

Young people with Type 1 Diabetes of non-white ethnicity and lower socioeconomic status have poorer glycaemic control in England and Wales

Amal R. Khanolkar^{1,2}, Rakesh Amin¹, David Taylor-Robinson^{3, 1}, Russell M. Viner¹, Justin T. Warner^{4*} and Terence Stephenson¹

Short title: Type 1 diabetes, ethnicity, deprivation and glycaemic control

1. Institute of Child Health, University College London (UCL), UK
2. Institute of Environmental Medicine, Karolinska Institutet, Sweden
3. Institute of Psychology, Health and Society, University of Liverpool, UK
4. Department of Child Health, University Hospital of Wales, Cardiff, UK,

*On behalf of the National Paediatric Diabetes Audit (NPDA) and the Royal College of Paediatrics and Child Health (RCPCH).

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.

Conflicts of Interest

The authors have no conflicts of interest to declare.

Word count - 3300

Corresponding author: Amal R. Khanolkar

Institute of Child Health, UCL

30, Guildford Street, London, UK

Ph: +44(0)20 79052938

a.khanolkar@ucl.ac.uk

Novelty statement

- This is the largest study in the UK to investigate associations between ethnicity, deprivation and glycaemic-control using a large nationally representative sample of children with type 1 diabetes, from all major ethnic groups.
- Black and mixed ethnicity children had poorest glycaemic-control. Greater deprivation was associated with worse glycaemic-control in all ethnicities.
- Ethnicity, deprivation and treatment regimen were independently associated with glycaemic control. Results suggest that [increasing insulin pump use in ethnic minority and more deprived children will help these groups achieve better glycaemic control.](#)

Abstract

Background The impact of ethnicity and socioeconomic status (SES) on glycaemic control during childhood Type 1 Diabetes (T1D) is poorly understood in England/Wales.

Methods We studied 18,478 participants with T1D (<19 years) attending diabetes clinics in England/Wales and included in 2012-13 National Paediatric Diabetes Audit. Self-identified ethnicity was categorized as white, Asian, black, mixed, other and 'not-stated' (did not to divulge ethnicity). A small area measure of SES was estimated from Index of Multiple Deprivation. Multiple linear regression was used to assess impact of ethnicity and SES on glycaemic control (mean HbA_{1c} levels) accounting for age, gender and diabetes duration. Impact of insulin pump use on the ethnicity/SES-HbA_{1c} associations was tested in 13,962 children.

Results All ethnic minorities had higher mean HbA_{1c} compared to white children, with largest differences observed in black and mixed ethnicities (8mmol/mol [2.9%], 95%CI 5-11 and 7mmol/mol [2.8%], 5-9 respectively). Lower SES was associated with higher mean HbA_{1c} with a dose effect. The lowest SES group had 7mmol/mol [2.8%] (6-8) higher mean HbA_{1c} compared to the highest SES group, adjusted for ethnicity. Estimates for ethnicity were attenuated but significant on adjustment for SES. Less non-white (white 20.3 vs Black 5.5%) and deprived (Least deprived 21.1 vs most deprived 13.2%) children were on insulin pump therapy. Ethnicity and SES remained significant predictors of HbA_{1c} after accounting for insulin pump use.

Conclusion The association between ethnicity and glycaemic control persists after adjustment for deprivation and pump use. An alternative approach to intensive insulin therapy might benefit these vulnerable children.

Keywords: Type 1 diabetes, children, ethnicity, socioeconomic status, glycosylated haemoglobin, England

Introduction

Inequalities in Type 1 Diabetes (T1D) treatment and disparities in glycaemic control in children and young people are well documented and are associated with severe short-term (hypoglycaemia and diabetic ketoacidosis) and long-term complications (nephropathy, retinopathy, neuropathy and cardiovascular disease) (1). Additionally, poor glycaemic control impacts adversely on family dynamics and tracks from childhood into adulthood increasing the lifetime risk of vascular complications and significantly reducing life expectancy (2, 3). Despite robust evidence showing the efficacy of intensive insulin regimens in improving outcomes, inequalities in treatments and outcomes related to T1D remain strong even in countries with tax-funded universal healthcare systems (4-8).

Research from outside the UK shows that ethnic minority children and those from lower Socioeconomic Status (SES) have consistently poorer glycaemic control (4, 6, 7, 9-11). Studies on health inequalities related to T1D treatment and glycaemic control in England and Wales have been conducted on smaller study samples often restricted to particular clinics or regions (4, 12-14). The role of ethnicity has not been comprehensively studied often due to low numbers of ethnic minorities. Some studies also showed contradictory results (14). Previously observed differences in glycaemic control between ethnic groups could be a reflection of greater deprivation in ethnic minorities and/or poorer access to intensive treatment regimens. Additionally, most of the evidence for inequalities in T1D health care processes, treatment and outcomes originate from North America, northern Europe and Australasia, where ethnic groupings and/or healthcare differ to the UK (5, 6, 15-18). Thus there is a strong need to investigate social and ethnic differences in glycaemic control in children in England and Wales.

