

# Factors Influencing Glycemic Control in Young People With Type 1 Diabetes in Scotland

## A population-based study (DIABAUD2)

SCOTTISH STUDY GROUP FOR THE CARE  
OF THE YOUNG DIABETIC

**OBJECTIVE** — To evaluate differences in HbA<sub>1c</sub> concentrations between centers and to assess the factors associated with glycemic control in young people with type 1 diabetes in Scotland.

**RESEARCH DESIGN AND METHODS** — Data on 1,755 patients (94% of those registered) were collected from 18 centers providing care to children <15 years of age. At every clinic visit, a duplicate HbA<sub>1c</sub> sample was measured in a reference laboratory, and clinical information was collected prospectively.

**RESULTS** — Average HbA<sub>1c</sub> concentration was 9.1% (range 5.0–15.0). The following significant associations with HbA<sub>1c</sub> level were identified: age, insulin regimen, BMI, season, social circumstances, and family history. HbA<sub>1c</sub> concentrations were significantly worse in older children (age 10–15 years 9.5% vs. other ages 8.8%,  $P < 0.001$ ), those using two injections per day (2/day 9.1% vs. 3/day 8.8%,  $P < 0.01$ ), children without both parents at home (9.4 vs. 9.0%,  $P < 0.001$ ), a sibling with diabetes (9.7% vs. no family history 9.1%,  $P < 0.001$ ). HbA<sub>1c</sub> concentration ranged from 8.1 to 10.2% between centers, after adjustment for factors associated with poor HbA<sub>1c</sub> ( $P < 0.001$ ).

**CONCLUSIONS** — The overall glycemic control of diabetic young people in Scotland is equivalent to a Diabetes Control and Complications Trial HbA<sub>1c</sub> concentration of 8.7%, placing the majority at a high risk of the complications of diabetes in adulthood. Although factors were significantly associated with poor HbA<sub>1c</sub>, adjustment for these did not explain the differences between centers. We suggest that factors not analyzed in DIABAUD2 (e.g., deployment of resources, organization of the clinical structure, strategies of care, and clinic philosophy) are the determinants of HbA<sub>1c</sub>. We speculate that the style of utilization of optimum resources is the key to achieving good glycemic control.

*Diabetes Care* 24:239–244, 2001

Since 1981, the Scottish Study Group for the Care of the Young Diabetic (SSGCYD) has acted as a forum for the organization of care for young people with diabetes in Scotland. All type 1 diabetic patients <15 years of

age are entered on a register (1) that conforms to quality-control guidelines (2). The SSGCYD is therefore in an ideal position to investigate the quality of care for and the patient response to diabetes in young patients in a defined national

population, against recommendations of clinical outcome (3–5).

The Diabetes Control and Complications Trial (DCCT) demonstrated that improved blood glucose control over a prolonged period significantly reduces the risk of developing the microvascular complications of type 1 diabetes (6). A strength of the DCCT was the use of a centralized laboratory HbA<sub>1c</sub> assay, overcoming criticisms of previous studies in which a confusing picture emerged because of differences in analytical techniques of measuring HbA<sub>1c</sub>. This confusion was highlighted by DIABAUD1, an audit of the management of young people with type 1 diabetes in Scotland (7), which revealed that in 1994, 10 different methodologies existed for HbA<sub>1c</sub> measurement. Although each center performed local quality assurance, there was considerable variation in the average HbA<sub>1c</sub> level for their patients, preventing comparison between centers of the accepted primary outcome measure of glycemic control (8). It prevented also the adoption in Scotland of a single target value HbA<sub>1c</sub> for young people with diabetes.

The DCCT suggested that switching to a more intensive insulin regimen (four or more daily injections or pump therapy), even in young people with type 1 diabetes, was the major factor in producing good glycemic control (6,9). However, after the publication of the study, debate ensued, suggesting that the “clinical support package” (i.e., intensive medical follow-up, additional nursing and dietetic input, and frequent contact) was the main reason for the improvement in glycemic control (10). This view received support with data from the Hvidøre Study Group (22 centers, with 2 from Scotland) (11), who used a single cross-sectional centralized HbA<sub>1c</sub> and showed that control deteriorated significantly throughout adolescence, despite increase insulin dose, and that the type of insulin regimen appeared to make no impact on this deterioration. There were, however, striking differences in the mean

From the Scottish Study Group for the Care of the Young Diabetic, the members of which are listed in the APPENDIX at the end of the article.

