glycemic Control 1 Year After Diabetes-Specific Family Conflict. The Mediating Role of Blood Glucose Monitoring Adherence. Arch Pediatri Adolesc Med. 2011; 165:624–629.
35. Dorchy H, Roggemans MP, Willems D. Glycated hemoglobin and related factors in diabetic children and adolescents under 18 years of age: a Belgian experience. Diabetes Care. 1997; 20:2–6. [PubMed: 9028684]
36. Shalitin S, Phillip M. Which factors predict glycemic control in children diagnosed with type 1 diabetes before 6.5 years of age? Acta Diabetol. 2012; 49:355–362. [PubMed: 21866397]
37. Muhammad BJ, Swift PG, Raymond NT, Botha JL. Partial remission phase of diabetes in children younger than age 10 years. Arch Dis Child. 1999; 80:367–369. [PubMed: 10086946]
38. Kordonouri O, Danne T, Enders I, Weber B. Does the long-term clinical course of type I diabetes mellitus differ in patients with prepubertal and pubertal onset? Results of the Berlin Retinopathy Study. Eur J Pediatr. 1998; 157:202–207. [PubMed: 9537486]
39. Sochett EB, Daneman D, Clarson C, Ehrlich RM. Factors affecting and patterns of residual insulin secretion during the first year of type 1 (insulin-dependent) diabetes mellitus in children. Diabetiologia. 1987; 30:453–459.
40. Anderson B, Ho J, Brackett J, Finkelstein D, Laffel L. Parental involvement in diabetes management tasks: Relationships to blood glucose monitoring adherence and metabolic control in young adolescents with insulin-dependent diabetes mellitus. J Pediatr. 1997; 130:257–265. [PubMed: 9042129]
41. Jacobson AM, Hauser ST, Wolfsdorf JI, et al. Psychologic predictors of compliance in children with recent onset of diabetes mellitus. J Pediatr. 1987; 110:805–811. [PubMed: 3572636]
42. Jorde R, Sundsfjord J. Intra-individual variability and longitudinal changes in glycaemic control in patients with Type 1 diabetes mellitus. Diabet Med. 2000; 17:451–456. [PubMed: 10975214]
43. Lewin AB, Heidgerken AD, Geffken GR, et al. The Relation Between Family Factors and Metabolic Control: The Role of Diabetes Adherence. J Pediatr Psychol. 2006; 31:174–183. [PubMed: 16467317]