The main aim of this study is to investigate whether ethnicity and SES are associated with glycaemic control in children with T1D using a nationwide population-based register that includes almost all T1D people under nineteen years of age representing all major ethnic groups with reliable measures of ethnicity and deprivation in England and Wales. We wanted to identify ethnic minority groups with the worst glycaemic control. Additionally, we wanted to investigate if ethnic differences in glycaemic control are independent of SES and treatment regimen and if ethnicity interacts with SES in its association with

glycemic control. Differences if any in glycaemic control between white and ethnic minority children that appear to be mediated by modifiable factors such as treatment regimen will provide opportunities for targeted interventions.

Methods

Data for this study was obtained from the National Paediatric Diabetes Audit (NPDA) for England and Wales(19). The NPDA is commissioned and sponsored by the Healthcare Quality Improvement Partnership as part of their National Clinical Audit Programme and was started in 2004 and reached 100% participation rate in 2012. It includes demographic and outcome data on almost all children with type 1 and type 2 diabetes under 19 years of age and treated at one of the 178 paediatric diabetic clinics. This study was based on data collected during the 2012-13 audit year (1st April 2012 – 31st March 2013). Inclusion criteria comprised: a diagnosis of T1D (for a minimum of six months to allow for stabilisation of diabetes control), the participant had to be <19 years of age on the first day of the audit, a minimum of one visit to a clinic during the audit year and have recorded information on ethnicity and postcode. During the 2012-13 audit year, 23,097 people <19 years of age were recorded as having T1D, of whom 19,122 people were eligible to be included in the study.

Study measurements

As per recommendations from the National Institute of Health and Care Excellence (NICE), a child with T1D is offered an integrated package of care by a multidisciplinary team at a clinic 2-4 times per year (more frequent when glycaemic control is poor). The team consists of paediatric endocrinologists/diabetologists, diabetes specialist nurses, dieticians and interpreters if needed. HbA_{1c} levels along with height and weight are meant to be recorded at each visit. Other clinical parameters such as blood lipids are recorded annually. All demographical and clinical parameters are measured systematically across all clinics enabling comparison.

The outcome of interest was glycaemic control measured by HbA_{1c} levels. HbA_{1c} values recorded as percentages were converted to mmol/mol using the formula: $(\text{HbA}_{1c} \text{ value in percentage} - 2.15) * 10.929$. We calculated mean HbA_{1c} values from all clinic visits in the audit year for each child. Both age at diagnosis and age at clinic visit were calculated by subtracting the date of diagnosis from date of birth and date of clinic visit from date of birth respectively. Duration of diabetes was calculated by subtracting the date at first visit

in the audit year from the date of diagnosis of T1D. Insulin treatment regimen; daily injections (non-pump therapy) or continuous subcutaneous insulin infusion (CSII) (pump therapy) was recorded at each visit. The first recorded entry in the audit year for ethnicity and treatment regimen was used in the analysis.

For this study the main predictors of diabetes control were ethnicity and SES. Participants (or their parents) were asked to self-identify their ethnicity when they visited a clinic. They were given the option to choose one of the fifteen categories as recommended by the Information Standards Board (ISB) for Health and Social Care. Participants were also given the option to decline identifying their ethnicity. For the purpose of this study, the fifteen ethnic categories were collapsed into six broad groups: white, Asian, black, mixed, other and 'not stated' (those who declined).

Socioeconomic status (SES) was derived from postcode using indices of Multiple Deprivation (IMD) 2010 for England, and Welsh Indices of Multiple Deprivation 2008 for Wales. Although these two countries use slightly differing indices to define deprivation, adjustment can be made to align the two techniques (20). The IMD is a small geographical area measure of deprivation. IMD is a multidimensional index 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 Super Output Areas (LSOA) with each area comprising 1500 individuals on average. IMD rank scores were grouped into quintiles for analysis, with the first and fifth quintiles corresponding to the least and most deprived respectively.

Of the 19,122 children eligible to be included, 24 lacked data on gender, 360 on SES, and 260 on HbA_{1c}. This left 18,478 children with T1D (96.6% of the eligible sample) with data on age, gender, diabetes duration, ethnicity and SES and were included in the main analysis to assess associations between ethnicity, SES and glycaemic control. A smaller sample of 13,962 participants with data on treatment regimen and all other covariates was included in a sub-analysis to assess the effects of treatment regimen on associations between ethnicity, SES and glycaemic control. We investigated whether the 4,516 excluded participants due to missing data on treatment regimen differed from the 13,962 children included in the sub-analysis.

