(all r = 0.9, P < 0.05), and log[1,5-AG] was negatively correlated with them (all r = -0.9, P < 0.05).

Conclusion: Modified hypoglycemia score was negatively correlated with 1,5-AG in well-controlled type 2 diabetes patients, but more number of subjects would be required to estimate the 1,5-AG as an independent determinant. However, in the subgroup analysis with insulin group, we could identified both the score and 1,5-AG levels were strongly correlated with indices of mean glucose and glycemic variability, which were also closely related with the hypoglycemia observed during the CGMS.

PO090

A LONGITUDINAL STUDY ON THE INTERRELATIONSHIPS BETWEEN HBA1C, FRUCTOSAMINE AND CONTINUOUS GLUCOSE MONITORING DERIVED MEAN BLOOD GLUCOSE IN TYPE 2 DM PATIENTS

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Background: The use of HbA_{1c} to assess glycaemic control in diabetes mellitus is virtually universal. Whilst most clinicians are familiar with HbA_{1c}, there are clinical settings where it is inaccurate (e.g. haemoglobinopathy). There is less familiarity with alternative measures of glycaemia, such as serum fructosamine (FRUC) and continuous glucose monitoring derived mean blood glucose (MBG). The primary aims of this study were to determine if FRUC and MBG correlated well with HbA_{1c} in Type 2 diabetes (T2DM) patients and to derive formulas to convert FRUC and MBG values to HbA_{1c}.

Method: 90 adult T2DM subjects were recruited in a controlled manner, with no more than 20 subjects from each of the following HbA_{1c} quintiles: 6 – 6.9%, 7 – 7.9%, 8 – 8.9%, 9 – 9.9%, ≥10%. Subjects with abnormal haemoglobin/MCV/MCH, current pregnancy or lactation, blood transfusion in the past 3 months, eGFR <60 ml/min, erythropoietin or haematinic therapy, abnormal serum albumin or abnormal liver enzymes were excluded. At baseline (month 0), after one (month 1) and two months (month 2), subjects had blood drawn for HbA_{1c} and FRUC. At month 0, 1 and 2, each subject had a continuous glucose monitoring device (iPro[™]2 with Enlite[™], Medtronic Minimed, USA) attached for 6 days (total of 18 days over 3 months). MBG values were downloaded after device removal.

Table 1. Pearson's correlation between HbA1c, FRUC and CGM-MBG $\,$

	Pearson Correlation, r								
	Month 0			Month 1			Month 2		
	MBG	$HbA_{\rm 1c}$	FRUC	MBG	HbA_{1c}	FRUC	MBG	$HbA_{\rm 1c}$	FRUC
MBG	1.00	0.63	0.50	1.00	0.65	0.40	1.00	0.62	0.51
HbA_{1c}	0.63	1.00	0.61	0.65	1.00	0.53	0.62	1.00	0.52
FRUC	0.50	0.61	1.00	0.40	0.53	1.00	0.51	0.52	1.00

*All ${\rm p}$ values are <0.01 and are not displayed for simplicity of viewing.

Result: 87 out of 90 subjects completed the study. 52% were male, mean age and diabetes duration were 57.4±9.9 years and 12.1±7.5 years respectively. Mean glycaemic control improved significantly over the study period: HbA_{1c} (%) decreased from 8.4±1.8 (Month 0) to 8.1±1.6 (Month 1), 7.9±1.4 (Month 2) [p = 0.029]. FRUC (umol/L) decreased from 350±84.8 (Month 0) to 336±87.8 (Month 1), 325±58.5 (Month 2) [p = 0.003]. MBG (mmol/L) decreased from 9.5±2.2 (Month 0) to 9.1±2.5 (Month 1), 8.7±2.5 (Month 2) [p=0.002]. There were strong correlations between HbA_{1c}, FRUC and MBG throughout the study period with Pearson's correlation r-values ranging from

0.4 to 0.65 (Table 1). The highest correlation was between HbA_{1c} and MBG (cumulative r=0.66). FRUC correlated better with HbA_{1c} than with MBG. The formulas derived from the linear correlations of each parameter were:

HbA_{1c} = $(0.034 \cdot FRUC) - 3.22 [r = 0.61, r^2 = 0.37]$,

HbA_{1c} = $(1.32 \cdot \text{MBG}) - 4.02$ [r = 0.65, r² = 0.42],

FRUC = $(21.6 \cdot MBG) + 131.6 [r = 0.51, r^2 = 0.26]$.

