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HbA1c Levels in Families of Ethnic Minorities with Early Onset Type 2 Diabetes: Glycaemic Control and Ethnicity

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Author's contribution

The sole author designed, analyzed and interpreted and prepared the manuscript.

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ABSTRACT

Aims: Ethnic differences and factors associated with high glycated haemoglobin A1c (HbA1c) levels in families of East Indian and West African descents with early onset type 2 diabetes mellitus (T2DM) were compared. Glycaemic control is affected by genetics and is strongly influenced by adherence to medication and cultural adaptations to instructions.
Study Design: A 12 months study.
Place and Duration of Study: Diabetes Mellitus Clinic, University of the West Indies Hospital, Mona, Jamaica, between September 2000 to October 2003.
Methodology: A 12 months analysis was done on 54 members of families of East Indian descent and 48 members of families of West African descent with early onset T2DM enrolled in the Maturity Onset Diabetes of the Young (MODY) study. HbA1c levels were measured at baseline, 6 and 12 months.
Results: The families of West African descent started at baseline with significantly higher HbA1c (11.0±1.5% versus 10.0±1.5% [97±17 mmol/l versus 86±16 mmol/l], *P*=0.05) than families of East Indian descent. Nevertheless their mean HbA1c level was significantly lower at 12 months

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(7.0±0.7% versus 7.9±0.5% [53±7 mmol/l versus 63±5 mmol/l], *P*=0.05). Variables such as access to healthcare, social status and education, age, body mass index (BMI), duration of diabetes and insulin and lipid therapies were similar in the ethnic groups. Lipid function was abnormal over 12 months in families of East Indian descent. Families of East Indian descent adhere less to medication guidelines and instructions.

Conclusion: Families of East Indian descent had poorer glycaemic control than those of West African descent. The poor control may be linked to genetics as seen in triglyceride and high density lipoprotein cholesterol profile over 12 months and may have been influenced by ethnic differences in adherence to medication and instruction following.

Keywords: Glycaemic control; diabetes mellitus; East Indian; African; disparities.

1. INTRODUCTION

Ethnic minorities in the United States of America (USA) are adversely affected by diabetes mellitus (DM) and its underlying complications such as nephropathy, neuropathy, retinopathy and lower limb amputations [1]. Repeated studies have shown that the prevalence of DM is higher in urban, migrant and populations of African origins in the Diaspora than in rural, non-immigrant, and population of non-African origin [2,3]. Indians from Asia also have a high prevalence of type 2 diabetes mellitus (T2DM) in comparison to Caucasians [4]. Whether the minorities are original natives or migrants, the disparity in DM rates and complications in comparison with Caucasians is frightening. Aboriginal patients in comparison to Euro-Canadian patients are nearly twice as likely to have highly elevated glycaemic levels [5]. American Indians are 2.2 times more likely to have DM than non-Hispanic whites [6]. In the USA the incidence and severity of retinopathy are higher in blacks or African Americans than in whites [7]. African Americans seem to be more disproportionately affected by kidney disease than Caucasians [8] and the rate of kidney failure in American Indians with DM is 1.9 times that of the general population [9]. Factors such as socioeconomic and poor access to healthcare may be driving the disparities [10-11]. However if these factors are to be given serious consideration, one needs to rationalize why DM rates are lower in migrant countries where these factors are also present than in other countries [3-4]. Proposed explanation for these disparities include low level of care in minorities clinics, reduced access to health care and racial/ethnic differences in medication adherence and monitoring [11-12]. Adverse health outcomes such as microvascular and macrovascular complications of DM can be reduced though effective glycaemic control. In T2DM, the risk of diabetic complications is linked to previous hyperglycaemia and any reduction in the risk is likely to reduce complications [13]. In the USA and Jamaica, the prevalence rates of DM are reported to be approximately 9.3% and 7.9% respectively [14-15]. Many studies have been done on glycaemic control in ethnic minorities with DM in the USA and Europe [10-12]. Few factors have been able to effectively explain why there are ethnic differences in glycaemic control [16-17]. An ethnicity and glycaemic control study would be novel in Jamaica and the objective was to compare the level of glycaemic control between members of two groups of ethnic minorities with early onset T2DM so as to better understand how ethnicity impacts glycaemia. The two groups had similar mean age of diagnosis, body mass index (BMI) and were on insulin for management of their DM. They, who had dyslipidaemia were managed on lipid lowering drugs.

