PRESS RELEASE

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How the Novel Antibody Felzartamab Impacts IgA Nephropathy

Highlights

- Through analyses of patient blood samples, scientists have revealed previously unknown mechanisms behind felzartamab's efficacy and tolerability in IgA nephropathy.
- Results from the study will be presented at ASN Kidney Week 2024 October 23–27.

San Diego, CA (October 25, 2024) — IgA nephropathy (IgAN) is an autoimmune kidney disease driven by immune cells that express a protein called CD38 on their surface. A recent Phase 2 trial revealed that felzartamab, an investigational anti-CD38 monoclonal antibody, helps to reduce proteinuria and maintain patients' kidney function. Investigators evaluated the molecular mechanisms underlying felzartamab's potential efficacy in IgAN. The findings will be presented at ASN Kidney Week 2024 October 23– 27.

It is hypothesized that CD38+ cells contribute to disease through the secretion of galactose-deficient IgA1 (Gd-IgA1) as well as anti-Gd-IgA1 antibodies, which subsequently form immune complexes that deposit in the kidneys and cause inflammation and downstream loss of kidney function.

In their analysis, researchers examined whole-blood and serum collected from patients with IgAN before, during, and after felzartamab treatment. Samples were assessed for immune cells, antibodies, and immunoglobulins including Gd-IgA1.

The analyses revealed that felzartamab induced rapid and durable depletion of Gd-IgA1 and total IgA antibodies. Patients who received 9 doses over a 6-month treatment period maintained Gd-IgA1 reduction for up to 9 months off-treatment. In addition, total IgA reduction was maintained for at least 18 months off-treatment. In contrast, the body's total IgG antibody titers were modestly reduced and recovered within 4 months off-treatment, suggesting the preservation of components of the body's adaptive immune response.

“These data further our understanding of the role of CD38+ antibody secreting cells in IgA nephropathy pathogenesis,” said corresponding author Millie Shah, PhD, Senior Scientist at Biogen. “By directly depleting these cells, felzartamab reduces the cellular drivers of disease and has the potential for durable clinical benefit without continuous dosing, potentially lowering patient burden and offering improved tolerability.”

Study: “Felzartamab durably reduces disease relevant biomarkers through targeting of CD38+ plasma cells and plasmablasts, the upstream drivers of IgA nephropathy (IgAN)”

Join ASN and approximately 12,000 other kidney professionals from across the globe at Kidney Week 2024 in San Diego, CA. The world's premier nephrology meeting, Kidney Week, provides participants with exciting and challenging opportunities to exchange knowledge, learn the latest scientific and medical advances, and listen to engaging and provocative discussions with leading experts in the field. Early programs begin on October 23, followed by the Annual Meeting from October 24-27. Follow the conversation at #KidneyWk.

About ASN
Since 1966, ASN has been leading the fight to prevent, treat, and cure kidney diseases throughout the world by educating health professionals and scientists, advancing research and innovation, communicating new knowledge and advocating for the highest quality care for patients. ASN has nearly 21,000 members representing 140 countries. For more information, visit www.asn-online.org and follow us on Facebook, X, LinkedIn, and Instagram.

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