biohaven®

BHVN
LISTED
NYSE

DAYS MATTER™

Corporate Presentation



January 2026

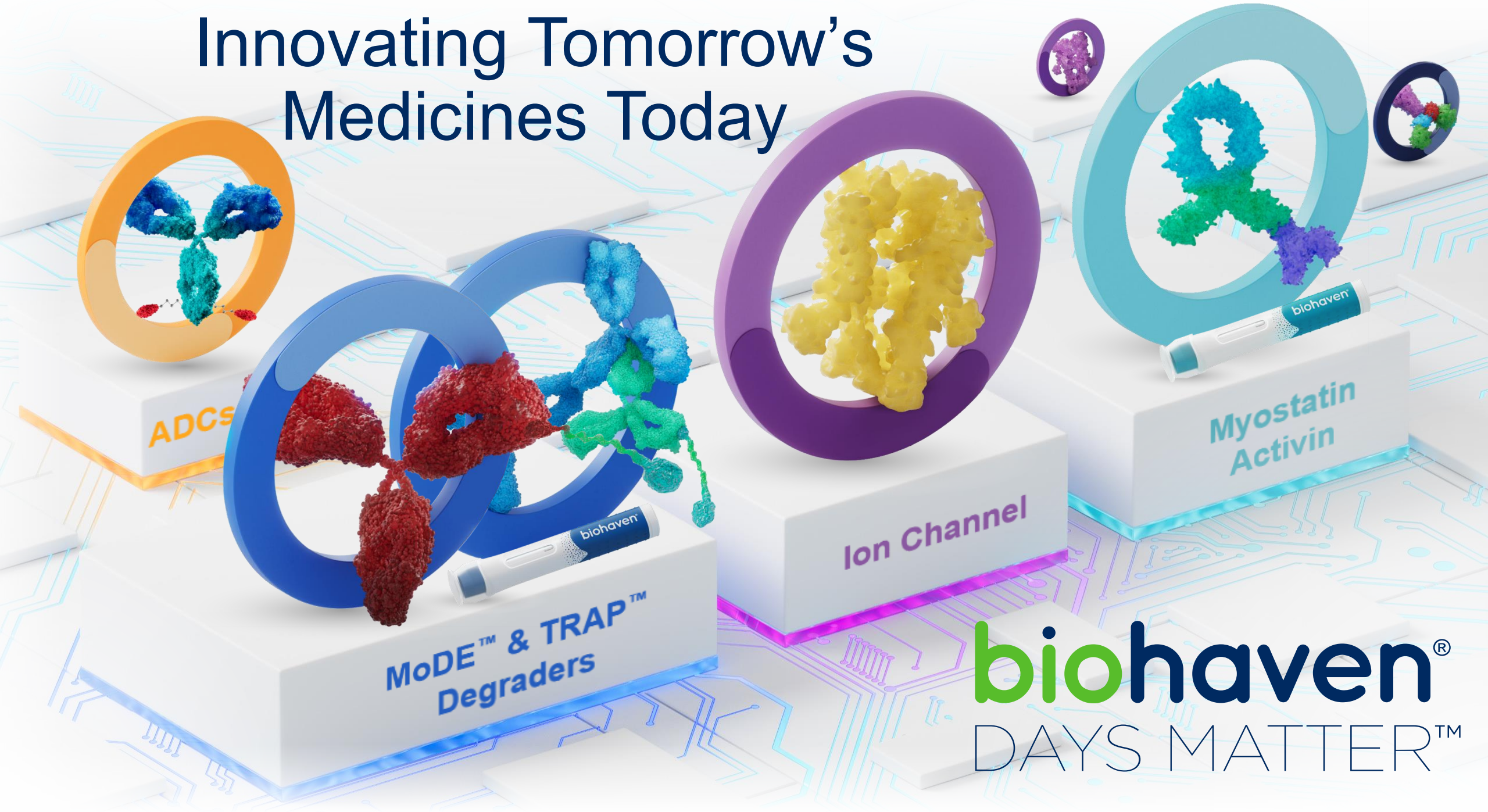
© 2026 Biohaven, Ltd. All rights reserved.

