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Spatially resolved transcriptomic profiling of mesangial regions reveals autophagy suppression in IgA nephropathy

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Objectives : IgA nephropathy (IgAN) is the most common primary glomerulonephritis and is characterized by mesangial IgA deposition with mesangial proliferation and matrix expansion. However, molecular alterations within mesangial regions remain incompletely understood.
Methods : Kidney biopsy samples from patients with IgAN (n=5), normal controls (n=3), and disease controls (diabetic kidney disease, DKD; n=3) were analyzed using spatially resolved laser-activated cell sorting (SLACS), a technique that enables selective isolation of region-specific cell populations from tissue sections. Mesangial cell-enriched regions within glomeruli were identified based on histologic features and selectively isolated. RNA sequencing was subsequently performed to characterize transcriptomic profiles. Differentially expressed genes (DEGs) were identified through pairwise comparisons, followed by Gene Ontology (GO) enrichment and gene network analyses.
Results : A total of 56 mesangial regions of interest (ROIs) were analyzed (Normal, n=9; DKD, n=16; IgAN, n=31). Differential expression analysis revealed 37 genes upregulated and 461 genes downregulated in IgAN compared with normal controls, indicating global transcriptional changes in IgAN mesangial regions. Notably, several key autophagy-related genes, including ATG7, BECN1, ULK1, and SIRT1, showed reduced expression in IgAN, suggesting impaired autophagy in mesangial cells. Gene network analysis further suggested potential involvement of KRAS-associated signaling linked to the PI3K–AKT–mTOR pathway associated with autophagy regulation. Compared with the disease control group (DKD), autophagy-related genes showed lower expression in IgAN.
Conclusions : This study provides spatially resolved transcriptomic profiling of mesangial cells in IgA nephropathy, revealing suppressed autophagy signatures compared with normal controls and DKD. These findings suggest that altered autophagy-related transcriptional profiles in mesangial cells may represent a molecular feature of IgAN and warrant further investigation.