WCN26-8548 HLA alleles and their association with chronic kidney disease in a Nigerian population [Abstract only]

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Methods: Six pediatric patients who received SGLT2 inhibitors for more than 18 months were analyzed. The underlying diseases were vesicoureteral reflux (n = 2), renal hypoplasia (n = 2), familial juvenile hyperuricemic nephropathy (n = 1), and Alport syndrome (n = 1). The median age at initiation was 16.0 years.

The annual change in estimated glomerular filtration rate (ΔeGFR) during the two years before treatment was compared with that observed after six months of SGLT2 inhibitor therapy.

Results: The mean baseline eGFR was 39.0 mL/min/1.73 m^2 (range: 21.7–72.6).

The mean ΔeGFR during the two years prior to treatment was −3.37 mL/min/1.73 m^2/year (range: −9.66 to −2.18).

During the first six months of therapy, ΔeGFR worsened in five cases; however, from six months onward, the mean ΔeGFR improved to −1.82 mL/min/1.73 m^2/year (range: −2.95 to 2.02), with improvement observed in all but one case.

The median rate of change was +66.1 % (range: −24.4 to 192.4 %), and three patients demonstrated an upward trend in eGFR.

Adverse events included one case of urinary tract infection and mild hypoglycemic symptoms in two cases.

The five patients showing ΔeGFR improvement had non-glomerular congenital renal diseases or CAKUT with negative or mild proteinuria.

Conclusion: Our findings suggest that SGLT2 inhibitors can exert renoprotective effects even in pediatric CKD patients with minimal proteinuria.

Because SGLT2 inhibitors are known to cause an initial transient decline in GFR ("initial dip") after therapy initiation, ΔeGFR was evaluated from six months onward, beyond this phase, and improvement was observed in five of the six cases.

I have no potential conflict of interest to disclose.

I did not use generative AI and AI-assisted technologies in the writing process.

WCN26-8548

HLA ALLELES AND THEIR ASSOCIATION WITH CHRONIC KIDNEY DISEASE IN A NIGERIAN POPULATION

(Article No. 104406)

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Introduction: Chronic kidney disease is a critical public health challenge in Nigeria, mirroring global concerns. It is a rapidly increasing health issue in both developed and developing countries. The interaction between the genetic and environmental factors may play a role in the development of end-stage renal disease. The HLA allele caries genetic information regarding the transmission of many pathologies, including that of the kidney, and as suggested in some studies, HLA alleles are now attributed to the possibility of developing ESRD, while some may be protective. There is no large-scale study that has mapped HLA alleles distributions in CKD patients or investigated their possible association with CKD. Therefore, this study aims to investigate the distribution of HLA alleles among CKD patients and their potential association with CKD in a Nigerian population.

Methods: This is a retrospective cross-sectional study that was carried out at a private health facility in Abuja, Nigeria. HLA data on CKD patients who were on work-up for kidney transplant were retrieved from the laboratory, while HLA data from the pool of prospective non-related donors were also retrieved for the analysis. The HLA typing from DNA samples extracted was performed by Polymerase Chain Reaction - Sequence-Specific Oligonucleotide Probing method. The HLA data was analysed using SPSS version 26 and p-value < 0.005 was considered statistically significant.

Results: The HLA results of a total of 846 recipients and 1017 donors were analyzed in this study. The distribution of HLA – A, HLA – B, and HLA – DR alleles are as shown in figures 1, 2 and 3. Among the samples studied, we identified 108 different alleles. It was observed that the HLA-B alleles were the most polymorphic (52 alleles) as compared to HLA-A (27 alleles), and DR (20 alleles). We used the logistic regression model to adjust for each allele's effect, considering the presence (or absence) of other alleles and to control for potential confounding factors caused by multiple HLA alleles. The following alleles had a statistically significant protective effect for the presence of CKD: HLA-A*23 (OR=0.78 [0.63 – 0.96]), HLA-A*29 (OR=0.47 [0.26 – 0.85]), HLA-B*49 (OR=0.58 [0.37 – 0.89]) and HLA-DR*15 (OR=0.79 [0.65 – 0.96]). On the other hand, alleles HLA-B*42 (OR=1.49 [1.20 – 2.03]), HLA-B*57 (OR=1.38 [1.02 – 1.86]), HLA-B*72 (OR=1.44 [1.04 – 2.00]), and HLA-DR*18 (OR=2.57 [1.70 – 3.89]) were found to have a statistically significant risk effect.

