Ethnic differences in early glycaemic control in childhood onset type 1 diabetes

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Short title: Ethnicity and early glycaemic control in children with type 1 diabetes

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Word count – (Max 3500)

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Abstract

Background Ethnic minorities with Type 1 Diabetes (T1D) have worse glycaemic control (higher $HbA1_c$) and greater risk for vascular complications. There is limited evidence on the impact of ethnicity on early glycaemic control when patients experience transient remission post-diagnosis. We used modelling techniques to examine the independent contribution of ethnicity on $HbA1_c$ trajectories during the first six months post-diagnosis.

Methods Data on 443 (50% female) children <19years of age, with T1D and attending one of three clinics in East London between Jan 2005 and Dec 2015 were included. Linear mixed-effects modelling was used to assess effects of ethnicity on longitudinal $HbA1_c$ trajectories during first six months post-diagnosis (1,028 $HbA1_c$ datapoints), adjusting for sex, age at diagnosis, socioeconomic status and pH at diagnosis. Growth curve modelling was used to identify discrete $HbA1_c$ trajectories by ethnicity.

Results All ethnic minorities had higher mean $HbA1_c$ at diagnosis compared to White children, most marked in Bangladeshi (9.7mmol/mol, 95% CI 5.1-14.3), Asian-Other (5.8mmol/mol, 95% CI 2.2-9.3) and Somali (5.2mmol/mol, 95% CI 0.1-10.2) children. During the first month, $HbA1_c$ decreased on average by 19.5mmol/mol (-21, -18) for all children. Population averaged $HbA1_c$ decreased between diagnosis and four months, followed by a gradual increase in $HbA1_c$ levels (mean difference of -30mmol/mol between diagnosis and six months). All ethnic groups had higher $HbA1_c$ levels throughout this time period compared to White children.

Conclusions Ethnic minorities have worse glycaemic control at diagnosis, with largest mean differences in Bangladeshi, Asian-Other and Somali children. These higher levels track into the first six months post-diagnosis.

Keywords Ethnicity, Socioeconomic status, child, type 1 diabetes, glycaemic control, $HbA1_c$

Introduction

Inequalities in paediatric type 1 diabetes (T1D) outcomes are well recognised. Children and young people (CYP) of lower socioeconomic status (SES) and ethnic minorities tend to have poorer glycaemic control and increased risk for acute life threatening complications (diabetic ketoacidosis and hypoglycaemia) and chronic vascular complications in later life (1-3). In a recently published study using national data on >95% of CYP with T1D in England and Wales, we found that all ethnic minorities had poorer glycaemic control (higher blood glycosylated haemoglobin [$HbA1_c$] concentrations) compared to White CYP (4). Lower SES was associated with poor glycaemic control in all ethnic groups but being poor and ethnic minority had significantly worse impact on glycaemic control than being poor and White.

Studies show that young patients from lower SES and ethnic minority backgrounds receive a later diagnosis of T1D and present with significantly worse symptoms at diagnosis (greater severity of diabetic ketoacidosis and higher $HbA1_c$ levels) (5, 6). A greater level of metabolic derangement at diagnosis may reflect factors such as poorer/late access to medical care (5, 7), cultural and/or biological differences between ethnic groups [ref] and also a lower level of residual pancreatic beta-cell function (8). There have been reports on associations between clinical presentation at diagnosis and subsequent early glycaemic control (9). Therefore if ethnic minorities present with worse clinical factors at diagnosis, one could hypothesise that the stabilisation of glycaemic control during the period of transient remission ('honeymoon phase') experienced by most newly diagnosed T1D patients, could also differ by ethnic background (10). However, this has not been comprehensively investigated in a multi-ethnic population in the UK. This is especially important to study as early glycaemic control during the first year post-diagnosis is linked to subsequent future control which may track into adulthood (11).

In order to identify those groups of CYP with poor glycaemic control based on clinical factors at presentation, we investigated ethnic differences in stabilisation of glycaemic control during the first six months post-diagnosis in a multi-ethnic population attending diabetes clinics in East London. Identification

of patients with initial and subsequent poor glycaemic control could help in devising targeted policies to help these CYP have better outcomes in the future.

