Introduction: Glomerular Filtration Rate (GFR) when measured (mGFR) is often found to be low among Living Kidney Donors (LKDs) in India. Isolated finding of low GFR with no other evidence of kidney disease or comorbidities like diabetes or hypertension was the most common reason why LKDs were considered ineligible to donate at our centre. The reason for low mGFR observed in Indians is not clear and multiple factors are proposed to play a role, including low dietary protein intake and low lean body mass. Malnutrition and low birth weight can result in low number of nephrons. There are no reports of number of glomeruli in healthy Indians. Hence this study was conducted to estimate the total number of glomeruli and single nephron GFR among LKDs at our centre.
Methods: In a prospective observational study, we enrolled LKDs who underwent donor nephrectomy at our centre. The renal cortical volume of both the kidneys were measured from the pre-donation Contrast Enhanced Computed Tomography images. We calculated the glomerular density from the pre-implantation kidney biopsy by morphometric analysis of needle core biopsy. Glomerular density was corrected for absence of capsule, presence of partially dissected glomeruli , missing glomerular tufts and tissue shrinkage based on validated methodology used in earlier studies. From the total renal cortical volume, glomerular density and the measured GFR, we calculated the total number of nephrons in each kidney and the single nephron GFR.
Results: Thirty-five LKDs were eligible and enrolled in the study. The mean age of the living kidney donors was 43.6±9.93 years. Our study population was heterogenous including LKDs from South and East

India. Out of the 35 donors, 29 (82.9%) were females. 14 (40%) LKDs had hypertension

(SBP>130 or DBP>80 mmHg) and 13 (37.1%) had prediabetes (IFG, IGT or HbA1c 5.7 – 6.4%). The mean BMI was 24.7±3.07. The mean GFR (CKD-EPI) was 105.97±16.64 ml/min/1.73m2 and mean normalized GFR by DTPA renal plasma clearance was 92.12 ± 14.71ml/min/1.73m2.The mean total cortical volume was 210,481.7±33570 mm3. The median number of total and nonsclerotic glomeruli among our LKDs were 651,178 (439,726-1,105,129) and 645,833 (439,7261,098,490) per kidney respectively. The median single nephron GFR was 52.13 (35.06-96.51) nanoliter/min. Among the various independent variables assessed, only serum uric acid had a statistically significant (p value - 0.029) negative association with the number of glomeruli.
Conclusions: The number of total glomeruli and non-sclerotic glomeruli among our LKDs were lower than the number of total glomeruli and non-sclerotic glomeruli reported among American and Japanese LKDs.

I have no potential conflict of interest to disclose.

WCN24-2381

PLASMA AND URINE INDOLELACTIC ACID: PROMISING BIOMARKERS FOR CHRONIC KIDNEY DISEASE AND INFLAMMATION STATUS

Hao Hong[*1]
[1]Suzhou, Soochow University, China

Introduction: Chronic kidney disease is one of the diseases which threaten the world, and patients' quality of life could be deteriorated with the disease progression. The pathogenesis of CKD involves inflammation, immune disorders, and oxidative stress. In fact, the specific pathogenesis of CKD has not been fully elaborated. It is of great significance to clarify the pathogenesis of CKD for the development and prognosis of the disease. Indolelactic acid is a protein-bound uric acid solute from tryptophan metabolism that belongs to the indole uremic solute family. Indolelactic acid is an agonist of the transcription factor aryl hydrocarbon receptor, promotes vascular inflammation, oxidative stress, and atherosclerosis, and plays a role in cardiovascular disease. Accumulation of urine toxins usually affects disease progression and prognosis in patients with CKD. Urine toxin is associated with multiple complications in patients with CKD and may be involved in cardiovascular events in patients with CKD, which has certainly raised our concerns about the accumulation of toxins in patients with CKD. At present, metabolomics methods are commonly used in clinical practice for the detection of urine toxins. This study intends to explore the changes of plasma and urine Indolelactic acid in patients with CKD and healthy people through LC-MS, and explore the relationship of inflammation and Indolelactic acid in patients with CKD and healthy people
Methods: Patients and Methods: A total of 47 patients with CKD and 30 healthy people were included. Clinical parameters were recorded. One-way ANOVA was performed for variables which had normal distributions and homogeneous variance. The rank sum test was performed for variables which had non-normal distributions. Pearson or Spearman correlation analysis was used for the correlations analysis. Binary logistic regression and ordinal logistic regression were used for the independent relationship of CKD. Receiver operating characteristic curve was used for the accuracy of diagnosis Youden's index was calculated for the best cut-off of the Receiver operating characteristic curve.
Results: The control group had higher levels of hemoglobin and albumin, and lower levels of Ln

