**Abstract**

**Purpose** Ethnic minority children are at a greater risk for type 2 diabetes (T2D). However current prevalence of T2D among children and young people is unknown in England and Wales. Additionally, little is known on glycaemic control in paediatric T2D globally.

**Methods** Using data from the National Paediatric Diabetes Audit (NPDA) for 2012-13 with >98% coverage of diabetes cases, we estimated 1. The overall, gender- and ethnic-specific prevalence of T2D in children and young people <16 years and 2. Whether ethnicity predicts glycaemic control (measured by mean HbA1c) in children and young people <19 years. Ethnicity was self-identified and categorised into

White, Asian, Black, Mixed, Other and ’Not-stated’. Multivariable linear regression was used to estimate differences in glycaemic control by ethnicity adjusting for socioeconomic status, age, diabetes duration and gender.

**Results** 307 children and young people <16 years of age were identified with T2D in the NPDA for 2012-13. Overall prevalence of T2D was 2.9/100,000. Females had a higher prevalence of T2D than males (4.3 vs. 1.5/100,000). The highest prevalence was found in Asian (12.2/100,000) followed by Mixed-ethnicity (4.4/100,000) females. Children of mixed-ethnicity had significantly higher mean HbA1c compared to White children (9.7% (83mmol/mol) vs. 7.8% (62mmol/mol), p<0.001, and adjusted mean difference of 4.2% (22.3mmol/mol), 95%CI 3.1-5.2% (10.9-33.7mmol/mol)), but there were no significant differences between the other ethnic minority groups.

**Conclusion** Children of all ethnic-minorities particularly females have an increased prevalence of T2D. Those belonging to mixed-ethnic backgrounds are at increased risk for poorer glycaemic control.

**Keywords** Type 2 diabetes, ethnicity, glycaemic control, HbA1c, childhood, adolescence, prevalence, England, Wales

Type 2 diabetes (T2D) remains relatively rare in children and adolescents (between 1-2% of all diabetes cases) (1, 2). However, with the ongoing obesity epidemic in this group and its associated adverse metabolic consequences, there has been concern about increasing incidence/prevalence of T2D and its comorbidities including hypertension, dyslipidaemia and impaired glucose intolerance leading to reduced life expectancy (1). There is considerable variability in incidence rates for T2D. In some countries, 25-40% of all new cases of diabetes in children are now diagnosed as obesity-related T2D with some studies reporting >85% of all children with T2D as overweight or obese (3). This is a complex disease to manage for young patients, their families and clinicians because of its comorbidities and requires significant lifestyle changes and consistent long-term management (4).

T2D is disproportionately more common among ethnic minority and low income children, which parallels similar disparities observed in obesity rates (1, 2, 5, 6). In adolescents, it is associated with elevated risk of future cardiovascular and other diabetes related complications which is different from long-term complications often associated with type 1 diabetes (7-11). These complications (such as dyslipidaemia and hypertension) are more common in ethnic minority and low income groups making them more vulnerable to increased morbidity and mortality in adulthood (2, 12).

T2D among children in Europe and the UK is relatively less common when compared to the USA, Canada, Mexico, Brazil, Australia and India (1, 13). However, studies to date suggest an increase in T2D incidence in many European countries including the UK (14, 15). Additionally, the composition and proportion of ethnic minority groups varies significantly between different countries due to historical, geographical and political reasons and patterns of immigration. This might affect both the overall- and ethnic-specific prevalence estimates of T2D in different high-income and multicultural countries. There has been little evidence in recent years to document ethnic differences in prevalence of T2D among children in England and Wales using nationally representative data (15). Additionally, not much is known on ethnic differences in glycaemic control in children with T2D globally. The main aims of this study are to document ethnic differences in the prevalence of T2D in children and young people in England and Wales in 2012-13 and to investigate any differences in glycaemic control between ethnic groups. Estimation of ethnic specific prevalences of T2D will help in framing future public health policies which might have to be more sensitive to the needs of particular ethnic minority groups.