The families who participated in the study were from the same socioeconomic background with similar levels of education and family income. These factors have been associated with disparity in glycaemic control in other studies [17-18]; the similarities of these factors in both groups therefore were designed to rule out the impact in this study. Since ethnic minorities in the Americas with DM usually have poorer glycaemic control than in their country of ancestry [2-4]. Some researchers hypothesized that glycaemic control could be influenced by cultural adaptations such as beliefs that might influence instruction following and medication adherence. An understanding of the levels of glycaemic control in families of ethnic minorities with DM will help inform about possible deterrents to proper control. Treatment options may thus be modified to be culturally specific so that different ethnic groups can achieve good glycaemic control.

The study therefore is a comparison of glycaemic control over 12 months between Jamaican

families of East Indian descent and West African descent with early onset T2DM.

2. PATIENTS AND METHODS

Probands and family members with early onset T2DM who were enrolled at the Diabetes Clinic at the University of the West Indies, Jamaica during 2000-2003 were recruited for the study. Early onset T2DM was defined as having a family history of DM in multi-generations with at least two first degree relatives diagnosed with T2DM before age 35 years of age and DM only on the maternal or paternal side of the family [19]. Exclusion criteria for the study were: Inability to self identify as of West African descent or East Indian descent, not born in Jamaica, non- ambulatory individual, diagnosed with heart and or kidney disease prior to registering for the study, blindness, a severe concurrent illness likely to limit life or require extensive systemic treatment, inadequate understanding or unwillingness to participate in the study and having diabetes not described as early onset T2DM [20].

Inclusion criteria were: Recruits who lived in or around the metropolitan area of the clinic and were able to walk, take public transportation or drive to the clinic. Participants had to self identify as of West African descent or East Indian descent with knowledge of family pedigree and family history of early onset T2DM. All participants were from families who were involved in the genome wide study for genes (GWSG) associated with maturity onset diabetes of the young (MODY). The GWSG in families did not find any of the common mutations associated with the MODY genes [20]. Five families consisting of 102 individuals spanning three generations participated in the study. Two families consisting of 48 individuals were of West African descent and three families consisting of 54 individuals were of East Indian descent. The participants were followed for 12 months.

Persons in the study were on insulin for the management of diabetes. Basal and pre-mixed insulin were used in the management of DM. Persons with abnormal lipid values were also managed with statins. The common drugs used for participants with hypertension were thiazides, reserpine and angiotensin converting enzyme (ACE) inhibitors. The participants attended clinic regularly and usually kept appointment dates. These guidelines were necessary as glycaemic variables were followed for 12 months. Collected information included age, sex, language and ancestral origin of parents and grandparents.

The study received approval from the Faculty of Medical Sciences, University of the West Indies Ethics Committee. Written informed consent was obtained from each participant.

Fasting blood samples were taken at baseline and each six months for glucose, insulin, triglyceride (TG) and total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), glycosylated haemoglobin (HbA1c) and creatinine. Plasma glucose was measured by the glucose oxidase method (Sigma-Aldrich, St Louis, MO, USA). Creatinine, TC and TG were measured in the RA-1000 autoanalyser (Technicon Instruments Corporation, Tarrytown, New York, USA). Fasting serum insulin was measured by the sandwich immunoassay method (Roche Diagnostics, Indianapolis, IN, USA). Beta cell function (HOMA-β) using the homeostasis model assessment was calculated as 20 x $insulin_0$ (μIU/mL) /fasting $glucose_0$ (mmol/L) -3.5 /22.5 and insulin resistance (HOMA-R) using the homeostasis model assessment was calculated as $insulin_0$ (μIU/mL) x fasting $glucose_0$ (mmol/L) /22.5 [21]. HDL-C was measured in the RA-1000 autoanalyser (Technicon Instruments Corporation, Tarrytown, New York, USA) after precipitating out the apo-B-containing lipoproteins. LDL-C was calculated according to Freidwald equation (TC–[VLDL-C ± HDL-C], VLDL-C: TG/5). HbA1c was measured using high performance liquid chromatography (TOSOH Lipoprotein Analytical System, Minato-Ku, Tokyo, Japan). Guidelines and recommendations for laboratory analysis in the diagnosis and management of diabetes were generally followed [22].

