Wednesday, August 1, 2018

Poster Session: 9:30 AM - 5:00 PM

Lipids/Lipoproteins

B-135

Sphingolipidomic Analysis Reveals Decreased Circulating Sphingomyelins and Ceramides in Sickle Cell Disease Patients

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Background: This study aimed to identify levels of C16-C24 sphingomyelin (CerP-Cho) and C16-C24 ceramide (CER) in serum obtained from SCD patients and controls. Circulating levels of neutral sphingomyelinase activity (N-SMase), ceramide-1-phosphate (C1P), sphingosine-1-phosphate (S1P) were also determined. **Methods:** Blood was collected from hemoglobin (Hb)A volunteers and homozygous HbSS patients. Serum levels of C16-C24 CerPCho and C16-C24 CER were determined by an optimized multiple reaction monitoring method using ultra fast-liquid chromatography coupled with tandem mass spectrometry. Serum activity of N-SMase was assayed by standard kit methods, C1P and S1P levels were determined by enzyme-linked immunosorbent assay. **Results:** A significant decrease was observed in serum levels of C18-C24 CerPCho and very-long-chain C22-C24 CERs in SCD patients compared to controls. A significant positive correlation was found between serum total cholesterol levels and C18-C24 CerPCho, C22-C24 CERs in SCD patients. Patients with SCD had significantly elevated serum activity of N-SMase, increased circulating levels of C1P and S1P compared to controls. **Conclusions**: Future studies are needed to understand the role of decreased CerPCho and CERs in the pathophysiology of SCD.

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The effect of obesity, vitamin D level and vitamin D receptor *Fok I* single nucleotide polymorphism on serum lipid profile in children and adolescents in West China

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Background: The most of obesity could lead to dyslipidemia. Obesity closely correlates with vitamin D and vitamin D receptor (VDR). The objective of this study is to reveal the effects of obesity, serum vitamin D level and VDR *Fok I* genotype on serum lipid profile of children and adolescents in west China. **Methods:**452 children and adolescents were recruited from West China Second University Hospital to participate in this cross-section study. All the participants were divided into two groups -- obese group and non-obese group according to the body mass index (BMI). Serum vitamin D level, serum lipid level and VDR *Fok I* gene polymorphism were detected in the laboratory. Based on the level of Serum vitamin D, all subjects were divided into three group, vitamin D normal group, vitamin D insufficiency group and vitamin D deficiency group. All children and adolescents were classified into TT genotype and C allele carriers on the basis of the different expression of VDR *Fok I* gene. The impact of obesity, vitamin D level and VDR *Fok I* genotype on lipid level was investigated by analysis of the experiment data. All data were analyzed by independent-samples T test and One-way ANOVA , and adjusted for age by covariance test. **Results:** 1. The concentrations of serum vitamin D in the obese group was lower than that in the non-obese group. The levels of serum TC, TG, HDL, LDL, Apo-A1, Apo-B, LDL/HDL, TG/HDL in the obese group were higher than those in the non-obese group (P=0.000, P=0.003, P=0.000, P=0.000, P=0.000 P=0.000, P=0.000). 2. There was no difference in genotype distribution and allele frequency of VDR *Fok I* site between Obese and non-obese group. 3. In all children and adolescents, there was no difference in serum vitamin D level and lipid profile between C allele carriers and TT genotype in non-obese group. However, in obese group, the C allele carriers had much lower concentrations of TC, TG, Apo-B, TC/TG, LDL/HDL, TG/HDL than TT genotype (P=0.000, P=0.017, P =0.000, P =0.009, P=0.033, P =0.020). 4. The concentration of HDL-C and Apo-A1 in the vitamin D deficiency group was significantly higher compared with the insufficiency and normal group (P=0.007, P=0.001; P=0.013, P=0.002). Moreover, the vitamin D insufficiency and deficiency group had higher concentration of TC and LDL-C compared with vitamin D normal group (P=0.025, P=0.012; P=0.044, P=0.032).5. In non-obese group, C allele carriers had higher TC, HDL, Apo-A1 in vitamin D deficiency group compared with TT genotype (P=0.039, P=0.025, P=0.009). In obese group, C allele carriers had lower concentrations of TC, TG, Apo-B, TC/HDL, LDL/HDL and TG/HDL in vitamin D deficiency group than TT genotype (P=0.009, P=0.011, P=0.001, P=0.000, P=0.007, P=0.008). **Conclusion:** The level of lipid is influenced by mutation of VDR Fok I gene, the level of vitamin D and obesity in children and adolescents in west China. The effect of the mutation of VDR genotype could be reinforced when the subjects had low concentration of vitamin D. The molecular mechanism of VDR genotype effect on lipid level requires a further research.

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Correlation of two Lipoprotein(a) assays, Lipoprotein(a) particle concentration and Lipoprotein(a) cholesterol assay

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Background: Lipoprotein(a) [Lp(a)] is consisted of modified LDL particle and an apolipoprotein(a) [apo(a)]. It is considered as a risk factor for cardiovascular event for many years, but most of the methods used in clinical laboratory are affected by apo(a) size heterogeneity and there are problems with size-sensitive results as well as standardization between assays. So we investigate the correlation and their characteristics two Lp(a) assays Tina-quant Lipoprotein(a) Gen.2 [LA(2)] (Roche Diagnostics GmbH) for Lp(a) particle concentration and Tina-quant Lipoprotein (a) (Latex) [LA(1)] for Lp(a) cholesterol assay. **Methods:** Total 400 consecutive clinical samples submitted for Lp(a) test from March to September, 2015, were studied. All the analysis were done with cobas c 501 analyzer (Roche Diagnostics GmbH) and more than 30 mg/dL of LA(1) and more than 75 nmol/L of LA(2) were considered as the clinical cut-off for the high risk group for atherosclerosis. For the correlation analysis of the results from LA(2) with LA(1), conversion factor, 0.4167 in the manufacture's insert was used. We performed the correlation analysis, kappa value, Fisher's exact test, and Student's t-test (when p values < 0.05 considered as statistically significant). All the statistical analyses were done with Analyse-it for Microsoft Excel (Ver. 4.65). **Results:** The correlation between two assays were not bad (Pearson's r = 0.7). And LA(1) results of 391 samples were larger than LA(2) converted, which showed statistical significance (P<0.00). Sixty-one samples among 400 samples were classified as high risk group in LA(2) and 114 samples were classified as high risk group in LA(1). The kappa value of high risk group of two assays was 0.62 (95% confidence interval 0.53-0.70). **Conclusions:** We observed that LA(1) results tended to be overestimated compared to LA(2) ($P<0.05$). And there were discrepancy between two assays for classifying high risk group for atherosclerosis. There are not enough clinical studies based on the difference between particle concentration and mass analysis. Also, standardization of Lp(a) is still far, many clinical guideline are still based on the older assay affected by apo(a) size heterogeneity and ethnic difference. So we suggested that the standardization should be done as soon as possible. And further studies including the establishment of the reference interval in the various ethnic group and clinical studies based on Lp(a) particle concentration are required.

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Performance characteristics of a novel direct assay for small, dense LDL cholesterol on Roche and Beckman chemistry analyzers

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Background: Small, dense LDL (sd LDL) is a highly atherogenic lipoprotein, and many clinical studies have strengthened the relationship between sd LDL-C level and CHD. We have developed a simple and fully-automated homogeneous assay for quantification of sd LDL-C (s LDL-EX"SEIKEN"). The assay received 510(k) clearance in August, 2017 based upon the performance data obtained on the Roche / Hitachi 917 analyzer. We further evaluated the assay performance on additional systems; Roche cobas c501 and Beckman AU5800. **Method:** Precision, linearity, limit of quantitation (LOQ) studies and a comparison study to Hitachi 917 were performed according to CLSI approved guideline EP05-A2, EP06-A, EP07-A2, EP17-A2 and EP09-A3, respectively. Equivalency between serum and plasma samples was evaluated using 40 paired samples. Reagent open vial stability was evaluated for up to 5 weeks. A calibration frequency was determined from results of weekly measurements up to 3 weeks by using the calibration curve obtained at the initial time point. **Result:** Within-laboratory %CV of the assay was 1.3-2.3% on Roche cobas c501, and

1.8-3.3% on Beckman AU5800. Linear assay range was confirmed up to 100 mg/dL for both analyzers. LOQ was established as 1.6 mg/dL and 0.6 mg/dL on Roche cobas c501 and Beckman AU5800, respectively, ensuring the lower measurement limit of the assay (4 mg/dL). Results with patient samples were substantially equivalent among Roche/Hitachi 917, Roche cobas c501 and Beckman AU 5800 (r>0.95). No sample matrix related bias was observed in a study with 40 paired serum and plasma samples. Reagent remained stable and functional in opened vials for 4 weeks and the calibration was stable for two weeks on both analyzers, comparably to Hitachi 917. **Conclusion:** The analytical performance of sd LDL-C assay was verified on Roche cobas series and Beckman AU series. These instrument families will be added on the list for this FDA-approved assay to lead expanded use in clinical laboratories.