Address correspondence and reprint requests to Dr. Stephen Greene, Senior Lecturer, Tayside Institute for Child Health, University of Dundee, Ninewells Hospital and Medical School, Dundee DD19SY, U.K. E-mail: s.a.greene@dundee.ac.uk.

Received for publication 11 May 2000 and accepted in revised form 9 October 2000.

**Abbreviations:** DCCT, Diabetes Control and Complications Trial; SDS, standard deviation score; SSGCYD, Scottish Study Group for the Care of the Young Diabetic.

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

Table 1—Comparison of first HbA<sub>1c</sub> results in subgroups of 1,609 children

	n (%)	Mean ± SD	P
Sex			
Male	855 (53.1)	8.99 ± 1.54	} 0.08
Female	754 (46.9)	9.13 ± 1.55	
Family history			
None	1,322 (82.2)	9.06 ± 1.57	} 0.003
Parent	142 (8.8)	8.80 ± 1.33	
Sibling only	54 (3.4)	9.72 ± 1.74	
Not known	91 (5.7)	9.03 ± 1.26	
Natural parents at home			
Yes	1,204 (74.8)	8.98 ± 1.48	} <0.001
No	346 (21.5)	9.37 ± 1.74	
Not known	59 (3.7)	8.80 ± 1.46	
Deprivation quintile of home address			
1 (affluent)	347 (21.8)	9.11 ± 1.51	} 0.96
2	333 (20.9)	9.05 ± 1.48	
3	332 (20.8)	9.03 ± 1.66	
4	300 (18.8)	9.08 ± 1.45	
5 (deprived)	283 (17.7)	9.02 ± 1.62	
Distance from home to clinic (km)			
<5	456 (29.4)	9.06 ± 1.57	} 0.14
5–9	428 (27.8)	8.93 ± 1.51	
10–19	381 (24.5)	9.06 ± 1.58	
≥20	288 (18.5)	9.21 ± 1.49	
Season			
September–November	936 (58.2)	9.06 ± 1.51	} 0.38
December–February	365 (22.7)	9.14 ± 1.57	
March–May	157 (9.8)	8.96 ± 1.71	
June–August	151 (9.4)	8.92 ± 1.52	
Age-group (years)			
>12	579 (36.0)	9.54 ± 1.74	} <0.001
8–12	607 (37.7)	8.93 ± 1.40	
4–8	351 (21.8)	8.57 ± 1.24	
<4	72 (4.5)	8.57 ± 1.32	
Puberty			
Prepubertal	801 (49.8)	8.79 ± 1.34	} <0.001
Pubertal/adult	533 (33.1)	9.50 ± 1.66	
Not known	275 (17.1)	8.97 ± 1.68	
Duration of diabetes			
>5 years	469 (29.1)	9.35 ± 1.39	} <0.001
18 months–5 years	643 (40.0)	9.15 ± 1.45	
6 months–18 months	279 (17.3)	8.92 ± 1.69	
<6 months	218 (13.5)	8.29 ± 1.68	
Insulin method			
Premixed	1351 (85.7)	9.05 ± 1.55	} 0.92
Self-titrated/both	225 (14.3)	9.04 ± 1.51	
Insulin regimen			
Two per day	1512 (94.0)	9.07 ± 1.54	} <0.001
One per day	29 (1.8)	8.01 ± 1.42	
Three per day	32 (2.0)	8.79 ± 1.12	
Four or more per day	30 (1.9)	9.79 ± 1.77	
Insulin dose per kg body weight			
<0.50	178 (11.1)	8.21 ± 1.72	} <0.001
0.50–0.74	440 (27.5)	8.91 ± 1.49	
0.75–0.99	584 (36.5)	9.16 ± 1.43	
1.00–1.24	281 (17.5)	9.42 ± 1.44	
>1.25	119 (7.4)	9.44 ± 1.67	
BMI (relative to national standard)			
<–2 SD	8 (0.5)	10.93 ± 1.82	} 0.002
–2 SD to –1 SD	58 (3.6)	9.30 ± 1.76	
–1 SD to +1 SD	1,032 (64.5)	9.08 ± 1.56	
+1 SD to +2 SD	407 (25.4)	8.97 ± 1.42	
>+2 SD	96 (6.0)	8.84 ± 1.52	

HbA<sub>1c</sub> concentrations between the various centers (HbA<sub>1c</sub> range 7.6–10.6%, DCCT equivalent +0.3%). No obvious factors associated with glycemic control emerged from this limited assessment of the patients' clinical management.

