Biohaven Reports Positive Clinical Biomarker and Patient Data: First MoDE and TRAP Extracellular Protein Degraders Achieve Deep, Rapid, Selective Lowering of Disease-Driving Antibodies in Graves' Disease and IgA Nephropathy

May 27, 2026

- *BHV-1300 demonstrated deep, rapid, and sustained lowering of pathogenic TSHR autoantibodies (TSHR-IgG1) in patients with Graves' hyperthyroidism receiving 1000 mg SC weekly, with mean reductions in TSHR-IgG1exceeding >80% over the 12-week study.*
  - *Participants with Graves' overt hyperthyroidism, confirmed by elevated baseline thyroid tests despite being treated with anti-thyroid drug therapy (ATD), experienced normalization of thyroid hormones within weeks; T4 normalization occurring at a median of 3 weeks and T3 at a median of 5 weeks after the first administration of BHV-1300.*
  - *These preliminary patient data from an ongoing Phase 1b study highlight deep lowering of TSHR-IgG1 with BHV-1300 as a potentially disease-modifying approach to Graves' disease.*
- *BHV-1400 achieved rapid, deep, selective, and sustained lowering of Gd-IgA1 in patients with IgA nephropathy (IgAN), differentiating Biohaven's leading TRAP degrader from the complement and BAFF/APRIL inhibitor competition.*
  - *Participants with IgAN administered BHV-1400 showed a rapid, > 60% mean reduction of Gd-IgA1 within hours, and a 70% mean reduction of within the first one-month of dosing. Observed reductions in Gd-IgA1 were deeper than has been reported with BAFF/APRIL inhibitors, APRIL-inhibitors, and CD38 inhibitors at these early time points.*
  - *Preliminary observations also demonstrated an increase in eGFR, decrease in spot UPCR, and a decrease in hematuria with one month or less of dosing.*
  - *BHV-1400 brings precision immunology to the treatment landscape of IgAN as it was rationally designed to selectively remove galactose-deficient IgA1 (Gd-IgA1), the pathogenic antibody driver of the disease while sparing healthy antibodies IgA, IgG, IgE, and IgM.*
  - *Designed to target the disease driver while preserving healthy immunoglobulins, the complement system, and cell-mediated and humoral immunity, BHV-1400 offers a differentiated approach to current immunosuppressive therapies approved or in development.*
- *Both BHV-1300 and BHV-1400 continue to demonstrate the paradigm shifting potential of its extracellular degrader platform to target pathogenic antibodies causing disease. BHV-1300 and BHV-1400 show a differentiated safety profile with no clinically significant increases in cholesterol, decreases in albumin, or increases in ALT, AST, or bilirubin. Most AEs were mild and self-resolving, there were no drug discontinuations throughout the patient studies, and no serious or severe AEs.*
- *Pivotal Phase 3 trials in Graves' Disease and IgA Nephropathy to initiate in the coming weeks by mid-2026. Both trials use a patient friendly, simple to self-administer autoinjector for BHV-1300 and BHV-1400.*

NEW HAVEN, Conn., May 27, 2026 /PRNewswire/ -- Biohaven Ltd. (NYSE: BHVN) ("Biohaven") a global clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of life-changing therapies with the potential to transform autoimmune diseases today reported compelling results regarding its proprietary extracellular MoDE™ (BHV-1300) and TRAP™ (BHV-1400) degrader platform. The new patient data from its BHV-1300 MoDE degrader for Graves' disease and BHV-1400 TRAP degrader for IgA nephropathy is being presented today at Biohaven's R&D and Analyst Day as part of the 2026 Yale Innovation Summit at the Yale School of Management in New Haven, Connecticut.

Tova Gardin, M.D., Chief Translational Officer of Biohaven, commented, "These new data in patients with Graves' disease and IgAN bridge scientific insights and Phase 1 findings – including pharmacodynamics and safety – to advance the programs into the upcoming Phase 3 studies planned for this summer. We continue to make good on our promise to translate the best of immunoscience into meaningful solutions for patients. Through focused innovation and execution, we have advanced the first MoDE and TRAP extracellular protein degraders, demonstrated their ability to rapidly and deeply reduce the antibodies that drive disease, and are encouraged by the biomarker data as well as early improvements in clinical outcomes noted by patients and investigators. As we enter pivotal trials in 2026 and continue to innovate with breakthrough science and precision tools, these data represent just the beginning of the impact we plan to deliver with our Biohaven proprietary extracellular degrader platform."