Forward-Looking Statement

This presentation includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, including statements about Biohaven Ltd. (the “Company”) and our planned and ongoing trials (including those for our taldefgrobep alfa, opakalim, BHV-2100, BHV-8000, BHV-1300, BHV-1400, BHV-1510 and BHV-1600 development programs), the timing of and the availability of data from our clinical trials, the timing and our decisions to proceed with our planned regulatory filings, the timing of and our ability to obtain regulatory approvals for our product candidates, the clinical potential utility of our product candidates, alone and as compared to other existing potential treatment options, and the potential advancement of our early phase programs including BHV-1310, BHV-1530 and BHV-1500. The use of certain words, including “continue”, “plan”, “will”, “believe”, “may”, “expect”, “anticipate” 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 and potential marketing approval and commercialization of our 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; the timing of planned interactions and filings with the Food and Drug Administration, including those regarding the resubmission of our new drug application for troriluzole for SCA; the timing and outcome of expected regulatory filings; complying with applicable U.S. regulatory requirements; the potential commercialization of Biohaven's product candidates; the potential for Biohaven’s product candidates to be first-in-class, best-in-class, best-in-clinic or best-in-category therapies; and the effectiveness and safety of Biohaven's product candidates, including open label clinical data in ongoing studies. You should, therefore, not rely on these forward-looking statements as representing our views as of any date subsequent to the date of this presentation. Additional important factors to be considered in connection with forward-looking statements are described in the Company’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”. This presentation also contains market data and other information based on industry publications, reports by market research firms or published independent sources. Some market data and information are also based on the Company’s good faith estimates, which are derived from management’s knowledge of its industry and such independent sources referred to above.

All images are actors unless otherwise noted. Biohaven is a registered trademark, and MoDE, TRAP and Days Matter are trademarks of Biohaven Therapeutics Ltd.

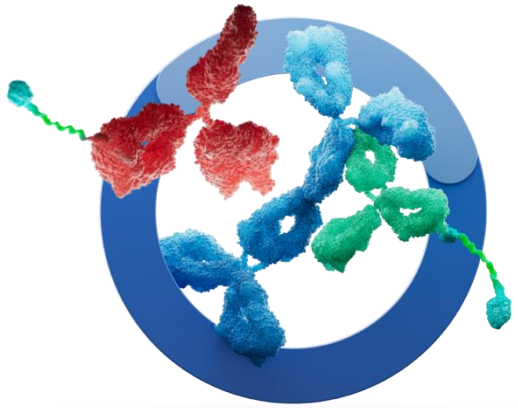
Innovating Tomorrow's Medicines Today



biohaven®

DAYS MATTER™

Diversified platforms delivering multiple innovative therapies because DAYS MATTER™