Methods

Design, setting and data source

We undertook a longitudinal cohort study of newly diagnosed patients with type 1 diabetes using data from three paediatric diabetes clinics that are part of the same Healthcare Trust (Barts Health NHS Trust) and located in East London, UK. The three clinics largely capture patients living in surrounding areas of East London, where up to 56% of the local population belongs to an ethnic minority with *c.* 50% of South Asian origin (primarily of Bangladeshi origin) and *c*. 40% of Black origin (primarily of Somali origin).

The study was restricted to children <19 years of age who received a diagnosis of type 1 diabetes between 1 Jan 2005 and 31 Dec 2015 and attended any one of the three clinics during the same period. Extensive clinical and sociodemographic data was collected prospectively, both at the time of diagnosis and during routine clinic visits. As per previous and current recommendations from the National Institute of Health and Care Excellence (NICE), a child with Type 1 diabetes is offered an integrated package of care by a multidisciplinary team at a paediatric diabetes clinic four times per year. The team consists of paediatric endocrinologists/diabetologists, diabetes specialist nurses, dieticians, psychologists and interpreters. $HbA1_c$ levels and height and weight are recorded at each visit. All demographical and clinical parameters are measured and electronically documented systematically across all three clinics enabling comparison. Out of 596 children diagnosed with type 1 diabetes during the study period, 571 (96%) children had data on sex, age at diagnosis, duration of diabetes, ethnicity and SES and were eligible to be included in the analysis.

Primary outcome, exposures and covariates

The outcome of interest was glycaemic control measured by $HbA1_c$ levels. $HbA1_c$ was measured at each visit using the point of care Siemens/Bayer DCA 2000+ Analyzer. $HbA1_c$ values recorded as percentages were converted to mmol/mol using the formula: ($HbA1_c$ value in % – 2.15) × 10.929.

The main exposures of interest were ethnicity and SES. Participants (or their parents) were asked to self-identify their ethnicity when they visited a clinic and we used the first recorded entries for ethnicity at the time of diagnosis. They were given the option to choose 1 of 15 categories or also the option to decline identifying their ethnicity. For the purpose of this study, the 15 ethnic categories were collapsed into six broad groups: White, mixed (any mixed ethnicity combination), Black, African-Somali, Bangladeshi and Asian-other (any Asian origin excluding Bangladeshi) which reflects the ethnic distribution of the study area in East London. The latter group included CYP mostly of Indian or Pakistani origin and a much smaller proportion originating from other Asian countries. The pH value (blood capillary samples) measured closest to initial presentation was used in the analysis.

SES was derived from postcode using Indices of Multiple Deprivation (IMD) 2010 for England. The IMD is a small geographical area measure of deprivation. It is multidimensional and scores are derived from a weighted combination of several indicators across seven distinct measures of deprivation including: income, employment, education skills and training, health, barriers to housing and services, living environment and crime [21]. It captures the 'relative' deprivation experienced by an individual living in an area. IMD scores are calculated at the level of lower-layer super output areas, with each area comprising 1500 individuals on average. IMD rank scores were grouped into quartiles for the analysis, with the first and fourth quartiles corresponding to the most and least deprived, respectively.

Other covariates adjusted for in the analysis included: age at diagnosis calculated by subtracting date of diagnosis from date of birth; age at clinic visit calculated by subtracting date of clinic visit from date of birth; duration of diabetes calculated in months by subtracting the date at first visit in the audit year from the date of diagnosis of Type 1 diabetes; which of the three Paediatric Diabetes clinics the child attended;

and pH levels recorded at diagnosis - used as an indicator of diabetic ketoacidosis severity at presentation, measured in a subgroup of patients.

Statistical analysis

Baseline characteristics were compared across all ethnic groups. Categorical variables were compared as frequencies using Chi2 or Fisher's Exact test. Mean differences in continuous variables by ethnicity were analysed using simple linear regression.