We compared the two groups on all covariates using univariable linear regression or Chi square tests for differences of proportions for continuous and categorical variables respectively.

Statistical analysis

Continuous variables are presented as mean values with standard deviations and categorical variables as frequencies. Associations between ethnicity and SES and other covariates were analysed using univariable linear regression or Chi square tests for differences of proportions for continuous and categorical variables respectively.

Multivariable linear regression models were fitted with mean HbA_{1c} as the principle outcome and ethnicity or SES as the primary predictor (Models 1 and 2 respectively) to assess associations. Model 3 included both predictors to assess mutually adjusted associations of ethnicity and SES.. All models were adjusted for child's age during the audit year (years), gender and diabetes duration (years). For linear regression analyses, assumptions of linearity for continuous variables and constant variance of the standardized residuals were assessed by plotting the residuals against the fitted values. Model fit of the three models was compared using R² which represents the proportion of variation in HbA_{1c} explained by the model.

Model 1, with SES as the main predictor, was then run stratified by ethnicity to assess whether the association between SES and HbA_{1c} were similar across all ethnic groups. Interactions between ethnicity and SES were tested using likelihood ratios tests. Robust standard errors allowing for clustering of children within clinics were used for all linear regression models.

A sub-analysis was performed using adjustments for the same covariates as in Model 3 above but restricted to the smaller study sample of 13,962 children with information on treatment regimen to assess if the latter could explain differences in glycaemic control by ethnicity or SES.

Further analysis was performed for the association between ethnicity, SES and HbA_{1c} (using the same adjustments for covariates as described above) by fitting linear multilevel models which take into account

random effects that vary across clinics. As multilevel modelling yielded the same results as multivariable linear regression, we present results from the former in Supplemental Table 1. All statistical analyses were conducted using STATA 13 (College Station, TX, USA).

The study protocol was reviewed by University College London (UCL) Research Ethics Committee which decided that ethics approval was not required. The NPDA has section 251 approval to collect patient identifiable information for the purpose of audit. For this study all participants were pseudonymised making them unidentifiable. The study is registered with the R&D office, Institute of Child Health, UCL, (Project number 14PP08).

Results

The mean age of the study sample was 12.76 years (range 1.3 to 19.0 years). Age at first visit in the audit year and age at diagnosis differed by ethnicity. On average, ethnic minority children were diagnosed younger than white children but differences were small and clinically not relevant. There was no statistically significant difference in diabetes duration between ethnic groups. In comparison to children of white ethnicity, children of all other ethnicities had higher mean HbA_{1c} levels with highest levels observed in black and mixed ethnicity groups (80mmol/mol [9.5%] and 79mmol/mol [9.4%] respectively, Table 1). In comparison to the white group, all ethnicities had higher proportions of children in the most deprived socioeconomic group (IMD quintile 5). More than half of all black children were in the most deprived quintile (52.6%, Table 1). Significant differences were observed in proportions of children on insulin pump therapy by ethnicity. For those with information on pump use, the white group had the largest proportion on insulin pump therapy (20.3%), whereas the black and 'not stated' groups had the lowest proportions (5.5% and 5.0% respectively).

On average age at first visit in the audit year was slightly lower in the most deprived groups (quintiles 4 and 5) compared to the least deprived group (quintile 1), however differences were clinically not relevant. There were no differences in diabetes duration across the quintiles of deprivation. We observed a strong positive association between deprivation and mean HbA_{1c} levels (the most deprived quintile had on average 7mmol/mol [2.8%] higher HbA_{1c} compared to the least deprived quintile (Table 2)). Similarly, the proportions of children on insulin pump therapy decreased with increasing deprivation (13.2% in the most deprived group were on insulin pump therapy compared to 21.2% in least deprived group, Table 2).

The 4,516 children excluded from sub-analysis because of missing data on treatment regimen did not differ significantly in age and mean HbA_{1c} from the 13,962 included in the analysis. The two groups differed significantly in age at diagnosis (7.53 vs 7.32 years, $P<0.05$), diabetes duration (5.26 vs 5.41 years, $P<0.005$) and mean IMD scores (20.85 vs 21.74, $P<0.05$) but differences were unlikely to be meaningful.