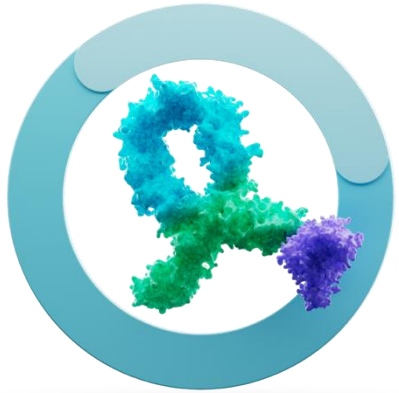
DEGRADERS

Selectively removing what harms while preserving what heals



ION CHANNEL

A modern Kv7 activator that offers efficacy without burdensome CNS side effects



MYOSTATIN ACTIVIN

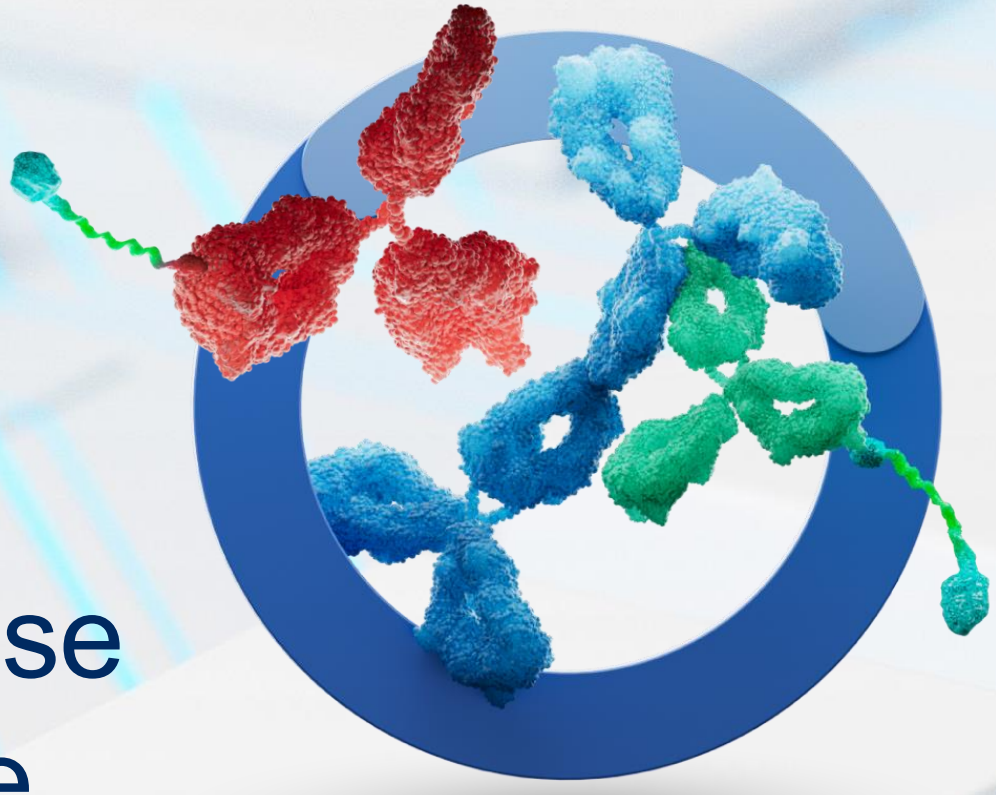
Targeting high quality weight loss, preserving muscle mass and reducing fat

Created a durable engine, built to scale, for near-term catalysts and long-term value

biohaven®

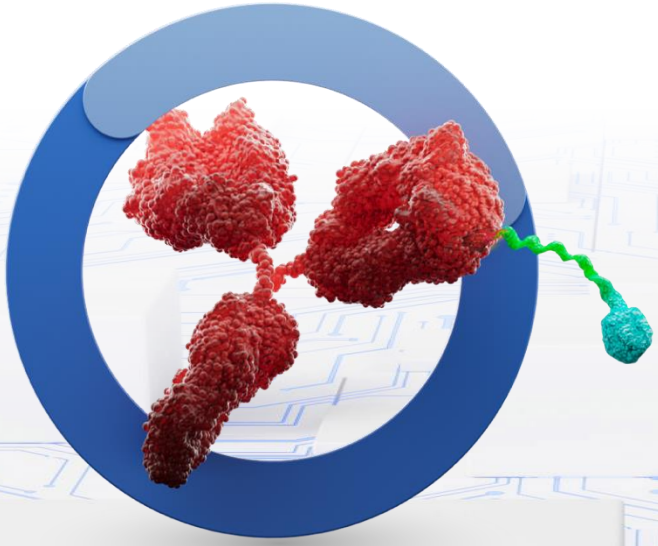
DEGRADERS:
MoDE™ AND TRAP™

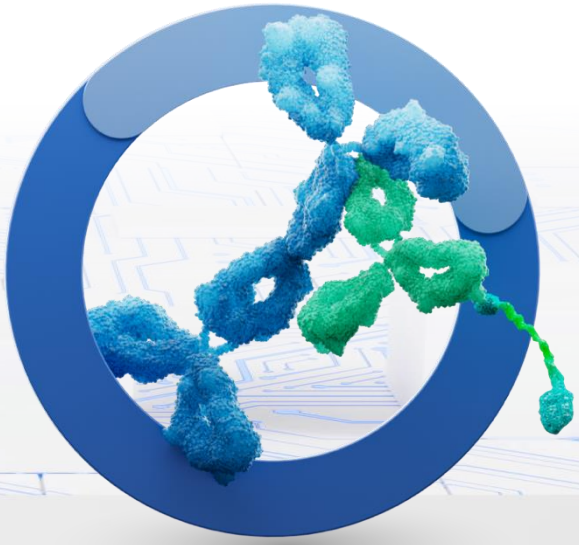
Targeting the Root Cause of Autoimmune Disease



OUR NOVEL DEGRADER PLATFORM

Targeting the Root Cause of Autoimmune Disease





MoDE™

Target a **class of proteins** implicated in pathogenesis of disease

TRAP™

Remove **specific disease-causing proteins and** leave the rest of immune system intact

KEY POINT

Revolutionary Yale-licensed technology to remove disease-causing proteins from the body