**RESEARCH DESIGN AND METHODS**

**Design, setting and data source**

Data for this cross-sectional study was obtained from the National Paediatric Diabetes Audit (NPDA) for England and Wales (16). The NPDA was started in 2002 and reached near 100% participation covering all 178 paediatric diabetes units in 2012. It includes demographic and outcome data on almost all children with all forms of diabetes <19 years old and treated at a specialist paediatric clinic. This study was based on the 2012-13 audit year (1st April 2012 – 31st March 2013). Inclusion criteria comprised: a diagnosis of T2D, <19 years old on the first day of the audit, a minimum of one visit to a clinic during the audit year and valid information on ethnicity and postcode of residence. As per recommendations from the National Institute of Health and Care Excellence, a patient with diabetes is offered integrated healthcare by a multidisciplinary team at a clinic four times/year. HbA1c levels, height and weight are recorded at each visit. All demographical and clinical parameters are recorded systematically across clinics enabling comparison. The analysis was conducted in two parts: 1. Estimation of prevalence of T2D by gender and ethnicity in all children <16 years and 2. A regression analysis analyzing association between ethnicity and glycaemic control (HbA1c) in all children <19 years.

**Prevalence calculation**

The analysis on prevalence of T2D was restricted to those children <16 years as some patients transfer to adult services at this time, potentially underestimating prevalence rates 16-18 year-olds. The numerator for analysis on prevalence included all cases of T2D prevalent in 2012-13 and <16 years of age with valid data on gender and ethnicity (Figure 1). Patients (or their parents) self-reported their ethnicity using one of the fifteen categories as recommended by the Information Standards Board for Health and Social Care. Participants were also given the option to decline identifying their ethnicity (‘Not Stated’ option). The fifteen ethnic categories were collapsed into six broader groups (listed in Supplemental Table 2): White (British, Irish and any other White background), Asian (comprising subjects of mostly South Asian origin), Black (subjects of Caribbean and African origin), Mixed (any form of mixed ethnic background), Other (including Chinese and any other ethnic background not listed above) and ‘Not Stated’. We excluded the ‘Not stated’ ethnic group from the analysis on prevalence of T2D by ethnicity as the national census does not include this category and we were unable to estimate the prevalence for this group. The denominators for the analysis on prevalence were obtained from 2011 national census (the most recent census).

All prevalence rates were expressed as cases per 100,000 children 0-15.99 years of age.

**Regression analysis**

**Outcome and independent variables**

HbA1c was used as a measure of overall T2D (glycaemic) control and the main outcome of interest. HbA1c values recorded as percentages were converted to mmol/mol using the formula: (HbA1c value in percentage-2.15) x 10.929. Where more than one HbA1c was recorded in the year, the mean was calculated for each individual. Independent variables included age, gender, diabetes duration, ethnicity and Socioeconomic Status (SES). 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 diabetes diagnosis. The first recorded entry for ethnicity in the audit year was used in the analysis. BMI was calculated as weight (in kilograms) divided by height (in metres) squared. Overweight and obesity in children was determined by using age- and sex-specific cut-offs proposed by the International Obesity Task Force (17). Age and sex appropriate BMI standard deviation scores or Z-scores were calculated as proposed by T.J. Cole et al (18). SES was derived from postcode using Indices of Multiple Deprivation (IMD) 2010 for England, and the Welsh Indices of Multiple Deprivation 2008 for Wales (19). 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 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 with each area comprising approximately 1,500 individuals. IMD rank scores were grouped into quartiles for analysis, with the first and fourth quartiles corresponding to the least and most deprived respectively.

***Missing data***

Overall, the level of missing data for all covariates was low; gender (0.2%), ethnicity (0.7%) and deprivation (3.4%). All subjects had valid data on age and diabetes duration. To minimize data loss, we imputed missing data using the multiple imputation chained equations (MICE) under a missing at random (MAR) assumption following published guidelines (22). We chose to only impute IMD rank scores. We did not impute ethnicity as this was the main exposure of interest. Regression analyses were run across 20 imputed datasets and parameters from each dataset were combined to obtain overall estimates using Rubin’s rules (23). Results from regression models with imputed data were near identical to those with observed data and we report the former.

***Statistical analysis***

Continuous variables are presented as mean values with standard deviations and categorical variables as frequencies. Associations between ethnicity, 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 HbA1c as the principal outcome and ethnicity or SES as the primary predictor (Models 1 and 2 respectively) to assess independent associations. Model 3 included both ethnicity and SES as covariates to assess associations with glycaemic control but adjusted for one another. 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. Robust standard errors allowing for clustering of children within clinics were used for all linear regression models. All statistical analyses were conducted using STATA 13 (College Station, TX, USA).