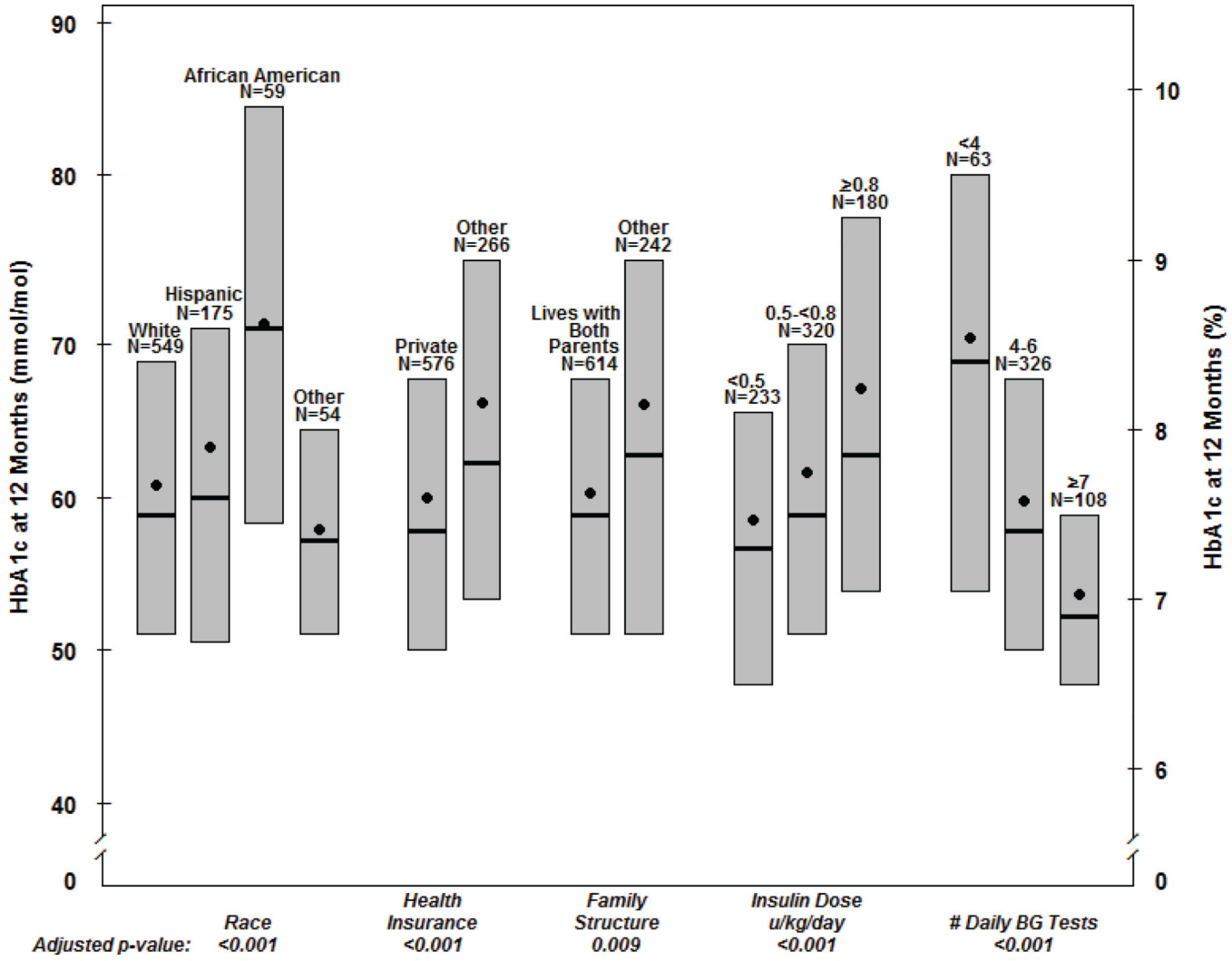


Figure 1. Factors Associated with HbA1c at One Year (N=857a)

Bottom and top of each box denote the 25th and 75th percentiles. Horizontal line inside each box denotes the median and the dot denotes the mean.

a. Number of participants with missing data: race/ethnicity (20), health insurance (15), family structure (1), insulin dose (124), # daily BG tests (360).

Table 1

Participant Characteristics at Diagnosis (N=857[a])

	%
Gender: Female	51%
Age (years)	
<5	19%
5–<12	56%
12–<19	25%
Mean ± SD	9.1 ± 4.1
Range	0.7 − 18.8
Tanner Stage[b]	
I	67%
II	11%
III	7%
IV	7%
V	7%
BMI Percentile[c]	
<85th	81%
85th–<95th (Overweight)	10%
≥ 95th (Obese)	9%
Race/Ethnicity	
White	66%
Hispanic or Latino	21%
Black/African American	7%
Other/More than one race	6%
Health Insurance	
Private	68%
Military/CHP/Medicaid/Medicare	30%
None	2%
Parent Education	
High school or less	32%
AA/BS/BA	44%
MS/MA or professional degree	23%
Family Income	
<$25,000	13%
$25,000−$49,999	19%
$50,000−$74,999	17%
$75,000−$99,999	14%
≥ $100,000	37%
Family Structure	
Lives with both parents	72%
HbA1c mmol/mol (%)	

	%
<86 (<10%)	28%
86−<119 (10–<13%)	45%
≥ 119 (≥ 13%)	27%
Mean ± SD	102 ± 25 (11.4 ± 2.3%)
Range	37−180 (5.5−18.6%)
Number of Positive Autoantibodies[d]	
0	5%
1	20%
2	39%
3	35%
DKA	33%

[a] Number of participants with missing data: Tanner stage (412), BMI percentile (361), race/ethnicity (20), health insurance (15), parent education (163), family income (280), family structure (1), HbA1c (39), DKA (27).

[b] Imputed as stage 1 for girls < 8 years of age and boys < 10 years of age.

[c] BMI percentile not calculated for n=36 participants less than 2 years of age.

[d] Limited to those tested for all three auto antibodies (510).

Table 2

Participant Characteristics at One Year (N=857[a])

	%
HbA1c mmol/mol (%)	
<53 (<7%)	29%
53–<64 (7–<8%)	34%
64–<75 (8–<9%)	20%
≥ 75 (≥ 9%)	17%
Mean ± SD	62 ± 16 (7.8 ± 1.5%)
Range	31–140 (5.0–15.0%)
Insulin Delivery	
Pump	34%
1–2 injections/day	7%
3–4 injections/day	45%
5 or more injections/day	14%
Median (IQR)[b]	4 (3 to 4)
Range[b]	1–12
Insulin Dose (units/kg/day)	
<0.5	32%
0.5–<0.8	44%
≥ 0.8	25%
Median (IQR)	0.6 (0.5 to 0.8)
Range	0.02–4.6
SMBG (# tests/day)	
1–3	13%
4–6	66%
≥ 7	22%
Median (IQR)	5 (4 to 6)
Range	2–18
Visits to Diabetes Team Providers through 1 Year	
1–3	12%
4–5	76%
≥ 6	11%
Median (IQR)	4 (4 to 5)
Range	1–9