B-139

Performance Evaluation of the Atellica CH Chol_2, D-HDL, DLDL, and Trig Assays

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Background: The purpose of the investigation was to evaluate the analytical performance of the Atellica® CH Chol_2, D-HDL, DLDL, and Trig Assays on the Atellica CH Analyzer. Measurement of these assays is used in assessing the risk of developing heart disease and monitoring patients with existing heart disease. The Atellica CH D-HDL Assay (D-HDL) and the Atellica CH DLDL Assay (DLDL) involve two reactions. The first reaction uses cholesterol esterase and cholesterol oxidase to isolate HDL or LDL cholesterol. The second reaction uses cholesterol esterase, cholesterol oxidase, and peroxidase—along with hydrogen peroxide, 4-aminoantipyrine, and a hydrogen peroxide-detecting molecule—to form a quinoneimine complex in a Trinder endpoint reaction. The absorbance of the complex is measured, and the intensity is directly proportional to the amount of cholesterol in the sample. The Atellica CH Chol_2 Assay (Chol_2) uses the same methodology; however, it involves only the second reaction. The Atellica CH Trig Assay (Trig) uses a similar methodology: the molecule is broken down to glycerol and fatty acids, eventually producing hydrogen peroxide. When the hydrogen peroxide is combined with 4-aminophenazone, 4-chlorophenol, and peroxidase, it produces a quinoneimine-dye complex. **Method:** Performance testing included precision and accuracy. Assay precision was evaluated using the Clinical and Laboratory Standards Institute (CLSI) guideline EP05-A3. Each sample was assayed in duplicate twice a day for 20 days. Method comparison studies were conducted according to CLSI EP09-A3, with patient-sample results compared to results from the ADVIA® 1800 Clinical Chemistry System. **Results:** For Chol_2, within-lab precision ranged from 0.8-1.3% CV in serum/plasma samples. For D-HDL, within-lab precision ranged from 0.7-2.0% CV in serum/plasma samples. For DLDL, within-lab precision ranged from 1.0-2.3% CV in serum/plasma samples. For Trig, within-lab precision ranged from 1.0-2.5% CV in serum/plasma samples. The Chol_2 serum method comparison study yielded a regression equation of y = 0.97x + 1 mg/dL with r = 0.997, versus the ADVIA 1800 Chol_2 Assay. The D-HDL serum method comparison study yielded a regression equation of y = 0.97x + 1.4 mg/dL with r = 0.999, versus the ADVIA 1800 D-HDL Assay. The DLDL serum method comparison study yielded a regression equation of y = 0.99x - 0.8 mg/dL with r = 1.000, versus the ADVIA 1800 DLDL Assay. The Trig serum method comparison study yielded a regression equation of y = 0.98x + 0.5 mg/dL with r = 0.999, versus the ADVIA 1800 TRIG_2 Assay. **Conclusions:** The Atellica CH Chol_2, D-HDL, DLDL, and Trig Assays tested on the Atellica CH Analyzer demonstrated acceptable precision. Method comparison results showed acceptable agreement with an on-market comparative analyzer.

B-140

Early Postoperative Changes of Sphingomyelins and Ceramides After Laparoscopic Sleeve Gastrectomy

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Background: This study aimed to determine early postoperative changes of serum sphingomyelin (SM) and ceramide (CER) species following laparoscopic sleeve gastrectomy (LSG). **Methods:** Twenty obese patients [mean body mass index (BMI) 45,64 ± 6,10 kg/m^2] underwent LSG and normal weight control patients (mean BMI 31,51 ± 6,21 kg/m^2) underwent laparoscopic cholecystectomy. Fasting blood samples were collected prior to surgery, at day 1 and day 30 after surgery. Circulating levels of C16-C24 SMs, C16-C24 CERs and sphingosine-1-phosphate (S1P) were determined by an optimized multiple reaction monitoring (MRM) method using ultra fast-liquid chromatography (UFLC) coupled with tandem mass spectrometry (MS/MS). Ceramide-1-phosphate (C1P) levels were determined by enzyme-linked immunosorbent assay (ELISA). Lipid profile, routine biochemical and hormone parameters were assayed by standard kit methods. Insulin sensitivity was evaluated using homeostatic model assessment for insulin resistance (HOMA IR). **Results:** A significant decrease was observed in serum levels of very-long-chain C24 SM, very-long-chain C22-C24 CERs and C1P in LSG patients after postoperation day 1 and day 30 compared to preoperation levels. At 30 days postsurgery, BMI was reduced by 11 %, fasting triglycerides were significantly decreased, and insulin sensitivity was increased compared to presurgery values. A significant positive correlation was found between HOMA-IR and serum levels of C22-C24 CERs in LSG patients. **Conclusions**: We conclude that very long chain CERs may mediate improved insulin sensitivity after LSG.

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Performance of Diazyme Laboratories, Inc. Lp(a) Assay Assay on the VITROS® 4600 Chemistry System and the VITROS® 5600 Integrated System.

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Background: The Diazyme Laboratories, Inc. Lp(a) Assay is intended as a latex particle enhanced immunoturbidimetric assay for the in vitro quantitative determination of lipoprotein(a) concentration in serum or EDTA plasma. Lipoprotein (a) is a cholesterol-rich lipoprotein particle found in human serum. There is substantial evidence linking lipoprotein (a) excess to a high risk for premature coronary heart disease (CHD), increased risk of myocardial infraction (MI), stroke, restenosis after angioplasty (PTCA) and coronary bypass procedures. The Diazyme Lipoprotein (a) Assay is based on a latex enhanced immunoturbidimetric methodology. Lp(a) in the sample binds to specific anti-Lp(a) antibody, which is coated on latex particles, and causes agglutination. The degree of the turbidity caused by agglutination can be measured optically at 700 nm and is proportional to the amount of Lp(a) in the sample. **Methods:** The performance of the Diazyme Lp(a) assay on the VITROS® 4600 Chemistry System and the VITROS® 5600 Integrated System was assessed on the VITROS MicroTip assay processing side of the MicroImmunoassay Center using 4.0 uL patient samples and the Diazyme Lp(a) reagents on the VITROS 4600/5600 Systems compared to predicate device Roche Hitachi 917 analyzer following CLSI: EP9-A2 guidelines. **Results:** The accuracy of Diazyme Lp(a) assay was evaluated with 80 patient serum samples (0.57 -108.34 mg/dL) on patients on the VITROS 4600 and VITROS 5600 System. Both showed excellent correlation with the Roche Hitachi 917. VITROS 4600 System R^2 value of 0.9918 with a slope= 0.9737, and y-intercept of +2.495. VITROS® 5600 System R^2 value of 0.9935 with a slope= 0.969, and y-intercept of -0.3492. A 20-day precision study conducted on the VITROS 4600 system at mean Lp(a) concentrations of 16.3 mg/dL and 47.9 mg/dL resulted in within-laboratory percent coefficient of variation (%CV) of 3.02 % and 1.74% respectively, for the VITROS 4600 System and 2.94% and 1.13% respectively, for the VITROS 5600 System. . The Limit of Quantification (LoQ) check for the VITROS® 4600 and VITROS 5600 Systems was found to be ≤5.44 mg/dL. At 17 mg/dL common interfering endogenous substances of ascorbic acid 10 mM, bilirubin 40 mg/dL, conjugated bilirubin 40 mg/dL, hemoglobin 1000 mg/dL and triglycerides 1000 mg/dL showed no significant interference (≤ 10%). **Conclusion:** The Diazyme Lp(a) assay run on the VITROS 4600 and VITROS 5600 Systems demonstrated excellent correlation with the Roche Hitachi 917 Clinical Chemistry Analyzer, exceptional precision, and low-end sensitivity. Additionally, the assay was free from interference by endogenous substances at clinically relevant Lp(a) concentrations.

