Biohaven Drives New Research in Graves' Disease: Phase 3 Pivotal Trial with BHV-1300 Underway and Three Scientific Posters at the 2026 American Thyroid Association Annual (ATA) Meeting

November 5, 2026

- Biohaven presents three posters at ATA, highlighting the patient experience, autoimmune biology of disease, and MoDE and TRAP platform, reflecting a research program built around a condition that remains strikingly understudied relative to its prevalence.
- No new therapy has been approved for Graves' disease in more than seventy years. Antithyroid drugs, radioactive iodine and thyroidectomy manage thyroid hormone output; none removes the autoantibody that causes the disease.
- Biohaven's Phase 3 trial of BHV-1300 in Graves' disease (NCT07661056) is enrolling at more than 55 active sites in the United States and Canada, with additional sites opening in Japan, UK, Australia, and EU. BHV-1300 is the first MoDE extracellular protein degrader to reach a pivotal study.
- In a Phase 1b study, weekly subcutaneous BHV-1300 1000 mg produced mean reductions in pathogenic anti-TSHR-IgG1 autoantibodies of approximately 80% by week 12, with normalization of free T4 at a median of 3 weeks and free T3 at a median of 5 weeks in patients who were hyperthyroid despite antithyroid drug therapy, while sparing IgG3.
- A pivotal trial, a key collaboration expanding the technology into Asia, and three scientific posters at ATA underscore the company's progress in advancing its Graves' disease program—from molecular science and autoimmune biology to the patient experience—in a disease that has seen no new therapy in seventy years.

NEW HAVEN, Conn., Nov. 5, 2026 /PRNewswire/ -- Biohaven Ltd. (NYSE: BHVN) (Biohaven) today announced continued progress across its Graves' disease program, including active enrollment in the pivotal Phase 3 trial of BHV-1300 and three scientific presentations at the 2026 American Thyroid Association (ATA) Annual Meeting in Philadelphia, Pennsylvania. Together they describe a program that extends from the molecule in pivotal testing, to the patient experience that defines the need for it, to the underlying autoantibody biology that Biohaven continues to characterize.

A Pivotal Trial Advances the Science for Patients

Graves' disease is diagnosed and driven by autoantibodies against the thyroid-stimulating hormone receptor (TSHR). Classical immunology work established that thyroid-stimulating activity in patients with Graves' disease is largely confined to the IgG1 subclass, in contrast to the unrestricted subclass distribution of other thyroid autoantibodies. BHV-1300 was selected for this disease on exactly that basis. As a small-molecule degrader of IgG1, IgG2 and IgG4, it engages the body's own hepatic clearance machinery to remove the pathogenic subclass that carries the disease-driving activity, while sparing IgG3, which mediates protection against bacteria, viruses and parasites.

BHV-1300 is built on Biohaven's MoDE™ extracellular protein degrader technology, exclusively licensed from Yale University, where it originated in the laboratory of David Spiegel, M.D., Ph.D. The principle is subtraction, not suppression: a MoDE extracellular protein degrader is designed to remove the disease-causing protein from the body rather than suppress its production or block its activity. One end of the bifunctional small molecule binds the target in circulation, and the other engages the asialoglycoprotein receptor (ASGPR), which is abundant on liver cells and is part of the body's natural system for clearing proteins from circulation. The target is then taken up by the liver cell and degraded in the lysosome, while ASGPR recycles to the cell surface. Dr. Spiegel was the first to patent targeted extracellular protein degraders that use ASGPR. Biohaven is pioneering the clinical use of an extracellular protein degrader that pairs a targeting mechanism with ASGPR in the liver to remove a disease-causing target in patients.

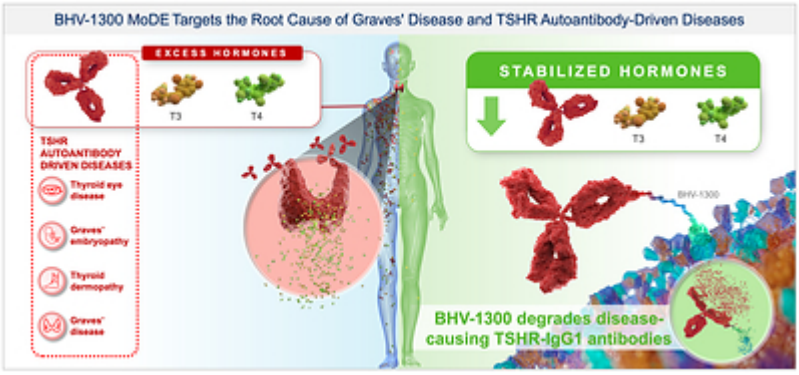


Figure 1. BHV-1300 degrades the disease-causing TSHR-IgG1 autoantibody that drives Graves' disease, stabilizing thyroid hormone levels and targeting the root autoimmune cause of Graves' disease and associated TSHR autoantibody-driven conditions, including thyroid eye disease, thyroid dermopathy and Graves' embryopathy.