MoDE™ and TRAP™ Degraders: Pioneering the First and Only Extracellular Degraders in the Clinic

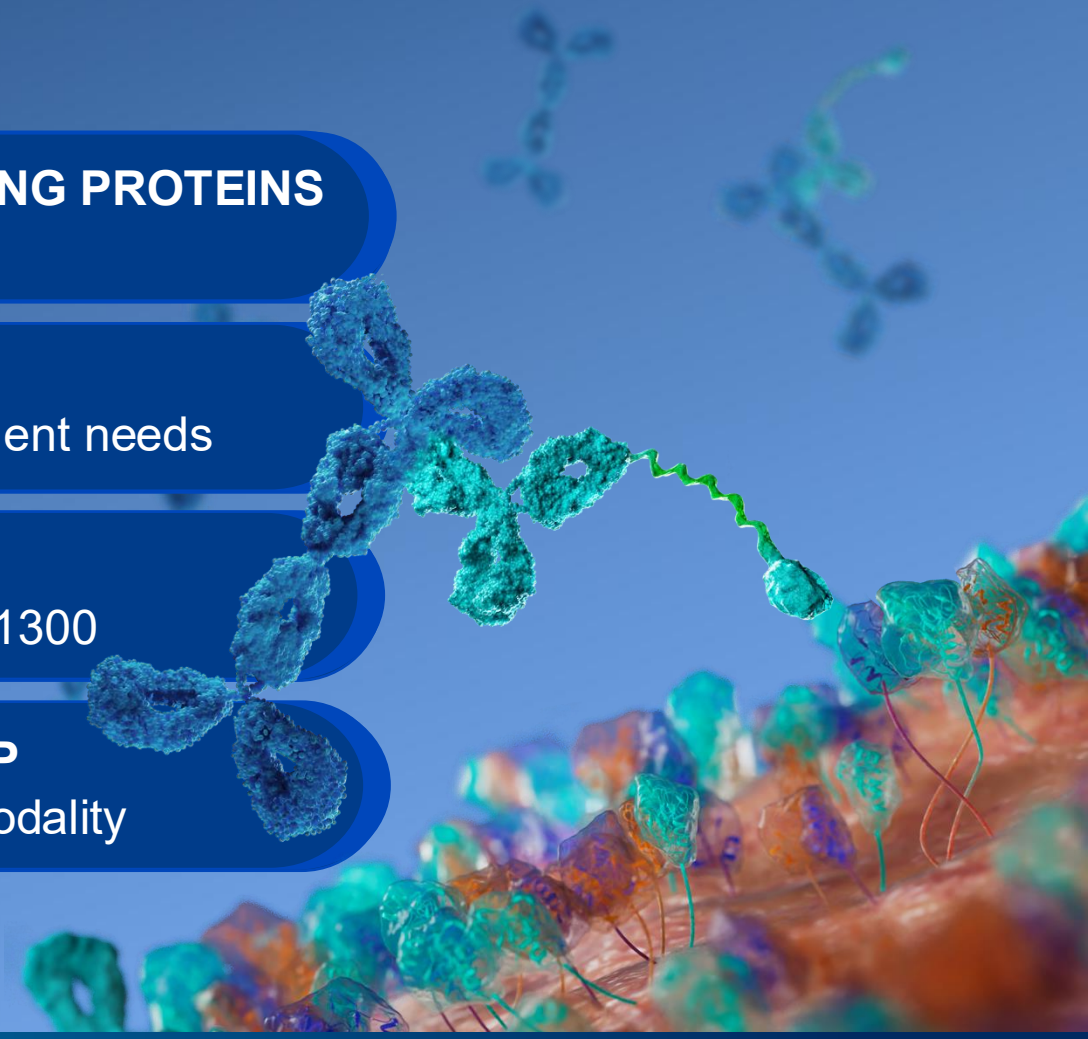


HIGHLY SELECTIVE TARGETING OF DISEASE-DRIVING PROTEINS
Validated in the clinic: Safe and well-tolerated

PATIENT FRIENDLY ADMINISTRATION
Easy-to-use autoinjector; rapid, selective, tunable for patient needs

POSITIONED TO INITIATE PIVOTAL TRIALS 2026
IgAN lead indication for BHV-1400 and Graves' for BHV-1300