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Postprandial GLP-1 response to a high-fat meal is blunted in obese adolescents with insulin resistance and metabolic dyslipidemia

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Background: Obesity and insulin resistance are becoming increasingly prevalent in adolescents and are commonly associated with dyslipidemia. Postprandial, rather than fasting, dyslipidemia independently predicts cardiovascular disease risk and is characterized by intestinal triglyceride-rich lipoprotein (TRL) overproduction. Co-secreted intestinal peptides, glucagon-like peptide 1 (GLP-1) and 2 (GLP-2), have been shown to attenuate and paradoxically augment intestinal TRL output, respectively, in both animal and human studies. We hypothesize that postprandial GLP-1 and GLP-2 responses are

altered in obese adolescents with insulin resistance and/or postprandial dyslipidemia. **Methods:** Normal weight (n=15; 8M/7F) and obese (n=15; 8M/7F) adolescents underwent an oral fat tolerance test (83% kcal from fat). Blood was collected at fasting and 1, 2, 4, and 6 hours following meal ingestion. The lipid profile, glucose, and insulin were measured on the Abbott ARCHITECT ci4100 analyzer. GLP-1 (active and total) and GLP-2 were measured by ELISA. The area under the curve (AUC) and incremental AUC (iAUC) of the postprandial profile was calculated using the trapezoidal method, with 0 and fasting values used as the baseline, respectively. Data were log-transformed prior to analysis if not normally distributed. AUC and iAUC were compared using independent samples t-test. Two-way, mixed analysis of variance (ANOVA) with time as a repeated measure, was used to test differences between each postpandial time point and fasting within each group, as well as test differences between groups at fasting and each postprandial time point. **Results:** Postprandial active GLP-1 (area under the curve (AUC), incremental AUC (iAUC)) and total GLP-1 (iAUC) were significantly lower in obese compared to normal weight adolescents, suggesting a blunted GLP-1 response to fat ingestion. However, when dividing the obese cohort by the presence of insulin resistance, postprandial active GLP-1 (AUC, iAUC, 2hour) and total GLP-1 (iAUC) were significantly lower only in obese adolescents with insulin resistance compared to normal weight. Furthermore, only obese adolescents with insulin resistance lacked a significant postprandial rise in active and total GLP-1. When dividing the obese cohort by the presence of postprandial dyslipidemia, postprandial active GLP-1 (AUC, 6 hour) was significantly lower only in obese subjects with postprandial dyslipidemia compared to normal weight adolescents. Postprandial GLP-2 tended to be higher in obese subjects, although this difference was not significant, even when obese adolescents with insulin resistant and postprandial dyslipidemia were each examined separately. However, a significant postprandial rise in GLP-2 was absent from obese subjects with insulin resistance and postprandial dyslipidemia, suggesting a blunted postprandial GLP-2 response in obese subjects with these metabolic conditions. **Conclusion:** The postprandial GLP-1 and GLP-2 response to a high-fat drink appears to be blunted in obese adolescents, but only in the presence of insulin resistance or postprandial dyslipidemia. However, it remains unknown if a blunted postprandial gut peptide response is a cause or consequence of the progression of these metabolic conditions in an obese state.

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Extreme eruptive Xanthomas Associated with Severe Hypertriglyceridemia, Diabetes Mellitus and Hypothyroidism: a Case Report

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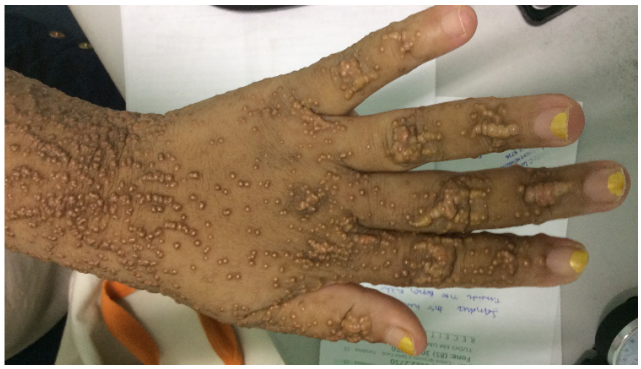
Case Report: A 37-year-old woman, with type 2 diabetes mellitus, presented with a pruritic skin rash of 3-months' duration. She had a history of total thyroidectomy for papillary thyroid cancer. Physical examination: multiple red-to-yellow papules, disseminated on both upper and lower extremities. A yellow deposit on the conjunctiva, and a yellowish discoloration of the distal ungeal portions were observed. Fasting blood sample: total cholesterol level of 1,252 mg/dL, a high density lipoprotein cholesterol of 34 mg/dL, a triglyceride level of 8,229 mg/dL, a glucose of 371 mg/dL (20.5 mmol/L), a glycated hemoglobin (HbA1c) of 11%, and a TSH of 41 mUI/mL. Punch biopsy of the skin lesions: lipid deposits and confirmed the diagnosis of Eruptive Xanthoma. Started on a 1,200-kcal diet, a full insulin regimen and fenofibrate 160 mg. The metformin dose of 2 g per day was maintained and the levothyroxin dose was uptitrated to 175 mcg. Six moths later, the diabetes and hypothyroidism were better controlled (HbA1c: 7,3% and TSH: 2,85 mUI/mL). The triglyceride levels dropped to 137 mg/dL, and the skin lesions were resolving.



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Polymorphic frequency of *APOE* gene from a clinical laboratory database

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Background: The apolipoprotein E gene (*APOE*) has an important role in lipoprotein metabolism. The study of the polymorphism variability allows in the determination of predisposition of some diseases such as Alzheimer's and cardiovascular diseases. Due to its clinical importance, the allelic distribution of this gene has been studied in different populations and ethnical groups in order to establish the diseases´ genetic profile. The objective of this study is to perform a yearlong (2017) data analysis to determine the allelic and genotypical frequencies of the *APOE* gene in samples tested in a Brazilian Clinical Laboratory. **Methods:** The data found were provided by ShiftLis software, used by our laboratory, and we conducted a descriptive analysis. The studied samples were processed by two distinctive methodologies, both using the same primers set described in the literature. The first of the two methodologies was a manual DNA extraction that utilizes Chelex®-100 Resin (BioRad), and processed in StepOne™ Real-time PCR System (Applied Biosystems) using Maxima SYBR Green/ROX qPCR Master Mix (Applied Biosystems). After the inicial technical validation there was a change in the methodology and we started using an automated DNA extraction MagNA Pure 96 System (Roche Life Science) and processed in Roche LightCycler®480 II (Roche Life Science) with LightCycler® 480 SYBR Green (Roche Life Science). **Results:** A total of 350 clinical samples were obtained in a period of one year, those being: 255 (72,9%) females, ages 6-86 (43.41±14.68) and 95 (27,1%) males, ages 8-83 (44.20±15.51). The analyzed samples came from nine different Brazilian cities: Manaus (56,5%), Brasilia (28%), Salvador (12%), Sao Jose dos Campos (1.4%), Palmas (0.9%), Campo Grande, Valparaiso, Ribeirao Preto and Campinas (0.3%). The highest allelic frequency found was E3 (80.3%), followed by E4 (15.1%) and finally E2 (4.6%). The largest genotypical group found was E3/E3 (64,9%), E3/E4 (24,0%), E2/E3 (6.9%), E4/E4 (2.3%), E2/E4 (1.7%) and the smallest one was E2/E2 (0.3%). The allelic frequencies obtained in this study were similar to the data of other Brazilian regions previously studied E3 (71.78 - 80.49%), E4 (13.41 - 22.88%) and E2 (2.73 - 6.63%) and from a recent worldwide study E3 (48 - 91%), E4 (6 - 40%) and E2 (<3 - >9%). **Conclusion:** This was the first study to analyze the polymorphic frequency of the *APOE* gene in a clinical laboratory, without the restriction of ethnical groups, ages, gender or previous known conditions, and also the first one to amass the results of six new Brazilian cities that haven´t been studied prior to this occasion, thus contributing to further genetic population studies.