The SSGCYD, therefore, established DIABAUD2 to assess glycemic control of young people with type 1 diabetes from different centers throughout Scotland using prospective measurements of HbA<sub>1c</sub> concentration in a central laboratory, to provide the first standardized national data within the U.K. to equate glycemic control to the DCCT, and to investigate the factors associated with glycemic control in this population.

## RESEARCH DESIGN AND METHODS

The study was approved by each appropriate local ethics committee, with voluntary participation after verbal and written explanation. Treatment occurred in 17 centers with a clinic (or group of clinics with the same lead clinician) that had 20 or more participating patients. Results from six small clinics (<20 patients) that were not linked to any larger clinic were combined to give the final center a total of 18 patients. Moreover, 12% of patients <15 years of age were seen in adult/adolescent clinics.

For the DIABAUD2 study period (August 1997–February 1999), all subjects who were <15 years of age by 18 August 1997 were eligible, and data were collected prospectively for every clinical visit during DIABAUD2. Patients were identified from the SSGCYD Register (based in Aberdeen, *n* = 1,861), allocated a unique number, and registered separately for DIABAUD2 (Dundee, *n* = 1,755, 94.3%) to maintain anonymity between the participating centers. There were 106 subjects who were not included in the study: 47 were lost (2.5%); 59 were accounted for but not used (3.2%). A minimum dataset was collected at the following times: 1) registration (sex, age, duration of diabetes, postcode address, family history of diabetes, family structure, and known complications of diabetes—microalbuminuria, retinopathy, neuropathy, necrobiosis lipoidica diabetorum, celiac disease, and thyroid disease, based on clinical determination) by each center (DIABAUD2 did not request a specific screening test); and 2) at each clinic visit during the study period (type and dose of insulin, occurrence of hypoglycemia and/or ketoacidosis since last clinic review, and weight and height).

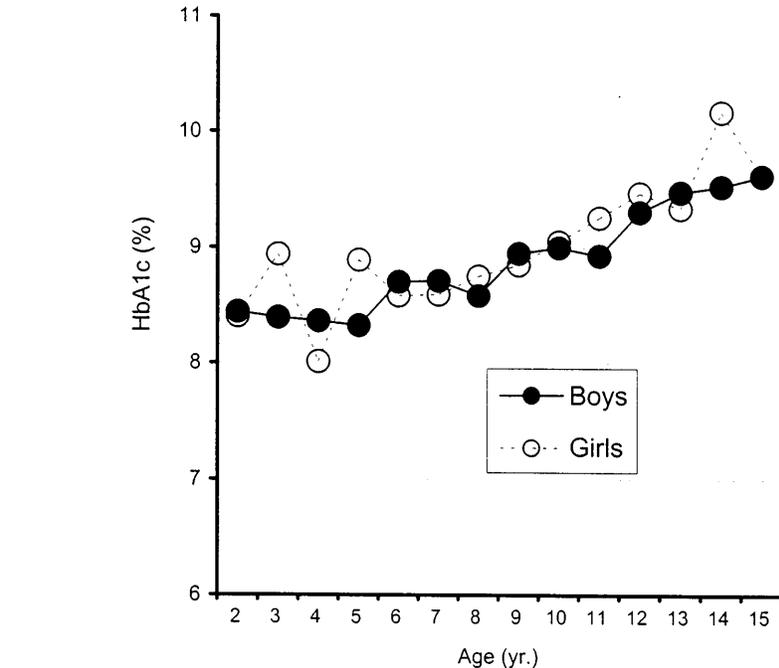
Data from the 1991 U.K. census were used to create a deprivation score for each of the postcode sectors based on the following: percentage of economically active men who are unemployed, percentage of individuals living in private households with no car, percentage of individuals living in overcrowded households (i.e., more than one person per room), and percentage of individuals in private households with an economically active head of household in a semiskilled or unskilled manual occupation (12). Distances between the child's home address and the clinic were calculated from grid coordinates obtained in the U.K. postcode address file.