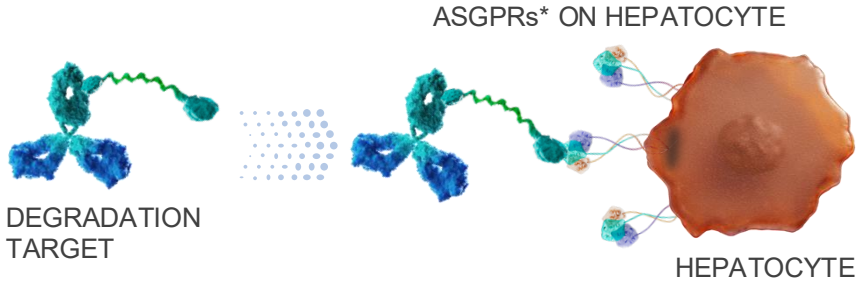
SCALABLE TO MULTIPLE TARGETS AND STRONG IP
Biohaven leads in technology and IP position with this modality



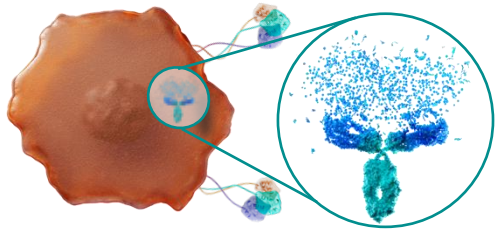
Innovation in Protein Degradation: Harnessing Hepatic ASGPR for Efficient and Safe Removal of Circulating Pathogenic Targets

DEGRADERS

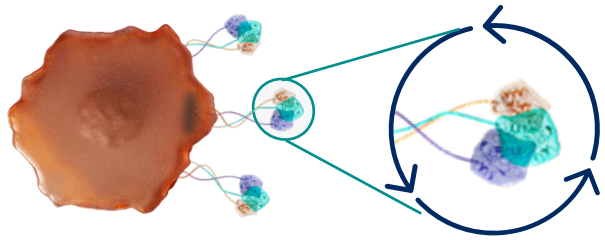
1. MoDE™ and TRAP™ degraders bind circulating target and efficiently deliver to ASGPR on hepatocytes



2. Internalized target is rapidly degraded in hepatic lysosomes



3. ASGPRs are rapidly recycled



BIFUNCTIONAL MoDE DEGRADERS



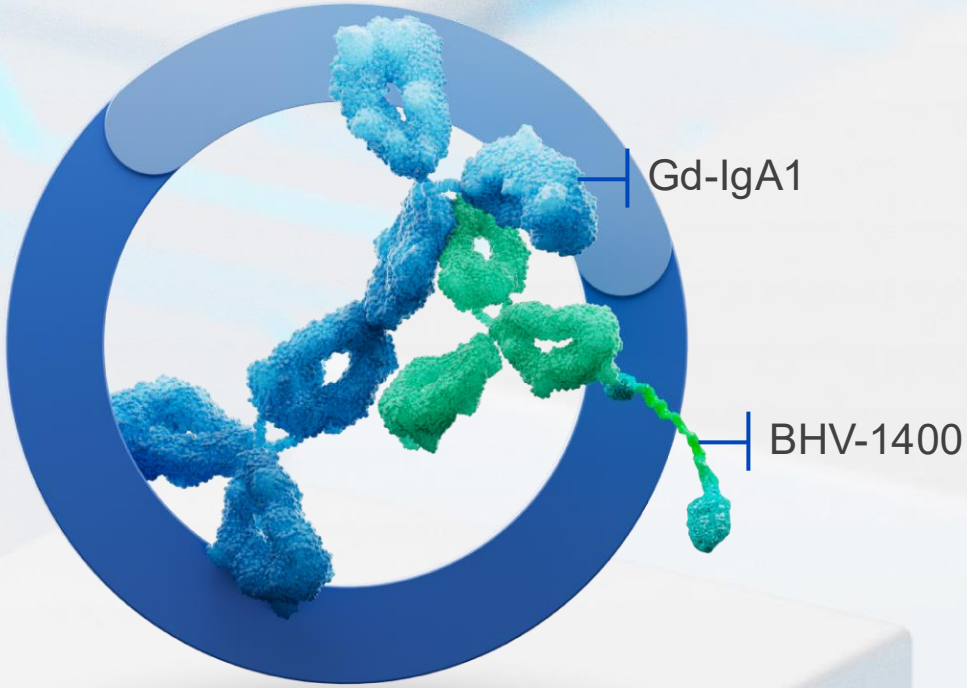
MoDE and TRAP optimized for patient friendly **autoinjector delivery**