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Comparison of calculated and directly measured low-density lipoprotein cholesterol at different triglyceride levels

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Background: Low-density lipoprotein cholesterol (LDL-C) is one of the major risk factors for coronary heart disease (CHD). LDL-C concentration is used in the National Cholesterol Education Program (NCEP) guideline for CHD risk categorization and therapeutic target. Therefore, accurate measurement of LDL-C is important for CHD patient management. Currently, calculation of LDL-C by the Friedewald equation is the most widely used laboratory method to determine LDL-C concentration. However, requirement of fasting and failure to accurately quantify LDL-C

when triglyceride (TG) greater than 400mg/dL limit its utilization. The aim of this study is to compare the serum LDL-C concentration measured by homogeneous enzymatic method ($LDL\text{-}C_D$) and LDL-C concentration calculated by Friedewald equation ($LDL\text{-}C_F$) and the novel equation based on the 180-cell table developed by the Johns Hopkins group ($LDL\text{-}C_{180}$). **Methods:** The lipid profile data were randomly retrieved from 257 patient serum samples. The determination of LDL-C concentrations by enzymatic method was performed on Beckman AU5800 analyzer. Lipid profiles (total cholesterol, HDL-C, and TG) measured by AU5800 were used to calculate LDL-C by the Friedewald equation (total cholesterol-HDL-TG/5) and the novel equation (total cholesterol -HDL-TG/adjustable factor based on the 180-cell table). Method comparison was done by EP Evaluator Software. **Results:** Of the 257 samples, 197 (76.7%) have TG level less than 200mg/dL, 34 (13.2%) have TG level between 200 and 400 mg/dL, and 25 (9.7%) have TG level greater than 400mg/dL. The overall comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_F$ showed the slope was 1.108 with intercept of -14.30, correlation coefficient of 0.8648, and the mean bias of -3.26. The overall comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_{180}$ showed the slope was 0.978 with intercept of 16.28, correlation coefficient of 0.9428 and the mean bias of 6.34. For the 197 samples with TG level less than 200 mg/dL, comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_F$ showed the slope was 0.943 with intercept of 6.59, correlation coefficient of 0.9353, and the mean bias of 0.69; comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_{180}$ showed the slope was 0.923 with intercept of 9.58, correlation coefficient of 0.9429, and the mean bias of 1.69. For the 34 samples with TG level between 200 and 400 mg/dL, comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_F$ showed the slope was 1.070 with intercept of -14.11, correlation coefficient of 0.8813, and the mean bias of -6.65; comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_{180}$ showed the slope was 0.936 with intercept of 12.55, correlation coefficient of 0.8827, and the mean bias of 5.82. For the 25 samples with TG level greater than 400 mg/dL, comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_F$ showed the slope was 1.523 with intercept of -78.89, correlation coefficient of 0.7913, and the mean bias of -29.74; comparison of $LDL\text{-}C_D$ with $LDL\text{-}C_{180}$ showed the slope was 1.022 with intercept of 14.16, correlation coefficient of 0.8851, and the mean bias of 16.24. **Conclusions:** These data demonstrate that LDL-C concentration measured by enzymatic method has the best correlation and least bias with both calculated LDL-C when TG level is less than 200mg/dL. When TG level is greater than 400 mg/dL, $LDL\text{-}C_D$ has a better correlation with $LDL\text{-}C_{180}$ than with $LDL\text{-}C_F$.

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Dyslipidemia and associated cardiovascular risk factors among young university students of Nepal

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Background: Cardiovascular diseases (CVDs) are one of the main causes of morbidity and mortality worldwide, atherosclerosis being the principal underlying cause of CVDs. Dyslipidemia is the most important risk factor for atherosclerosis. Although CVDs are not observed in childhood, cardiac risk factors such as dyslipidemia are present in children and they remain silent until adulthood. Cardiovascular risk factors are on the rise in the Nepalese population and recent observation has shown a significant elevation of risk factors in the youth population. The purpose of this study was to assess dyslipidemia and associated cardiovascular risk factors among young university students of Nepal **Methods:** A cross sectional study was carried out in Institute of Medicine (IOM), Tribhuwan University Teaching Hospital. The study was conducted from 20th February 2017 to 20th July 2017.The study population encompassed 280 undergraduate students aged 17-24 (156 males and 124 females) during the time frame. Recruitment was done randomly and eligible participants were selected if they were healthy, physically active and taking no medications known to influence lipid metabolism. An interview-based questionnaire was designed and information about age, sex, smoking and alcohol consumption was collected. Body mass index and waist-to-hip ratio of all participants were calculated. Venous blood samples were obtained from the antecubital vein in suitable vacutainers after 12 hours overnight fasting. Triglycerides (TG), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL) were analyzed enzymatically and the low-density lipoprotein cholesterol (LDL) was calculated using Friedwald's formula. All the estimations were done using the autoanalyser (BT 1500) **Results:** Overall, dyslipidemia was seen as Hypercholesterolemia in 31 (11.1%), elevated low-density lipoprotein in 34 (12.1%), low high-density lipoprotein in 95 (33.9%) and hypertriglyceridemia in 39 (13.9%). Risk factors found to be significantly associated with Hypercholesterolemia were a history of smoking ($p< 0.01$, OR: 3.6) and binge drinking ($p < 0.001$, OR: 8). Sex ($p < 0.05$, OR: 0.41), binge drinking ($p <0.001$, OR: 5.1) and smoking ($p<0.001$, OR: 11.3) were found to be significantly associated with elevated LDL. Sex ($p <0.05$, OR: 0.45), Binge drinking ($p<0.05$, OR: 1.67) and smoking ($p<0.05$, OR: 3.5) were significantly associated with hypertriglyceridemia. There was no statistically significant association between risk factors and the low HDL. Multivariate Logistic regression analyses showed that current smoking and binge drinking were significant predictors of Hypercholesterolemia and elevated LDL. In females, only the serum LDL level was significantly correlated with BMI however, serum TC and LDL levels both significantly correlated with WHR. Meanwhile, in the case of males, all TC, TG, LDL, HDL levels significantly correlated with BMI and WHR. **Conclusion:** The prevalence of dyslipidemia was high in young Nepalese university students (17-24 years). Early detection of dyslipidemia and long-term prevention of cardiovascular disease by controlling the risk factors should begin in youth. Increasing student awareness of importance of controlling alcohol consumption, quitting smoking and increasing physical activity is of paramount importance to reduce dyslipidemia prevalence and prevent cardiovascular disease.

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Determination of serum triglyceride by Isotope Dilution Liquid Chromatography Tandem Mass Spectrometry

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Background: There are some reference methods to determine serum total glycerol and triglyceride all by isotope dilution gas chromatography mass spectrometry which are time consuming and complicated. A need exists for a simple reference method that can be easily adopted to verify the accuracy of serum triglyceride measurements, especially with different measurement principles. Just as serum triglyceride concentrations are generally determined from total glycerol with or without a subtraction of free glycerol. So candidate reference methods involving isotope dilution liquid chromatography tandem mass spectrometry (ID-LC/MS/MS) for total glycerol and free glycerol were established. The triglyceride concentration was the difference between total glycerol and free glycerol. **Methods:** An isotopically labeled internal standard, [$^{13}C_3$]-glycerol, was added to serum, protein precipitation, and derivatization by benzoyl chloride to prepare samples for LC/MS/MS analysis using electrospray for ionization (ESI). For total glycerol, hydrolysis was conducted after adding the internal standard. For separation, a Nova-Pak C18 column was used with a mobile phase consisting of 10 mmol/L ammonium formate in water-acetonitrile (20:80 by volume) for positive ions. The quantitative ion transitions of $[M+NH4]^+$ at m/z 422.2→283.2 and m/z 425.2→286.2 were monitored for glycerol and [$^{13}C_3$]-glycerol, respectively. The qualitative ion transitions were at m/z 422.2→105.1 and m/z 425.2→108.1, respectively. The method was calibrated with linear regression using five-point calibration curves. **Results:** The correlation coefficients between the peak area ratios and glycerol concentrations were 0.9999 and higher. The within-run coefficients of variation (*CV*) for serum total glycerol analysis averaged 0.52% (ranged 0.3%~1.02%) and the total *CV* 0.73% (0.49%~1.27%). Results on certified reference materials (SRM 909b Level I and Level II, SRM 1951b Level I and Level II, SRM 909c, GBW 09146 and GBW 09147) showed an averaged bias of 0.32% (0%~0.97%). **Conclusion: I**sotope dilution LC/MS/MS method for serum triglyceride has been developed. This method was been used in international laboratory comparison including RELA (IFCC) and Cholesterol Reference Method Laboratory Network(CRMLN,US. CDC). Results showed that this ID-LC/MS/MS method was well-characterized for serum glycerides with a theoretically sound approach, demonstrated good accuracy and precision, and low susceptibility to interferences qualifies as a candidate reference method. Use of this reference method as an accuracy base may reduce the apparent biases in routine methods along with the high interlaboratory imprecision.

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Developing a modified low-density lipoprotein (M-LDL-C) Friedewald's equation as a substitute for direct LDL-c measure in the Ghanaian Setting

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Background: An elevated concentration of low density lipoprotein cholesterol (LDL-c) is a well-known atherogenic risk factor with a high predictive value for coronary heart disease. Though a number of homogenous assay are available for estimation of LDL-c, use of calculated LDL-c by Friedewald's formula (FF) is common in Ghanaian laboratories for logistic reasons. Several novel formulae (Martin's, Vojovic's and Anandaraja's formula) have been reported to outperform the Friedewald formula. This study therefore validated existing formulae and derived a more accurate formula to determine LDL-c in a Ghanaian population.

