Commentary

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APOL1 Variants and Kidney Function in Middle-Aged Adults: The ARIC Study

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Description

In the United States, individuals experience a disproportionately higher prevalence of Chronic Kidney Disease (CKD) compared to white individuals. This increased risk has been associated with variants in the Apolipoprotein L1 (APOL1) gene. Two specific APOL1 variants, prevalent in around 12%-15% of black Americans and rare among those of European descent, are linked to a more rapid decline in kidney function among CKD patients who carry both variants (high-risk genotype) compared to those with one or none of these variants (low-risk genotype). As a result, healthcare providers are now screening black CKD patients with proteinuria for these APOL1 variants. Additionally, APOL1 genetic testing has been used to assess risk in black individuals considering kidney donation, aiming to better understand their susceptibility to future kidney disease. However, it is important to note that not all individuals with the high-risk APOL1 genotype will develop kidney disease; additional factors are usually required. It remains unclear whether kidney donation itself triggers the progression to CKD or End-Stage Kidney Disease (ESKD) in some Living Kidney Donors (LKD) with the high-risk APOL1 genotype. Current evidence, primarily from a single small study, suggests that kidney donation may not significantly impact the development of APOL1-related kidney disease. To comprehensively assess the influence of the high-risk APOL1 genotype on post-donation outcomes, it is essential to establish the baseline risk of kidney disease in healthy individuals carrying two APOL1 kidney risk variants. The lifetime risk of developing End-Stage Kidney Disease (ESKD) varies based on race, gender and age. The progression of kidney disease associated with the APOL1 gene also shows age-related differences. Studies have found that among healthy black individuals aged 18–30 years, there is a connection between the number of APOL1 kidney risk variants and the 25-year risk of Chronic Kidney Disease (CKD). However, in Black individuals aged 45–64 years, despite a high prevalence of pre-existing diabetes and hypertension, the influence of APOL1 kidney risk variants on CKD risk appears to be less pronounced.

Kidney disease

This information would be beneficial for counselling middle-aged black individuals with high-risk APOL1 genotypes who are potential living kidney donors and for unaffected family members of patients with APOL1-related kidney disease. Our hypothesis was that having a high-risk APOL1 genotype would not negatively affect the long-term kidney function of those who reach middle age with good health and normal kidney function. We analysed data from the Atherosclerosis Risk in Communities (ARIC) study to identify a group of healthy individuals aged 45–64 years and examined how self-reported race and APOL1 genotype influence long-term kidney function. The lifetime risk of developing End-Stage Kidney Disease (ESKD) varies based on race, gender and age. The progression of kidney disease associated with the APOL1 gene also shows age-related differences. Studies have found that among healthy black individuals aged 18–30 years, there is a connection between the number of APOL1 kidney risk variants and the 25-year risk of Chronic Kidney Disease (CKD). However, in black individuals aged 45–64 years, despite a high prevalence of pre-existing diabetes and hypertension, the influence of APOL1 kidney risk variants on CKD risk appears to be less pronounced. This information would be beneficial for counselling middle-aged black individuals with high-risk APOL1 genotypes who are potential living kidney donors and for unaffected family members of patients with APOL1-related kidney disease.

Metabolic pathways

While significant progress has been made in identifying the molecular and metabolic pathways involved in human kidney disease, there remains a pressing need for innovative therapies to prevent the necessity of renal replacement therapy. Developing new medicines faces challenges, including difficulties in translating findings from rodent models of kidney disease to clinical applications in human patients, as well as gaps in our understanding of the diverse renal cell types and the specific molecular and metabolic pathways driving kidney disease in humans. The adult human kidney comprises over 24 distinct cell types, including tubuloepithelial, stromal, immune cells and it is believed that the interplay between these intrinsic renal cells and immune cells plays a crucial role in developing effective kidney disease therapies. Although cutting-edge multiplex imaging technologies such as imaging mass cytometry, multiplexed ion beam imaging, and codetection by indexing are currently being employed to comprehensively characterize human kidneys with unprecedented detail, there remains a scarcity of knowledge about the intrinsic renal cell types

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implicated in kidney disease progression and renal inflammation in humans. By gaining a deeper understanding of the cellular composition of adult kidneys and the alterations in the renal microenvironment following injury, valuable insights into the complex pathophysiology of kidney disease in humans can be attained. Bioactive lipids are key metabolites crucial for generating Adenosine Triphosphate (ATP) to support physiological processes in the kidney. Dysregulated lipid metabolism has been found to contribute to renal inflammation, epithelial cell death and fibrosis. Despite recent progress in understanding the roles of lipids and associated enzymes in kidney disorders, along with detailed knowledge of the human kidney's cellular landscape at the single-cell level, there remains a scarcity of comprehensive multi-omics data that can identify and validate metabolic pathways driving human acute kidney injury and chronic kidney disease. Given the significant role of the immune system in kidney injury, a thorough examination of changes in immune cell populations and their connection to affected metabolic pathways post-injury may be crucial for discovering new therapeutic targets in kidney disease.