Key Highlights from the Phase 1b Graves' Disease Patient Experience with BHV-1300:

- Weekly administration of BHV-1300 1000 mg achieved mean reductions of pathogenic TSHR-IgG1 of >80% by week 12 in patients with Graves' hyperthyroidism (see Figure 1), a deeper reduction in pathogenic TSHR-IgG1 than has been demonstrated with the FcRn inhibitor competition.
- Patients with elevated thyroid hormones experienced a normalization of free T4 at a median of 3 weeks and a normalization of free T3 within a median of 5 weeks (see Figure 2). Patients reported improvements in classic symptoms of

hyperthyroidism including palpitations, fatigue, diarrhea, diaphoresis, tremor, and mood.

- BHV-1300 has been safe and well-tolerated through 12 weeks of dosing. Most AE's have been mild and self-resolving, there have been no drug-discontinuations, and no serious or severe AEs. There have been no clinically significant increases in cholesterol, decreases in albumin, or increases in ALT, AST or bilirubin. Critically, BHV-1300 preserved IgG3 and demonstrated no clinically significant reductions in IgG3, IgA, IgE, or IgM relative to baseline, maintaining the immune protection that FcRn inhibitors — which lower all IgG subclasses including IgG3 — have not demonstrated

Malini Gupta, M.D., ECNU, FACE, Director of G2Endo- Endocrinology in Memphis, the 2025 AACE Thyroid Chair and investigator in the BHV-1300 study, commented, "We are excited by these initial data in patients with Graves' disease, which demonstrate BHV-1300's ability to profoundly reduce TSHR autoantibodies and to normalize thyroid function in patients who have limited options today." Dr. Gupta added, "The strong link between TSHR-IgG1 lowering and clinical response positions BHV-1300 as a potential first-and-best-in-class therapy for Graves' disease. We see substantial unmet need in this population with no current disease modifying therapy and are excited for our patients to participate in the BHV-1300 pivotal Phase 3 study this year."

The positive Phase1b results in patients with Graves' disease who were hyperthyroid despite concurrent treatment with ATDs build on the observed dose-response relationship of BHV-1300 and literature that suggests deeper TSHR-IgG1 reductions are known to drive meaningfully higher efficacy compared to more modest lowering. Additional biomarker and pharmacokinetic data (n=8) regarding Graves' patients who were and were not hyperthyroid at baseline will be presented at the Biohaven R&D Day and will be available on the company's website. Based upon the pharmacokinetic and biomarker results with BHV-1300 in this Phase 1b study, Biohaven is advancing to a pivotal trial in Graves' disease in the coming weeks. The planned study design is a double-blind placebo-controlled study in adults with Graves' hyperthyroidism evaluating normalization of T3, T4, and TSH at 26 weeks absent an antithyroid drug.

Figure 1: BHV-1300 rapidly and robustly reduced TSHR autoantibodies and normalized free T4 and free T3 in consecutively treated hyperthyroid patients (n=3) who were already on ATDs