Methods: We estimated 1518 lipid profiles from the outpatient department of the Cardiothoracic Centre of the Korle-bu Teaching Hospital. We evaluated three formulae (Friedewald, Anandaraji, and Martin's) and compared these to direct measurement of LDL-c across triglyceride (TG) high density lipoprotein (HDL-c) and total cholesterol (TC) ranges using a reagent kit obtained from Human Diagnostic Worldwide, Germany and URIT 8210 automatic chemistry analyzer. Using values of lipoproteins from the initial measurements in our population, a new modified Friedewald's LDL-c (M-LDL-C) equation was derived by replacing the term 2.2 with 4.0. Receiver operator characteristic (ROC) and linear regression were performed. Data was analysed using STATA version 12.0 **Results:** The mean LDL-c concentration measured by enzyme-based direct homogenous assay (D-LDL-c) and that calculated by Friedewald's formula (F-LDL-c), Martin's formula (N-LDL-c), Anandaraja's formula (A-LDL-c) and modified Friedewald's LDL-c (M-LDL-C) formula were 2.47±0.71 mmol/L, 2.76±1.05mmol/L, 2.74±1.04 mmol/L, 2.99±1.02 mmol/L and 2.97±1.08 mmol/L respectively. D-LDL-c levels were significantly lower compared to F-LDL-c, N-LDL-c, A-LDL-c and M-LDL-C ($p < 0.001$) using the Student paired t-test. The F-LDL-c equation showed a significantly strong positive correlation with A-LDL-c ($r=0.898$, $p<0.0001$), N-LDL-c ($r=0.991$, $p<0.0001$) and M-LDL-c ($r=0.989$, $p<0.0001$), but a weak positive correlation with LDL-c ($r=0.481$, $p<0.0001$). Analysis on ROC curve showed a better diagnostic accuracy for M-LDL-c (AUC=0.81) and N-LDL-C (AUC=0.81) followed by F-LDL-c (AUC=0.80) and A-LDL-c (AUC=0.77) based on a D-LDL-c cut-off >2.5mmol/L. Bland-Altman graphs showed a definite agreement between mean and differences of the calculation formulae and D-LDL-C with 95% of values lying with in±0.50 SD limits. **Conclusion:** The modified LDL-c (M-LDL-c) equation could serve as a better substitute for both D-LDL-c and F-LDL-c equation in the Ghanaian settings.

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Performance Evaluation of the Atellica CH Apolipoprotein A-1 and Apolipoprotein B Assays

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Background: The purpose of the investigation was to evaluate the analytical performance of the Atellica® CH **Apolipoprotein A-1 (APO A1)** and **Apolipoprotein B (APO B)** Assays on the Atellica CH Analyzer. Measurements of APO A1 and APO B are used in assessing arteriosclerosis development and the severity of coronary artery stenosis. APO A1 and APO B use a PEG-enhanced immunoturbidimetric methodology. APO A1 or APO B in the sample forms an insoluble complex with a specific antiserum, which is measured turbidimetrically. **Method:** Performance testing included precision and accuracy. Assay precision was evaluated using the Clinical and Laboratory Standards Institute (CLSI) guideline EP05-A3. Each sample was assayed in duplicate twice a day for 20 days. Method comparison studies were conducted according to CLSI EP09-A3, with patient sample results compared to results from the ADVIA® 1800 Clinical Chemistry System. **Results:** For APO A1, within-lab precision ranged from 1.7-2.4% CV in serum/plasma samples. For APO B, within-lab precision ranged from 2.8-6.1% CV in serum/plasma samples. The APO A1 serum method-comparison study yielded a regression equation of $y = 1.05x - 1$ mg/dL with $r = 0.989$, versus the ADVIA 1800 APO A1 Assay. The APO B serum method comparison study yielded a regression equation of $y = 0.99x - 4$ mg/dL with $r = 0.999$, versus the ADVIA 1800 APO B Assay. **Conclusions:** The Atellica CH APO A1 and APO B Assays tested on the Atellica CH Analyzer demonstrated acceptable precision. Method comparison results showed acceptable agreement with an on-market comparative analyzer.

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LDL-cholesterol determination by measured LDL-cholesterol versus Martin equation for triglycerides levels above 400 mg/dL

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Background: The determination of the low-density lipoprotein cholesterol (LDL-C) is classically obtained by Friedewald equation. This equation assumes a fixed factor of 5 for the ratio of triglycerides to very low-density lipoprotein cholesterol. The Friedewald equation should not be used when plasma triglycerides concentration exceeds 400 mg/dL. A novel method for estimating LDL-C reported by Martin et al has advocated use of a newly derived equation to estimate LDL-C that is intended to correct for this limitation in the Friedewald calculation and improve LDL-C estimation even when triglycerides values are >400 mg/dL. The novel LDL-C is calculated using an adjustable factor determined on the basis of an individual patient's triglycerides and non-HDL-Cholesterol (non-HDL-C). The aim of this study was to compare the LDL-C results using the Martin equation versus directly measured LDL-C for triglycerides values above 400 mg/dL. **Methods:** The results of 319 directly measured LDL-C previously released in a laboratory routine were recalculated using the equation of Martin et al. The total cholesterol, HDL-C, LDL-C and triglycerides were carried out on the Roche 8000 analyzer (Roche Diagnostics GmbH, Germany) using reagents from Roche. The mean triglycerides value was 570 ± 211 mg/dL ranging from 401 to 1888 mg/dL. **Results:** The values of measured LDL-cholesterol and obtained by calculation were respectively: Mean ± SD: 132 ± 49 mg/dL and 128 ± 44 mg/dL; Median: 129 mg/dL and 126 mg/dL; First Quartile (box-plot): 99 mg/dL and 100 mg/dL; Third Quartile (box-plot): 163 mg/dL and 155 mg/dL; The linear regression equation considering measured versus calculated LDL-C was: $y = 0.851x + 15.889$ ($R^2 = 0.8906$). **Conclusion:** The calculation of LDL-C using the Martin equation showed a good correlation with directly measured LDL-C results, a fact that encouraged us to implement in the routine standard lipid profile of our Service.

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Performance characteristics of a novel direct assay for small, dense LDL cholesterol, and results from a reference range study

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Background: Small, dense LDL (sd LDL) is a highly atherogenic lipoprotein, and many clinical studies have strengthened the relationship between sd LDL-C level and CHD. We have developed a simple and fully-automated homogeneous method for quantification of sd LDL-C (s LDL-EX"SEIKEN"). A series of basic performance studies as well as reference range study were performed using the s LDL-EX "SEIKEN" on the Roche Diagnostics Hitachi 917 analyzer. This kit received 510(k) clearance in August, 2017 **Method:** Precision, linearity, interference, limit of quantitation (LOQ) and correlation with ultracentrifugation method (UC) were performed according to CLSI approved guideline EP05-A2, EP06-A, EP07-A2, EP17-A2 and EP09-A3. Matrix comparison was carried out using 47 paired serum and plasma samples. Reference range study was conducted in accordance with EP28-A3c. Subjects were recruited from two geographical regions in the US. Based on Adult Treatment Panel III (ATPIII) guideline, subjects were partitioned by age and gender. **Result:** Within-laboratory %CV was 1.3 to 4.1%. Linear assay range was confirmed up to 100 mg/dL. Established LOQ was 1.14 mg/dL, which is below the lower measurement limit (4 mg/dL). No interference of hemoglobin, bilirubin, chyle, statins, fibrates was found against sd LDL-C values. The results were in good correlation between this kit and the recognized UC reference method (slope: 1.028 [95%CI 0.932 - 1.127], intercept: -1.38 [95%CI -5.73 - 1.80]). Age differences associated with the sd LDL-C level were significant in both genders ($p = 0.0030$ in males and $p < 0.0001$ in females). No significant difference was observed in the sd LDL-C level between males and females ($p = 0.7564$). According to the CLSI guideline, the normal range was defined as the 2.5th percentile value to the 97.5th percentile value, younger group was 12.7 to 48.3 mg/dL and older group was 12.6 to 51.7 mg/dL. **Conclusion:** The sd LDL-C assay demonstrated good analytical performance. This FDA-approved assay is promising for application in routine clinical practice as an IVD product.