Each person on each visit was asked to name important influences on medication adherence and compliance with instructions. Body mass index (BMI) was calculated as body weight in kilograms (kg) divided by height in metres squared (m^2). Values ≥ 25.0 kg/m² of BMI were considered as overweight, while obesity was defined as BMI ≥ 30 kg/m² [23]. Blood pressure was determined by using the average of three sequential measurements taken on the day that baseline, 6 and 12 months assessments were done. Hypertension was defined by using current guidelines as increased systolic (≥ 130 mmHg) and/or diastolic (≥ 80 mmHg) blood pressure [24] using measurement of clinic blood pressure

(CBP). The patients' dockets were reviewed at baseline, 6 and 12 months to note if mentions were made by the attending physicians of macrovascular complications (coronary artery disease, peripheral arterial disease, and stroke) and microvascular complications (diabetic nephropathy, neuropathy, and retinopathy) [25].

2.1 Statistical Analysis

Statistics were computed using IBM SPSS, Statistics 20 (IBM, Boston, MA, USA). Frequency tables and accompanying histograms were produced and examined with related descriptive statistics (means, medians, modes, and standard deviations) for all participants. This information along with the Kolmogorov-Smirnov test and one-sample test were used to assess the variables approximation to normality. Such results were used to determine whether a parametric test should be used for comparison of means and medians and the independent t test was used to compare the mean differences of these two groups. Data are generally presented as mean ± SD or as median (interquartile range). Significance is noted at two-tailed values of $P \leq 0.05$.

3. RESULTS AND DISCUSSION

3.1 Families of East Indian Descent (Table 1)

In the study there were 54 participants of East Indian descent at baseline, 49 at 6 months and 47 remained until the end of the study. The differences in participants at the different stages were accounted for by migration and death.

There was no significant change in BMI values over the three testing periods.

Fasting plasma glucose showed a non-significant increase and decrease from baseline to 6 months and 6 to 12 months respectively. The decrease in postprandial plasma glucose from baseline to 6 months was not significant but the decrease from 6 to 12 months was significant ($P<0.001$). HbA1c decreased non-significantly from baseline to 6 months. However, the decrease from 6 to 12 months was significant ($P<0.01$). There were no significant changes in fasting insulin and TC from baseline to 6 months and from 6 months to 12 months. HDL-C value did not change significantly from baseline to 6 months but showed a significant ($P<0.05$) increase from 6 to 12 months. LDL-C showed no significant change from baseline to 6 months but a significant (P=0.05) decrease from 6 to 12 months. There was a significant ($P<0.01$) decrease in TG value from baseline to 6 months but a significant ($P = 0.05$) increase from 6 to 12 months.

Creatinine in serum decreased significantly ($P<0.05$) from baseline to 6 months and then increased significantly ($P<0.05$) from 6 to 12 months.

HOMA- β showed no significant change from baseline to 6 months, a significant ($P<0.05$) increase from 6 months to 12 months and no significant increase from baseline to 12 months.

HOMA-R showed no significant change from baseline to 6 months but showed significant ($P=0.05$) decreases from 6 to 12 months and baseline to 12 months. Systolic and diastolic CBP levels showed significant ($P \leq 0.01$) decreases from 6 to 12 months and baseline to 12 months. At the beginning of the study most of the group (65%) was prescribed premixed insulin and 35% basal insulin, at 6 months 60% of the group was on premixed insulin given ≥ 2 times daily. At 12 months > 60% of patients was still on premixed insulin given ≥ 2 times daily. There was a significant ($P < 0.01$) decrease in compliance and instruction following from baseline to 6 months and a significant ($P<0.05$) increase from 6 to 12 months. The decrease and increase seemed to have lead to a non-significant change overall in medication compliance and instruction following from baseline to12 months. There was no diabetes complication at the beginning of the study or at 6 months. However at 12 months, two members of the East Indian families developed nephropathy and were referred to a specialist clinic.