Both ethnicity and SES were independently and consistently associated with HbA_{1c} levels. However, the model with only SES explained slightly more of the variance in HbA_{1c} level compared to the model with only ethnicity ($R^2=0.09$ and 0.07 respectively). Compared to the white group, all ethnic minorities (except the 'not stated' group) had higher mean HbA_{1c} after adjustment for age, gender and diabetes duration. Largest differences were observed in black and mixed ethnic children (8mmol/mol [2.9%], 95% CI 5.07-10.60 and 7mmol/mol [2.8%], 4.55-9.08 respectively, after adjustment for age, gender and diabetes duration, Model 1, Table 3). SES was significantly associated with glycaemic control with a strong apparent dose-effect across quintiles. In the regression model with SES only, being in quintiles 2 to 5 was associated with higher mean HbA_{1c}, with the most deprived group (quintile 5) having the highest HbA_{1c} (on average 7mmol/mol [2.8%] higher HbA_{1c} compared to the least deprived group, Model 2, Table 3). However, after controlling for SES, (i.e. the model which included both ethnicity and SES, Model 3, Table 3), the estimates for all ethnic groups were attenuated considerably and were no longer significant for the 'other' group. In contrast, the estimates for deprivation quintiles were only marginally attenuated on adjustment for ethnicity (Model 3, Table 3).

The association between SES and glycaemic control was similar across ethnic groups in stratified analysis, i.e. having a lower SES was associated with higher mean HbA_{1c} irrespective of ethnicity. However, being in the lowest SES groups (quintiles 4 and 5) and of Asian, mixed, other and 'not stated' ethnicity was associated with significantly higher mean HbA_{1c} compared to being in the lowest SES groups and having white ethnicity (Table 4). For example, being white and in the lowest SES group (quintile 5) was associated with 6mmol/mol (2.7%) higher HbA_{1c} when compared to the highest SES group (quintile 1). However, being in the lowest SES group and belonging to Asian (7mmol/mol [2.8%], 2.52-11.28), mixed (11mmol/mol [3.2%], 6.31-16.21) and 'not stated' (10mmol/mol [3.1%], 7.67-12.65) ethnic groups was associated with much higher HbA_{1c} levels (Table 4). The interaction test between ethnicity and SES was statistically significant ($P=0.006$).

In models restricted to those with information on insulin pump therapy ($N=13,962$), both ethnicity and SES remained independently associated with HbA_{1c} levels with a pattern very similar to that observed in

regression models with the entire study population. However, on adjustment for pump therapy being in the Asian group or in deprivation quintile 2 was no longer significantly associated with higher HbA_{1c} (Model 4, Table 5). Adjustment for pump therapy marginally attenuated the estimates for the black and the more deprived groups (quintiles 3 to 5). On average children on insulin pump therapy had lower mean HbA_{1c} compared to those on other therapies (-5 [-2.6%], -6.16 -3.67), Model 4, Table 5.

Discussion

Results from this study indicate that minority ethnicity, deprivation and access to insulin pump therapy are each independently associated with poorer glycaemic control in this large national sample. Black and mixed ethnicity children had the poorest glycaemic control. Low SES was associated with worse glycaemic control in all ethnicities. Whilst the estimates of ethnicity, SES and treatment regimen are smaller in the mutually adjusted models, they remain for black and mixed children and are attenuated for Asian children. This suggests that for Asians, deprivation and lack of access to pumps maybe the main way in which ethnic group membership affects glycaemic control. However, the observed poorer glycaemic control in black and mixed children is probably a result of factors not accounted for, such as cultural/lifestyle and/or those relating to healthcare access above and beyond insulin pump use.

Few UK studies have examined the combined effect of ethnicity and SES on glycaemic control in children. These studies were smaller in size, restricted to fewer ethnicities, small geographical regions and clinics (4, 12, 13). Due to small sample sizes, studies also combined all ethnic minorities into one group for comparisons with the white group (12). Consistent with our findings, previous UK studies found that children of African or black ethnicity and those with greater deprivation (lower SES) had the highest mean HbA_{1c} levels or worse glycaemic control (4, 13). However, ours is the first to show the inverse association between SES and glycaemic control is present with a dose-effect in most ethnic groups in England and Wales.

This is the largest study to date in the UK to have analysed ethnic and SES differences in glycaemic control in children with T1D. It is the first study to analyse differences in glycaemic control in all six major ethnic groups corresponding to official standard ethnicity classifications. Additionally, ethnicity is self-identified which is considered the 'gold standard' in studies on ethnicity and health (22). The IMD scores have been shown to be associated with several health outcomes in previous studies and is considered to be the standard benchmark for UK governmental health and social policy (23).

The NPDA data is collected annually by participating paediatric diabetes clinics. Each clinic submits data on all participants under their care to a centralised database which helps minimize selection bias. Although the

NPDA cannot verify whether 100% of children with diabetes in England and Wales are included in the audit, it is estimated to represent in excess of 95% of cases and is nationally representative.

