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IL-10 Signaling in Lupus Nephritis: Implications for Disease Progression

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Objectives : IL-10 is a key immunosuppressive cytokine with therapeutic potential in immune regulation. However, in lupus nephritis (LN), an autoimmune kidney disease, IL-10 signaling paradoxically deviates from its suppressive role, suggesting a need for further investigation.

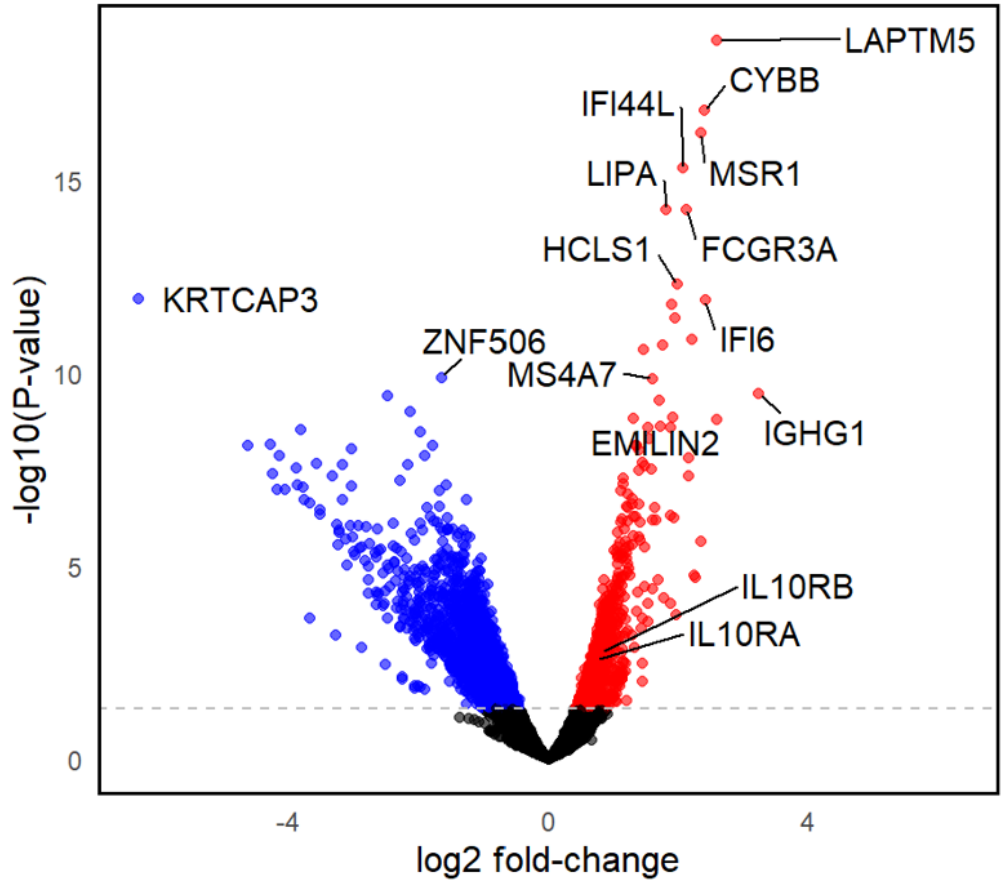
Methods : We performed spatial transcriptomics analysis on kidney tissues from three LN class IV patients with kidney failure, 11 healthy individuals, and 20 other LN patients, including those with class IV LN but with an indolent disease course. Findings from human samples were validated in an *in vivo* model using LN TG mice treated with a target biomarker.

Results : Spatial transcriptomics revealed upregulation of glomerular IL10RA and IL10RB in LN class IV patients who progressed to kidney failure compared to other groups. To explore the mechanisms underlying IL-10's role in poor prognosis, we conducted *in vivo* experiments using TG mice. Mice received intraperitoneal injections of recombinant IL-10 (rIL-10), while a control group was treated with dexamethasone. The rIL-10-treated mice developed severe proteinuria and fibrotic kidney pathology, which was alleviated by dexamethasone treatment. Furthermore, renal expression of IL10RA and IL10RB was upregulated in rIL-10-treated mice. We also observed an increase in granulocytic myeloid-derived suppressor cells (G-MDSCs) in the kidneys of rIL-10-treated mice, which was reduced following dexamethasone treatment. G-MDSCs in IL-10-treated mice exhibited enhanced interactions with diverse kidney cell types. In the spleen, the frequency of plasma cells and plasmablasts increased, while B cell populations declined.

Conclusions : IL-10 is implicated in the exacerbation of LN. G-MDSCs may play a pathogenic role in disease progression, and IL-10 appears to promote B cell differentiation into plasma cells, altering kidney myeloid cell populations. These findings suggest that IL-10 contributes to LN progression through immune modulation in both renal and systemic compartments.

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Normal Control vs LN class IV (kidney failure)



LN_IL10_KSN2025_figure1.png