(creatinine) and Ln (BUN). Plasma and urine ILA had significant differences between the patients with CKD and control group, and showed an increased trend with the progression of renal function. Plasma ILA and urine ILA were positively correlated ($r = 0.51$, $P < 0.01$). Plasma ILA had positive correlations with age, BMI, creatinine, BUN, triglycerides, and uric acid, and negative correlations with hemoglobin. Urine ILA had positive correlations with age, creatinine, BUN, and uric acid, and a negative correlation with hemoglobin and albumin. Before adjustment for confounding, CKD was associated with plasma ILA (OR = 5.20, $P < 0.01$) and urine ILA (OR = 5.24, $P < 0.01$). After adjustment for confounding, ordinal logistic regression indicated that CKD had a significant relationship with plasma ILA (OR = 4.49, $P < 0.01$), urine ILA (OR =

2.14, $P < 0.01$), BUN (OR = 1.43, $P < 0.01$), and hemoglobin (OR = 0.95, $P < 0.05$). ROC curve showed that plasma ILA and urine ILA were reliable predictions of CKD. CKD was associated with three inflammatory factors: plasma ILA (OR = 5.92, $P < 0.01$), urine ILA (OR = 2.79, $P < 0.01$), and Hs-CRP (OR = 2.45, $P < 0.01$).
Conclusions: Plasma and urine ILA have potential use as biomarkers for CKD, and inflammation status.

I have no potential conflict of interest to disclose.

WCN24-2457

AIR POLLUTION AND KIDNEY DISEASES IN SÃO PAULO CITY

Iara da Silva[*1], Alejandro Herman Delgado Peralta[2], Mario Eduardo Gavidia-Calderón[2], Maria de Fatima Andrade[2], Lucia Andrade[3], Edmilson Dias de Freitas[2], Alessandra Tammaro[4], Jesper Kers[4]
[1]São Paulo, Department of Atmospheric Sciences, Brazil; [2]Department of Atmospheric Sciences, Institute of Astronomy, Geophysics and Atmospheric Sciences, University of São Paulo, São Paulo; [3]Division of Nephrology, School of Medicine, University of São Paulo, São Paulo; [4]Department of Pathology, Amsterdam Infection & Immunity Institute, University of Amsterdam, Amsterdam

Introduction: Air pollution is a widespread problem that affects all areas of society because poor air quality results in an increase in the incidence of various diseases that are influenced by the environment, notable among which are kidney diseases. Studies indicate that prolonged exposure to moderate-tohigh concentrations of particulate matter (PM) can decrease renal function, promoting the progression of chronic kidney diseases and related problems. Despite emission controls on the road transport sector, people of the Metropolitan Area of São Paulo (MASP) have been exposed to air pollutants concentrations above the WMO guidelines for many years, especially surface ozone and fine particles. Therefore, the objective of this study was to explore the impact of air pollution on glomerular and chronic kidney diseases in the city of São Paulo, Brazil, through the use of statistical models.
Methods: Meteorological and air quality data were obtained from monitoring stations operated by the São Paulo State Environmental Protection Agency. The variables analyzed included average daily temperatures, relative humidity (%), PM < 2.5 µm (PM2.5) concentration, and daily hospital admissions in São Paulo City during the 2011–2021 period. Hospitalization records were extracted from the public health care system and from private facilities affiliated with the public health care system. The patients investigated were diagnosed with diseases directly or indirectly linked to kidney problems, as defined in the International Classification of Diseases, 10th revision. Daily hospitalization admissions were categorized by patient age and sex. Regression analysis, employing the Generalized Additive Model in conjunction with negative binomial distribution of exponential probability, was performed in a time series. We examined temperature, relative humidity, and PM2.5 concentrations for each group. Two other variables - holiday and weekday data - were introduced into the model. Given the nonlinearity of the effects of exposure to PM, we assessed the

cumulative impact of exposure in the medium term. We also employed the distributed lag non-linear models, which provides a modeling framework that simultaneously considers the response to exposure and the effects of time lags. The lag considered was up to 2,000 days (approximately 5 years) for all groups, (PM Kidney Consortium, FAPESP, NWO).

Results: These preliminary results show that the risks of exposure to air pollution are most clearly defined (relative risk, 1.04; 95% CI:1.02-1.05) in adult women (18 < age > 60 years), which could be attributed to hormonal or physiological factors. For younger women, the risk is not yet established, whereas the risk in older women became evident after ≥ 5 years of exposure. For almost all groups, exposure to PM2.5 concentrations > 10 μg/m^3 for a prolonged period (more than 2,000 days) was found to increase the risk of developing one of the diseases analyzed. Men, especially older men, tended to be more susceptible (relative risk ≤ 1.05; 95% CI: 1.01-1.06).