Stabilisation of glycaemic control during the first six months post-diagnosis was assessed using linear longitudinal mixed effects models, which allow comparison of population average $HbA1_c$ levels and change over time for the different ethnic categories while controlling for potential covariates. We approximated time trends using a quadratic model for time since diagnosis as this provided a better statistical fit than a linear model. Ethnicity, SES, age at diagnosis, sex and diabetes clinic were entered as time-constant predictors. We constructed a series of models using the *xtmixed* commands in Stata 13. The first model (Model 1) was an unadjusted growth model using time since diagnosis (disease duration) as the temporal metric. Subsequent models were additionally adjusted for our hypothesised predictors; sex and age at diagnosis in years (Model 2), ethnicity (Model 3), SES (Model 4) and diabetes clinic (Model 5). We tested for a potential interaction between ethnicity and duration to assess whether $HbA1_c$ trajectories differed by ethnic group. We estimated all model parameters by maximum likelihood. We used generalised likelihood ratio statistics, -2 log-likelihood (-2LL), Aikake information criterion (AIC) and sample-adjusted Bayesian information criterion (BIC) to compare model fit between subsequent nested models, and Wald statistics to test hypotheses about model parameters. We plotted quadratic growth curves at the group level (i.e. ethnicity) to visualise the fit of the model. All analysis were run in Stata 13 (College Station, Texas).

We ran models 3 and 5 above in a subgroup of patients with data on pH levels at diagnosis to assess any change to the observed ethnicity – HbA1c associations.

Results

Of the 571 subjects eligible to be included, 89 did not have any recorded data during the first six months post-diagnosis. Additionally, 39 had missing data on HbA1c during the first six months post-diagnosis leaving 443 (78% of the eligible population) children with 1,028 measurements of $HbA1_c$ during the first six months and data on all covariates that were included in the analysis. The mean number of $HbA1_c$ measurements per child during the first six months was 2.3 (range 1 to 7).

Characteristics of the study population at baseline and six months post-diagnosis

50% of the study population were female. Mean $HbA1_c$ at diagnosis of T1D was 93.7mmol/mol. All ethnic groups had relatively high $HbA1_c$ levels at diagnosis with the highest mean levels observed in Bangladeshi children (99.9mmol/mol (SD 24.1), Table 1). Mean deprivation score and the proportion of subjects in the lowest SES quartile (i.e. the most deprived) differed significantly by ethnic group, with the African-Somali, mixed and Black groups having the lowest mean deprivation scores and largest proportions of CYP in the most deprived SES quartile (Table 1). There were no significant differences in $HbA1_c$ at diagnosis by SES quartiles. We observed no significant differences in gender, age at diagnosis and pH by ethnicity. $HbA1_c$ decreased by an average of 32.4mmol/mol during the first six months post-diagnosis.

Longitudinal modelling of $HbA1_c$ trajectories

Table 2 shows the regression parameters from longitudinal modelling. We detected a significant difference in $HbA1_c$ when comparing ethnic minority children to White children. All ethnic minority groups had higher mean $HbA1_c$ levels at diagnosis compared to the White group. However, differences were statistically significant only in the Bangladeshi (9.1mmol/mol, 95%CI 4.5-13.6) and Asian-other (5.8mmol/mol, 2.3-9.4, Table 2) groups which exhibited the largest mean differences in $HbA1_c$ levels at diagnosis compared to White children. Adjustment for SES slightly enlarged the estimates for all ethnic groups, and the estimate

for the African-Somali group reached statistical significance (5.2mmol/mol, 0.1-10.2, Table 2). However, SES, age at diagnosis and gender were not significantly associated with $HbA1_c$ levels at diagnosis. We also observed a significant difference in $HbA1_c$ at diagnosis by diabetes clinic.