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Clinical significance of measurement by novel direct assay for small, dense LDL cholesterol for CHD risk assessment

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Background: Small, dense LDL (sd LDL) is an atherogenic lipoprotein, and many clinical studies have strengthened the relationship between sd LDL-cholesterol(C) level and incident CHD. We have developed a simple and technically conventional method for quantification of sd LDL-C (s LDL-EX"SEIKEN"). The clinical cutoff value was established by Multi-Ethnic Study of Atherosclerosis (MESA) and was validated using the cohort of Atherosclerosis Risk In Community (ARIC) study. This kit received 510(k) clearance in August, 2017. **Method:** The Adult Treatment Panel III has generally selected the 75th percentile value for LDL-C as being associated with high risk of CHD. Based on this principle, the 75th percentile value was selected as a clinical cutoff in normolipidemic and dislipidemic subjects who showed no signs of CHD or diabetes mellitus at baseline (n =

3,938) in MESA. The established cutoff value was validated using individuals who participated in ARIC study Visit 4 (1996-1998). The study population was recruited from four U.S. communities, and the subjects who did not have CHD at the baseline (n=10,290) were followed for a maximum of 16 years' period. Proportional hazards regression analyses were used to investigate the association of incident CHD, defined as hospitalized myocardial infarction, fatal CHD, or cardiac procedure. Hazard ratios (HRs) of incident CHD were adjusted for Model 1: age, sex and race; and Model 2: Model 1 variables + ever smoker, BMI, hypertension, HDL-C, triglycerides (log-transformed), lipid-lowering medications, diabetes and hs-CRP (log-transformed). **Result:** In MESA, the 75th percentile value of sd LDL-C was analyzed as 48.4 mg/dL in the subjects with no CHD or diabetes mellitus, and this was rounded to 50.0 mg/dL as a clinical cutoff. Absolute risk and Cox proportional hazards regression analyses were used to investigate the association of incident CHD. Subjects were dichotomized by their baseline levels of sd LDL-C, and analyses were conducted using the group with sd LDL-C <50.0 mg/dL as reference. In Model 1, individuals in the higher sd LDL-C group showed approximately a 1.6-fold higher risk for incident CHD compared to the reference group (HR 1.55; 95% CI 1.39-1.73). In Model 2, risk for incident CHD was somewhat attenuated, but remained significant (HR 1.26; 95% CI 1.10-1.43). **Conclusion:** The cutoff value of sd LDL-C established as 50 mg/dL was validated for its clinical use in predicting the risk of CHD. This FDA-approved assay is promising for application in routine clinical practice as an IVD product.

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Lipoprotein Particle Stability in Serum for Nuclear Magnetic Resonance Analysis

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Background: Lipoprotein particles consist of cholesterol and other lipids characterized according to their composition, density, size and biological function. It is also known that independent of cholesterol content, lipoprotein particles, including their subclasses, can vary with respect to density, size and lipid content. Associations between the blood concentrations of these particles and increased coronary heart disease (CHD) risk, insulin resistance, diabetes mellitus and metabolic syndrome are well established. Consequently, lipoprotein-lipid profiling may better identify individuals with an increased risk of CHD. Methods for analyzing lipoprotein particles include gel electrophoresis, density gradient ultracentrifugation, ion mobility analysis, and nuclear magnetic resonance (NMR) spectroscopy. The stability of lipoproteins in biological samples is of importance in clinical settings. The purpose of this study was to assess the stability of lipoprotein particles in serum for analysis by NMR. **Methods:** Deidentified residual serum specimens sent to ARUP Laboratories for routine testing were used. Lipoprotein particle concentrations and sizes were measured using the AXINON® lipoFIT® test system incorporating the Bruker Ascend™ 600 Avance III HD NMR platform (numares AG, Regensburg, Germany) according to the AXINON test kit protocol. The University of Utah's Institutional Review Board approved this study. The lipoproteins evaluated were high-density lipoprotein particle number (HDL-p), large high-density lipoprotein particle number (LHDL-p), low-density lipoprotein particle number (LDL-p), small low-density lipoprotein particle number (SLDL-p), large very-low-density lipoprotein particle number (LVLDL-p), high-density lipoprotein particle size (HDL-s), low-density lipoprotein particle size (LDL-s) and very-low-density lipoprotein size (VLDL-s). Conditions included room temperature, refrigerated, frozen (-20 °C) and freeze/thaw cycles. **Results:** Summarized in the table below. **Conclusions:** Lipoprotein particles HDL-p, LHDL-p, LDL-p, SLDL-p, LVLDL-p, HDL-s, LDL-s and VLDL-s demonstrate reasonable stability in serum at room and refrigerated temperatures for analysis by NMR. However, untimely degradation is possible for LHDL-p, SLDL-p and especially, LVLDL-p for specimens stored frozen at -20 °C.

Lipoprotein Particle Stability

	Room Temperature	Refrigerated	Frozen (-20 °C)	Freeze/Thaw
HDL-p	min 48 hours	min 30 days	min 3 months	min 3 cycles
LHDL-p	min 48 hours	min 30 days	1 month	min 3 cycles
LDL-p	min 48 hours	min 30 days	min 3 months	min 3 cycles
SLDL-p	min 48 hours	min 30 days	2 months	min 3 cycles
LVLDL-p	min 48 hours	min 30 days	1 week	2 cycles
HDL-s	min 48 hours	min 30 days	min 3 months	min 3 cycles
LDL-s	min 48 hours	min 30 days	min 3 months	min 3 cycles
VLDL-s	min 48 hours	min 30 days	min 3 months	min 3 cycles

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Possible Concern of Erythrocytes with Reverse Cholesterol Transport

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Background: High-density lipoprotein (HDL) plays a main role in the reverse cholesterol transport (RCT) by taking up cholesterol (cholesterol efflux) from foam cells and carrying it to the liver. Recent studies have pointed out that cholesterol accumulation within the intima and expansion of the necrotic core are induced also by intraplaque hemorrhage and lysis of erythrocytes at the lesion site. One report showed blood cholesterol in mouse was possibly transported by erythrocytes at higher level when apolipoprotein A-I (apoA-I), the main apolipoprotein in HDL, was knocked down. Therefore, we investigated the participation of erythrocytes in RCT according to interacting with apoA-I and HDL. **Methods:** Cholesterol efflux capacity (CEC) was measured as we have previously described. THP-1 cells (human acute monocytic leukemia cell line) were stimulated by phorbol myristate acetate and differentiated into macrophages. Cells were then incubated with acetylated low-density lipoprotein for foam cell formation, ^{3}H-cholesterol as a tracer and LXR activator T0901317 for enhancing the expression of cholesterol transporters. To evaluate CEC, apoA-I or HDL as a conventional cholesterol accepter was incubated with foam cells for 4 hours in the presence or absence of various amounts of erythrocytes and the radioactivity in the medium, erythrocyte and cell lysate were measured. CEC was defined as the percentage of radioactivity distributed to the medium (apoA-I or HDL) and erythrocytes. Cholesterol transferred between apoA-I (or HDL) and erythrocytes was also determined by the incubation of ^{3}H-cholesterol acquired apoA-I (or HDL) and erythrocytes, obtained by CEC assays, with fresh erythrocytes and apoA-I (or HDL), respectively. **Results:** In the CEC assay including both apoA-I (or HDL) and various amounts of erythrocytes, the percentages of radioactivity in erythrocytes increased in a dose dependent manner, while those in medium including apoA-I or HDL decreased contrastively. Next, cholesterol transferred between apoA-I (or HDL) and erythrocytes was investigated using ^{3}H-cholesterol acquired apoA-I (or HDL) and erythrocytes obtained by CEC assay. The radioactivity of apoA-I and HDL in the medium decreased by approx. 81% and 51% after incubating with fresh erythrocytes, respectively. On the opposite direction, some ^{3}H-cholesterol acquired by erythrocyte transferred to fresh HDL but almost not to fresh apoA-I. Further, human serum albumin (HAS) tends to facilitate cholesterol efflux of erythrocytes without regard to existence of apoA-I or HDL; however, HAS did not affect cholesterol exchange between erythrocytes and apoA-I (or HDL). **Conclusions:** Erythrocytes might facilitate cholesterol efflux and increase CEC by receiving cholesterol which apoA-I and HDL take up from foam cells. Actually, cholesterol transport from ^{3}H-cholesterol acquired apoA-I and HDL to erythrocytes was observed in the condition without foam cells; however, its efficiency in apoA-I was higher than that in HDL. Cholesterol transport from ^{3}H-cholesterol acquired erythrocytes to HDL was also observed, but not to apoA-I. These could indicate that free apoA-I, a minor part of total apoA-I, plays as a predominant cholesterol accepter in cholesterol efflux according to a collaboration with erythrocytes. Consequently, erythrocyte may play an important role in RCT as a temporary cholesterol storeroom.