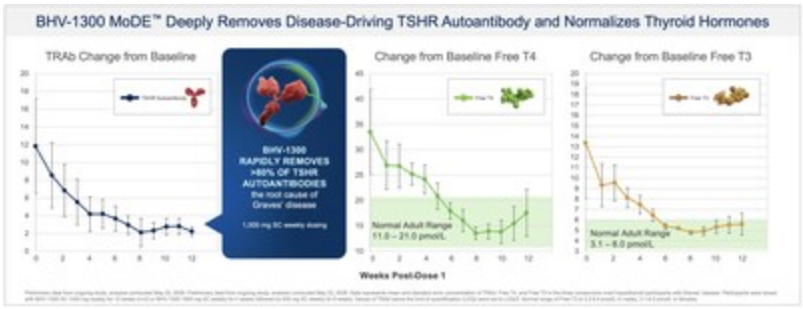
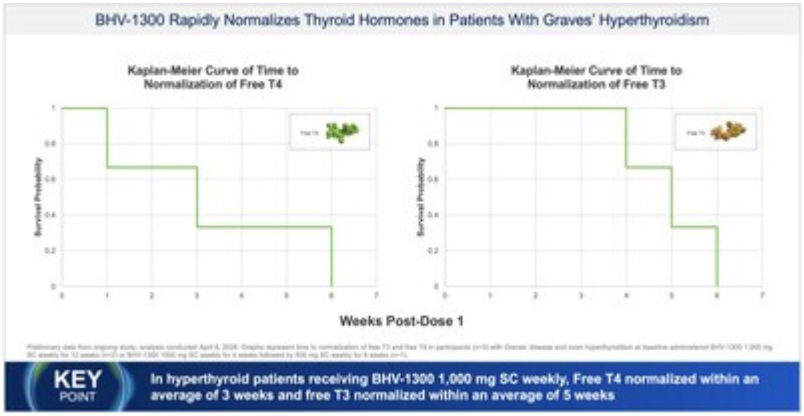


Figure 2: Time to normalization of thyroid hormones after treatment with BHV-1300 in consecutively treated patients (n=3) with hyperthyroidism at baseline despite concurrent use of antithyroid drugs.



Key Highlights from the Phase 1b IgA Nephropathy Disease Patient Experience with BHV-1400:

- Administration of BHV-1400, the Company's first TRAP, galactose-deficient IgA1 (Gd-IgA1) TRAP degrader for the treatment of IgA nephropathy (IgAN) achieved deep, rapid, and sustained mean reductions of pathogenic Gd-IgA1 of >60% within 48 hours of subcutaneous administration and 70% within the one month of dosing in patients with IgAN (see Figure 3). These reductions are deeper than those demonstrated by BAFF-APRIL inhibitors, APRIL inhibitors, and CD38 inhibitors at this early time point.

- Reductions in Gd-IgA1 were associated with resolution of hematuria, decreases in spot UPCR, and increases in eGFR (see Figures 4-6). Participants also subjectively reported improvements in fatigue.

- BHV-1400 has been safe and well-tolerated throughout one month of dosing. Most AE's have been mild and self-resolving, there have been no drug-discontinuations, and no serious or severe AEs. There have been no clinically significant

increases in ALT, AST or bilirubin, and no clinically significant reductions in other immunoglobulins relative to baseline.

- Effects were selective, with no clinically significant reductions observed in other immunoglobulins: IgA, IgG, IgE, or IgM.

Dr. Jonathan Barratt, the Mayer Professor of Renal Medicine at University of Leicester and leading expert in the treatment of IgAN, commented on the new Phase 1b patient data, "These new data, which include data from my own patients, represent the next frontier of innovation for patients with IgA nephropathy. Currently approved treatments broadly suppress the immune system, and disease recurs upon their cessation, necessitating long term, potentially life long, treatment. There is a need for rapid acting and safe and rapid therapies that can be utilized throughout a patient's lifetime. The data I presented today show consistency between Phase 1 healthy subjects and patients with IgAN in BHV-1400's ability to deeply remove pathogenic Gd-IgA1. This reduction in Gd-IgA1 is associated with an immediate impact on patient outcomes rapidly without immunosuppression. I remain excited about BHV-1400, based on the Phase 1 patient data, as it rapidly and specifically targeted the fundamental abnormality in IgA nephropathy while sparing the rest of the immune system. It has the potential to take away the major driver for immune complex formation while leaving other antibodies unaffected, which means it potentially has efficacy with unrivaled safety."

Figure 3: BHV-1400 rapidly and robustly reduced Gd-IgA1 in patient with IgAN.