Previous studies showed that T1D children from single parent households have consistently worse glycaemic control compared to those living with both parents, a factor we were unable to account for in our analyses (24, 25). The NPDA does not collect data on physical activity and diet which might explain the observed differences. We had significant missing data on treatment regimen. However, as sensitivity analyses revealed, we believe our results from the sub-analysis on treatment regimen can be generalized to the entire study population.

The category 'Not Stated' is used when an individual has been asked for but declined to provide information on their ethnicity. However, the NPDA cannot verify that this is the case and it may contain individuals where the ethnicity is unknown. This group appeared to be similar to the white group when compared on mean HbA_{1c}, age at diagnosis, age at visit, proportion of boys and SES. We observed no significant differences between 'Not stated' and white groups in regression models indicating that the former is composed of mostly white children. However, in the stratified analysis, children with 'not stated' ethnicity and belonging to the lowest SES groups had much higher mean HbA_{1c} compared to white children of the same SES groups (10mmol/mol [3.1%] vs 6mmol/mol [2.7%] respectively). In all likelihood, this group is a heterogeneous mix of children belonging to different ethnicities.

The observed independent association between ethnicity and glycaemic control in this study population could be attributed to cultural and lifestyle differences between ethnicities such as diet and physical activity which impact on glycaemic control (26). As previously reported, certain ethnic groups might favour a particular treatment regimen and older participants might be more reluctant to change to new therapies (4). Cultural barriers might lead to less effective communication between healthcare providers and families of ethnic minority diabetic children, especially for those that do not have English as their first language. Another explanation is the evident lower insulin pump use among ethnic minorities and lower SES groups (27). However, accounting for pump use only marginally attenuated the observed ethnicity/SES estimates. Observed differences could also reflect in part, biological differences between ethnic groups such as that

related to haemoglobin glycation (28, 29). Also higher cumulative stress in ethnic minorities might lead to alterations in allostatic load (physiologic response to chronic exposure to stress), which in turn influences cortisol secretion (30). This can lead to differences in glucose regulation (31).

The National Health Service provides free medical care in the country and thus direct costs of T1D treatment should have no bearing on patient's families and, theoretically, income should not be an impediment for access to treatment. For lower SES families, cost of transport to and from clinics is reimbursed. However, the relative economic impact of the carer taking time off work is likely to be greater in low income families and such losses are not reimbursed. However, the strong differences observed in glycaemic control between the lowest and highest SES groups despite free access to healthcare points to possible cultural differences in how treatment methods are managed at home or by their clinical team and/or to barriers in access to better treatments. Additionally, it appears that SES is a stronger determinant of glycaemic control than ethnicity as it was consistently evident in all ethnicities.

In order to improve glycaemic control, better consideration of the needs of all ethnic groups and those belonging to lower SES need to be taken into account. This could involve strengthening the implementation of insulin pump therapy, reviewing how healthcare professionals interact with patients and their families, and a deeper understanding of cultural difference in attitudes to disease management. Further studies are needed to better understand underlying mechanisms which could explain poorer glycaemic control in black and mixed ethnicity children.

Acknowledgements

We would like to acknowledge the paediatric diabetes clinics who submit data to the NPDA and participants and families for making this study possible. We would also like to thank the RCPCH and HQIP for facilitating access to the NPDA data needed for this study.

Transparency declaration

The lead author (Amal R. Khanolkar) affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