That design has translated to patients. In a Phase 1b study, BHV-1300 lowered TSHR-IgG1 by approximately 80% by week 12 and normalized free T4 at a median of 3 weeks and free T3 at a median of 5 weeks in individuals who remained hyperthyroid on antithyroid drug therapy, while preserving IgG3, IgA, IgM and IgE. Nearly 200 individuals have now received MoDE and TRAP degraders in Phase 1 testing, with favorable tolerability and most adverse events mild and self-resolving.

The Phase 3 study is a randomized, double-blind, placebo-controlled trial in approximately 300 adults, evaluating restoration of normal thyroid function at week 26 in the absence of an antithyroid drug. It is open at more than 55 sites across the United States and Canada, enrollment is underway, and additional sites are activating through the fourth quarter of 2026 and the first quarter of 2027.

Extending the Reach of the Technology

In October 2026, Ono Pharmaceutical licensed the MoDE pan IgG degrader portfolio, led by BHV-1300 and including next-generation degraders BHV-1310 and BHV-1320, in Japan, South Korea, Taiwan and ASEAN. Biohaven received $80 million upfront, is eligible for a further $20 million on a milestone anticipated within a year, and will receive royalties of approximately twenty percent on net sales in the territory. Biohaven retains all rights outside the licensed territory, including the United States, Europe and China, and retains worldwide rights to its TRAP™ degraders. The collaboration carries extracellular protein degradation to patients in markets Biohaven does not serve directly, and the proceeds are reinvested in the research engine behind it.

Documenting the Burden of Graves' Disease and the Lived Experience of Patients With the Disease

Graves' disease affects roughly 1% of the population and is four to ten times more common in women than in men, yet the lived experience remains understudied. Biohaven conducted a patient interview study, designed to elicit the burden of disease, in adults with Graves' disease who had been treated with an antithyroid drug within the prior two years and had not undergone radioactive iodine ablation or thyroidectomy.

A series of semi-structured interviews were conducted, and findings were coded into symptom and impact concepts. Symptoms were highly concurrant: 13 of 14 participants (93%) reported two or more symptoms at once, 10 (71%) reported five or more, and one reported 11, spanning cardiac, constitutional, ocular, neuropsychiatric, gastrointestinal and dermatologic systems in the same individuals. Fatigue was most common at 93%. Even among the 8 participants (57%) with well controlled thyroid levels all had symptoms, and 6 described them as recurring or persistent. Every participant (100%) reported an emotional impact, most often anxiety (64%), depression (57%) and effects on self-image (43%).

Using the same interview data, reported symptoms and impacts were mapped against the ThyPRO-39 short form, item by item, to assess the coverage of this scale developed for a range of benign thyroid disorders to the specific experiences of patients with Graves' Disease. Concepts mapped to 31 of 38 items and every scale had at least one patient endorsed item, supporting its patient-centricity in this population.

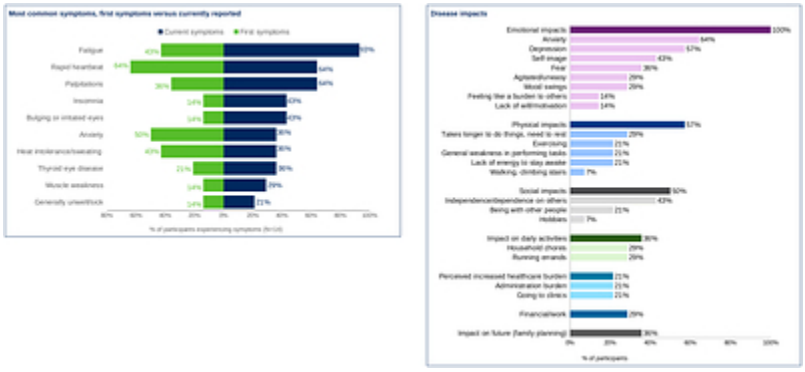


Figure 2. Most common symptoms reported by participants with Graves' disease, first symptoms versus currently reported (left), and disease impacts across emotional, physical, social, daily activity, healthcare, financial and family planning domains (right). N=14.