At each visit when a blood sample was taken for routine clinical practice, a duplicate sample was taken (5  $\mu$ l Bio-Rad HbA<sub>1c</sub> Capillary Collection System), which was sent to a laboratory in Edinburgh for analysis ( $n = 6,187$  HbA<sub>1c</sub> assays). Samples are stable for 28 days using this system (11). The following were received: clinic visit forms ( $n = 7,931$ ), HbA<sub>1c</sub> results ( $n = 6,187$ ), and first-visit forms with central laboratory HbA<sub>1c</sub> measurement ( $n = 1,609$ ). The central laboratory received 7,449 forms and 7,028 samples, with <2% error rates; 98% of samples reached the laboratory within 10 days of sampling. HbA<sub>1c</sub> was analyzed by a Bio-Rad variant analyzer, BioRex 70 ion-exchange column chromatography (locally derived reference range 5.0–6.5%). The laboratory's variant analyzer was traceable to the DCCT under the National Glycohaemoglobin Standardisation Program. The relationship was given by the following:  $DIABAUD2 = 0.951 \times DCCT + 0.632$  ( $r = 0.985$ ). DIABAUD2 HbA<sub>1c</sub> concentrations of 6.6 and 8.5% correspond to DCCT HbA<sub>1c</sub> concentrations of 6.3 and 8.3%, respectively. The between-run coefficient of variation was 1.2% at HbA<sub>1c</sub> 5.4% and 1.8% at HbA<sub>1c</sub> 10.8%.

### Statistical analysis

The DIABAUD2 database was validated against the register to check on completeness of coverage of the DIABAUD2 study and individual items of data (e.g., date of birth). The reference data for height and weight were from the U.K. National Growth standard (13). Height, weight, and BMI were expressed as standard deviation scores (SDSs).

Factors likely to be associated with glycemic control were investigated by an independent sample *t* test, one-way analysis of variance, and simple regression analysis.



**Figure 1**—Rise in HbA<sub>1c</sub> concentration in type 1 diabetic children <15 years of age in Scotland. Glycemic control was significantly worse in the older child (average HbA<sub>1c</sub> level age range 10–15 years 9.5% vs. all other ages 8.6%),  $P < 0.001$ . No significant differences were found between boys and girls.

Multiple regression analysis was then used to compare differences in mean HbA<sub>1c</sub> between centers, adjusting for characteristics that were found to be significantly associated with control but over which the clinical team had no influence (age, sex, and diabetes duration). From this model, we derived a predicted HbA<sub>1c</sub> level for each subject given his or her characteristics. The HbA<sub>1c</sub> values of individual subjects were then expressed as deviations from predicted values. Finally, these deviations were compared between centers and ranked by mean deviation while maintaining anonymity.

**RESULTS** — The characteristics of 1,609 children are given in Table 1. The average age of the patients was 10.2 years, the average duration of disease was 3.7 years, and male subjects comprised 53% of the total. There was a family history of type 1 diabetes in first-degree relatives in 12.0% of children: mother (2.9%), father (6.4%), and sibling (4.4%). In 22% of families, one or both parents were not living at home.

Both boys and girls showed a small but significant ( $P < 0.001$ ) excess in height relative to national standards with mean height SDS (95% CI) of 0.18 (0.11–0.25) and 0.12 (0.04–0.20), respectively. The BMI of boys and girls was greater than that of the normal

population, with no sex difference: the mean BMI SDS (95% CI) for boys was 0.61 (0.55–0.67); for girls, it was 0.57 (0.50–0.64). There was a low incidence of persistent microalbuminuria (0.7%), retinopathy (0.8%), thyroid disease (0.7%), celiac disease (0.1%), and necrobiosis lipodica (0.1%). The absence of one or both parents was associated with poorer control. A family history of type 1 diabetes was associated with significantly poorer glycemic control, but only when there was a sibling with diabetes. HbA<sub>1c</sub> levels were significantly better during the summer months.