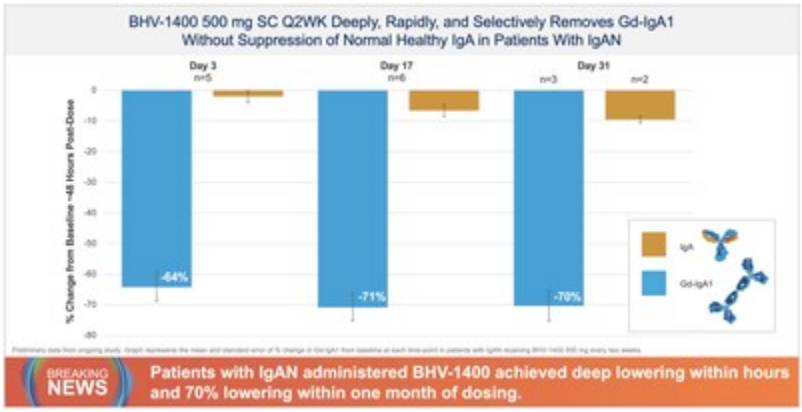
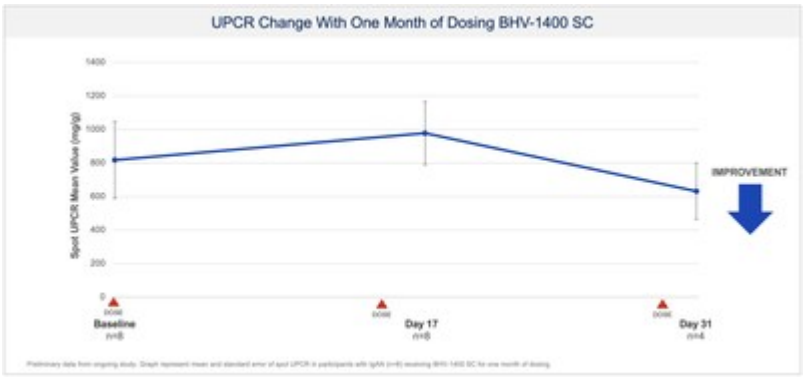


Figure 4: Associated changes to eGFR in IgAN patients administered one month of treatment with BHV-1400.

Figure 5: Associated changes to UPCR in IgAN patients administered one month of treatment with BHV-1400.



Based upon the rapid and deep reductions of Gd-IgA1 observed with subcutaneous BHV-1400, Biohaven plans to study BHV-1400 in patients with IgAN, initiating a pivotal trial in mid-2026 designed to support regulatory approval, evaluating urine protein-creatinine ratio (UPCR) and eGFR at 1 year.

About Graves' Disease

Graves' disease is the most common cause of hyperthyroidism, driven by autoantibodies stimulating the TSH receptor. A relapsing and remitting

condition, Graves' disease affects 1% of the global population.

Driven by an autoantibody (TSHR-IgG1) that binds the TSH receptor, Graves' disease causes multi-organ symptoms and is complicated by associated TSHR-IgG1 driven conditions: thyroid eye disease, neonatal Graves' disease, and pretibial myxedema. Current treatment options are limited to thyroid organ removal or ablation or anti-thyroid drugs, none of which target the underlying autoantibody driving the disease. Standard ATDs do not target the underlying driver of disease and leave patients still symptomatic and at risk for development of thyroid eye disease and pretibial myxedema. Among patients with Graves' disease recently exposed to antithyroid drugs, 93% report multiple ongoing symptoms, with 72% reporting five or more persistent symptoms. With no new FDA approved therapy in over seventy years, there is a high unmet need for a novel therapy targeting the root cause of disease.

Biohaven's Lead MoDE degrader, BHV-1300, is poised to advance into its pivotal trial in Graves' disease mid-2026.

The presentation slides from Biohaven's R&D Day for the TRAP and MoDE degraders and its other platforms will be available on the Events and Presentations page of the Biohaven website just prior to their presentations.