All ethnic groups experienced an initial decrease in $HbA1_c$ levels during the first four months post-diagnosis followed by a slight gradual increase between months four and six, with a significant quadratic term in all models (Figure 1, Table 2). $HbA1_c$ levels decreased by an average 19.5mmol/mol during the first month after diagnosis for the entire group. Likelihood ratio tests showed that each subsequent model had a statistically better fit than the preceding model with fewer covariates. Model 5 with all covariates had the best fit (-2LL=8634 and AIC=8673, Table 3). In the final Model 5 with all covariates, 76% of the variation in $HbA1_c$ was due to individual differences, Intra-class Coefficient, ICC = 0.76, Table 2).

The model testing for an interaction between ethnicity and duration found no statistically significant interaction, indicating no evidence of a difference by ethnicity in post-diagnosis $HbA1_c$ trajectories over time (data not shown).

Multilevel modelling for change in $HbA1_c$ with pH at diagnosis

Models on pH at diagnosis and $HbA1_c$ were restricted to a smaller sample of 338 children with 764 $HbA1_c$ datapoints (average 2.3 $HbA1_c$ datapoints per subject, range 1 to 6) as pH at diagnosis was not documented in all cases.

All ethnic minorities had higher mean $HbA1_c$ at diagnosis compared to White children as observed in models comprising the entire study sample (N=443). However, estimates for the mixed and Black groups previously not significant were observed to be significantly different from the White group. pH at diagnosis was inversely associated with $HbA1_c$ at diagnosis. For every one unit increase in pH levels (for e.g. from 7.2 to 7.3), $HbA1_c$ decreased by an adjusted average of -15.6mmol/mol (-25.1, -6.2, Table 3).

Discussion

Our aim was to analyse glycaemic control ($HbA1_c$) during the first six months after diagnosis using longitudinal modelling to determine whether $HbA1_c$ trajectories differed by ethnicity. As expected, we observed a general and rapid improvement in $HbA1_c$ levels for the entire study sample that reached a nadir around four months post-diagnosis. This was followed by a slight gradual increase in $HbA1_c$ levels between four and six months post-diagnosis. We found that all ethnic minority children presented with poorer $HbA1_c$ at diagnosis compared to White children and the magnitude of this difference did not change over the study period. An additional aim was to assess the effect of pH (a marker of disease severity at diagnosis) on glycaemic control during the first six months after diagnosis. We found that while pH at diagnosis was negatively associated with $HbA1_c$ during the first month post-diagnosis, it did not affect the observed ethnicity-$HbA1_c$ growth trajectories.

Comparisons with the literature:

Few studies and none from the UK have investigated early glycaemic control (control during the first year after diagnosis) using longitudinal data from diagnosis onwards. Most did not investigate the impact of ethnicity on early glycaemic control, did not use advanced methods to analyse longitudinal data (such as mixed effects models) or had longer follow-up periods without focussing on the transient remission ('honeymoon phase') period as in this study. Our finding of initial very high $HbA1_c$ levels at diagnosis, followed by a steep decline is reported in other studies with comparable study populations (age at diagnosis and proportion of female subjects). However, those studies that reported $HbA1_c$ levels at diagnosis and during the first year post-diagnosis (including datapoints at six months post-diagnosis) did not investigate trends in glycaemic control and were restricted to study populations of White ethnicity limiting comparisons (9, 12-14). Chase et al found a steep decline in $HbA1_c$ levels at 2-4 months followed by a gradual increase similar to our findings (15). Studies that analysed data using longitudinal modelling with a focus on ethnic differences in metabolic control had a much longer follow-up and thus did not report exclusively on the first six months post-diagnosis when most patients undergo transient remission (16, 17).

Initial high $HbA1_c$ levels at diagnosis and lower levels at six months reported elsewhere are similar to that observed in our study.

There have been conflicting reports on the association of age at diagnosis and gender with $HbA1_c$ levels at diagnosis and during follow-up. Some studies report that females and older children have increased $HbA1_c$ levels at diagnosis and during follow-up, whereas others show an interaction between gender and age at diagnosis on subsequent $HbA1_c$ levels during the first year post-diagnosis (12, 14, 17). We observed no association between either gender or age at diagnosis with initial and subsequent $HbA1_c$ levels.

