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SBP1-associated pathogenesis in IgA nephropathy

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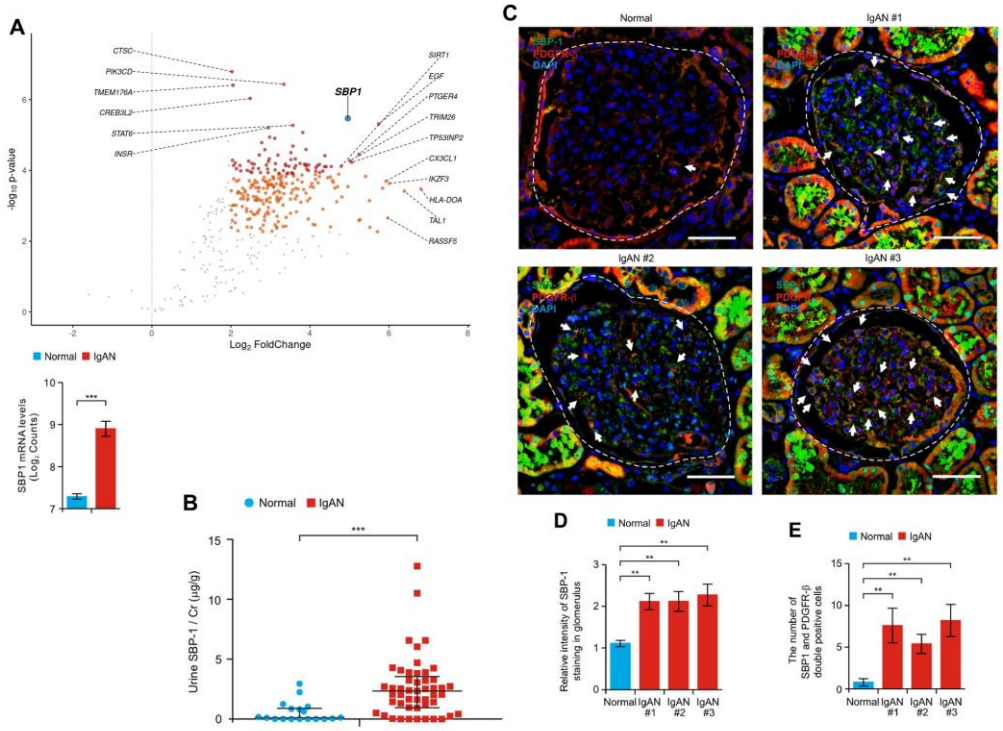
Objectives : Glomerular mesangial cells are important contributors to the early progression of IgA nephropathy (IgAN). Selenium-binding protein 1 (SBP1) is a highly conserved protein that covalently binds selenium, which is important for redox signaling in the endocrine and immune systems. The role of SBP1 has been described in selenium metabolism and redox regulation. We investigated the role of SBP1 in renal mesangial cells during the pathogenesis of IgA nephropathy.
Methods : We used the Nanostring nCounter gene expression profiler to measure the mRNA expression of kidney samples from the normal group and IgA nephropathy group. Also, we measured urine SBP1 levels in both groups with normal and IgA nephropathy. To explore the role of SBP1 in the pathogenesis of IgA nephropathy, we investigated mitochondrial respiration and ATP production in SBP1-overexpressed human renal mesangial cells using a Seahorse XF96e bioanalyzer.
Results : SBP1 mRNA expression was increased alongside other inflammation-associated mRNA in patients with IgA nephropathy. Urine SBP1 levels were also increased in patients with IgA nephropathy. Increased intensity of SBP1 was observed in the immunofluorescence study, alongside the elevated number of colocalization of SBP1 and PDGFR-b, a marker for mesangial cells. SBP1-overexpressed mesangial cells showed increased mitochondrial respiration and ATP production, accompanying increased levels of other pro-inflammatory and proliferative cytokines. These results suggested that SBP1 regulates cellular proliferation and inflammation within glomerular mesangial cells during the pathogenesis of IgA nephropathy.
Conclusions : In summary, our study demonstrated that SBP1 promotes cellular proliferation and pro-inflammatory responses in glomerular mesangial cells, as a molecular mechanism of IgA nephropathy.

Figure1_KSN.png



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