**Robustness tests**

We repeated the main analysis (Model 3) but restricted to those children with a mean HbA1c<=13.1% or 120mmol/mol (N=376) children to assess whether differences in glycaemic control by ethnicity or SES remained the same after excluding very high values of HbA1c (>13.1% or 120mmol/mol). This was done to ensure that observed differences in mean HbA1c between the White and ethnic minority groups was not due to very high values (outliers) of HbA1c. We also repeated the analysis fitting multilevel regression models which take into account potential clustering by clinics. As multilevel modelling yielded almost identical results as multivariable linear regression, we present results from the former in Supplemental Table 1.

**Ethics**

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

**RESULTS**

During the 2012-13 audit year, 443 children and young people (1.8% of all forms of diabetes) <19 years were recorded as having T2D in the NPDA. Of these 443, one lacked data on gender, three on ethnicity and 35 on HbA1c leaving 404 children with T2D (91% of the eligible sample) with data on age, gender, diabetes duration, ethnicity and SES who were included in the analysis to assess associations between ethnicity, SES and glycaemic control (Figure 1). Data on missing values of IMD scores for 13 children were imputed.

Table 1 shows the study population stratified by ethnicity. The mean age at diagnosis of T2D was 13 years and the mean age at first visit in the audit year was 14.9 years. There were no significant differences in either age at diagnosis or age at first visit between ethnic groups. More than 70% of study population with T2D were female in all ethnic groups with the exception of the ’Other’ and ’Mixed’ groups. The mean BMI for the study population was 30.2 kg/m2 and the vast majority of children (>80%) across all ethnic groups were overweight or obese (BMI ≥25kg/m2).

*Prevalence of type 2 diabetes*

307 (224 or 73% females) children and young people <16 years of age with valid data on age, gender and ethnicity were included in the analysis on prevalence. The overall prevalence of T2D in children <16years was 2.9 (95% CI 2.6-3.2) per 100,000. Prevalence of T2D differed significantly between ethnic groups (Table 2). The highest prevalence of T2D was observed in Asians (8.0 (6.2-9.6)/100,000) followed by the ‘Other’ (6.0, (2.9-11.6)/100,000) and Mixed (3.8, (2.4-5.7)/100,000) ethnic groups (Table 2). The White group had the lowest prevalence of T2D (1.4, (1.2-1.7)/100,000). Overall, females had a higher prevalence compared to males (4.3 vs. 1.5/100,000, Table 2). The higher prevalence of T2D in female was observed in all ethnic groups with the exception of the ‘Other’ group (Table 2). The highest prevalence of T2D was found in Asian females (12.2, (9.5-15.6)/100,000), whereas White males had the lowest prevalence (0.6, (0.4-0.9)/100,000), Table 2.

*Ethnic differences in glycaemic control*

Table 3 shows the results of regression analysis. Univariate analysis revealed that the ’Mixed’ ethnic group had significantly higher mean HbA1c (9.8% (83mmol/mol)) compared to all other ethnic groups. Differences in mean HbA1cwere relatively small between the other ethnic groups. Additionally, the ‘Mixed’ ethnic group was the only group where the majority (67%) of subjects were classified has having poor glycaemic control (HbA1c≥58mmol/mol). In multivariable linear regression models, mixed ethnicity children were the only ethnic group to have statistically significant differences in mean HbA1c compared to those of White ethnicity. On average, HbA1c was 4.2% or 22.3 mmol/mol (95% CI 3.1-5.2% or 10.9-33.7mmol/mol) higher in the ’Mixed’ ethnic group compared to White group after adjustment for covariates (Table 3). Asians and the ’Other’ ethnic group also had higher mean HbA1c values but these differences were not statistically significant (Table 3).

SES was not associated with glycaemic control in either univariate analysis or in the regression models.

*Robustness checks*

The observed higher mean HbA1c in ‘Mixed’ children was similar but with a slightly attenuated point estimate (3.8% or 17.5mmol/mol, 3-4.6% or 7.9-27.1) and remained statistically significant in the regression model restricted to those with HbA1c<13.2% (121mmol/mol). Repeating the analysis using multilevel regression modelling taking into account potential clustering at the clinic level made no difference to the conclusions (Supplemental Table 1).

**CONCLUSIONS**

Using a near complete sample of all children with T2D in England and Wales in 2012/13, we found ethnic minority children had higher prevalence of T2D compared to White children. Highest prevalence estimates were observed in Asian and Mixed-ethnicity females, who had a greater than five-fold and nearly two-fold increased prevalence of T2D compared to White female children respectively. With the exception of the Mixed ethnicity group, other ethnic minorities were not at increased risk for worse glycaemic control compared to the White group. The Mixed ethnic group had substantially higher mean HbA1c corresponding to ~1SD above the mean for the entire study population. This was despite the Mixed ethnic group having the second lowest mean BMI compared to other groups (Table 1).