3.2 Families of West African descent (Table 2)

There were 48 participants of West African descent in the study at baseline, 46 at 6 months and 45 at 12 months. They, who did not continue in the study migrated or died. There were significant ($P \leq 0.01$) decreases in fasting and postprandial plasma glucose, and HbA1c from baseline to 6 months, from 6 to 12 months and from baseline to 12 months in this group. Fasting insulin decreased significantly ($P<0.01$) from baseline to 6 months and showed a non-significant increase from 6 to 12 months and a significant ($P<0.05$) decrease from baseline to 12

months. There was a significant (P<0.05) decrease in TC from baseline to 6 months and no significant change from 6 to 12 months. Significant (P<0.05) increases and decreases were seen in HDL-C and LDL-C from 6 to 12 months and baseline to 12 months respectively. There was a significant (P<0.01) decrease in TG value from baseline to 6 months, no change shown from 6 to 12 months but a significant (P<0.01) decrease from baseline to 12 months. There was no significant change in BMI value over the 12 months period.

HOMA-β showed no significant (P=0.12) change from baseline to 6 months but showed significant (P≤0.01) increases from 6 to 12 months and baseline to 12 months. HOMA-R showed significant (P <0.01) decreases over the three testing periods. There were significant (P<0.05) decreases in systolic CBP from baseline to 12 months. There was no significant change in diastolic CBP from baseline to 6 months but a significant (P<0.05) decrease was seen from 6 to 12 months. At the beginning of the study most of the group (67%) was prescribed premixed insulin, and only 33% was on basal insulin, at 6 months approximately 58% of the group was on premixed insulin given ≥ 2 times daily. At 12 months approximately 58% of patients remained on premixed insulin given ≥ 2 times daily. There was no significant change in medication compliance and instruction following over the three periods of testing. No incidences of diabetes complications were in the group over the 12 months of the study.

3.3 Differences between the Two Ethnic Groups Tables 1 and 2

The mean ages of the families of the two ethnic groups were similar. The durations of DM were 1-13 years in families of East Indian descent and 2-14 years in families of West African descent indicating that there was no significant difference in the duration of the disease.

Table 1. Clinical characteristics of families of East Indian descent with Early Onset Type 2 diabetes

Parameter	Baseline	6 months	12 months	P_1 value	P_2 value	P_3 value
M/F	20/34	19/30	18/29			
Age at diagnosis (years)	31.4±2.7					
Duration of diabetes (years)	1-13					
FPG (mmol/l)	10.1±1.1	10.4±0.8	9.5±0.9	-	-	-
PPG after 2 hours (mmol/l)	16.6±4.6	15.4±2.5	11.0±0.9	--	**	**
HbA1c (mmol/l)	86±16	79±8	63±5	_	**	**
%	10.0±1.5	9.4±0.7	7.9±0.5	--	**	**
FI (mU/L)	9.2±2.3	8.7±1.5	8.5±0.7	-	-	-
Total cholesterol (mmol/l)	5.7±0.4	5.6±0.6	5.6±0.5	-	-	-
HDL-C (mmol/l)	1.3±0.1	1.3±0.2	1.4±0.2	-	*	*
LDL-C (mmol/l)	2.4±0.4	2.4±0.2	2.3±0.5	-	*	*
Triglyceride (mmol/l)	2.0±0.5	1.8±0.3	1.9±0.3	**	-	-
BMI (kg/m^2)	26.2±1.5	25.9±1.7	25.5±0.8	-	-	-
Beta cell function (%)	25.7±3.1	23.2±1.8	27.4±3.2	-	*	-
Insulin Resistance	4.1±0.5	4.0±0.4	3.4 ±0.2	-	*	*
Creatinine (umol/l)	80.8±8.4	76.1±9.1	90.5±10.2	*	*	*
Systolic (mmHg)	132.0±5.1	130.4±2.9	125.4±5.6	-	**	**
Diastolic (mmHg)	89.2±5.1	87.8±0.1	81.5±4.5	-	**	**
Units of insulin per day	57(23-69)	60(34-100)	59(32-79)	-	-	-
Compliance (%)	98	92	96	**	*	-
Incidence of diabetes complications	0	0	2			

*Longitudinal assessment of biochemical parameters at baseline, 6 months and 12 months = no significant change, *,** =significant (P=0.05 and P≤0.01 respectively), P_1= significance of baseline vs 6 month values, P_2 = significance of 6 month vs 12 month values, P_3 = significance of baseline vs 12 month values in families of East Indian descent. M/F = male female ratio, FPG = fasting plasma glucose, PPG= postprandial plasma glucose, FI = fasting insulin, BMI = body mass index, HOMA-β = β cell function, HOMA- R= insulin resistance, HDL-C = high density lipoprotein cholesterol, LDL-C =low density lipoprotein cholesterol. Units of insulin per day is reported as median (interquartile range)*