References

1. Stanescu DE, Lord K, Lipman TH. The Epidemiology of Type 1 Diabetes in Children. *Endocrin Metab Clin.* 2012;41(4):679-+.
2. Aiello LP, Group DER. Diabetic retinopathy and other ocular findings in the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. *Diabetes care.* 2014;37(1):17-23.
3. Nordwall M, Abrahamsson M, Dhir M, Fredrikson M, Ludvigsson J, Arnqvist HJ. Impact of HbA1c, followed from onset of type 1 diabetes, on the development of severe retinopathy and nephropathy: the VISS Study (Vascular Diabetic Complications in Southeast Sweden). *Diabetes care.* 2015;38(2):308-15.
4. Thompson RJ, Agostini K, Potts L, Luscombe J, Christie D, Viner R, et al. Deprivation and ethnicity impact on diabetes control and use of treatment regimen. *Diabetic Med.* 2013;30(4):491-4.
5. Cutfield SW, Derraik JGB, Reed PW, Hofman PL, Jefferies C, Cutfield WS. Early Markers of Glycaemic Control in Children with Type 1 Diabetes Mellitus. *Plos One.* 2011;6(9).
6. Fredheim S, Delli A, Rida H, Drivvoll AK, Skrivarhaug T, Bjarnason R, et al. Equal access to health care may diminish the differences in outcome between native and immigrant patients with type 1 diabetes. *Pediatr Diabetes.* 2014;15(7):519-27.
7. Deladoey J, Henderson M, Geoffroy L. Linear Association Between Household Income and Metabolic Control in Children With Insulin-Dependent Diabetes Mellitus Despite Free Access to Health Care. *J Clin Endocr Metab.* 2013;98(5):E882-E5.
8. Lachin JM, Orchard TJ, Nathan DM, Group DER. Update on cardiovascular outcomes at 30 years of the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. *Diabetes care.* 2014;37(1):39-43.
9. Zuijdwijk CS, Cuerden M, Mahmud FH. Social Determinants of Health on Glycemic Control in Pediatric Type 1 Diabetes. *J Pediatr-U.S.* 2013;162(4):730-5.
10. Springer D, Dziura J, Tamborlane WV, Steffen AT, Ahern JH, Vincent M, et al. Optimal control of type 1 diabetes mellitus in youth receiving intensive treatment. *J Pediatr-U.S.* 2006;149(2):227-32.
11. Carter PJ, Cutfield WS, Hofman PL, Gunn AJ, Wilson DA, Reed PW, et al. Ethnicity and social deprivation independently influence metabolic control in children with type 1 diabetes. *Diabetologia.* 2008;51(10):1835-42.
12. Hine P, Senniappan S, Sankar V, Amin R. Deprivation impedes success of insulin intensification in children and adolescents with Type 1 diabetes; longitudinal linear mixed modelling of a retrospective observational cohort. *Diabetic Med.* 2011;28(3):338-44.
13. Dias RP, Brown F, Wyatt C, Cheema S, Allgrove J, Amin R. The effect of insulin intensification in children and young persons with Type 1 diabetes differs in relation to ethnic group; a prospective observational study. *Diabetic Med.* 2013;30(4):495-501.
14. Baumer JH, Hunt LP, Shield JPH. Social disadvantage, family composition, and diabetes mellitus: prevalence and outcome. *Arch Dis Child.* 1998;79(5):427-30.
15. Hatherly K, Smith L, Overland J, Johnston C, Brown-Singh L, Waller D, et al. Glycemic control and type 1 diabetes: the differential impact of model of care and income. *Pediatr Diabetes.* 2011;12(2):115-9.
16. Willi SM, Miller KM, DiMeglio LA, Klingensmith GJ, Simmons JH, Tamborlane WV, et al. Racial-ethnic disparities in management and outcomes among children with type 1 diabetes. *Pediatrics.* 2015;135(3):424-34.
17. Jacobsen JJ, Black MH, Li BH, Reynolds K, Lawrence JM. Race/ethnicity and measures of glycaemia in the year after diagnosis among youth with type 1 and type 2 diabetes mellitus. *J Diabetes Complicat.* 2014;28(3):279-85.
18. Wintergerst KA, Hinkle KM, Barnes CN, Omoruyi AO, Foster MB. The impact of health insurance coverage on pediatric diabetes management. *Diabetes Res Clin Pr.* 2010;90(1):40-4.
19. National Paediatric Diabetes Audit Report: Royal College of Paediatrics and Child Health; 2015 [cited 2015]. Available from: <http://www.rcpch.ac.uk/child-health/standards-care/clinical-audit-and-quality-improvement/national-paediatric-diabetes-au-1>.
20. G.A. PRAA. UK indices of multiple deprivation – a way to make comparisons across constituent countries easier. Office of National Statistics, 2012.