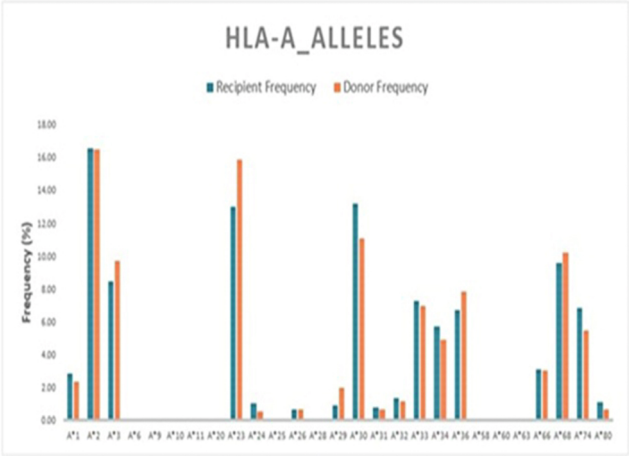

Figure 1. Graphical representation of human leukocyte antigen-A alleles frequencies.

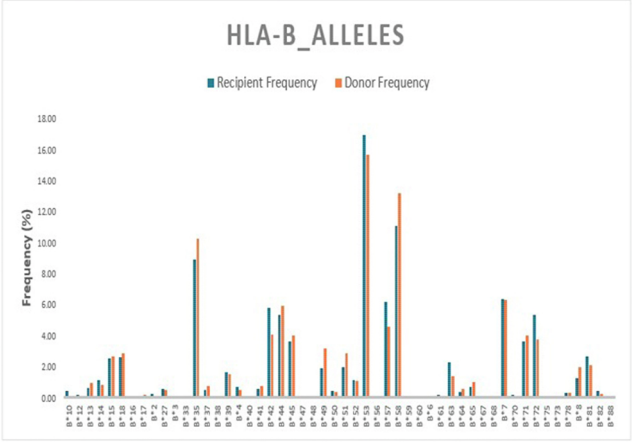

Figure 2. Graphical representation of human leukocyte antigen-B alleles frequencies.

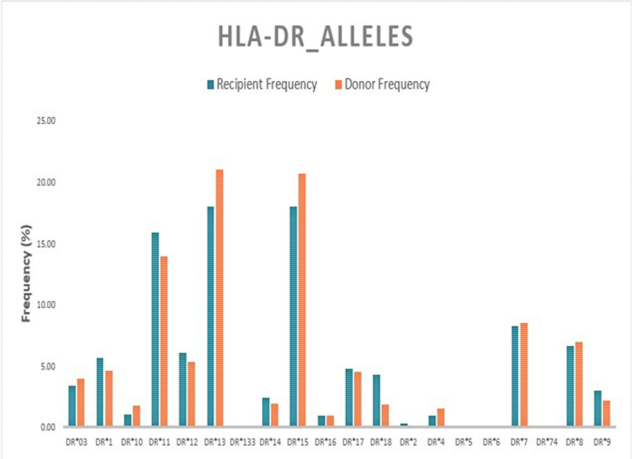

Figure 3. Graphical representation of human leukocyte antigen-DR alleles frequencies

Conclusion: The study's results contribute to a better understanding of HLA allele variability and its influence on CKD susceptibility, which may have implications for donor-recipient matching and improving transplant outcomes. Additionally, this study serves as a background study for the design and conduct of larger studies to find other genetic predisposition to the development of CKD in Nigerians.

I have no potential conflict of interest to disclose.