Table 2. Clinical characteristics of families of West African descent with Early Onset Type 2 diabetes

Parameter	Baseline	6 months	12 months	P_1 value	P_2 value	P_3 value
M/F	19/29	17/29	17/28			
Age at diagnosis (years)	32.9±3.3					
Duration of diabetes (years)	2-14					
FPG (mmol/l)	10.9±1.5	9.1±0.5	7.4±0.7	**	* *	**
PPG after 2 hours (mmol/l)	15.6±3.6	12.4±2.9	10.0±0.9	*	**	**
HbA1c (mmol/l)	97±17	70±8	53±7	**	**	**
%	11.0±1.5	8.6±0.8	7.0±0.7	**	**	**
FI (mU/L)	10.2±2.5	8.9±1.5	9.1±0.8	**	_	**
Total cholesterol (mmol/l)	5.6±0.4	5.3±0.6	5.2±0.5	*	-	**
HDL-C (mmol/l)	1.4±0.1	1.4±0.2	1.5±0.1	-	*	*
LDL-C (mmol/l)	2.3±0.3	2.3±0.2	2.1±0.5	-	*	*
Triglyceride (mmol/l)	1.8±0.5	1.6±0.2	1.6±0.2	*	-	**
BMI (kg/m^2)	27.1±0.2	26.9±0.3	26.3±0.9	-	-	-
Beta cell function (%)	25.8±2.0	29.2±4.1	43.3±6.1	-	**	**
Insulin resistance	4.9±1.0	3.6±0.5	2.9±0.4	**	**	**
Creatinine (umol/l)	100.2+ 5.4	88.1±7.9	100.9±10.8	*	**	-
Systolic (mmHg)	139.0±8.8	135.4±7.9	129.2±6.6	*	**	**
Diastolic (mmHg)	90.2±5.1	88.9±0.9	83.5±4.6	-	*	**
Units of insulin per day	56 (32-69)	60(33-100)	59(31-90)	-	-	-
Compliance (%)	99	96	98	-	-	-
Incidence of diabetes complications	0	0	0			

*Longitudinal assessment of biochemical parameters at baseline, 6 months and 12 months = no significant change, *,** =significant (P=0.05 and P≤0.01 respectively), P_1= significance of baseline vs 6 month values,P_2 = significance of 6 month vs 12 month values, P_3 = significance of baseline vs 12 month values in families of East Indian descent. M/F = male female ratio, FPG = fasting plasma glucose, PPG= postprandial plasma glucose, FI = fasting insulin, BMI = body mass index, HOMA-β = β cell function, HOMA- R= insulin resistance, HDL-C = high density lipoprotein cholesterol, LDL-C =low density lipoprotein cholesterol. Units of insulin per day is reported as median (interquartile range)*

No significant differences in BMI values between the groups were also noted. There was also no significant difference in fasting plasma glucose levels between both groups at the beginning of the study. However there were significant (P <0.05) differences in fasting plasma glucose at 6 months and 12 months between groups. There were significant (P<0.05) differences in post prandial plasma glucose values between groups at 6 months and 12 months. There were significant (P<0.05) differences in HbA1c values between the two groups at baseline, 6 months and 12 months. HDL-C values were significantly (P<0.01) lower at baseline, 6 and 12 months in the East Indian group than in the West African group while TG values at baseline and at 12 months were significantly (P<0.05) higher in the East Indian group than in the West African group.

Serum creatinine values were significantly (P<0.05) higher during the three periods of testing in families of West African descent than in those of East Indian descent. At the start of the study HOMA-β was approximately the same in the two ethnic groups. However there was an increase in HOMA-β at 6 months in the families of West African descent but a decrease in the families of East Indian descent. At 12 months HOMA-β almost doubled the value of baseline in the West African group.

HOMA-R showed a steady decrease over 12 months in both groups but the decrease in HOMA-R at 12 months was more striking in the families of West African descent (2.9±0.4) than in families of East Indian descent (3.4±0.2). Systolic CBP values were significantly(P<0.01) higher for baseline, 6 and 12 months in the West African group than in the East Indian group. There were no significant differences in diastolic CBP between the two groups at baseline, 6 and 12 months. Both groups started the study with most

of their members on pre-mixed insulin and this pattern followed throughout the study. Therefore there was no difference in medications for diseases between the groups. Medication adherence and instruction following were greater in families of West African descent than families of East Indian descent. The families of East Indian descent had two members who developed nephropathy.