Conclusions: In conclusion, exposure to air pollution seems to increase the incidence of glomerular and chronic kidney disease.

I have no potential conflict of interest to disclose.

WCN24-2483

ASSESSMENT OF RENAL FUNCTION BY DIFFERENT GFR ESTIMATION METHODS IN A RURAL POPULATION OF BANGLADESH

MAA Chowdhury*[1], MM Iqbal[2], Md Nasir Uddin[3], KS Alam[3], SK B anerjee[4], Sohel Reza Choudhury[5], Dipal K. Adhikary[4], Rana M. Hossain[6], Md Zahid Hassan[7], MS Hassan[8], MN Islam[9], Sarwar Iqbal[10], MI Arslan[11]

[1]Dhaka, Nephrology, Bangladesh; [2]Nephrology, NIKDU & KDRG, Dhaka; [3]Nephrology, NIKDU, Dhaka; [4]Cardiology, BSMMU, Dhaka; [5]Epidemiology And Research, NHFH&RI, Dhaka; [6]Nephrology, BSMMU, Dhaka; [7]Physiology and Molecular Biology, BUHS, Dhaka; [8]Immunology, BIRDEM, Dhaka; [9]Nephrology, DMCH, Dhaka; [10]Nephrology, KDRG, Dhaka; [11]Biochemistry, KDRG, Dhaka

Introduction: Proper measurement of renal function is important for diagnosis and stratification of kidney disease. Though several methods have been used to estimate the glomerular filtration rate, results have been variable depending on the population studied. This study was undertaken to estimate and compare different creatinine and cystatin C based GFRs to see their agreement with each other.

Methods: This cross-sectional study was conducted on random 222 participants of a rural area. Detail medical history was taken along with available relevant investigations from each patient. The eGFR was estimated using creatinine-based C-G (Cockcroft-Gault), MDRD, CKD-EPI methods and creatinine and cystatin C based formula methods.

Results: Mean age of the study population was 41±12 years with a majority in 31-50 years of age (59%) where male were 53%. Hypertensive subjects were 11% and diabetes was present in 7%. Mean eGFR measured by creatinine based MDRD, C&G (Cockcroft-Gault) and CKD-EPI were 117±25, 102±30 and 119±20 mL/min/1.73 m2 (p=NS). Mean eGFR measured by cystatin C based equation was 107±16 and by both creatinine and cystatin C was 105±20 mL/min/1.73 m2 (p=NS). Comparisons of eGFR by Bland-Altman plot in the study subjects showed some bias between all methods. Here data were skewed and a wide range for the limit of agreement.

The distribution of the frequency of CKD stages varied significantly when each of the formulas applied by creatinine and cystatin C based methods. The stage distribution pattern by MDRD formula of G1, G2 and G3 was 86.5%, 12.6% & 0.9% ; by C-G 68.0%, 22.1% & 9.9% and by CKD-EPI 88.7%, 10.4% & 0.9%. The pattern for cystatin C based formula found only stage G1 and G2 of 87.8% &12.2% . By applying both creatinine and cystatin C it was 76.6% & 23.4%.

Conclusions: GFR estimation methods revealed wide variations when the distribution pattern of CKD stages was done by different creatinine and cystatin C based formulas. Therefore, these GFR estimation methods need to be compared with an ideal measured method to identify the appropriate one.

I have no potential conflict of interest to disclose.

WCN24-2501

BODY MASS INDEX AND MORTALITY RISK IN A CHRONIC KIDNEY DISEASE 1-5ND COHORT

Alejandro Ferreiro*[1], Pablo Rios[2], Ricardo Silvariño[3], José Santiago[3], Laura Solá[3], Graciela Suarez[3], Liliana Gadola[3]

[1]Montevideo, Montevideo, Uruguay; [2]Montevideo, PROGRAMA SALUD RENAL URUGUAY, Montevideo; [3]Montevideo, Comisión Asesora en Salud Renal, Montevideo

Introduction: Body mass index (BMI) evaluation is very important in chronic kidney disease (CKD) patients, as a screening tool. According to WHO(1) overweight (BMI 25 a 29,9 Kg/m2) and obesity (BMI ≥30 Kg/m2) increased the mortality risk vs normal weight (BMI18,5 a 24,9 Kg/m2), among the general population. In the oldest persons, this relation changes and the least mortality risk is associated with the highest BMI (22-26.9 Kg/m2)(2) . In CKD 5D patients an inverse association has been described, but in earlier CKD stages it is not well defined. The Uruguayan National Renal Healthcare Program (NRHP-U) registry included CKD adult patients stages 1-5 no D, median age 70 years.

