Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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Future Perspectives: Antigen Discovery, Multi-Omics, and Translational Immunology

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Glomerular diseases encompass a heterogeneous spectrum of immune-mediated nephropathies whose pathomechanisms remain incompletely understood at the cellular and spatial level. Herein, we are sharing our experiences on profiling of human glomerular disease with various multiomics platform. In refractory LN class IV, paired spatial and single-cell analyses revealed marked upregulation of IL-10 receptor expression compared to remission cases. In vivo validation with recombinant murine IL-10 confirmed significant myeloid cell expansion in treated animals. These myeloid cells function as central mediators of glomerular immune recruitment through the SPP1–CD44 signaling axis with proximal tubular epithelial cells, positioning IL-10 hypersensitivity as a key pathological amplifier in treatment-resistant LN. In MN, peripheral blood multiome profiling combined with GeoMx kidney spatial data revealed that refractory cases exhibit reduced B cell CD83 expression—favoring autoreactive B cell activation—alongside significant Treg depletion, together constituting a dual immune checkpoint failure that sustains anti-PLA2R autoantibody-mediated glomerular injury. Collectively, our findings demonstrate that multi-omics approach can uncover disease-specific molecular programs that will reclassfy conventional clinicopathologic disgnostic framework. This work provides a foundation for transitioning from morphology-based to molecularly informed disease classification.

Keywords: spatial transcriptomics, lupus nephritis, membranous nephropathy, single-cell RNA sequencing, molecular classification