Strengths and limitations:

We used a well powered sample and methods allowed inclusion of a large number of longitudinal datapoints. The study sample was drawn from East London where the majority (67%) belonged to an ethnic minority. This enabled us to study for the first time, paediatric glycaemic control in specific ethnic minority groups such as the Bangladeshi and Somali groups. Such vulnerable groups often get overlooked as they are analysed in combination with other ethnic groups masking potential underlying differences. Ethnicity was self-identified which is considered to be the 'gold standard' in studies on ethnicity and health (18).

Our study also has certain limitations. The study sample was drawn from three paediatric diabetes clinics which operate together as a network since 2012 and results may not be generalizable to the rest of the country. However, we feel results can be generalised to ethnically diverse urban populations in other parts of the UK. One cannot exclude the possibility of residual confounding due to other factors known to impact on glycaemic control at diagnosis and during early follow-up which may interact with ethnicity including family structure, family history of diabetes, pubertal status and incidence of severe hypoglycaemia and diabetic ketoacidosis. Our finding of no association between SES and metabolic control at diagnosis was unexpected. This could be explained by the fact that a significant proportion of the study sample was highly deprived (reflecting the neighbourhood from which the sample was drawn) leading to low variability in SES. Black and mixed ethnic children have increased risk for poor metabolic control (4). However, the statistically nonsignificant estimates for both groups, while in the right direction, could be due to low

statistical power. We were unable to calculate the proportion of subjects undergoing remission at different time points by known methods as we lacked the information needed (insulin dose-adjusted $HbA1_c$ or total daily insulin dose/kg body weight/day).

Several factors may explain the observed ethnic differences in metabolic control at diagnosis and during early follow-up including physiological (greater loss of insulin producing beta cells in some ethnic groups and greater disease severity at diagnosis) and psychosocial (later presentation at diagnosis, attitudes towards understanding disease symptoms and seeking care at later stages) factors. Studies indicate that younger children often present with greater acidosis, experience a more rapid loss of insulin producing β-cells and worse symptoms at diagnosis (19). However, we found no differences in age at diagnosis by ethnicity that could explain the observed differences in $HbA1_c$ at diagnosis.

Our outcomes are timely given long term data from the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications (DCCT/EDC) recently reported by Orchard and colleagues (20). During 30 years of follow-up, less cardiovascular disease events occurred in the former intensive treatment group subjects compared with the former conventional treatment group subjects. The lower $HbA1_c$ levels during the DCCT/EDIC, achieved through intensive insulin therapy, statistically account for all of the observed treatment effect on cardiovascular disease risk. Whilst similar long term data are required for childhood onset T1D, the DCCT/EDC outcomes indicate that achieving very early target HbA1c has important benefits in later life. The mechanisms for the ethnic differences observed in our study require further investigation.

Conclusions:

Ethnic minority children with type 1 diabetes presented with worse glycaemic control (higher mean $HbA1_c$ levels) at diagnosis compared to White children. These differences by ethnicity were sustained throughout the study period including the 'honeymoon' phase when all groups experienced a steep decline in $HbA1_c$ levels. Our study highlights the importance for formulating interventions early on, at the time of diagnosis

and during the first few months to ensure that differences in glycaemic control are reduced. This is especially important as people with poor early glycaemic control are more likely to have poor control later in life.

Acknowledgements

This study and the Policy Research Unit in the Health of Children, Young People and Families (CPRU, GOS Institute of Child Health, UCL) is funded by the Department of Health Policy Research Programme. This is an independent report commissioned and funded by the Department of Health. The views expressed are not necessarily those of the Department.

We thank members of the Policy Research Unit for the health of children, young people and families: Catherine Law, Amanda Edwards, Ruth Gilbert, Steve Morris, Helen Roberts, Cathy Street and Miranda Wolpert.

We would also like to acknowledge the diabetes teams of Barts Health NHS Trust and especially Dr Claire Hughes (Royal London Hospital), Dr Abdul Moodambail (Newham University Hospital) and Dr Paramita Cifelli (Whipps Cross Hospital) for approving the study and facilitating access to data.

