Results: Mean eGFR decreased with higher POx quartiles. eGFR modified the associations of POx with CKD progression (P=0.01) and death (P=0.02). In participants with eGFR≥45, higher POx quartiles were associated with CKD progression after adjusting for demographic factors, comorbidities, medications, lab values (including hemoglobin, serum albumin, urine protein-to-creatinine ratio), and eGFR (Q3 vs Q1: HR 2.07, 95% CI 1.12-3.82; Q4 vs Q1: HR 2.23, 95% CI 1.24-3.99). Higher POx was associated with death in participants with eGFR≥45 after multivariable adjustment (Q4 vs Q1, HR 1.94, 95% CI 1.10-3.44). POx doubling was associated with a 34% increased risk of CKD progression and 28% increased risk of death (Table 1A). In those with eGFR<45, higher POx was associated with CKD progression after adjusting for demographic factors, comorbidities, medications, and lab values. Adjusting for eGFR attenuated these associations, with higher POx trending towards being protective of CKD progression. Associations of POx and death were not significant after adjusting for covariates and trended towards being protective after adjusting for eGFR (Table 1B). Sensitivity analyses adjusting for 24-hour urinary oxalate did not change these associations.

Conclusions: Higher plasma oxalate may be an independent risk factor for CKD progression/ESKD and death in persons with eGFR≥45.

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PO2252

Relationship Between 24-Hour Urinary Oxalate and Incident CKD Among Patients with and Without Underlying Gastrointestinal Disease

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Background: Hyperoxaluria may result from intake of high oxalate foods or enhanced intestinal absorption of dietary oxalate caused by gastrointestinal (GI) disorders with underlying malabsorption, including Crohn’s disease, short bowel syndrome, gastric bypass surgery, and chronic pancreatitis. Hyperoxaluria has been associated with negative outcomes, including kidney stones and chronic kidney disease (CKD), but larger studies are needed.

Methods: This is a longitudinal retrospective observational cohort study of patients in the US who have completed at least one 24-hour urine collection analyzed by a central laboratory during the study period of January 2013 through December 2020. Outcome and covariate data were drawn from a multi-source data cloud containing deterministically linked, de-identified, and up-to-date healthcare claims and electronic medical records (EMR) data. Malabsorption was defined by the presence of a relevant ICD 9/10 or CPT code. The association between categories of urinary oxalate (UOx) and incident CKD was modeled using logistic regression.

Results: 762,337 individuals age ≥18y with at least one 24-hr urine collection were identified. At least 6 months of baseline and 6 months of follow-up data (median follow-up time: 37.6 months; IQRR: 20.4, 56.0) were available for 447,958. Of these, N=12,522 (2.8%) had an underlying malabsorptive condition preceding the index urine test. 426,896 patients had no evidence of CKD at baseline and were eligible for analysis of incident CKD. After adjusting for baseline urine calcium, urine citrate, age, sex, race, BMI, tobacco use, hypertension, diabetes, malabsorption, and CVD, a significant association was found between baseline UOx and the development of incident CKD was observed. Compared with patients with UOx<20 mg/d, the odds of developing incident CKD increased for 20-29 mg/d (OR: 1.22, 95% CI: 1.15, 1.30) through 80+ mg/d (OR: 1.67, 95% CI: 1.51, 1.86) and was statistically significant for each UOx category.

Conclusions: In this large population of patients with hyperoxaluria, the risk of incident CKD increased with increasing 24-hr urine oxalate excretion. Future studies should examine whether reducing urinary oxalate diminishes the risk of developing CKD.

Funding: Commercial Support - Synlogic Inc.

PO2253

Associations of CKD Risk Factors and Longitudinal Changes in Urine Biomarkers of Kidney Tubules Among Women Living with HIV

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Background: Among women with HIV (WWH), urine biomarkers of tubule dysfunction and injury allow detection of antiretroviral toxicity and prediction of CKD risk and mortality. However, risk factors for changes in urine biomarkers are unclear.

Methods: We assessed traditional and infection-related CKD risk factors and measured 14 urine biomarkers at baseline and at follow-up (median 2.5 years) among WWH in the Women’s Interagency HIV Study. We used simultaneously adjusted multivariable linear regression models to evaluate the associations of CKD risk factors with changes in biomarker levels concurrently.

Results: Of the 647 women in this analysis, 67% were Black, median age at baseline was 45 years and eGFR was 104 ml/min/1.73m². Each CKD risk factor associated with distinct changes in urine biomarkers (Figure). For example, baseline hemoglobin a1c (HbA1c) associated with worse tubular injury (higher interleukin-18 [IL-18]), proximal tubular reabsorptive dysfunction (higher alpha-1 microglobulin), tubular reserve (lower umodulin) and heightened immune response to injury (higher chitinase-3-like protein [YKL-40]). HIV viremia associated with worsening proximal tubular reabsorptive dysfunction (higher beta-2 microglobulin [β2m]), and immune response to injury (higher YKL-40). Hepatitis C virus co-infection associated with worsening proximal tubular reabsorptive dysfunction (higher beta-2 microglobulin [β2m]), and immune response to injury (higher YKL-40), whereas HIV viremia associated with worsening markers of tubular and glomerular injury (higher KIM-1 and albumin, respectively).

Conclusions: CKD risk factors associated with unique patterns of biomarker changes among WWH, suggesting that longitudinal biomarker measurements may help in detecting and monitoring kidney disease in WWH.

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PO2254

Correlation Between Urinary Sodium and Protein Excretion in CKD

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Background: Urinary protein excretion often fluctuates in patients with chronic kidney disease (CKD). We aimed to establish a correlation between spot urine sodium (Na) measurement as a surrogate marker of 24hr urine excretion based on Kawasaki formula, and therefore, sodium intake, and urinary protein excretion. We hypothesize that urinary Na excretion may affect urinary protein excretion.

Methods: This was a retrospective study involving 213 US veterans with CKD followed in the Albany VA/MC nephrology clinic for the period of 2 years. Patients with cirrhosis, end-stage renal disease, and renal transplant were excluded. Simultaneous measurements of serum Na, Creatinine (Cr) and urine Na, Cr and protein were performed on 2 separate visits. Kawasaki formula was used to estimate 24-hour urine Na excretion. Proteinuria was calculated using urine protein to creatinine ratio (UPCR). Correlations among percent change in estimated 24th urine Na and UPCR were determined with linear regression model.

Results: The mean age a SD of the cohort was 74.4 ± 9.5 years. Mean estimated GFR was 47.4 ml/min/1.73m² and UPCR was 1.0 g/g. About 97% of subjects were male and 51% had diabetes. Using multivariable linear regression, we found that weight, height,