There were no important differences in the finding of analyses based on the first HbA<sub>1c</sub> result and the average of all available HbA<sub>1c</sub> results, and, therefore, we present data from first available HbA<sub>1c</sub>. The overall median HbA<sub>1c</sub> level was 8.9%: HbA<sub>1c</sub> <7.0% in 6.9% of patients, 7.0–8.9% in 43.2% of patients, 9.0–10.9% in 39.2% of patients, and >11.0% in 10.7% of patients. The rise with age is shown in Fig. 1. Median HbA<sub>1c</sub> was significantly worse in the older child (age >12 years 9.5% vs. all other ages 8.8%), and HbA<sub>1c</sub> concentration was best during the first 6 months after diagnosis (8.3 vs. 9.2%). The following were not significantly associated with glycemic control: insulin type (self-titrated vs. premixed)

Table 2—Coefficients for variables in regression analyses of HbA<sub>1c</sub> (%)

	Before adjustment (n = 1,609 maximum)			After adjustment for age-group, sex duration, BMI score, broken home, family history, season and center (n = 1,601 maximum)		
	Effect	95% CI	P	Effect	95% CI	P
Age						
<4 vs. >12 years	-0.97	(-1.33 to -0.60)	} <0.001	-0.56	(-0.94 to -0.18)	} <0.001
4–8 vs. >12 years	-0.97	(-1.16 to -0.78)		-0.81	(-1.01 to -0.60)	
8–12 vs. >12 years	-0.60	(-0.77 to -0.43)		-0.58	(-0.75 to -0.41)	
Sex						
Female vs. male	0.13	(-0.02 to 0.29)	0.08	0.14	(-0.01 to 0.28)	0.06
Duration						
<6 months vs. >5 years	-1.06	(-1.30 to -0.81)	} <0.001	-0.76	(-1.01 to -0.50)	} <0.001
6–18 months vs. >5 years	-0.43	(-0.65 to -0.20)		-0.18	(-0.41 to 0.04)	
18 months–5 years vs. >5 years	-0.20	(-0.37 to -0.01)		0.03	(-0.15 to 0.21)	
Insulin type						
Self-titrated or both vs. premixed	-0.01	(-0.23 to 0.21)	0.92	-0.15	(-0.42 to 0.12)	0.27
Regimen						
One per day vs. two per day	-1.06	(-1.63 to -0.50)	} <0.001	-0.63	(-1.18 to -0.09)	} 0.002
Three per day vs. two per day	-0.28	(-0.81 to 0.26)		-0.74	(-1.25 to -0.22)	
Four or more per day vs. two per day	0.73	(0.17 to 1.28)		0.34	(-0.19 to 0.87)	
Insulin dose per unit/kg	1.03	(0.79 to 1.27)	<0.001	0.35	(0.07 to 0.63)	0.02
BMI per SDS	-0.16	(-0.24 to -0.07)	<0.001	-0.12	(-0.19 to -0.04)	0.005
Natural parents at home						
Yes vs. no	0.39	(0.21 to 0.58)	} <0.001	0.38	(0.20 to 0.50)	} <0.001
NK vs. no	-0.18	(-0.58 to 0.22)		-0.27	(-0.72 to 0.17)	
Family history						
Parent vs. none	-0.26	(-0.53 to 0.01)	} 0.003	-0.19	(-0.44 to 0.07)	} 0.002
Sibling vs. none	0.66	(0.24 to 1.08)		0.70	(0.30 to 1.09)	
NK vs. none	-0.03	(-0.35 to 0.30)		0.02	(-0.34 to 0.38)	

NK, not known.

or deprivation score by postcode sector. No significant association was found between mean HbA<sub>1c</sub> and distance from home to clinic, although the highest mean HbA<sub>1c</sub> was observed in children who traveled >20 km. Various clinical and demographic factors were related to HbA<sub>1c</sub> levels, and the results of the multiple regression analysis with HbA<sub>1c</sub> as the response variable are shown in Table 2. Of all subjects, 94% were on two injections per day. After adjustment for confounding variables, this regimen was associated with significantly poorer HbA<sub>1c</sub> concentration than one or three injections per day, although the worst HbA<sub>1c</sub> results were observed in those receiving four or more injections per day. There was a significant negative relationship between HbA<sub>1c</sub> level and BMI.