Conclusion: Our results indicate that HbA_{1c} , FRUC and MBG have a high degree of correlation in T2DM patients without anaemia, gestation, renal and liver disease. Significant changes in glycaemia could be detected by all three modalities over 1 month intervals. The formulae derived to describe the relationships between HbA1c, FRUC and MBG may be used to guide clinicians in interpreting results.

PO091

TRIGLYCERIDES/HDL-CHOLESTEROL RATIO IS CORRELATED WITH INSULIN RESISTANCE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Background: Diabetes has long been recognized to be an independent risk factor for cardiovascular disease (CVD). Cardiovascular complications are now the leading causes of diabetes-related morbidity and mortality. Prognosis of patients with diabetes is highly dependent on the presence of CVD. Insulin resistance (IR) plays an important role for development and progression of diabetes, and also may act as a predictor for development of CVD. Insulin resistance is thought to be a common finding in several metabolic disorders, such as type 2 diabetes mellitus (T2DM), impaired glucose tolerance (IGT), metabolic syndrome (MetS), hypertension, hypertriglyceridemia, low HDL cholesterol, hypercholesterolemia, hyperuricemia, obesity, and low serum testosterone. Evaluations of IR and β -cell function are important for understanding the disease status and selection of pharmacologic treatment. Triglyceride/HDL-cholesterol (TG/HDL) ratio is a new surrogate marker for IR. Diagnostic value of TG/HDL ratio is as good as HOMA-IR and could be used as an indicator of IR in clinical setting. Because IR plays important role in development of CVD and the TG/HDL-c ratio was significantly associated with these conditions, we conducted this study to determine whether the TG/HDL-c ratio is associated with IR in patients with T2DM. HOMA-IR value more than 2.0 is considered to be positive for IR.

Method: This is a cross-sectional study, which includes men and women with T2DM who were on routine follow up in private out patient diabetic clinic. Informed consent was obtained from all patients. Exclusion criteria for this study are: history of alcohol use, history of having cardiovascular disease or cerebrovascular disease. Patients with end stage renal disease or on dialysis and with active hepatic disease are also excluded from the study. Insulin Resistance was measured using HOMA-IR and TG/HDL ratio measured using simple laboratory measurement. TG/HDL-c ratio more than 3.5 for male and more than 2.5 for female is considered abnormal. Statistical analysis was performed using SPSS 17.0 and Pearson's correlation test.

Result: a total of 227 subjects, 165 males and 62 females were enrolled. Mean value of TG/HDL-c ratio was 4.56, mean value of HOMA-IR was 6.3967. From the statistical analysis we found that TG/HDL-c ratio was significantly associated with HOMA-IR (p < 0.05. 95% CI) respectively.

Conclusion: In our study, TG/HDL-c ratio was significantly correlated with IR in patients with T2DM.

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PO092

FASTING PLASMA INSULIN LEVEL IS CORRELATED WITH THE BETA CELLS FUNCTION IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Background: Insulin Resistance (IR) is a common finding in diabetes mellitus and may serve as a measure of efficacy of therapies (exercise, exogenous insulin, sulfonylureas, and PPAR gamma agonists) for diabetes mellitus and as a possible marker for risk of developing type 2 diabetes mellitus (T2DM). Insulin Resistance is widely believed to be able to be predicted using measurement of plasma insulin level and usually marked by existence of fasting hyperinsulinemia. However, recently fasting hyperinsulinemia itself was found to have a primary pathogenic role in the development of diabetes, independent of insulin resistance. Further analyses revealed that individuals with a high relative fasting plasma insulin concentration (for their degree of adiposity and insulin resistance) are at increased risk for a decline in early phase insulin secretion, but not in insulin sensitivity, before the onset of diabetes. Since beta cell dysfunction is the core of the pathogenesis of T2DM, therefore the aim of this study is to determine whether fasting plasma insulin level correlates with residual beta-cell function.