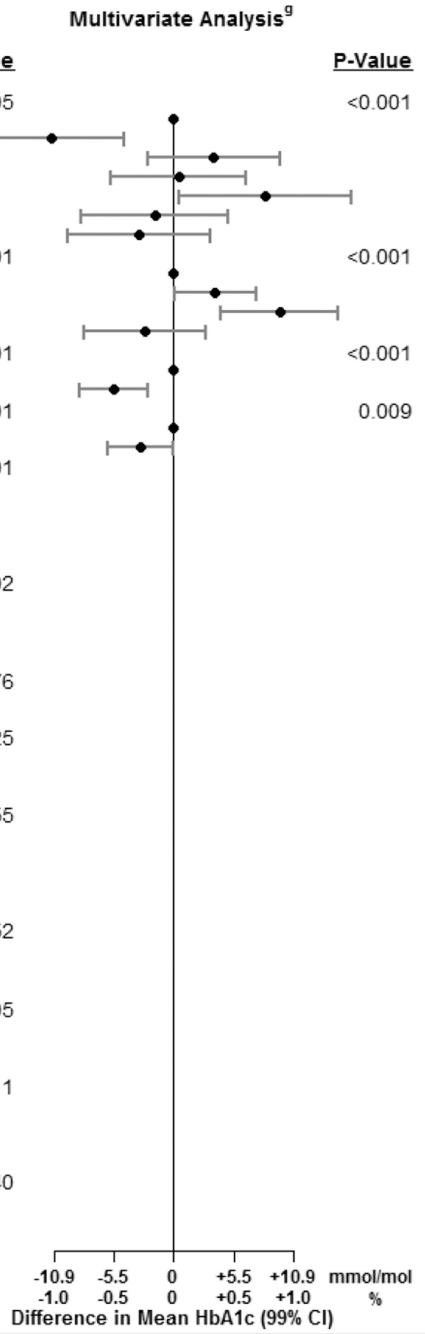
[a] Number of participants with missing data: number of injections (305), insulin dose (124), blood glucose monitoring (360).

[b] Limited to those not on an insulin pump (552).

Table 3

Factors at Diagnosis Associated with HbA1c at One Year (N+857[a])

	N	HbA1c Mean ± SD mmol/mol	%	Univariate Analysis P-Value	Multivariate Analysis[g] P-Value
Overall	857	62 ± 16	7.8 ± 1.5		
Clinical Site				0.005	<0.001
A	53	65 ± 17	8.1 ± 1.6		
B	127	53 ± 13	7.0 ± 1.2		
C	237	66 ± 15	8.2 ± 1.3		
D	179	62 ± 17	7.9 ± 1.5		
E	46	69 ± 21	8.5 ± 1.9		
F	109	59 ± 15	7.6 ± 1.4		
G	106	58 ± 13	7.5 ± 1.2		
Race/Ethnicity				< 0.001	<0.001
White (non-Hispanic)	549	60 ± 15	7.7 ± 1.3		
Hispanic or Latino	175	63 ± 19	7.9 ± 1.7		
Black/African American	59	71 ± 20	8.6 ± 1.8		
Other/More than One Race	54	58 ± 11	7.4 ± 1.0		
Health Insurance				<0.001	<0.001
Other	266	66 ± 18	8.2 ± 1.6		
Private	576	60 ± 14	7.6 ± 1.3		
Family Structure				<0.001	0.009
Other[b]	242	66 ± 19	8.2 ± 1.8		
Living with Both Parents	614	60 ± 14	7.6 ± 1.3		
Household Income[c]				<0.001	
<$25,000	75	66 ± 21	8.2 ± 1.9		
$25,000-$49,999	111	67 ± 17	8.3 ± 1.6		
$50,000-$74,999	97	64 ± 16	8.0 ± 1.5		
$75,000-$99,999	81	60 ± 12	7.6 ± 1.1		
≥ $100,000	213	59 ± 13	7.5 ± 1.2		
Parent Education[c]				0.002	
High school or Less	225	63 ± 17	7.9 ± 1.6		
AA	103	63 ± 14	7.9 ± 1.3		
BS/BA	204	62 ± 16	7.8 ± 1.4		
MS/MA or PhD	162	58 ± 13	7.4 ± 1.2		
Gender				0.76	
Female	436	62 ± 16	7.8 ± 1.5		
Male	421	61 ± 15	7.8 ± 1.4		
Age (years)[d]				0.25	
<5	165	62 ± 12	7.9 ± 1.1		
5-<12	481	61 ± 14	7.8 ± 1.3		
12-<19	211	61 ± 21	7.7 ± 2.0		
Tanner Stage[c,e]				0.55	
I	299	60 ± 14	7.7 ± 1.3		
II	51	61 ± 18	7.8 ± 1.6		
III	32	56 ± 17	7.3 ± 1.5		
IV	31	60 ± 21	7.6 ± 2.0		
V	32	60 ± 16	7.6 ± 1.5		
BMI Percentile[d]				0.52	
<85th	401	60 ± 15	7.6 ± 1.4		
85th - <95th (Overweight)	52	63 ± 15	7.9 ± 1.4		
≥ 95th (Obese)	43	62 ± 14	7.8 ± 1.3		
DKA at Diagnosis				0.005	
No	555	60 ± 15	7.7 ± 1.4		
Yes	275	64 ± 16	8.0 ± 1.5		
HbA1c at Diagnosis[d] mmol/mol (%)				0.11	
<86 (<10%)	226	60 ± 14	7.7 ± 1.3		
86-<119 (10-<13%)	368	61 ± 15	7.8 ± 1.4		
≥ 119 (≥ 13%)	224	62 ± 18	7.8 ± 1.6		
Number of Positive Autoantibodies[c,f]				0.40	
0	27	60 ± 21	7.6 ± 1.9		
1	102	59 ± 12	7.5 ± 1.1		
2	200	62 ± 17	7.8 ± 1.6		
3	181	61 ± 15	7.7 ± 1.4		