This study is the first to calculate the prevalence of paediatric T2D in all major ethnic groups in England and Wales. Two previous studies analysed ethnic differences in T2D prevalence in England and Wales using nationally representative data collected by the British Paediatric Surveillance Unit (BPSU) (14, 24). Ehtisham et al reported that South Asian children had a higher prevalence compared to White children but the study was restricted to two ethnic groups. Similar to these studies, we found the majority of T2D cases were ethnic minority females. However, an alarming observation is the substantial 15 fold increase in overall T2D prevalence from 0.2/100,000 in 2000 to 3/100,000 in 2012-13 in <16 year-olds (24). BPSU surveys have high response rates (>90%), are considered to have high ascertainment relying on paediatricians’ and diabetes specialist nurses reporting and are duplicated in countries around the world (14). Additionally, it would be unlikely that a child/young person with T2D who would not be under the care of a paediatrician early in the last decade, making the BPSU survey data comparable to the NPDA.

Our results are consistent with international studies in that ethnic minority children have increased prevalence of T2D. The average age at diagnosis in this study (13 years) is comparable to that reported elsewhere (25). A US study reported an overall prevalence of 0.22/1,000 in <20year olds which is much higher than that in our study (26). Our prevalence estimates are similar to those reported in Germany and Sweden (27, 28).

To our knowledge this is the first study in the UK to report ethnic differences in glycaemic control in a national cohort of children with T2D. A smaller American study with 103 subjects but restricted to two ethnic groups reported that non-White adolescents had poorer glycaemic control compared to White adolescents (29).

Additionally, we analysed differences in prevalence and glycaemic control in all six major ethnic groups corresponding to official standard ethnicity classifications. Ethnicity is self-identified which is considered the ‘gold standard’ in studies on ethnicity and health (30). The IMD scores have been shown to be associated with several health outcomes is previous studies and is considered to be the standard benchmark for UK governmental health and social policy (31).

A strength of our analysis is the representative dataset, with near-complete coverage of England and Wales. The NPDA data is collected annually by participating 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 T2D in England and Wales are included in the audit, it is estimated to represent in excess of 95% of cases and is thus nationally representative.

This study has certain limitations. The NPDA cannot validate the diagnosis of T2D made by clinicians and it is possible that a certain proportion of cases such as Maturity Onset Diabetes of the Young (MODY) could have been misclassified as T2D. However, MODY affects less <2% of all diabetes cases and any misclassification should not affect the results. Nearly 20% of all subjects chose not to divulge their ethnicity and were grouped in the ‘Not-Stated’ category. This could lead to an underestimation of ethnic-specific prevalence rates. The ‘Not-stated’ group is more likely a heterogeneous mix of children belonging to all ethnic backgrounds and thus underestimation of prevalence rates is likely to be minimal. The small sample sizes in some ethnic minority groups could limit potential differences in glycaemic control from the white group. We were unable to account for family history of T2D, a known risk factor, as this information is not recorded. Given that the majority of children with T2D are overweight or obese, future studies need to investigate the potential modifying effects of lifestyle factors (such as physical activity and diet) which are likely to vary between ethnic groups (29, 32). The use of area-based deprivation scores such as the IMD makes the assumption that all residents in a specific area have the same attributes of SES associated with that area. This is probably less likely (the ecological fallacy), but it has been shown that area-based SES indicators such as the IMD are reasonable proxies in the absence of individual-based SES indicators (33). In fact the IMD is recommended for use in tracking health inequalities in UK government statistics and allows much finer small area resolution, down to the level of census lower layer super output areas (LLSOAs) which contain on average 1,400 people. Lastly, the absolute number of children with T2D is small and any increase over time is likely to appear considerable when presented as an ‘x-fold’ increase.

Our finding of a 15 fold increase in the prevalence of diabetes is a cause for concern. It shows that T2D is no longer an exceptionally rare disease among children and disproportionately affects ethnic minorities. This will significantly impact healthcare delivery as ethnic-specific programs should be designed to identify children at greater risk for T2D, including intervention programs that take into account diet, afterschool activities and neighbourhood characteristics. The latter is important as certain ethnic groups are more likely to live in deprived areas often associated with a poorer choice of amenities (restaurants, supermarkets, recreational spaces – giving rise to the ‘obesogenic environment’) (34). The lack of association between SES and glycaemic control is counterintuitive. We expected lower SES children to have worse glycaemic control similar to that seen in children with type 1 diabetes and in adults with T2D (35). One possible explanation for this could be low statistical power due to the small numbers in ethnic minority groups.