*Stylistic representation
ASGPR, asialoglycoprotein receptor

biohaven®

DEGRADERS:
BHV-1400 TRAP™

IgA Nephropathy (IgAN)



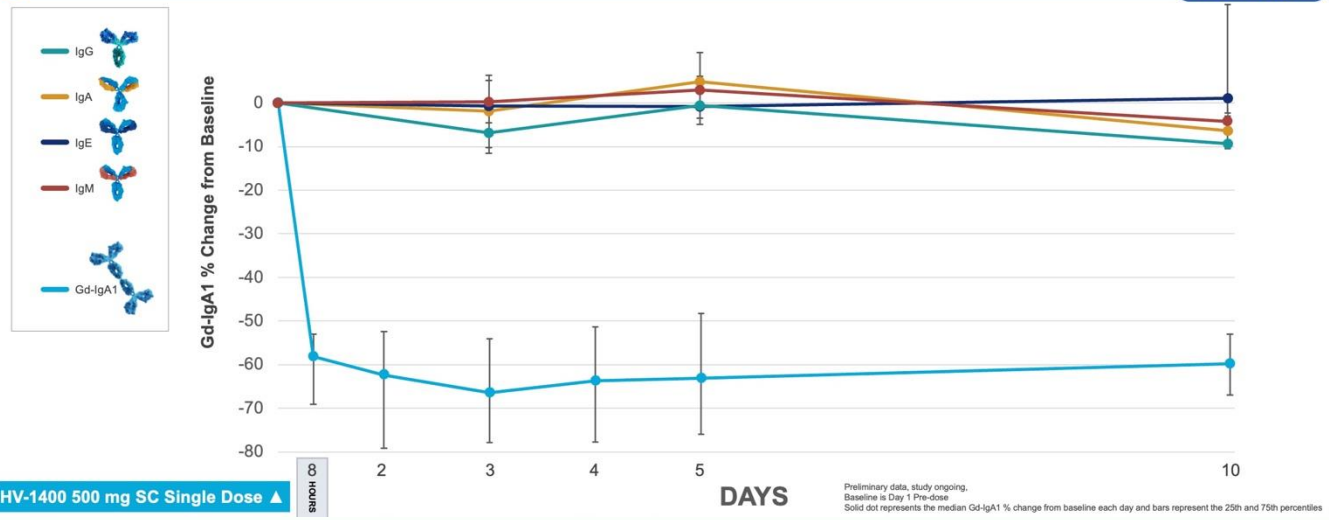
Potential of BHV-1400 in IgAN Recognized by Nephrology Community

DEGRADERS

The future? IgA Degradation

BHV-1400: Single Subcutaneous Dose Delivers *Rapid, Selective, Deep and Sustained* Removal of Gd-IgA1

DEGRADERS



https://ir.biohaven.com/news-releases/news-release-details/biohaven-highlights-innovation-and-advancement-across-mode-and