Funding

This work was supported by the Children's Policy Research Unit (CPRU), UCL, (funding reference 10090001), which is funded by the Department of Health Policy Research Programme and supported by the National Institute for Health Research Biomedical Research Centre at Great Ormond Street Hospital for Children NHS Foundation Trust and University College London.

Table 1. Characteristics of the study population (N=443) diagnosed with type 1 diabetes between 1 Jan 2005 and 31 Dec 2015. Values are means (standard deviations) or proportions.

	Ethnic group							
Characteristics N (%)	**White** 164 (37)	**Mixed** 37 (8)	**Black** 60 (14)	**African-Somali** 39 (9)	**Bangladeshi** 45 (10)	**Asian-Other** 98 (22)	**Total** 443 (100)	**p Value[a]**
Girls (%)	46	38	47	54	64	55	50	0.14
Age at diagnosis (years)	9.1 (4.5)	7.7 (4.0)	9.2 (3.9)	7.7 (4.2)	9.3 (4.2)	9.1 (4.6)	8.9 (4.4)	0.2
$HbA1_c$ at diagnosis (mmol/mol)[b]	92.1 (20.8)	88.8 (22.1)	95.2 (25.4)	91.8 (21.6)	99.9 (24.1)	93.2 (32.5)	93.7 (24.1)	0.62
$HbA1_c$ at 6 months (mmol/mol)[c]	58.9 (14.1)	60.8 (14.7)	68.1 (26.1)	64.7 (16.9)	61.2 (19.5)	60.4 (17.9)	61.3 (17.9)	0.15
Mean difference in $HbA1_c$ between diagnosis and 6 months (mmol/mol)	33.2	28	27.1	27.1	38.7	32.8	32.4	
pH at diagnosis[d]	7.58 (0.173)	7.271 (0.146)	7.270 (0.152)	7.311 (0.150)	7.296 (0.154)	7.272 (0.163)	7.272 (0.161)	0.68
Index of multiple deprivation score	10537 (7303)	5973 (3588)	6166 (3953)	5035 (2911)	6855 (5324)	8665 (5178)	8291 (6029)	<0.001
Index of multiple deprivation score (median)	8720	5094	5023	4950	5554	7574	6743	
Proportion in lowest quartile	18	38	38	49	36	20	28	<0.001
Number of clinic visits	2.6 (1.3)	2.8 (1.6)	2.7 (1.5)	2.5 (1.3)	2.6 (1.3)	2.9 (1.7)	2.7 (1.5)	0.11

[a]Test for difference in means or proportions [b]202 $HbA1_c$ values recorded during the first one month post-diagnosis [c]285 $HbA1_c$ values recorded between the 5th and 6th months post-diagnosis. [d]Smaller sample, N=338

Table 2. Multilevel model for change in $HbA1_c$ during the first six months post-diagnosis

