

APOL1 risk allele variants are absent in Indian Patients with Chronic Kidney Disease

Ashok Kumar Yadav¹, Vivek Kumar¹, Nisha Sinha¹, Vivekanand Jha^{1,2}

¹Department of Nephrology, Postgraduate Institute of Medical Education and Research,
Chandigarh, India,

²George Institute for Global Health, 219-221 Splendor Forum, Jasola District Center, New
Delhi, [India](#)

Corresponding address:

Prof Vivekanand Jha, George Institute for Global Health, 219-221 Splendor Forum, Jasola
District Center, New Delhi 110 025, INDIA Email: vjha@pginephro.org

To the Editor: Apolipoprotein L1 (*APOL1*) gene risk allele variants (G1 and G2) are strongly associated with focal segmental glomerulosclerosis (FSGS), human immunodeficiency virus associated nephropathy (HIVAN) and development as well as progression of chronic kidney disease (CKD) in subjects with African ancestry.¹ India is home to one-sixth of world population and has witnessed a rapid rise in burden of CKD, including that of unknown etiology. Out of many postulated mechanisms, the role of genetic predisposition has been suggested. However, published reports have failed to show *APOL1* high risk variants in any other population.² Though an *APOL1* null individual has been described from India, he did not have kidney disease.³ As there are no data on frequency of *APOL1* risk alleles in the Indian population, we decided to analyze *APOL1* G1 and G2 variants by direct sequencing of 302 base pair fragment in exon region in 159 subjects with CKD enrolled in the pilot phase of Indian Chronic Kidney Disease (ICKD) study.⁴

The causes of CKD and selected clinical parameters in the study population are listed in table 1. Importantly, the cause of CKD could not be ascertained in almost one third of subjects. We did not find *APOL1* risk alleles (G1 and G2) in any subject in our study population. Our findings suggest that *APOL1* is unlikely to explain the development ~~and/or progression~~ of CKD in Indian subjects in our cohort.

Table 1: Characteristics of study population (n=159)

| Parameter | Value |
|---|--------------------------|
| Age (years) | 45.3 ₀ ±13.52 |
| Gender (M/F) | 100/59 |
| Body mass index (kg/m ²) | 24.98±5.28 |
| Serum creatinine (mg/dl) | 1.75±0.63 |
| CKD-EPI eGFR (ml/min/1.73m ²) | 51.32±36.30 |
| Hemoglobin (g/dl) | 11.82±2.10 |
| Serum calcium (mg/dl) | 9.31±0.85 |
| Serum inorganic phosphorus (mg/dl) | 3.87±0.95 |
| Serum albumin (mg/dl) | 4.44±0.79 |
| Duration of CKD (months) | 41.46±43.42 |
| | |
| Cause of CKD | |
| Unknown | 55 (34.6) |
| Chronic interstitial nephritis | 22 (13.8) |
| Diabetic kidney disease | 21 (13.2) |
| Membranous nephropathy | 13 (8.2) |
| Immunoglobulin A nephropathy | 10 (6.3) |
| Polycystic kidney disease | 9 (5.6) |
| Focal segmental glomerulosclerosis | 6 (3.8) |
| Hypertensive nephrosclerosis | 6 (3.8) |
| Others | 17 (10.7) |

CKD: chronic kidney disease; CKD-EPI: chronic kidney disease epidemiology collaboration;
eGFR: estimated glomerular filtration rate. Figures in parentheses are percentages

References:

1. Limou S, Nelson GW, Kopp JB, *et al.* APOL1 kidney risk alleles: population genetics and disease associations. *Adv Chronic Kidney Dis* 2014; **21**: 426-433.
2. Kopp JB, Nelson GW, Sampath K, *et al.* APOL1 genetic variants in focal segmental glomerulosclerosis and HIV-associated nephropathy. *J Am Soc Nephrol* 2011; **22**: 2129-2137.
3. Johnstone DB, Shegokar V, Nihalani D, *et al.* APOL1 null alleles from a rural village in India do not correlate with glomerulosclerosis. *PLoS One* 2012; **7**: e51546.
4. Kumar V, Yadav AK, Gang S, *et al.* The Indian Chronic Kidney Disease (ICKD) Study: Design and Methods. *Nephrology (Carlton)* 2016.