"Patients told us something our standard measures may not have fully captured. The people who said their thyroid levels were well controlled were still describing fatigue, palpitations, anxiety and effects on how they see themselves," said Michele Potashman, Ph.D., Head of Clinical Outcomes Assessments and Health Economics at Biohaven. "Biochemical control is not the same as feeling well. What these interviews make clear is how much the daily lives of patients are still impacted while their disease is considered managed". As the autoantibody that drives Graves' disease may still be present in patients who have achieved biochemical control, therapies that address the direct cause of the disease are of great interest to patients.

Characterizing the Autoantibody Biology

Alongside the clinical and patient-experience work, Biohaven continues to investigate the autoantibody biology underlying Graves' disease and its associated conditions. The third poster presents the preclinical characterization of BHV-1440, a TRAP degrader engineered to bind anti-TSHR autoantibodies and direct them to ASGPR for lysosomal degradation.

BHV-1440 bound the patient-derived anti-TSHR antibody M22 and ASGPR simultaneously, inducing formation of a ternary complex and driving internalization of M22 in primary human hepatocytes across three donors with an average EC50 of 0.73 nM. In a mouse passive-transfer model, a single subcutaneous dose of BHV-1440 reduced circulating M22 within two hours and returned elevated T4 to baseline levels by 24 hours. The tissue findings are the most informative: within 30 minutes of dosing, anti-TSHR antibody signal fell in thyroid tissue while hepatic signal rose and colocalized with the lysosomal marker LAMP1, tracing the antibody from its site of action to its site of disposal. Moreover, BHV-1440 restored follicular histology toward normal. In patient samples, a biotinylated BHV-1440 analog depleted anti-TSHR IgG to the levels seen in healthy controls.

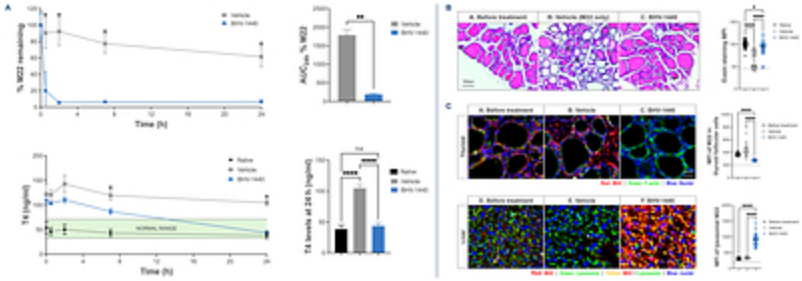


Figure 3. Preclinical activity of BHV-1440. (A) A single subcutaneous dose rapidly depletes circulating anti-TSHR antibody M22 and normalizes T4 levels over 24 hours. (B) Subcutaneous BHV-1440 restores eosin staining of follicular lumens and normalizes thyroid histology. (C) BHV-1440 reduces M22 signal in thyroid tissue with a corresponding increase in hepatic lysosomal M22.

"Graves' disease has gone more than seventy years without a new approved therapy, despite affecting millions of patients. We are committed to changing that with a comprehensive strategy that elevates the science and the patient experience," said Tova Gardin, M.D., MPP, Chief Translational Officer of Biohaven. "We are advancing a pivotal trial of an investigational therapy designed to address the pathogenic autoantibody at the root of the disease, while simultaneously advancing research to better define the patient experience, strengthen how it is measured, and deepen our understanding of the underlying biology. Our goal is to deliver meaningful progress for a patient community that has waited far too long."

Presentation Details

Poster 1	The Under-Recognized Burden of Graves' Disease: Insights Obtained from Participant Interviews. Presenting author: Michele Potashman, Ph.D., Biohaven.
Poster 2	Qualitative Interview-Based Validation of the ThyPRO-39 Short Form in Graves' Disease: Assessing the Coverage of Signs, Symptoms, and Impacts. Presenting author: Michele Potashman, Ph.D., Biohaven.
Poster 3	BHV-1440: Novel TRAP™ Degrader for the Treatment of Graves' Disease Selectively Removes TSHR Autoantibodies from Circulation and Thyroid Tissue, Restoring Normal Thyroid Function in Preclinical Models of Graves' Disease. Presenting author: Mariano Oppikofer, Ph.D., Biohaven.
Meeting	2026 ATA Annual Meeting, November 4 to 7, 2026
Location	Philadelphia, Pennsylvania

About Graves' Disease

Graves' disease is the most common cause of hyperthyroidism and is driven by autoantibodies that stimulate the TSH receptor. It affects roughly 1% of the global population and is four to ten times more common in women than in men. The same autoantibody biology underlies associated conditions including thyroid eye disease, neonatal Graves' disease and pretibial myxedema. Antithyroid drugs have been the mainstay of treatment for more than seventy years; radioactive iodine and thyroidectomy commit patients to lifelong hypothyroidism management. None of these options targets the autoantibody that causes the disease, and relapse after antithyroid drug withdrawal is common and tracks with residual TSH receptor antibody levels.