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WCN26-8557

PEMP: A HIGHLY AUTOMATED SUPER-RESOLUTION PLATFORM FOR QUANTITATIVE ANALYSIS OF PODOCYTE FOOT PROCEÖSS MORPHOLOGY ACROSS RESEARCH AND CLINICAL APPLICATIONS

(Article No. 104407)

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Introduction: Podocyte foot process (FP) morphology is a pivotal determinant of glomerular filtration barrier integrity. Owing to their nanoscale dimensions, FPs have traditionally been analyzed by electron microscopy (EM), a labor-intensive and low-throughput technique that limits large-scale or comparative studies. To overcome these constraints, we developed the Podocyte Exact Morphology Measurement Procedure (PEMP)—a super-resolution microscopy (structured illumination microscopy, SIM)-based method enabling precise, reproducible, and largely automated quantification of FP morphology, particularly filtration slit density (FSD), in standard paraffin-embedded kidney tissue.
Methods: PEMP combines multichannel immunofluorescence labeling (e.g., nephrin/podocin, synaptopodin/integrin α3) with SIM imaging to capture nanoscale structural detail. A proprietary software and AI-assisted analysis pipeline developed at NIPOKA performs fully automated FSD quantification, minimizing observer bias and ensuring consistent cross-sample comparisons. Except for immunofluorescence staining and slide loading, the workflow is fully automated, making PEMP readily applicable to both research and diagnostic laboratories
Results: We validated PEMP across diverse settings. In nephrotoxic serum (NTS) injury models, PEMP detected a significant reduction in FSD within 24 hours post-injury—preceding detectable proteinuria. In a translational feasibility study of over 135 human kidney biopsies encompassing various glomerular diseases, FSD values correlated with clinical and functional parameters, offering enhanced morphometric insights compared to EM. Furthermore, in a murine aging cohort, PEMP revealed a progressive, age-dependent decline in FSD and increased glomerular diameter, highlighting its sensitivity to both pathological and physiological remodeling.
Conclusion: Together, these findings establish PEMP as a robust, scalable, and reproducible platform for quantitative analysis of podocyte architecture in preclinical and clinical contexts. Its compatibility with routine histology, high throughput, and minimal user input position PEMP as a next-generation tool for glomerular pathology, with significant potential for early disease detection, longitudinal monitoring, and aging research in translational nephrology.

I have potential conflict of interest to disclose.
I am the CEO of NIPOKA

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WCN26-8801

THE KIDNEY FAILURE RISK EQUATION CALIBRATION AND VALIDATION ACROSS DIVERSE POPULATIONS: A SYSTEMATIC REVIEW

(Article No. 104408)

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Introduction: Chronic kidney disease (CKD) is a public health concern affecting approximately 10% of the global population. It is associated with multiple cardiovascular and non-cardiovascular complications, and with increased risks of death and hospitalization. The burden of CKD is rising and is projected to become the fifth leading cause of death by 2040. To improve assessment, monitoring, and referral to nephrologist, several tools have been developed to predict progression to kidney failure. Among them, the Kidney Failure Risk Equation (KFRE) has demonstrated excellent discrimination and can accurately predict kidney failure risk across diverse CKD populations. This systematic review synthesizes evidence on the external validation of KFRE across age subgroups, CKD etiologies, and comorbidities in highly complex patients.
Methods: We conducted a systematic review in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. We searched PubMed, Web of Science, Google Scholar, Semantic Scholar, the Cochrane Library, SciELO, Dialnet, and DOAJ for peer-reviewed articles in English and Spanish to identify all studies validating the KFRE in different age groups, CKD etiologies, and comorbidities since the original development study by Tangri et al. (2011). Risk of bias and certainty of evidence were assessed using PROBAST (Prediction model Risk Of Bias ASsessment Tool) and the GRADE framework, respectively. When the mean (SD) age was not reported, it was estimated from the median (IQR) using the method described by Wan et al. (2014).
Results: Thirty studies were included (Figure 1). The mean age of the evaluated populations was 69.5 ± 10.2 years. Across analyses, the KFRE showed high discrimination (c-statistic/AUC >0.70). Diabetic kidney disease (28.8%) and hypertensive kidney disease (29.3%) were the most frequent CKD etiologies analyzed. Other etiologies—such as autosomal dominant polycystic kidney disease (ADPKD, 1.3%) and chronic tubulointerstitial nephropathy (CTIN, 2.5%)—were underrepresented ($p < 0.0001$). The most common comorbidities were diabetes mellitus (31%), hypertension (56%), vascular disease (6.1%), and heart failure (HF) (3.5%). Only one study directly evaluated a population with frailty (0.58%). Overall, only 9 studies (30%) were rated as low risk of bias (Figure 2), and the certainty of findings was moderate in 47% and low in 53% of studies, indicating that more than half warrant cautious interpretation.