KIDNEY WEEK 2025

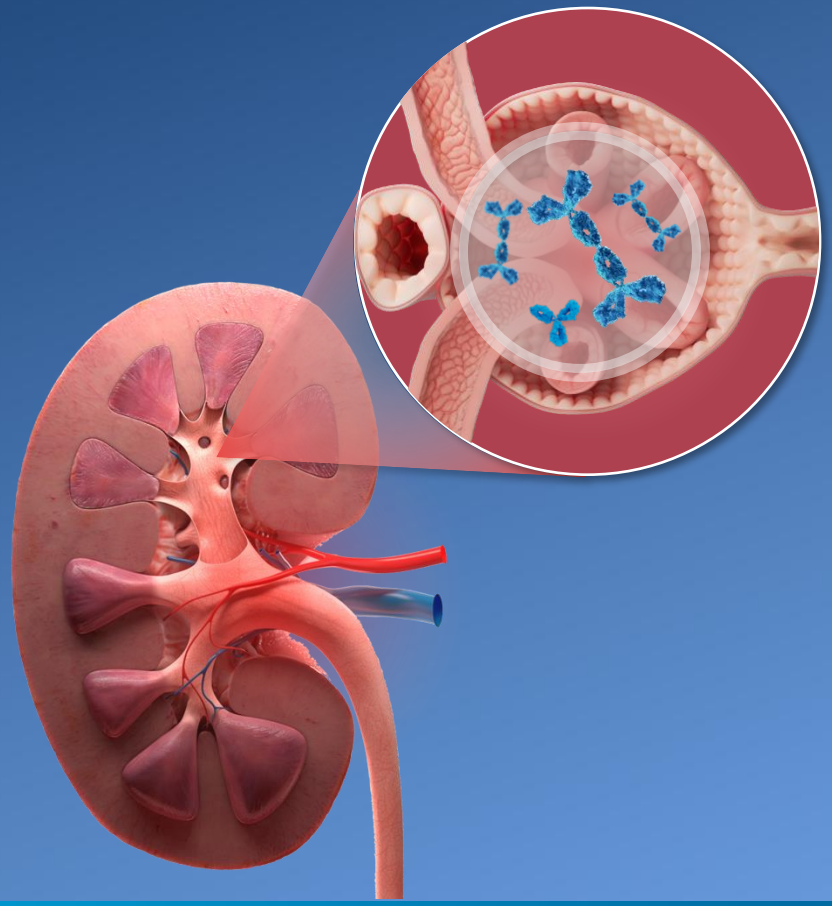
KEY POINT

BHV-1400 highlighted in opening plenary state-of-the-art lecture, “Yes, We Can…Cure Kidney Disease” at American Society of Nephrology 2025

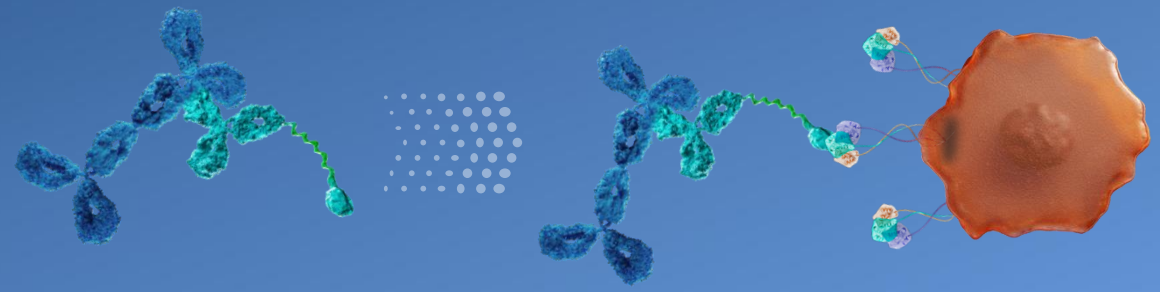
BHV-1400 Directly Targets the Disease-Driver of IgA Nephropathy

DEGRADERS

1 **Gd-IgA1** forms in excess, binds to antibodies forming immune complexes, deposits in the kidney and causes inflammation and fibrosis, ultimately leading to renal failure

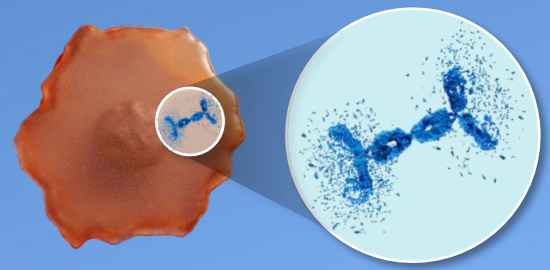


2 **TRAP™** degrader BHV-1400 selectively binds Gd-IgA1 and its complexes and redirects these to hepatocytes for removal



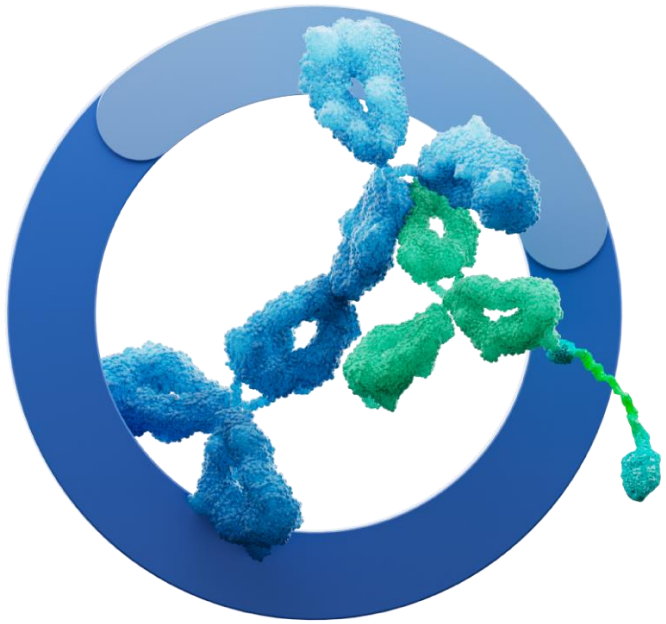
3

- Gd-IgA1 bound to BHV-1400 is rapidly degraded when BHV-1400 binds to ASGPR
- Healthy immunoglobulins: IgG, IgA, IgE and IgM are preserved