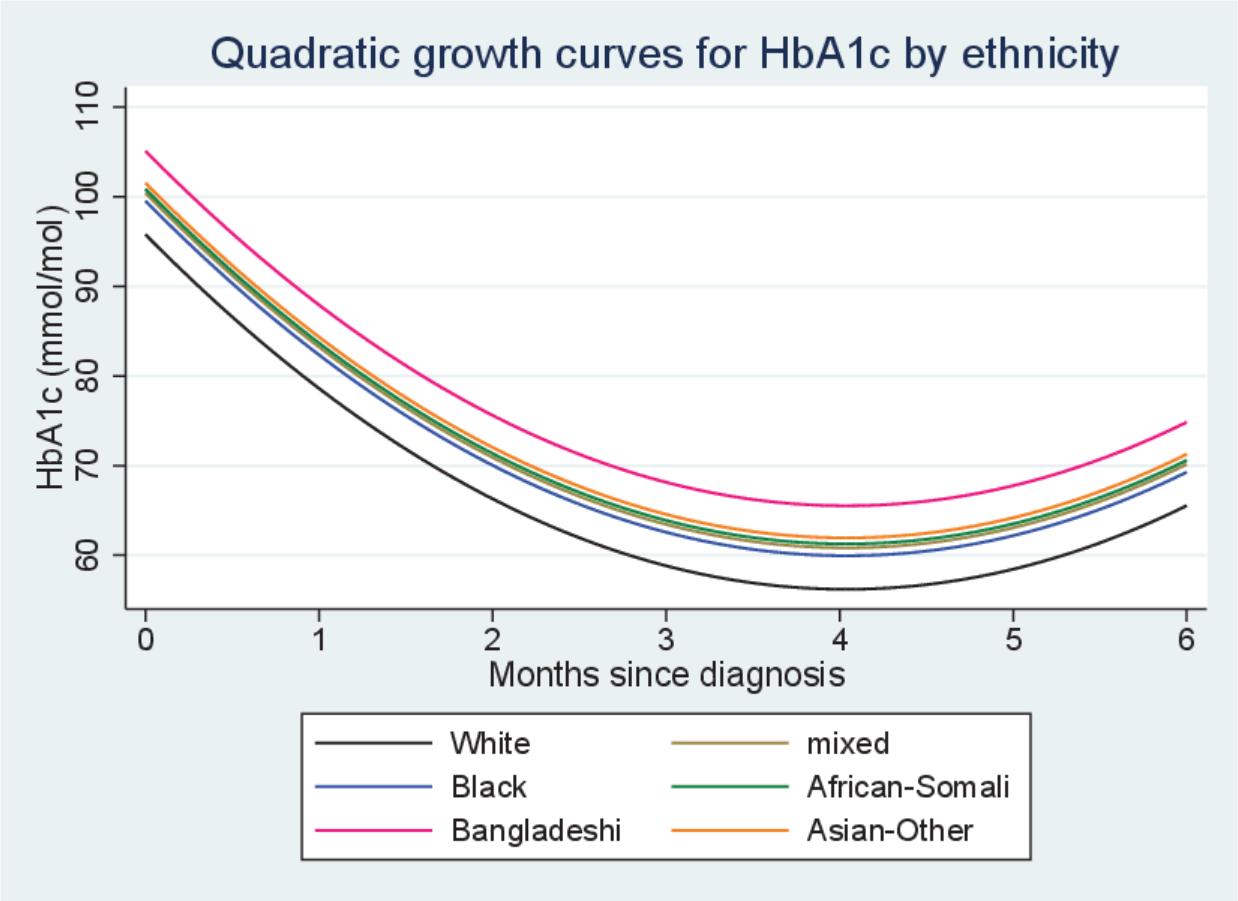
Covariates	**Model1: Growth Model**	**Model 2: Plus age at diagnosis**	**Model 3: Plus Ethnicity**	**Model 4: Plus Socioeconomic Status**	**Model 5: Plus Paediatric diabetes clinic**
Fixed effects					
Constant/intercept	**95.1 (92.4, 97.7)**	**97. 9 (92.5, 103.4)**	**93.5 (87.8, 99.3)**	**91.1 (84.9, 97.2)**	**90.4 (84.3, 96.5)**
Duration Linear	**-19.6 (-21.1, -18.1)**	**-19.6 (-21.1, -18.1)**	**-19.6 (-21.1, -18.1)**	**-19.6 (-21.1, -18.1)**	**-19.6 (-21.2, -18.1)**
Quadratic	**2.4 (2.2, 2.7)**	**2.4 (2.2, 2.7)**	**2.4 (2.2, 2.7)**	**2.4 (2.2, 2.7)**	**2.4 (2.2, 2.7)**
Sex Male		Ref	Ref	Ref	Ref
Female		-2.5 (-5.2, 0.1)	-1.9 (-4.6, 0.7)	-1.9 (-4.5, 0.7)	-1.9 (-4.5, 0.7)
Age at diagnosis		0.1 (-0.2, 0.4)	0.1 (-0.2, 0.4)	0.1 (-0.2, 0.4)	0.1 (-0.3, 0.3)
Ethnicity					
White			Ref	Ref	Ref
Mixed			4.1 (-0.9, 9.1)	4.6 (-0.4, 9.7)	3.9 (-1.0, 8.9)
Black			3.3 (-0.9, 7.4)	3.7 (-0.5, 8.0)	4.0 (-0.1, 8.1)
African-Somali			4.1 (-0.8, 9.1)	**5.1 (0.1, 10.1)**	4.5 (-0.5, 9.4)
Bangladeshi			**8.8 (4.3, 13.3)**	**9.3 (4.7, 14.0)**	**9.4 (4.8, 13.9)**
Asian-Other			**5.8 (2.3, 9.4)**	**5.7 (2.2, 9.3)**	**3.9 (0.2, 7.6)**
Socioeconomic status					
Quartile 1 (poorest)				Ref	Ref
Quartile 2				3.2 (-0.3, 6.7)	2.6 (-0.8, 6.1)
Quartile 3				3.9 (0.2, 7.6)	2.5 (-1.1, 6.3)
Quartile 4 (richest)				2.5 (-1.5, 6.5)	2.7 (-1.3, 6.7)
Paediatric Diabetes Clinic					
Clinic 1					Ref
Clinic 2					1.7 (-2.1, 5.4)
Clinic 3					**6.1 (2.8, 9.3)**