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Impact of fasting time on abnormal flagging rates when clinical decision limits are applied to total cholesterol, HDL-C, LDL-C (calculated), non-HDL-C, and triglycerides

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Background: Use of non-fasting lipids is gaining widespread acceptance for cardiovascular risk-stratification. However, many dyslipidemia guidelines have yet to provide recommendations for clinical cutoffs for non-fasting total cholesterol, HDL-C, LDL-C, non-HDL-C, and triglycerides and few studies have examined the impact of fasting time on the kurtosis and skewness of the distribution of lipid results and abnormal flagging rates. We hypothesized that variations in meal composition may broaden the distribution of lipid results and impact flagging rates in non-fasting specimens. Our objectives were to identify the proportion of patients presenting for lipid testing in a non-fasting state and to assess the impact of fasting time on: 1) the median concentration, kurtosis and skewness of results; 2) the abnormal flagging rate when clinical cutoffs are applied. **Methods:** A retrospective cross-sectional review of adult (≥18 years of age) lipid results (N=261,645 males and 280,651 females) obtained over a four-month period was performed. Abnormal flagging rates were based on the following desirable clinical cutoffs: total cholesterol<200 mg/dL; male HDL-C<40 mg/dL, female HDL-C<50 mg/dL; LDL-C<135 mg/dL; non-HDL-C<166 mg/dL; and triglycerides<150 mg/dL. Total cholesterol, HDL-C, and triglycerides were measured while LDL-C and non-HDL-C were calculated. Fasting duration was obtained by patient-report and results were partitioned by sex without age distinction. The flagging rate, calculated as the number of results exceeding the clinical cutoff/total number of results for that fasting time, was calculated for each hour of fasting. **Results:** Although mandatory fasting requirements for lipid measurements had been removed over two years prior to the study, only 35% of males and 37% of females presented for phlebotomy in a non-fasting state (0-11 hours for cholesterols, 0-7 hours for triglycerides). Consistent with previous studies, the median concentrations of total cholesterol, HDL-C, and LDL-C differed statistically but not clinically across fasting times (0-16 hours). No significant change was seen in non-HDL-C. Additionally, regardless of fasting status, the kurtosis and skewness of the distributions for these analytes remained consistent. However, the median concentration of triglycerides increased by ~20% and the distribution curve broadened between 0-7 hours as compared to 8-16 hours fasting. For all analytes, these changes were mirrored in their flagging rates. For males, between fasting and non-fasting, the average flagging rate changed from 27.8% to 28.1% for cholesterol; 21.4% to 24.1% for HDL-C; 18.1% to 15.9% for LDL-C; 18.3% to 19.3% for non-HDL-C; and 33.0% to 46.9% for triglycerides. Similarly, for females, between fasting and non-fasting, the average flagging rate changed from 38.1% to 37.9% for cholesterol; 26.9% to 28.2% for HDL-C, 20.2% to 17.9% for LDL-C, 17.8% to 17.9% for non-HDL-C, and 24.2% to 36.1% for triglycerides. In total, for our patient cohort, ~286,000 lipid test results, or ~13%, would be differentially flagged if all patients presented as non-fasting as compared to fasting. **Conclusion:** Dyslipidemia guidelines should consider providing adjusted clinical decision limits for non-fasting lipids, in particular LDL-C (if calculated) in order to improve clinical sensitivity. Distinct limits should be provided for fasting and non-fasting triglycerides to avoid substantial increases in abnormal flags in non-fasting specimens.

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High prevalence of diabetes mellitus type 2 among participants of Hipercol Ceará Program

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Background: The Familial Hypercholesterolemia (FH) is a genetic disease. It causes high LDL-cholesterol levels and high cardiovascular disease (CVD) risk. The Type 2 Diabetes Mellitus (T2DM) is an additional cardiovascular risk which can also occur in FH patients. Few studies on the prevalence of T2DM among FH patients have been reported. Data from international studies show a prevalence of T2DM among FH patients of less than 10%. In Brazil, the prevalence of T2DM is about 12.1% (SBD, 2016) and among FH cases, 14.7% (Santos et al., 2014). However, data about T2DM prevalence among FH patients is unknown. **Methods:** A cross-scetional study of patients evaluated in "Programa Genético de Rastreamento Ativo de Hipercolesterolemia Familiar do Ceará - HIPERCOL CEARÁ" at reference service from 2013 to 2017. The patients evaluated had a level of LDL- cholesterol level above 210 mg/dL, besides that clinical characteristics, comorbidities, lifestyle and family history to early CVD. A molecular study was performed for gene mutation analysis (LDL receptor, ApoB protein and inhibitor protein PSK9). The first degree relatives (FDR) of genetic confirmed cases were also evaluated. **Results:** A total of 122 participants were evaluated; 34 had LDL receptor gene mutation and 24 was previously diagnosed for T2DM (mean age=57.8 (±12.4) years; female gender= 83.3%). About origin, 18 (75%) lived in the capital, 4 were from interior state and 2 (8.3%) lived in Fortaleza Metropolitan Region. The T2DM prevalence among the index cases was 87.5% and 12.5% in their relatives. About the suspect cases, the T2DM prevalence was 19.7%. When considering only the confirmed cases, the T2DM prevalence was 20.8%. **Conclusion:** It is observed a high prevalence of T2DM in the evaluated population being superior to that described in another Brazilian population. A larger study is needed, but these data may suggest the need for early detection of T2DM among FH patients.

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Lipid profile, Oxidative Stress & Anti-Oxidative Status of Paraoxonase Enzyme: Comparative Approach in Nepalese Obese & Non-obese People

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Background
Obesity is a condition of excessive body fat accumulation adversely affecting health. It is associated with lipoprotein peroxidation leading to atherogenesis. Paraoxonase enzyme associated with high–density lipoprotein (HDL-PON) exerts a protective effect against oxidative damage of circulating cells and lipoproteins.
Objective
We investigated the relationship between lipid profile, total peroxide (measure of oxidative stress) & serum paraoxonase (HDL associated) aryl esterase activity (measure of antioxidative status) in non–obese and obese Nepalese people.
Methods
Anthropometric variables including BMI, serum lipids, total peroxide and PON ARA were measured in, and compared between, consenting age-matched (33.75±0.82 yrs.) non-obese (n=105) and obese (n=105) subjects. The data are as mean±SE.
Results
The concentrations of total cholesterol (obese 188.63±6.71 Vs non-obese 147.56±3.24, mg/dl, p<0.001), triglycerides (obese 189.12±9.96 Vs non-obese 134.53±6.69, mg/dl, p<0.001) and LDL cholesterol (obese 109.97±6.06 Vs non-obese 75.75±2.90, mg/dl, p<0.001) were significantly high in obese subjects. Serum PON ARA (obese 124.23±9 Vs non-obese 184.10±13.80, µmol/min/ml, p<0.01) and HDL (obese 40.82±0.66 Vs non-obese 44.88±1.19, mg/dl, p<0.01) were significantly lower and total peroxide level (obese 19.29±0.54 Vs non-obese 12.77±0.25, µmol H_2O_2/liter, p<0.001) was higher in obese than in non-obese. Negative correlation found between PON ARA and total peroxide level confirms the relation between paraoxonase activity and lipoprotein lipid-peroxidation.
Conclusion
The findings suggest an increase in the level of bad cholesterol along with reduced level of good cholesterol, increase level of oxidative stress in obesity, which is associated with a decrease in HDL-PON activity and increased risk of cardiovascular diseases among people with obesity in least developed country, Nepal. This study would suggest guiding the development of policies to reduce the burgeoning issue of obesity & thus the burden of metabolic syndrome & non-communicable diseases in least developed country in Asia, like Nepal.

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Performance Evaluation of an Automated Assay for the Measurement of LPL and HL Activity

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Background: Lipoprotein lipase (LPL) hydrolyzes triglycerides (TGs) into chylomicrons and VLDL particles during lipoprotein metabolism. Similarly, hepatic lipase (HL) is synthesized by hepatocytes and hydrolyzes TGs and phospholipids