3.4 Discussion

Indians and people of African descent living in the Americas have higher DM and mortality and morbidity associated rates compared to Caucasians [1-4]. Varied reasons have been given for the disparities including lack of access to proper health care and socio-economic constraints that prevent early visits to clinics before the health related problems become chronic [10-14]. The two groups of ethnic families studied were predominately poor and lived in the same metropolitan area in Jamaica that the clinic for DM is located. Family members and some of the participants had been involved in an earlier genome wide study for the MODY genes [20]. The participants were seen by the same group of healthcare workers specializing in diabetes care. Anti-hypertensive drugs commonly prescribed for those with hypertension were thiazides, reserpine and ACE inhibitors. Statins were generally used along with dietary counseling to manage dyslipidaemia. Basal and premixed insulin were prescribed for glycaemic control. However, Humulin 70/30 appeared to be the premixed insulin formulation most commonly prescribed. The American Association of Clinical Endocrinologists recommends insulin therapy for T2DM patients with initial HbA1c values >9.0% [26]. In both groups mean baseline HbA1c was above 9.0% and the treatment regimen seemed to follow standard practices for persons with T2DM who are at risk of developing micro and macrovascular complications [27]. Therefore the difference between the two groups may not be due to the differences of therapies for the disease. The prescribed drugs are usually available at reduced cost to patients who access care at the University Hospital of the West Indies. All participants in the study had less than a high school level of education and tend to shop for food at the local shops and market in the general area. However, there were no significant differences in levels of education and socio-economic variables which could be used to explain the difference in glycaemic control seen between the two ethnic groups. There was no significant difference in fasting plasma glucose levels between both ethnic groups at the beginning of the study. At 6 and 12 months there were significant ($P < 0.01$) differences in both fasting and postprandial plasma glucose values between groups with the East Indian families having significantly higher fasting and postprandial glucose values at 12 months. HbA1c was significantly lower at 12 months in the families of West African descent compared with in families of East Indian descent (7.0±0.7% versus 7.9±0.5% [53±7 mmol/l versus 63±5 mmol/l], P=0.05). The families of West African descent had significantly lower TC, TG and LDL-C and higher HDL-C values over the entire 12 months of the study than those of East Indian descent. The significantly (P=0.05) higher HbA1c levels at 6 and 12 months in the families of East Indian descent than those of West African descent might have impacted the lipid levels in this group. Uncontrolled glycaemia modifies HDL-C through increased glycation of the protein component which reduces the antioxidant effects of HDL-C and leads to increased LDL-C instead of HDL-C [28,29]. Gatti et al. in a study in 2009 [30] reinforced the link between dyslipidaemia and poor glycaemic control by indicating that high TG and low HDL-C signal poor glycaemic control in some DM patients. The families of East Indian descent have a characteristic lipid profile of elevated TG, and LDL-C and, reduced HDL-C, which were shown as characteristics of some persons with uncontrolled T2DM [31]. The prolonged elevated TG over twelve months in the East Indian group is a feature of a particular type of dyslipidaemia seen in some groups with T2DM. A previous study done on lipid profile of these two ethnic groups in Jamaica showed that persons of East Indian descent had higher glucose and TG levels than those of West African descent [32]. Since factors such as BMI, age, duration of DM and current treatment that have previously been linked to both glycaemic control and dyslipidaemia [33] were not significantly different in both groups, it seemed that the poorer glycaemic control and dyslipidaemia seen in the East Indians may be linked to ethnicity. This study is not the first to link glycaemic control and dyslipidaemia to ethnicity. Glycaemic control and dyslipidaemia have also been linked to ethnicity in a study of Malaysians [34]. The researcher reasoned that the high TG which is a feature of dyslipidaemia seen in the East Indians as reported in a previous study [30] may have impacted β cell function which in turn did not response adequately to the glycaemic challenge. High TG