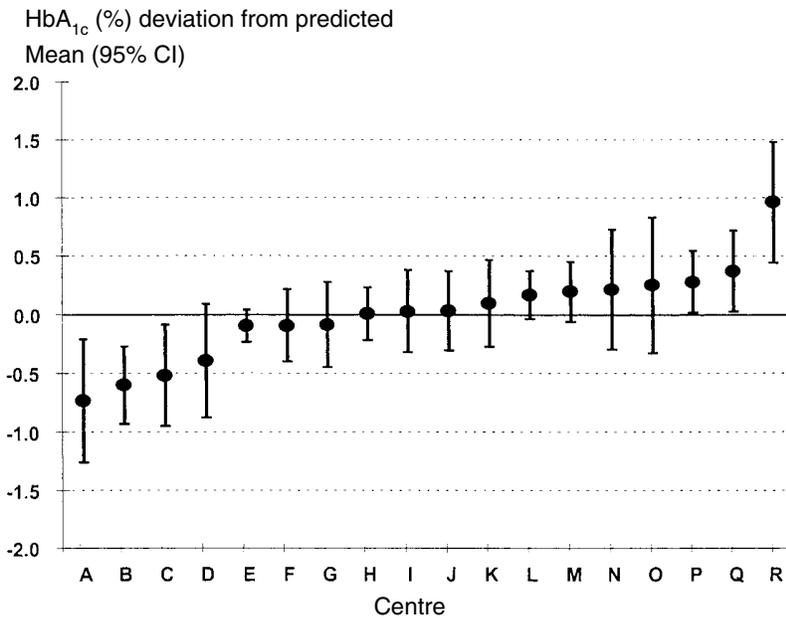
There were statistically significant differences in mean HbA<sub>1c</sub> levels between the 18 centers ( $P < 0.001$ ), and these differences remained significant after adjustment for selected factors that were found: age, sex, duration of diabetes, BMI score, family history, family structure, and season. Each

child's HbA<sub>1c</sub> concentration was expressed as a deviation from the value predicted from these factors, and Fig. 2 shows the mean of these deviations by center. When these selected factors were included in the regression model, they explained ~12% of the patient-to-patient variation in HbA<sub>1c</sub> level (rising to 16% if a center was included in the model). Clearly, the majority of patient-to-patient variation in HbA<sub>1c</sub> levels remains to be explained by other factors not considered in this analysis. The number of patients per center achieving an HbA<sub>1c</sub> <8.5% was calculated and was found to range from 10 to 67%. Only 3 of 18 centers achieved >50% of patients with HbA<sub>1c</sub> <8.5%, the equivalent average value obtained by the intensive group of adolescents in the DCCT.

**CONCLUSIONS** — DIABAUD2 is the first U.K. national study on the influences of glycemic control in young people with type 1 diabetes. The unexpected finding was the significant and independent effect on glycemic control of the center delivering care.

DIABAUD1 (1994) failed to make inter-center comparisons because of the disparate methodologies for the assessment of HbA<sub>1c</sub> concentration. Clinical governance requires that U.K. National Health Service organizations have in place processes for monitoring and improving clinical quality as well as setting frameworks and standards for treatment and care (14–17). Therefore, an aim of DIABAUD2 was to provide evidence from which standards of care could be developed and to demonstrate the ability of the SSGCYD to construct and manage an effective monitoring system of clinical care. DIABAUD2 was developed and completed over 3 years, with an ascertainment rate of 94% of children <15 years of age. A robust methodology for collection of data was developed, using the SSGCYD Register of existing and newly presenting patients. DIABAUD2 is the first U.K. study to compare average HbA<sub>1c</sub> levels in a defined population against the DCCT standard.

The overall glycemic control was 8.9% (DCCT equivalent 9.1%), which equates to



**Figure 2**—Differences among centers (expressed as the mean of the deviations from predicted values in units of HbA<sub>1c</sub>) adjusted for factors known to influence glycemic control. If there were no center effects, all values would lie on the zero line.

the nonintensive arm of the DCCT and is well above the accepted target value (6,18). However, the applicability of the DCCT parameters to a population of children has yet to be established. The adolescent cohort of the DCCT had an average HbA<sub>1c</sub> concentration of 10.1% on conventional therapy and 8.2% on intensive treatment (9). The overall glycemic control in Scotland is similar to the only other national published data: Denmark (mean HbA<sub>1c</sub> 9.1%) (19) and France (mean HbA<sub>1c</sub> 9.0%) (20). However, both of these studies had ascertainment rates well below 70% of the young diabetic population.

Significant associations with HbA<sub>1c</sub> were identified (age, sex, insulin regimen, BMI, season, social circumstances, and family history). However, these associations accounted for <12% of the patient-to-patient variation in HbA<sub>1c</sub> levels. Inequalities between clinics in HbA<sub>1c</sub> concentration were demonstrated after adjustment for factors associated with poor glycemic control, with significant and major differences in average HbA<sub>1c</sub> level between clinical centers. Although formal analysis has not been undertaken, a survey of the resources in each clinical center (i.e., distribution of medical staff, variation in performing of clinical tasks, offering 24-h contact and home care, and differences in methodologies for investi-

gations) has not shown a clear pattern of effect on HbA<sub>1c</sub>.