in chylomicron remnants, intermediate density lipoproteins and HDLs. LPL deficiency leads to hypertriglyceridemia with accumulation of chylomicrons. HL deficiency leads to hypercholesterolemia, hypertriglyceridemia and accumulation of β-VLDLs, chylomicron remnants, IDLs, TG-rich LDLs and HDLs. The conventional method for measuring LPL and HL activity uses ^{3}H- or ^{14}C-labeled trioleoyl glycerol and is not suitable for routine clinical measurement. A novel assay has been developed which is applicable to automated clinical analyzers. Here, we evaluated the performance of the new LPL and HL activity assay method in human post-heparin EDTA plasma (PHP) using the cobas c501 autoanalyzer. **Methods:** LPL and HL activities were measured colorimetrically using two different channels on the cobas c501 autoanalyzer (Roche Diagnostics). The first channel contained apoCII, a cofactor required for LPL activity, and measured combined LPL and HL activities. The second channel lacked apoCII, measuring only HL activity. LPL activity was calculated from the difference between the two channels. The performance of the two channels as well as the calculated LPL activity were evaluated and validated using PHP through several experiments, including precision, linearity (2 samples), recovery (2 samples spiked to 3 different levels), sensitivity, reference interval (20 subjects) and stability (3 single donors). The within-run precision (WRP) and between-run precision (BRP) were evaluated using three in-house plasma controls with three different concentrations of LPL+HL and HL activities. **Results:** For precision of both activity channels, coefficients of variation (CV) for all controls ranged from 0.9 – 4.5% for WRP, and 2.8 – 7.4% for BRP. For linearity, acceptable results ranged from 82.9 – 119.5% of targets with up to 16-fold dilution for both activity channels. For recovery, post-heparin plasma spiked into pre-heparin plasma demonstrated acceptable recovery of LPL and HL activities ranging from 100.0 – 117.1% of targets. The sensitivities for LPL+HL, HL and LPL activities in PHP were identified at 5, 17 and 11 U/L, respectively, with precision of ≤ 20%CV. For the reference interval, LPL and HL activities were measured in 20 PHP samples from normal healthy volunteers. These results aligned with the range obtained by the manufacturer with values of 42 – 209 U/L and 198 – 859 U/L for LPL and HL activity, respectively. Accuracy was also verified using commercially available control samples and ranged from 90.0 – 108.6% of target. For short-term stability in post-heparin plasma, LPL+HL and HL activities were stable at 2 – 8 °C for up to 3 days, up to 1 day at room temperature, and stable for 3 additional freeze-thaw cycles. **Conclusion:** The utility of the LPL/HTGL Activity was demonstrated with sufficient analytical performance. Overall, this assay on an automated platform is ideal for measuring LPL and HL activities in clinical trials.

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Dapagliflozin decreases sd LDL-C and increases HDL2-C in patients with type 2 diabetes comparison with sitagliptin

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BACKGROUND: Several recent studies have reported that sodium-glucose co-transporter-2 (SGLT-2) inhibitors increase both low-density lipoprotein (LDL) and high-density lipoprotein (HDL)-cholesterol (C). In this study, we determined the effect of SGLT-2 inhibitors on LDL and HDL-C subspecies. **METHODS**: Single center, open-label, randomized, prospective design was employed. 80 patients with type 2 diabetes taking prescribed oral agents were allocated to receive SGLT-2 inhibitors, dapagliflozin (n=40) or dipeptidyl peptidase-4 inhibitor, sitagliptin (n=40) as add-on treatment. Fasting blood samples were collected before and 12 weeks after this intervention. Small, dense (sd) LDL-C, large buoyant (lb) LDL-C, HDL2-C, and HDL3-C were measured using our established homogeneous assays. **RESULTS**: Dapagliflozin and sitagliptin comparably decreased HbA1c (0.75 and 0.63%, respectively). Dapagliflozin significantly decreased body weight, systolic blood pressure, plasma triglycerides and liver transaminases, and increased adiponectin; sitagliptin did not affect these measurements. For the patients with dapagliflozin treatment, no significant change was observed in their LDL-C and apolipoprotein (apo) B levels, whilst their HDL-C and apo AI were increased. Interestingly, however, we found that sd LDL-C decreased by 20% and lb LDL-C increased by 18%. The level of lb LDL-C was remarkably elevated (53%) in individuals (n=20) with elevated LDL-C by dapagliflozin, whilst sd LDL-C remained suppressed (20%). Dapagliflozin increased HDL2-C by 18% without affecting HDL3-C. Sitagliptin did not alter plasma lipids or lipoprotein subspecies. **CONCLUSIONS**: Dapagliflozin suppressed potent atherogenic sd LDL-C and increased HDL2-C, a favorable cardiometabolic marker. Although LDL-C levels are elevated by treatment with dapagliflozin, this was due to increased concentrations of the less atherogenic lb LDL-C. However, these findings were not observed after treatment with sitagliptin.

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Direct Lipoprotein Measurements and Cardiovascular Disease Risk

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Objectives: Cardiovascular disease (CVD) is a major cause of death and disability in the United States. The standard American Heart Association (AHA) model for CVD risk assessment includes age, gender, systolic blood pressure, use of blood pressure medication, history of diabetes, history of current smoking, total cholesterol (TC), and high density lipoprotein cholesterol (HDL-C). Elevated serum levels of low density lipoprotein cholesterol (LDL-C), small dense LDL-C (sdLDL-C), remnant lipoprotein cholesterol (RLP-C), and lipoprotein (a) or Lp(a) and low HDL-C have all been associated with an increased risk of CVD, including coronary heart disease, stroke, peripheral vascular disease, coronary revascularization, and CVD mortality. Our objectives were to assess direct measurements of these lipoproteins as compared to standard risk factors in the prospective Framingham Offspring Study. **Methods:** Stored frozen plasma samples (-80 degrees C) obtained after an overnight fast from male and female participants free of all CVD at cycle 6 of the Framingham Offspring Study were used (n=3,147, mean age 58 years). A total of 677 subjects or 21.5% developed a CVD endpoint over a 16 year period of follow-up. TC, HDL-C, direct LDL-C, sdLDL-C, RLP-C, Lp(a), and high sensitivity C reactive protein (hsCRP) were measured by standardized automated analysis. All assays had within and between run coefficients of variation of < 5%. Estimated LDL-C was calculated as: total cholesterol - HDL-C - TG/5 provided subjects had fasting triglyceride values < 400 mg/dL. Statistical analysis included logistic regression, multivariate modeling, and net reclassification. **Results**: For CVD risk on univariate analysis significant factors with p values in parentheses in order of significance were: age (8.1×10^{-41}), hypertension (3.2×10^{-23}), HDL-C (4.2×10^{-16}), sdLDL-C (4.2×10^{-14}), hypertension treatment (1.5×10^{-14}), gender (1.7×10^{-10}), diabetes (5.1×10^{-9}), direct LDL-C (8.2×10^{-9}), body mass index (9.2×10^{-7}), calculated LDL-C (6.2×10^{-6}), RLP-C (8.0×10^{-4}) cholesterol medication (1.8×10^{-4}), total cholesterol (0.00081) smoking (0.0024), hsCRP (0.005), and Lp(a) (0.024). On multivariate analysis sdLDL-C, direct LDL-C, hsCRP, and Lp(a) were all still significant using the model including all standard risk factors. All four parameters significantly improved the model C statistic and net risk reclassification. **Conclusions:** Our data indicate that: 1) HDL-C and sdLDL-C are the most significant lipoprotein predictors of CVD; 2) calculated LDL-C underestimates direct LDL-C levels, 3) direct LDL-C is significantly better than calculated LDL-C in CVD risk prediction; 4) HDL-C, sdLDL-C, direct LDL-C, RLP-C, and Lp(a) are all significant lipoprotein particles contributing to CVD risk, and 5) with all standard risk factors in the model sdLDL-C, direct LDL-C, Lp(a), and hsCRP all add significant information above and beyond the standard model in predicting CVD risk prospectively over about a 16 year period in the Framingham Offspring Study. We conclude that these parameters should be measured in all patients with CVD, diabetes, or those with an AHA calculated 10 year CVD risk of ≥ 7.5% (i.e patients targeted by AHA for diet and statin treatment).

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Fasting versus Non-Fasting Lipid Panels

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Background: In 2016 a European guideline was introduced by Nordestgaard *et al.*, advising for patients not to fast before having their lipid levels tested[1]. Non-fasting lipid testing is more convenient for the patient, could reduce laboratory costs by spreading tests on a workday, but also may better represent a typical lipid load throughout the day, as was explained in the AACCs' January/February *Clinical Laboratory News*. The aim of our study was to retrospectively measure whether the introduction of a non-fasting policy for lipid testing in our hospital changed the test results, including the LDL-C (low-density lipoprotein-cholesterol) calculated with the Friedewald formula. **Methods:** Of each lipid test (Total Cholesterol, HDL-C, Triglycerides, and LDL-C) we compared at least 10,000 results before the introduction of the non-fasting policy for lipid testing to at least 10,000 results after. Differences of the mean, median and standard deviations were calculated with EP Evaluator. **Results:** The fasting versus non-fasting lipid panels showed a very good correlation. Differences in the mean and median were very small (0,009-0,111mmol/L and 0,00-0,09mmol/L, respectively) and not significant. Box-plots are presented in the figure. Results are in mmol/L. **Con-**

clusion: In concordance with previous studies, the non-fasting lipid panels in our hospital do not significantly differ from fasting lipid panels. Because of the advantages of non-fasting lipid testing, this is the method of choice. **References:** Nordestgaard *et al.* 2016. Clin Chem 62:7, 930-946 and CLN Stat. Feb.1.2018.