Cytokines found to contribute to IgA nephropathy

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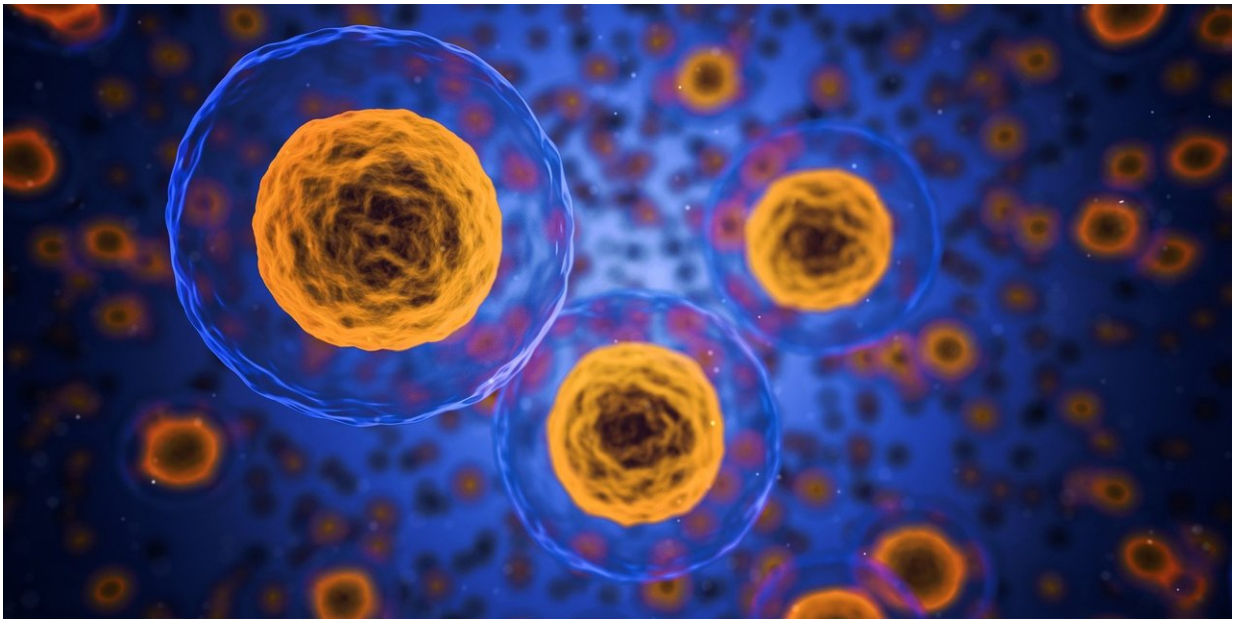

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Some cytokines can enhance the production of aberrantly O-glycosylated IgA1, the main autoantigen in IgA nephropathy. The mechanism behind this observation is described in the *Journal of Interferon & Cytokine Research (JICR)*.

Immunoglobulin A (IgA) nephropathy is the most common primary glomerulonephritis worldwide. There is no disease-specific treatment,

and up to 40% of patients will progress to kidney failure. IgA nephropathy is an autoimmune disease in which the kidneys are injured as a result of circulating immune complexes that contain aberrantly O-glycosylated IgA1 bound by IgG autoantibodies.

Colin Reily, Ph.D., from the University of Alabama at Birmingham, and coauthors, present their view of the pathogenesis of IgA nephropathy and review how abnormal cytokine signaling in IgA1-producing cells can contribute to the disease process. The authors discuss the human IgA structure and glycosylation, genetics of IgA nephropathy, and the role of cytokines in B cell development and differentiation, and cytokines and production of aberrantly glycosylated IgA1. They also provide an update on clinical trials that use some of these cytokines as therapeutic targets and discuss future directions in IgA nephropathy.

"In a thoughtful and comprehensive review, Reily and colleagues discuss how cytokines contribute to the disease process by enhancing the production of aberrantly glycosylated IgA1. The authors also review current clinical trials of IgA nephropathy based on cytokine-targeting therapeutic approaches. This is a must-read article for all researchers in this important area of research," says *Journal of Interferon & Cytokine Research* Editor-in-Chief David L. (Woody) Woodland, Ph.D.

More information: Taylor Person et al, Cytokines and Production of Aberrantly O-Glycosylated IgA1, the Main Autoantigen in IgA Nephropathy, *Journal of Interferon & Cytokine Research* (2022). DOI: 10.1089/jir.2022.0039

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