Given that significant differences in HbA<sub>1c</sub> levels between centers remained after adjustment for factors found to influence the individual child's glycemic control, it appears that other factors not considered in DIABAUD2 must explain the differences in HbA<sub>1c</sub> levels between centers. Debate within the SSGCYD in light of the results suggests that deployment of the resources, organization of the clinic, and strategies of medical care may explain some of the different performance levels of the various centers. The "best" center has a policy of frequent contact (both medical and nursing), with at least monthly formal advice (more if required), together with a rapid "troubleshooting" service and frequent change in insulin regimen with no fixed "favorite" and the aim of a near-normal target for HbA<sub>1c</sub> concentration (<7.5%). We speculate that the style of utilization of optimum resources is the key factor in achieving good glycemic control.

The lower HbA<sub>1c</sub> level in the first 6 months after diagnosis is likely to be due to endogenous insulin secretion. In Scotland, the only insulin regimen associated with a lower HbA<sub>1c</sub> level was one comprising three injections per day. However, for unexplained reasons of "fashion," the vast majority of patients were treated by twice-daily premixed insulin. We must be cau-

tious in the interpretation of this finding from the multivariate analysis: it may be accounted for by other unmeasured variables (e.g., selection of compliant patients for multiple-dose regimens). Nevertheless, other cross-sectional studies have failed to show a benefit from multiple-injection regimens in young people (11,19,21). The regimen of three injections per day should be tested prospectively to determine whether it improves glycemic control in this cohort, particularly as the use of this regimen appears to be rising.

In summary, this study has provided national data on glycemic control, illustrated a number of important associations with glycemic control, and demonstrated that considerable variation exists between diabetes centers within one country in the level of HbA<sub>1c</sub> achieved. In winter, the teenager with 5 years' duration of disease who takes two injections per day, with a high insulin dose, who has a diabetic sibling, and who comes from a "broken home" appears to be the stereotype of the young person with poor glycemic control and is at probable high risk of long-term effects of diabetes. However, it appears that within Scotland, certain clinical centers can influence more young people with type 1 diabetes who are in their care to maintain glycemic control within the desired target range. The challenge for all of us is to emulate these centers.

**Acknowledgments**— The membership of the SSGCYD, an independent medical body, is grateful for the previous and continuing financial support from Novo Nordisk, U.K. The DIABAUD2 Project was supported by the Clinical Research and Audit Group of the Scottish Executive and Novo Nordisk U.K.

The project was conceived and delivered by the membership of the SSGCYD and guided by the DIABAUD2 Steering Committee.

**APPENDIX**— DIABAUD2 was undertaken with the support, direction, and participation of the following members of the SSGCYD (\*DIABAUD2 Steering Committee): V. Alexander (Dundee); A. Blair (Kirkcaldy); M. Blair (Kilmarnock); I. Campbell (Kirkcaldy); A. Collier (Ayrshire); J. Croll\* (Dundee); A. Connacher (Perth); I. Craigie (Glasgow); G. Farmer\* (Inverness); M. Fisher (Paisley); S. Gallacher (Glasgow); S. Gray (Livingston); S. Greene\* (Dundee); A. Harrower (Airdrie); A. Jaapp (Kilmarnock); R. Jung (Dundee); C. Kelnar (Edinburgh);

J. Lawrence (Dumfries); G. Leese (Dundee); P. Leslie (Melrose); M. Louden (Airdrie); A. MacCuish (Glasgow); D. Matthews (Stonehouse); S. MacRury (Inverness); M. McGregor (Carlisle); J. McKnight (Edinburgh); H. McLaren (Glasgow); B. McSporran\* (Aberdeen); A. Morris (Dundee); L. Murchison (Aberdeen); R. Newton (Dundee); K. Noyes (Edinburgh); E. O'Brien (Carlisle); A. Patrick (Edinburgh); C. Patterson\* (Belfast); D. Pearson (Aberdeen); N. Peden (Falkirk); P. Rae (Edinburgh); S. Reith (Stirling); K. Robertson\* (Glasgow); D. Rooney (Glasgow); I. Ruthven (Kilmarnock); C. Shepherd (Paisley); J. Schulga\* (Stirling); P. Smail\* (Aberdeen); M. Small\* (Glasgow); J. Steel (Kirkcaldy); R. Thomson (Dumfries); J. Walker\* (Edinburgh); and N. Waugh (Aberdeen).