[a.] Number of participants with missing data: race/ethnicity (20), health insurance (15), family structure (1), household income (280), parent education (163), Tanner stage (412), BMI percentile (361), DKA (27) and HbA1c (39).

[a.] "Other" could be lives with mother, lives with father, splits time with mother and father, lives with legal guardian who is not parent, lives away at school or other.

[c.] Analyzed as an ordinal variable.

[d.] Analyzed as a continuous variable. Categories are for display purposes in this table.

[e.] Missing values imputed as stage I for girls < 8 years of age and boys < 10 years of age.

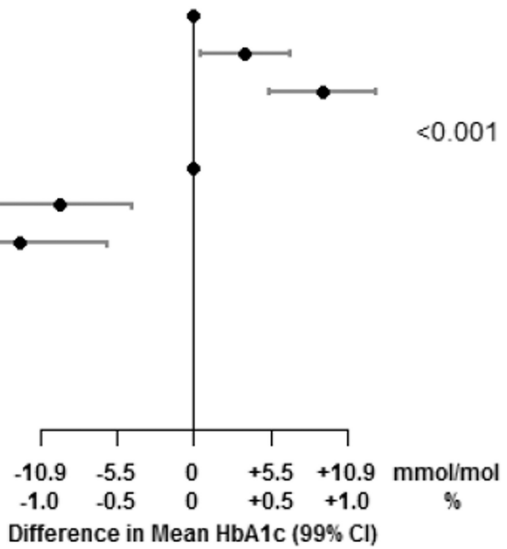
[f.] Limited to those tested for all three auto antibodies (510).

[g.] The multivariate model contains all factors with an adjusted p-value <0.10 to account for potential confounding, but only p-values <0.01 are considered statistically significant in this analysis. Factors with blank entries in the multivariate columns were excluded from the model because p ≥0.10.

Table 4

Factors at One Year Associated with HbA1c at One Year (N±857[a])

		Univariate Analysis			Multivariate Analysis[b]
		HbA1c Mean ± SD			
	N	mmol/mol	%	P-Value	P-Value
Overall	857	62 ± 16	7.8 ± 1.5		
Insulin Dose (u/kg/day)[c]				<0.001	<0.001
<0.5	233	58 ± 15	7.5 ± 1.4		
0.5-0.8	320	61 ± 15	7.8 ± 1.3		
≥ 0.8	180	67 ± 18	8.2 ± 1.7		
SMBG (# tests/day)				<0.001	<0.001
1-3	63	70 ± 23	8.5 ± 2.1		
4-6	326	59 ± 15	7.6 ± 1.3		
≥ 7	108	53 ± 10	7.0 ± 0.9		
Visits to Diabetes Team Providers through 1 Year[c,d]				0.10	
1-3	106	63 ± 20	8.0 ± 1.8		
4-5	654	61 ± 15	7.8 ± 1.4		
≥ 6	97	60 ± 15	7.6 ± 1.4		



[a] Number of participants with missing data: insulin dose (124) and blood glucose monitoring (360).

[b] Also adjusted for all factors (p<0.10) from diagnosis model in Table 3.

[c] Analyzed as continuous variables. Categories are for display purposes in this table.

[d] Visits during the first 3 weeks following diagnosis were excluded due to differences among centers in initial diabetes education.