About BHV-1300

BHV-1300 is an investigational small molecule and potential first-in-class extracellular IgG degrader, rationally designed to leverage the body's natural hepatic clearance mechanisms to selectively target and remove IgG1, IgG2 and IgG4, the principal drivers of many immune-mediated diseases. BHV-1300 spares IgG3 to preserve patient immune protection against bacteria, viruses and parasites, does not accelerate clearance of co-administered biologic therapies, and avoids the class effects of cholesterol elevation, albumin reduction and headache associated with FcRn inhibitors. In ongoing Phase 1 studies, subcutaneously administered BHV-1300 produced rapid, deep and sustained reductions in total IgG of up to 87%, with a favorable safety and tolerability profile observed to date, and demonstrated tunable dosing suitable for both acute and chronic disease management. BHV-1300 is designed for convenient self-administration via a patient-friendly autoinjector and is currently being evaluated in a pivotal Phase 3 trial in Graves' disease, with additional programs being pursued in other autoimmune conditions.

About BHV-1440

BHV-1440 is an investigational TRAP degrader designed to selectively bind anti-TSHR autoantibodies and direct them to liver cells for lysosomal degradation. BHV-1440 is in preclinical research, has not been approved by any regulator, and its safety and efficacy have not been established.

About the MoDE and TRAP Degrader Platforms

Originating from research in the laboratory of Dr. David Spiegel at Yale University, Biohaven's MoDE platform is the first clinically validated technology enabling the targeted degradation of disease-causing proteins in the extracellular space. Each MoDE is a bispecific small molecule that binds a pathogenic target and directs it to the liver through the asialoglycoprotein receptor (ASGPR) for rapid and deep degradation via the body's own endosomal and lysosomal pathways. This approach offers a differentiated profile relative to FcRn-targeting antibodies, including rapid onset of action, deep and tunable lowering, patient-friendly subcutaneous self-administration, and the potential to be combined with standard-of-care biologic therapies. Biohaven's next-generation TRAP degraders extend the platform with highly selective molecules, each engineered to remove a single specific disease-causing protein. Together, the MoDE and TRAP platforms form the foundation of a broad, diverse and rapidly expanding pipeline of

precision immunology therapeutics with the potential to address a wide range of immune-mediated and other diseases.

About Biohaven

Biohaven Ltd. (NYSE: BHVN) is a biopharmaceutical company focused on the discovery, development, and commercialization of life-changing treatments across immunology, neuroscience, and oncology. Biohaven is advancing an innovative portfolio of therapeutics built on proven drug development experience and multiple proprietary platforms, including Kv7 ion channel modulation for epilepsy; MoDE and TRAP extracellular protein degradation for immunological diseases; next generation ADC platform for oncology; and myostatin inhibition for neuromuscular and metabolic diseases, including obesity. For more information, visit www.biohaven.com.

Forward-Looking Statements

This news release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including statements about the potential of BHV-1300, BHV-1440 and other MoDE and TRAP degraders, the conduct, timing, site activation, enrollment and outcome of the Phase 3 trial of BHV-1300 in Graves' disease, the development and commercialization of Biohaven's product candidates, the anticipated benefits of the collaboration with Ono Pharmaceutical including the receipt of milestone and royalty payments, and the future development of Biohaven's degrader programs. Words such as "designed to," "intends," "potential," "believe," "may," "will" and "continue" identify forward-looking statements. These statements are not guarantees of future performance and involve risks and uncertainties, including whether planned clinical sites activate on the anticipated timeline, whether enrollment proceeds as expected, whether preclinical results will be reproduced in humans, whether early clinical findings will be confirmed in larger controlled trials, the outcomes of clinical trials, decisions by the U.S. Food and Drug Administration and other regulators, the availability of funding and partners, and the other risks described in Biohaven's filings with the U.S. Securities and Exchange Commission, including the sections titled "Risk Factors." Qualitative interview studies described in this release were conducted in a small, self-reported sample and are not designed to estimate prevalence or to support efficacy claims. Forward-looking statements speak only as of the date of this release, and Biohaven undertakes no obligation to update them except as required by law.

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