#### References

- Rangasami JJ, Greenwood DC, McSporran B, Smail PJ, Patterson CC, Waugh NR: Rising incidence of type 1 diabetes in Scottish Children, 1984–93. *Arch Dis Child* 77:210–213, 1997
- Diabetes Epidemiology Research International: Preventing insulin dependent diabetes mellitus. *Br Med J* 295:479–481, 1987
- Diabetes Care and Research in Europe: St. Vincent Declaration. *Diabet Med* 7:360, 1990
- International Society for Paediatric and Adolescent Diabetes (ISPAD): *Consensus Guidelines for the Management of Insulin Dependent (Type 1) Diabetes Mellitus (IDDM) in Childhood and Adolescence*. London, Freund Publishing House, 1995
- The Scottish Intercollegiate Guidelines Network: *Report on Good Practice in the Care of Children and Young People with Diabetes: SIGN 10*. Edinburgh, U.K. Royal College of Physicians, 1996
- 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* 329:977–986, 1993
- Robertson KJ, Greene SA, on behalf of the Scottish Study Group for the Care of the Young Diabetic: *A Clinical Audit of the Management of Young People With Type 1 Diabetes in Scotland*. Clinical Resource and Audit Group, Scottish Office, 1994
- Greene SA: Perspectives of quality control in diabetes treatment at the end of the century: facts and visions. *Horm Res* 50 (Suppl. 1):103–105, 1998
- Diabetes Control and Complications Trial: Effect of intensive diabetes treatment on the development and progression of long-term complications in adolescents with insulin-dependent diabetes mellitus. *J Pediatr* 125:177–188, 1994
- Boulton A: DCCT: implications for diabetes care in the UK (Editorial). *Diabet Med* 10:687, 1993
- Mortensen HB, Hougaard P: Comparison of metabolic control in a cross-sectional study of 2,873 children and adolescents with IDDM from 18 countries: the Hvidovre Study Group on Childhood Diabetes. *Diabetes Care* 20:714–720, 1997
- Carstairs V, Morris R: Deprivation and mortality: an alternative to social class? *Commun Med* 11:210–219, 1989
- Freeman JV, Cole TJ, Chinn S, Jones PR, White EM, Preece MA: Cross sectional stature and weight reference curves for the UK. *Arch Dis Child* 73:17–24, 1995
- The Scottish Intercollegiate Guidelines Network: *Report on a Recommended Minimum Dataset for Collection in People With Diabetes: SIGN 25*. Edinburgh, U.K. Royal College of Physicians, 1998
- British Diabetic Association: *Report on Good Practice in Children and Adolescents With Diabetes*. London, British Diabetic Association, 1998
- Clinical Governance: National Health Service, Management Executive Letter, The Scottish Office (Edinburgh) MEL 75, 1998
- Greene SA: Diabetes mellitus in childhood and adolescence. In *Textbook of Diabetes*. Vol. 87. Pickup J, Williams G, Eds. Oxford, U.K., Blackwell Scientific Publications, 1991, p. 866–883
- Pound N, Sturrock NDC, Jeffcoate WJ: Age related changes in glycosylated haemoglobin in patients with insulin dependent diabetes mellitus. *Diabet Med* 13:510–513, 1996
- Mortensen HB, Marinelli K, Norgaard K, on behalf of the Danish Study Group of Diabetes in Childhood: A nationwide cross-sectional study of urinary albumin excretion rate, arterial blood pressure and blood glucose control in Danish children with type 1 diabetes mellitus. *Diabet Med* 7:887–897, 1990
- Rosilio M, Cotton JB, Wieliczko MC, Gendreau B, Carel JC, Couvaras O, Ser N, Bougneres PF, Gillet P, Soskin S, Garandeau P, Stuckens C, Le luyer B, Jos J, Bony-Trifunovic H, Bertrand AM, Leturcq F, Lafuma A: 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* 21:1146–1153, 1998
- Shirish C, Shah MD, John I, Malone MD: A randomized trial of intensive insulin therapy in newly diagnosed insulin-dependent diabetes mellitus. *N Engl J Med* 320:550–558, 1989