The aim of the study was to evaluate, in the NRHP-U CKD cohort, the association between BMI and mortality (all-causes and cardiovascular), according to age and CKD stages.

Methods: A retrospective analysis of the NRHP-U CKD patients was done. The registry included patients 19 years or older, with weight and height data. The variables studied were sex, age, CKD stage, proteinuria, blood pressure, diabetes, smoking, and cardiovascular comorbidities. Follow-up time was calculated between NRHP-U inclusion and the final date (death date, kidney replacement therapy starting date, or June 30th, 2023). BMI were categorized in: <18.5, 18,5 a 22.49, 22.5 a 24.99, 25 a 27.49, 27.5 a 29.99, 30 a 32.49, 32.5 a 34.99, 35 a 37.49, 37.5 a 39.99 y ≥ 40 Kg/m^2. The Hazard Ratio (HR) risk for all-cause and cardiovascular death was calculated with the Cox proportional risk model adjusted to the above-mentioned variables. Each BMI category HR was calculated with the lower risk category as the reference. All patients signed informed consent and the study was approved by the Ethics Committee.

Results: There were included 21183 patients, mean age (pc 25-75) 70 years (60-77), male sex 58%, CKD stage 1, 2, 3, 4 y 5 (%): 6.6, 11.5, 58.5, 19.7 y 3.1%. Proteinuria/albuminuria < 0.3 gr/day: 78.8%, between 0.3 y 0.99 gr/d: 10.7% and ≥ 1 gr/d: 9.0%. Diabetes: 38.3%, smoking: 6.8%, cardiovascular comorbidity: 30.2%, systolic blood pressure (SBP) < 120 mmHg: 17.2%, 120 - 139 mmHg: 42.3%, BMI: <18.5: 0.9%, 18,5 a 22.49: 8.2%, 22.5 a 24.99: 13.6%, 25 a 27.49: 18.5%, 27.5 a 29.99: 18.9%, 30 a 32.49: 15.5%, 32.5 a 34.99: 10.3%, 35 a 37.49: 5.8%, 37.5 a 39.99: 3.3% y ≥ 40 Kg/m^2: 5%. Follow-up time, median 5.71 years (pc25-75: 3.33 – 9.58). All-cause death: 8.025 (37.9%) (rate 6.0/100 pts-year), cardiovascular deaths 2353 (11.1%), rate 1.76/100 pts-year. The BMI categories with lower all-cause and cardiovascular death risk are shown in Table 1. The BMI category 27.5 and 32.49 Kg/m^2 showed a significantly lower all-cause mortality risk (Figure 1) In the group older than 70 years, the lower death risk is associated with a BMI of 30 to 32.4 Kg/m^2. significantly different from the highest (BMI ≥ 37.5 Kg/m^2: HR 1.23) and the lowest categories (BMI < 25 Kg/m^2: HR 1.38).

Conclusions: In the Uruguayan CKD 1-5ND patient cohort, a BMI between 27.5 and 32.49 Kg/m^2, was associated with the lower all-cause and cardiovascular death risk.

I have no potential conflict of interest to disclose.

WCN24-2515

TOLERANCE OF HIGH DOSE LAMIVUDINE IN PATIENTS WITH CHRONIC KIDNEY DISEASE LIVING WITH HIV

Faryal Raza*[1], Eijaaz Ahmed[2], Regina Dahaye[2], Evaline Muro[3], Kajiru Kilonzo[2]

[1]Moshi, Internal Medicine, Tanzania; [2]Internal Medicine, KCMC, Moshi; [3]Pharmacology, KCMC, Moshi

Introduction: Lamivudine is a nucleoside reverse transcriptase inhibitor (NRTI) widely used for the treatment of HIV (Human Immunodeficiency Virus) infection in combination with other antiretrovirals. It is an effective agent with a long intracellular half-life, allowing for once or twice daily dosing. It also has one of the best tolerability and long-term safety profiles among all antiretroviral agents. However, lamivudine is only available in combinational fixed doses, which can pose a challenge for patients with chronic kidney disease (CKD). CKD is a common comorbidity in HIV-infected patients, with a prevalence of 6.4% in a global study. Patients with CKD require dose adjustments of lamivudine to avoid toxicity. In Sub-Saharan Africa, where the prevalence of CKD is highest, patients with HIV and CKD often do not receive the renal-adjusted doses of lamivudine recommended by national guidelines. This is due to a lack of accessibility to single-agent lamivudine.