About BHV-1300

BHV-1300, first MoDE, is a small molecule, extracellular IgG degrader, rationally designed to leverage the body's natural hepatic clearance mechanisms to selectively target and remove IgG1, IgG2, and IgG4, the underlying cause of many immune-mediated diseases. BHV-1300 is mechanistically differentiated from FcRn inhibitors in multiple important ways: first, it selectively targets the disease-causing IgG subclasses (IgG1, IgG2, IgG4) while sparing IgG3, which mediates protection against bacteria, viruses, and parasites and which has not been demonstrated with FcRn inhibitors; second, as a small molecule, BHV-1300 does not interact with FcRn and therefore does not accelerate the clearance of co-administered Fc-containing biologic therapies — a meaningful clinical limitation of the FcRn inhibitor class; third, it avoids the off-target effects of cholesterol elevation, albumin reduction, and headache associated with first-generation FcRn inhibitors; and fourth, it is delivered in a convenient self-administered autoinjector.

About IgA Nephropathy

IgA nephropathy is the leading cause of glomerular disease globally and is commonly diagnosed in individuals in their second and third decades of life, with most individuals progressing to renal failure over the ensuing 10-15 years. As a disease of the immune system, IgA nephropathy frequently returns even after renal transplant. While the 2021 Kidney Disease Improving Global Outcomes (KDIGO) treatment guidelines recommended only standard chronic kidney disease treatments, the 2025 guidelines emphasize the importance of treating the underlying immune disease by removing aberrant forms of IgA. "Galactose deficient IgA1 is the fundamental abnormality in IgA nephropathy," Dr. Barratt explained, "It's a group of IgA molecules that have changes to the sugars on the IgA1 hinge region that fundamentally change the way this antibody behaves. It promotes immune complex formation and it's these immune complexes that cause glomerular injury and damage and promote loss of kidney function."

About BHV-1400

BHV-1400, Biohaven's lead TRAP extracellular degrader, rapidly and selectively removes Gd-IgA1, the fundamental abnormality in IgA nephropathy, without adversely affecting any other immunoglobulins or components of protective immunity. BHV-1400's precision approach is differentiated from all existing and investigational therapies for IgAN: complement inhibitors, BAFF/APRIL inhibitors and non-selective inhibitors of IgA all suppress immunity. BHV-1400 has been safe and well-tolerated across the ongoing Phase 1b study in IgAN patients. Most adverse events (AEs) were mild and self-resolving, there were no discontinuations due to AEs related to study drug, and there were no serious or severe AEs related to drug. There were no clinically significant increases in ALT, AST or bilirubin, no clinically significant reductions in albumin and no clinically significant increases in cholesterol relative to placebo over the one month dosing period. There were no clinically significant reductions in other immunoglobulins including IgG, IgA, IgE, or IgM relative to baseline.

About Biohaven

Biohaven is a biopharmaceutical company focused on the discovery, development, and commercialization of life-changing treatments in key therapeutic areas, including immunology, neuroscience, and oncology. The Company is advancing its innovative portfolio of therapeutics, leveraging its proven drug development experience and multiple proprietary drug development platforms. Biohaven's extensive clinical and preclinical programs include Kv7 ion channel modulation for epilepsy; MoDE and TRAP extracellular protein degradation for immunological diseases; and myostatin inhibition for neuromuscular and metabolic diseases, including obesity. For more information, visit www.biohavenpharma.com.

Forward-Looking Statements

This news release includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. The use of certain words, including "continue", "plan", "will", "believe", "may", "expect", "potentially", "groundbreaking" and similar expressions, is intended to identify forward-looking statements. Investors are cautioned that any forward-looking statements, including statements regarding the future development, timing to and potential marketing approval and commercialization of development candidates, are not guarantees of future performance or results and involve substantial risks and uncertainties. Actual results, developments and events may differ materially from those in the forward-looking statements as a result of various factors including: the expected timing, commencement and outcomes of Biohaven's planned and ongoing clinical trials, including the studies of BHV-1300 and BHV-1400; the timing of planned interactions and filings with the FDA; the timing and outcome of expected regulatory filings; complying with applicable US regulatory requirements; the potential commercialization of Biohaven's product candidates; and the effectiveness and safety of Biohaven's product candidates. Additional important factors to be considered in connection with forward-looking statements are described in Biohaven's filings with the Securities and Exchange Commission, including within the sections titled "Risk Factors" and "Management's Discussion and Analysis of Financial Condition and Results of Operations". The forward-looking statements are made as of the date of this news release, and Biohaven does not undertake any obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

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