Interclass Correlation (ICC)	0.77	0.77	0.76	0.76	0.76
Goodness of fit					
Aikake information criterion (AIC)	8690.614	8690.926	8682.079	8683.001	8673.485
Bayesian information criterion (BIC)	8725.161	8735.345	8751.175	8766.902	8767.257
-2LL	8676	8672	8654	8648	8635

Table 3. Multilevel model for change in $HbA1_c$ during the first six months post-diagnosis adjusted for pH levels at diagnosis

Covariates	**Model 1**	**Model 2:**
Fixed effects		
Constant/intercept	**202.1 (134.2, 270.9)**	**203.7 (136.4, 271.1)**
Duration Linear	**-20.6 (-22.5, -18.8)**	**-20.8 (-22.6, -18.9)**
Quadratic	**2.5 (2.3, 2.8)**	**2.6 (2.3, 2.8)**
Sex Male	Ref	Ref
Female	-0.5 (-3.5, 2.5)	-0.4 (-3.3, 2.6)
Age at diagnosis	0.1 (-0.2, 0.5)	0.1 (-0.3, 0.4)
pH at diagnosis	**-15.2 (-24.7, -5.7)**	**-15.7 (-25.1, -6.3)**
Ethnicity		
White	Ref	Ref
Mixed	**7.1 (1.7, 12.6)**	**6.5 (1.1, 11.9)**
Black	**4.2 (0.2, 8.8)**	**4.6 (0.9, 9.1)**
African-Somali	4.7 (-0.9, 10.3)	5.2 (-0.4, 10.9)
Bangladeshi	**10.1 (4.9, 15.3)**	**10.6 (5.4, 15.7)**
Asian-Other	**7.5 (3.4, 11.5)**	**5.3 (1.8, 9.4)**
Socioeconomic status		
Quartile 1 (poorest)		
Quartile 2		2.2 (-1.7, 6.1)
Quartile 3		1.9 (-2.1, 5.9)
Quartile 4 (richest)		2.7 (-1.9, 7.3)
Paediatric Diabetes Clinic		
Clinic 1		Ref
Clinic 2		1.9 (-2.5, 6.3)
Clinic 3		**6.1 (2.4, 9.6)**
Goodness of fit		
Aikake information criterion (AIC)	6434.644	6430.969
Bayesian information criterion (BIC)	6504.184	6523.688

Figure 1. Predicted glycaemic control ($HbA1_c$) trajectories during the first six months post-diagnosis by ethnicity.



Trajectories are estimated for a sample group with a mean age of diagnosis = 9 years and IMD = quartile 3.

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