There is limited but growing evidence for increased risk of behavioural (substance abuse, suicide attempts), emotional, stress and health issues in adolescents of mixed ethnicity (36, 37). However, evidence is contradictory, varies by country of residence and the combination of ethnic group-origin of mixed ethnicity children (37). Mixed ethnicity children represent a small but growing proportion of the population (4.9% of all <20 years olds identified as being mixed in the 2011 UK National census). Within this group, the largest proportion is those identifying as being mixed White and Black-Caribbean (35.5%), followed by mixed White and Asian (28.9%) and mixed White and ‘other’ (21.4%). The smallest group is the mixed White and Black-African (14.2%). Identity issues leading to social isolation, poorer self-confidence, poorer academic performance and family dynamics could explain worse health outcomes this group. Such factors will vary between different mixed ethnicity groups due to differences in SES, place of residence and cultural factors. One possible explanation for greater risk behaviour in mixed ethnicity children is the struggle to find a positive identity (an important factor for increasing resiliency) especially during puberty and adolescence (36). Resulting issues like low self-esteem, not feeing accepted, lack of confidence can contribute to risky behaviour making it difficult to manage chronic conditions like diabetes (and also lead to risk factors for such diseases). A study on the British MBC study found that households with mixed-ethnicity children were socioeconomically better-off when compared with their ethnic counterparts with non-mixed children, but were on average poorer than White households (38). The only exception was households with mixed-Indian children that had the highest income among all ethnic groups. This suggests a need to analyse the associations presented in this study separately for different mixed-ethnicity groups, which we were unable to do due to small numbers in this category.

This study shows a substantial increase in prevalence of T2D in England and Wales over the past decade. The most vulnerable children are ethnic minorities, specifically females of Asian, Mixed and Black ethnicity. More needs to be done to be able to identify these vulnerable populations before the actual onset of T2D.

**REFERENCES**

[1] Pulgaron ER, Delamater AM. Obesity and Type 2 Diabetes in Children: Epidemiology and Treatment. Curr Diabetes Rep 2014;14.

[2] Dean HJ, Sellers EA. Children have type 2 diabetes too: an historical perspective. Biochem Cell Biol 2015:1-5.

[3] Kaufman FR, Shaw J. Type 2 diabetes in youth: rates, antecedents, treatment, problems and prevention. Pediatr Diabetes 2007;8 Suppl 9:4-6.

[4] Copeland KC, Zeitler P, Geffner M, et al. Characteristics of Adolescents and Youth with Recent-Onset Type 2 Diabetes: The TODAY Cohort at Baseline. J Clin Endocr Metab 2011;96:159-167.

[5] Protudjer JL, Dumontet J, McGavock JM. My voice: a grounded theory analysis of the lived experience of type 2 diabetes in adolescence. Can J Diabetes 2014;38:229-236.

[6] Naughton MJ, Ruggiero AM, Lawrence JM, et al. Health-related quality of life of children and adolescents with type 1 or type 2 diabetes mellitus. Arch Pediat Adol Med 2008;162:649-657.

[7] Burns SF, Lee S, Bacha F, et al. Pre-diabetes in overweight youth and early atherogenic risk. Metabolism 2014;63:1528-1535.

[8] Levitsky LL, Danis RP, Drews KL, et al. Retinopathy in Youth With Type 2 Diabetes Participating in the TODAY Clinical Trial. Diabetes Care 2013;36:1772-1774.

[9] Weinstock RS, Caprio S, Copeland KC, et al. Lipid and Inflammatory Cardiovascular Risk Worsens Over 3 Years in Youth With Type 2 Diabetes The TODAY clinical trial. Diabetes Care 2013;36:1758-1764.

[10] Lynch J, Elghormli L, Fisher L, et al. Rapid Rise in Hypertension and Nephropathy in Youth With Type 2 Diabetes The TODAY clinical trial. Diabetes Care 2013;36:1735-1741.

[11] Bacha F, Gungor N, Lee S, et al. Progressive deterioration of beta-cell function in obese youth with type 2 diabetes. Pediatr Diabetes 2013;14:106-111.

[12] Bacha F, Gungor N, Lee S, et al. Type 2 diabetes in youth: are there racial differences in beta-cell responsiveness relative to insulin sensitivity? Pediatric Diabetes 2012;13:259-265.