21. The English Indices Of Deprivation 2010. Government DfCaL; 2011.
22. Bhopal R. Ethnicity, race, and health in multicultural societies. Foundations for better epidemiology, public health, and health care.: Oxford University Press; 2008.
23. Niggebrugge A, Haynes R, Jones A, Lovett A, Harvey I. The index of multiple deprivation 2000 access domain: a useful indicator for public health? *Social science & medicine*. 2005;60(12):2743-53.
24. Swift EE, Chen RS, Hershberger A, Holmes CS. Demographic risk factors, mediators, and moderators in youths' diabetes metabolic control. *Ann Behav Med*. 2006;32(1):39-49.
25. Thompson SJ, Auslander WF, White NH. Influence of family structure on health among youths with diabetes. *Health Soc Work*. 2001;26(1):7-14.
26. Kummer S, Stahl-Pehe A, Castillo K, Bachle C, Graf C, Strassburger K, et al. Health Behaviour in Children and Adolescents with Type 1 Diabetes Compared to a Representative Reference Population. *Plos One*. 2014;9(11).
27. Senniappan S, Hine P, Tang W, Campbell J, Bone M, Sankar V, et al. The effect of socioeconomic deprivation on efficacy of continuous subcutaneous insulin infusion: a retrospective paediatric case-controlled survey. *Eur J Pediatr*. 2012;171(1):59-65.
28. Ziemer DC, Kolm P, Weintraub WS, Vaccarino V, Rhee MK, Twombly JG, et al. Glucose-Independent, Black-White Differences in Hemoglobin A(1c) Levels. *Ann Intern Med*. 2010;152(12):770-+.
29. Maruthur NM, Kao WHL, Clark JM, Brancati FL, Cheng CY, Pankow JS, et al. Does Genetic Ancestry Explain Higher Values of Glycated Hemoglobin in African Americans? *Diabetes*. 2011;60(9):2434-8.
30. Szanton SL, Gill JM, Allen JK. Allostatic load: A mechanism of socioeconomic health disparities? *Biol Res Nurs*. 2005;7(1):7-15.
31. Weigensberg MJ, Toledo-Corral CM, Goran MI. Association between the metabolic syndrome and serum cortisol in overweight Latino youth. *J Clin Endocr Metab*. 2008;93(4):1372-8.

Table 1. Characteristics of the 18,478 children and young people with T1D included in the study by ethnicity

Characteristics	Ethnicity							P Value**
	White N=13,582	Asian N=874	Black N=323	Mixed N=441	Other N=248	Not Stated N=3,010	Total N=18,478	
Age at visit* (years)	12.86 (3.62)	12.08 (3.86)	11.90 (3.81)	12.02 (3.93)	12.04 (3.93)	12.73 (3.64)	12.76 (3.64)	<0.0001
Age at diagnosis (years)	7.42 (3.87)	6.60 (3.68)	6.85 (3.96)	6.84 (3.81)	6.80 (3.96)	7.57 (3.823)	7.37 (3.86)	<0.0001
Boys (%)	52.9	49.5	47.4	51.9	46.8	51.8	52.4	<0.05
Diabetes duration (years)	5.44 (3.57)	5.48 (3.60)	5.05 (3.30)	5.18 (3.55)	5.24 (3.57)	5.15 (3.50)	5.38 (3.55)	NS
Mean HbA1c (mmol/mol)	73 (17)	76 (17)	80 (19)	79 (20)	76 (18)	73 (17)	74 (17)	<0.0001
Mean HbA1c (%)	8.8 (3.7)	9.1 (3.7)	9.5 (3.9)	9.4 (4.0)	9.1 (3.8)	8.8 (3.7)	8.9 (3.7)	<0.0001
Socioeconomic status (IMD score)***	20.31 (14.92)	31.56 (16.80)	35.47 (13.78)	27.91 (16.2)	32.31 (16.84)	20.78 (15.45)	21.52 (15.53)	<0.0001
Proportion in most deprived SES group (IMD quintile 5)	17.1	39.8	52.6	36.3	44	16.4	19.4	<0.0001
Insulin pump therapy (%)*	20.3	12.1	5.5	17.4	18.8	5.0	17.3	<0.0001

Values are means (SD or percentages)

*Age at first clinic visit in the audit year

**P values are for a test of equal means or proportions

***A lower IMD score indicates lower deprivation (or higher socioeconomic status)

NS – Not statistically Significant.

*Proportions shown are for a smaller sample of 13,962 children.

Table 2. Characteristics of 18,478 children and young people with T1D by Socioeconomic Status (IMD quintile)

Characteristics	Socioeconomic status – Index of Multiple Deprivation (IMD)						P Value**
	Quintile 1 N=3,755	Quintile 2 N=3,737	Quintile 3 N=3,708	Quintile 4 N=3,684	Quintile 5 N=3,594	Total N=18,478	
Age at visit* (years)	12.87 (3.58)	12.86 (3.65)	12.87 (3.63)	12.62 (3.75)	12.55 (3.67)	12.76 (3.66)	<0.001
Age at diagnosis (years)	7.58 (3.9)	7.45 (3.87)	7.39 (3.82)	7.22 (3.90)	7.23 (3.83)	7.37 (3.86)	<0.001
Boys (%)	53.1	52.7	52.2	52.4	51.4	52.4	NS
Diabetes duration (years)	5.28 (3.53)	5.40 (3.54)	5.48 (3.58)	5.40 (3.58)	5.31 (3.51)	5.38 (3.55)	NS
Mean HbA1c (mmol/mol)	70 (15)	72 (16)	74 (18)	76 (18)	77 (18)	74 (17)	<0.0001
Mean HbA1c (%)	8.6 (3.5)	8.7 (3.6)	8.9 (3.8)	9.1 (3.8)	9.2 (3.8)	8.9 (3.7)	<0.0001
Insulin pump therapy (%)*	21.2	20.9	16.9	14.3	13.2	17.3	<0.0001

Values are means (SD or percentages).