Method: The study was a cross-sectional study, which had enrolled men and women subjects with type 2 diabetes (T2DM) that were on routine follow up in a private outpatient diabetic clinic. The study included T2DM patients with age >40 years old. Informed consents were obtained from all patients. Exclusion criteria for the study group were: history of alcohol use, history having cardiovascular or cerebrovascular disease. Patients with end stage renal disease or on dialysis and with active hepatitis disease were also excluded from the study. Fasting Plasma Insulin Levels were measured as well as beta-cell function using HOMA-B. Fasting plasma insulin was considered within normal range if the value was <25mIU/L, above that was considered hyperinsulinemia. Statistical analysis was performed using SPSS for Windows 17.0 and Spearman's correlation rank test.

Result: A total of 206 subjects were enrolled, consisting of 144 (69.9%) males and 62 (30.1%) females. Mean laboratory result for fasting plasma insulin level was 13.7 ± 4.1 , while mean result for HOMA-B was 65.17 ± 3.34 . Fasting plasma insulin level is significantly correlated with HOMA-B (p < 0.05 95% CI), respectively.

Conclusion: Fasting plasma insulin level was significantly correlated with beta cell function, however further study is needed to clarify.

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PO093

ASSOCIATION BETWEEN RED BLOOD CELL DEFORMABILITY AND DIABETIC RETINOPATHY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

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Background: Red blood cell (RBC) deformability is an ability of RBC to change shape under stress. RBC deformability has been demonstrated to be impaired in diabetes mellitus. But, little is known about the association between impaired RBC deformability and type 2 diabetes mellitus (T2DM). The aim of this study was to determine the influence of RBC deformability on T2DM.

Method: We conducted a cross-sectional study with 198 patients with T2DM who visited in Yeungnam university hospital from Mar. to Jul. 2014. Patients with end stage renal disease and who are taking a pentoxifylline and ginkgo biloba were excluded. RBC deformability was measured by using a Rheoscan-D (Rheo-Meditech, Seoul, Korea), and expressed as elongation index (EI). The EI was measured at 3 Pa. We divided the EI into quartile (Q1, Q2, Q3, Q4 from lowest to highest EI).

Result: 193 patients (mean age 59.82±12.29 years, M:F = 100:93) were finally included. EI had significantly negative correlation with the levels of glycated hemoglobin, and positive correlation with HOMA-B, respectively (β –23.52, p = 0.01 and β 520.03, p = 0.02, respectively). Patients with micro complications had lower EI compared with patietns without complications (EI 0.303623 vs. 0.310637, p = 0.01). Of them, patients with retinopathy had lower EI compared with patients without retinopathy (EI 0.300449 vs. 0.309653, p = 0.00), whereas patients with nephropathy or neuropathy and macro complications had no significant difference in EI. After adjustment for age, sex, hypertension, smoking, and lipids, lower EI remained significantly associated with the prevalence of diabetic retinopathy (Odd rati! o for Q1 compared with Q4, 4.16; 95% confidence interval, 1.43–12.13). Conclusion: In patients with T2DM, there are significant relationship between RBC deformability and glycemic control, beta cell function and diabetic retinopathy. These results suggest that decreased RBC deformabiliy is a useful surrogate marker for predicting diabetic retinopathy.

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Healthcare delivery

PO094

INSULIN PUMP THERAPY PROGRAM FOR CHILDREN IN KAZAKHSTAN – PUBLIC/PRIVATE PARTNERSHIP

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