impacts β cell function as elevated TG is associated with pancreatitis [35]. Hypertriglyceridaemia associated pancreatitis might explain why a significant reduction in insulin resistance from baseline to 12 months in families of East Indian descent did not result in increased β cell function and lower fasting and post prandial glucose values. Beta cell response to glucose challenge is also linked to ethnicity and genetics. The improvement in β cell function over 12 months was almost two fold that of baseline value in the families of West African descent and this improvement possibly led to lower fasting and postprandial glucose value at 12 months than those of East Indian descent. Genetics might have limited the action of β cells to the glucose challenge in the East Indians. Donath et al. [36] proposed that genetics might play a part in the response of the β cells to hyperglycaemia with certain groups unable to respond effectively. In a comparative study of South Asians with other racial/ethnic groups in the USA including African Americans, the data showed that the Asians had lower β cell function and higher insulin resistance than the other ethnic groups [37]. These findings along with the findings from our study suggested that Asians which include Indians may have lower β cell function and are unable to compensate for higher glucose levels which may lead to poorer glycaemic control than other ethnic groups. Data also show that Asians experience earlier decline in β-cell function, compared with other ethnic groups. Some studies showed that the prevalence of insulin resistance in Asian men was threefold to fourfold greater than lean men of the other ethnic groups despite lifestyle and BMI similarities. When β-cell function was assessed in a subgroup of South Asian and Caucasian men, it was observed that that Asians had a 30% increase in basal β-cell responsiveness; however, this increase in β-cell function was not sufficient to compensate for the degree of insulin resistance [38]. In 2003 at the end of the study, the families of East Indian descent in Jamaica had higher DM related mortality compared with the families of West African descent (15% versus 8%). An international study however indicated that persons from India appear to be of greater risk for DM than any other ethnic groups [39] and are more prone to DM related complications [40]. It would be interesting to compare the glycaemic control in Afro-Caribbean and Indians with atypical T2DM in the USA and United Kingdom to the glycaemic control seen in West Africans and East Indians with early onset T2DM in Jamaica. Cross border and cross cultural data might help to unravel the disparities in glycaemic associated with certain ethnic groups worldwide. Although ethnicity and genetics may have played significant roles in glycaemic control in both groups; not fully complying with medication guidelines and instructions could have negatively impacted glycaemic control in families of East Indian descent. The families of West African descent were willing to follow instructions and prescription guidelines regardless of the age or ethnicity of the healthcare workers. The researcher reasoned that cultural/ethnic differences in medication adherence and instruction following as seen in another study [41] might be influencing the disparity in glycaemic control. The researcher also reasoned that families of African descent might feel more comfortable in the DM clinic since Jamaica's population is predominately of African descent [42] and, it would be that more likely the attending physician or healthcare worker would be of the same ethnicity as the patient. Two members of East Indian descent developed nephropathy at 12 months. These members were older and had diabetes over 10 years. Duration of diabetes is linked to macro and microvascular complications [13], however there is also a strong association between glycaemia as measured by HbA1c and risk of developing nephropathy [25]. Poor glycaemic control, serum lipid levels and genetics are some of the risk factors for the development of diabetic nephropathy [42]. The families of East Indian descent presented these factors more so than the families of West African descent and these factors may have accounted for the two incidences of nephropathy in the East Indians. Quite possibly more incidences could have developed in these families however some of them were managed with ACE inhibitors which have been shown to decrease the risk of developing nephropathy and cardiovascular events in patients with T2DM [27]. The study showed that evidence based clinical recommenddations for diabetes care are important [43]. A limitation of the study was that prescriptions and instructions through standard for certain conditions at the DM clinic in Jamaica may still vary by healthcare worker. Diet was not followed in the cohort but tend to be standard for the families in the study as groups from similar social classes in Jamaica tend to access foods which are cheap from shops and the municipal market in the area that they live in. The participants from the two groups reported DM related mortality rates. However the figures could not be verified from the dockets at the DM clinic. The researcher also

noted that some members of the two ethnic families were not recruited for the study because of major DM related complications that pre-dated the study.

4. CONCLUSION

The families of East Indian descent had poorer glycaemic control at 12 months than those of West African descent. The poor control may be linked to genetics as seen in triglyceride and HDL profile over 12 months but may have been influenced by ethnic differences in medication adherence and instruction following.

CONSENT

All participants signed an informed consent form.

ETHICAL APPROVAL

Ethical approval was granted by UHWI Ethics Committee.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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