*Age at first clinic visit in the audit year

**P values are for a test of equal means or proportions

NS – Not statistically Significant.

*Proportions shown are for a smaller sample of 13,962 children.

Table 3. Results from multivariate linear regression – assessing effects of ethnicity and SES on glycaemic control in children with type 1 diabetes in England and Wales in 2012-13

	Model 1 ^a	Model 2 ^b	Model 3 ^c
Ethnicity	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)
White	Reference	-	Reference
Asian	4 2-5	-	2 0-3
Black	8 5-11	-	5 3-8
Mixed	7 5-9	-	6 3-8
Other	3 1-6	-	1 (-)1-4
Not Stated	0 (-)1-2	-	0 (-)1-2
Socioeconomic Status (IMD quintile)			
Quintile 1	-	Reference	Reference
Quintile 2	-	2 1-2	2 1-2
Quintile 3	-	4 3-5	4 3-5
Quintile 4	-	6 5-8	6 5-7
Quintile 5	-	7 6-8	7 6-8

^aModel 1: adjusted for age, gender, diabetes duration and ethnicity, R²=0.07

^bModel 2: adjusted for age, gender, diabetes duration and socioeconomic status, R²=0.09

^cModel 3: adjusted for age, gender, diabetes duration, ethnicity and socioeconomic status, R²=0.10

Text in bold indicates statistical significance at p<0.05

Table 4. Results from multivariate linear regression – assessing effects of SES on glycaemic control in children with type 1 diabetes in England and Wales in 2012-13, analysis stratified by ethnic group

	Ethnicity					
	White	Asian	Black	Mixed	Other	Not stated
Socioeconomic Status (IMD quintile)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)
Quintile 1	Reference	Reference	Reference	Reference	Reference	Reference
Quintile 2	1 1-2	2 (-)3-7	2 (-)12-15	(-)1 (-)6-5	7 (-)3-17	2 0-4
Quintile 3	4 3-4	6 2-11	6 (-)6-18	4 (-)2-10	6 (-)4-16	5 3-7
Quintile 4	6 5-7	5 0-10	7 (-)5-18	12 6-18	10 1-19	7 5-10
Quintile 5	6 5-7	7 3-11	4 (-)6-14	11 6-16	9 0-18	10 8-13
<i>P</i> *	<0.001	<0.001	N.S.	<0.001	N.S.	<0.001

All models adjusted for age, gender and diabetes duration

*Test for trend

N.S. – Statistically Not Significant

Text in bold indicates statistical significance at $p < 0.05$

Table 5. Results from multivariate linear regression – assessing effects of ethnicity and SES on glycaemic control in 13,962 children with type 1 diabetes in England and Wales in 2012-13 and data on treatment regimen

	Model 1 ^a	Model 2 ^b	Model 3 ^c	Model 4 ^d
Ethnicity	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)	HbA1c (mmol/mol) difference from reference (95 % CI)
White	Reference	-	Reference	Reference
Asian	3 1-4	-	1 (-)0-3	1 (-)1-3
Black	7 5-10	-	5 2-8	5 2-8
Mixed	7 4-9	-	6 3-8	5 3-8
Other	3 0-6	-	1 (-)1-4	1 (-)2-4
Not Stated	1 (-)1.03-2.37	-	1 (-)1-2	0 (-)2-1
Socioeconomic Status (IMD quintile)				
Quintile 1	-	Reference	Reference	Reference
Quintile 2	-	1 0-2	1 0-2	1 0-2
Quintile 3	-	3 2-4	3 2-4	3 2-4
Quintile 4	-	6 5-7	5 4-7	5 4-6
Quintile 5	-	7 5-8	6 5-7	6 5-7
Insulin pump				
No	-	-	-	Reference
Yes	-	-	-	(-)5 (-)6-(-)4

^aModel 1: adjusted for age, gender, diabetes duration and ethnicity, $R^2=0.07$

^bModel 2: adjusted for age, gender, diabetes duration and socioeconomic status, $R^2=0.08$

^cModel 3: adjusted for age, gender, diabetes duration, ethnicity and socioeconomic status, , $R^2=0.09$

^dModel 4: adjusted for age, gender, diabetes duration, ethnicity and socioeconomic status and pump therapy, $R^2=0.10$

Text in bold indicates statistical significance at $p<0.05$