[13] Karres J, Pratt V, Guettier JM, et al. Joining forces: a call for greater collaboration to study new medicines in children and adolescents with type 2 diabetes. Diabetes Care 2014;37:2665-2667.

[14] Haines L, Wan KC, Lynn R, et al. Rising incidence of type 2 diabetes in children in the U.K. Diabetes Care 2007;30:1097-1101.

[15] Holman N, Young B, Gadsby R. Current prevalence of Type 1 and Type 2 diabetes in adults and children in the UK. Diabet Med 2015.

[16] National Paediatric Diabetes Audit Report. Available at: <http://www.rcpch.ac.uk/child-health/standards-care/clinical-audit-and-quality-improvement/national-paediatric-diabetes-au-12015>.

[17] Cole TJ, Bellizzi MC, Flegal KM, et al. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ 2000;320:1240-1243.

[18] T C. User's guide to lmsGrowth. 2015.

[19] GOV.UK. English indices of deprivation 2010. Available at: <https://www.gov.uk/government/statistics/english-indices-of-deprivation-2010> Accessed 10 August 2015.

[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; 2011.

[22] Sterne JA, White IR, Carlin JB, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. Bmj 2009;338:b2393.

[23] Schafer JL. Multiple imputation: a primer. Statistical methods in medical research 1999;8:3-15.

[24] Ehtisham S, Hattersley AT, Dunger DB, et al. First UK survey of paediatric type 2 diabetes and MODY. Arch Dis Child 2004;89:526-529.

[25] Rosenbloom AL, Silverstein JH, Amemiya S, et al. ISPAD Clinical Practice Consensus Guidelines 2006-2007. Type 2 diabetes mellitus in the child and adolescent. Pediatr Diabetes 2008;9:512-526.

[26] Liese AD, D'Agostino RB, Hamman RF, et al. The burden of diabetes mellitus among US youth: Prevalence estimates from the SEARCH for Diabetes in Youth Study. Pediatrics 2006;118:1510-1518.

[27] Neu A, Feldhahn L, Ehehalt S, et al. Type 2 diabetes mellitus in children and adolescents is still a rare disease in Germany: a population-based assessment of the prevalence of type 2 diabetes and MODY in patients aged 0-20 years. Pediatric Diabetes 2009;10:468-473.

[28] Zachrisson I, Tibell C, Bang P, et al. Prevalence of Type 2 diabetes among known cases of diabetes aged 0-18 years in Sweden. Diabetologia 2003;46:A25-A26.

[29] Rothman RL, Mulvaney S, Elasy TA, et al. Self-management behaviors, racial disparities, and glycemic control among adolescents with type 2 diabetes. Pediatrics 2008;121:E912-E919.

[30] Bhopal R. Ethnicity, race, and health in multicultural societies. Foundations for better epidemiology, public health, and health care.: Oxford University Press, 2008.

[31] Niggebrugge A, Haynes R, Jones A, et al. The index of multiple deprivation 2000 access domain: a useful indicator for public health? Social science & medicine 2005;60:2743-2753.

[32] Kriska A, Delahanty L, Edelstein S, et al. Sedentary behavior and physical activity in youth with recent onset of type 2 diabetes. Pediatrics 2013;131:e850-856.

[33] Krieger N, Chen JT, Waterman PD, et al. Geocoding and monitoring of US socioeconomic inequalities in mortality and cancer incidence: Does the choice of area-based measure and geographic level matter? The Public Health Disparities Geocoding Project. American Journal of Epidemiology 2002;156:471-482.

[34] Patterson R, Risby A, Chan MY. Consumption of takeaway and fast food in a deprived inner London Borough: are they associated with childhood obesity? BMJ Open 2012;2.

[35] Khanolkar AR, Amin R, Taylor-Robinson D, et al. Young people with Type 1 diabetes of non-white ethnicity and lower socio-economic status have poorer glycaemic control in England and Wales. Diabet Med 2016.

[36] Wong SS, Sugimoto-Matsuda JJ, Chang JY, et al. Ethnic Differences in Risk Factors For Suicide Among American High School Students, 2009: The Vulnerability of Multiracial and Pacific Islander Adolescents. Arch Suicide Res 2012;16:159-173.

[37] Schlabach S. The Importance of Family, Race, and Gender for Multiracial Adolescent Well-being. Fam Relat 2013;62:154-174.

[38] Panico L, Nazroo JY. The social and economic circumstances of mixed ethnicity children in the UK: findings from the Millennium Cohort Study. Ethnic Racial Stud 2011;34:1421-1444.