Updated KDIGO Guidelines: Treat Earlier and Target Pathogenic Gd-IgA1

IgAN Treatment Is Now Defined by Targeting of **Gd-IgA1**



New KDIGO 2025 Guidance		BHV-1400
First-line intervention now targets pathogenic Gd-IgA1 for disease modification	»	Designed to selectively remove pathogenic Gd-IgA1, the root cause of IgAN
Proteinuria threshold lowered to >0.5 g/g	»	Meaningfully expands the first-line addressable population
Early, disease-specific therapy recommended	»	Positions BHV-1400 as a potential foundational therapy

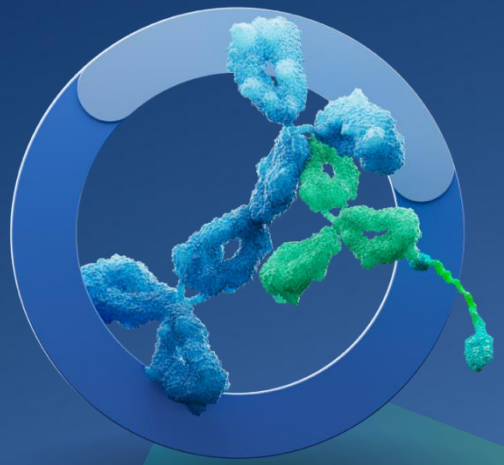
KDIGO: Kidney Disease Improving Global Outcomes; https://kdigo.org/wp-content/uploads/2024/08/KDIGO-2025-IgAN-IgAV-Guideline.pdf

KEY POINT

BHV-1400 is the only therapy in clinical development that directly and selectively removes Gd-IgA1

BHV-1400

ADVANCING A THERAPY FOR
ALL STAGES OF DISEASE





EARLY

Across the Spectrum

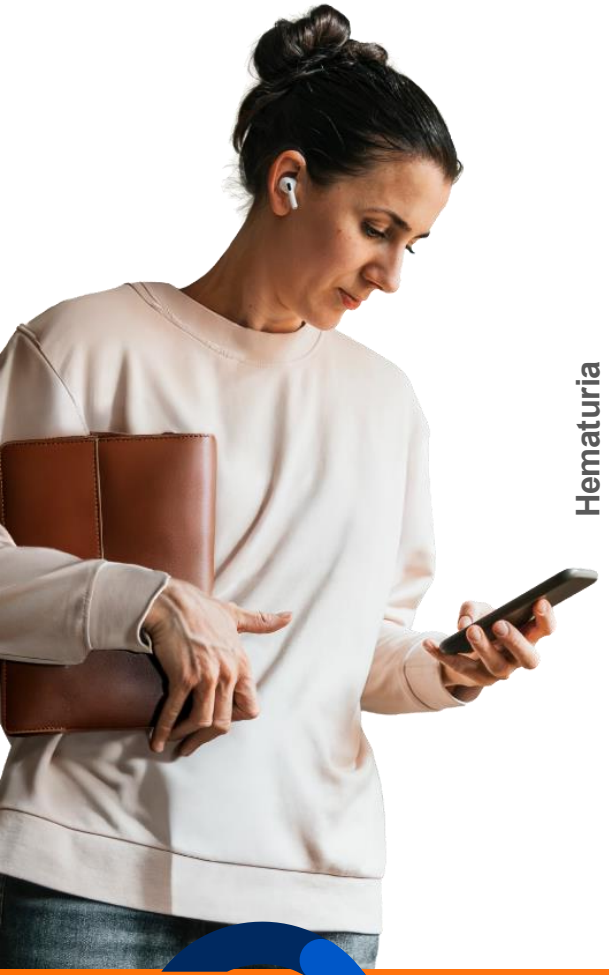
ADVANCED

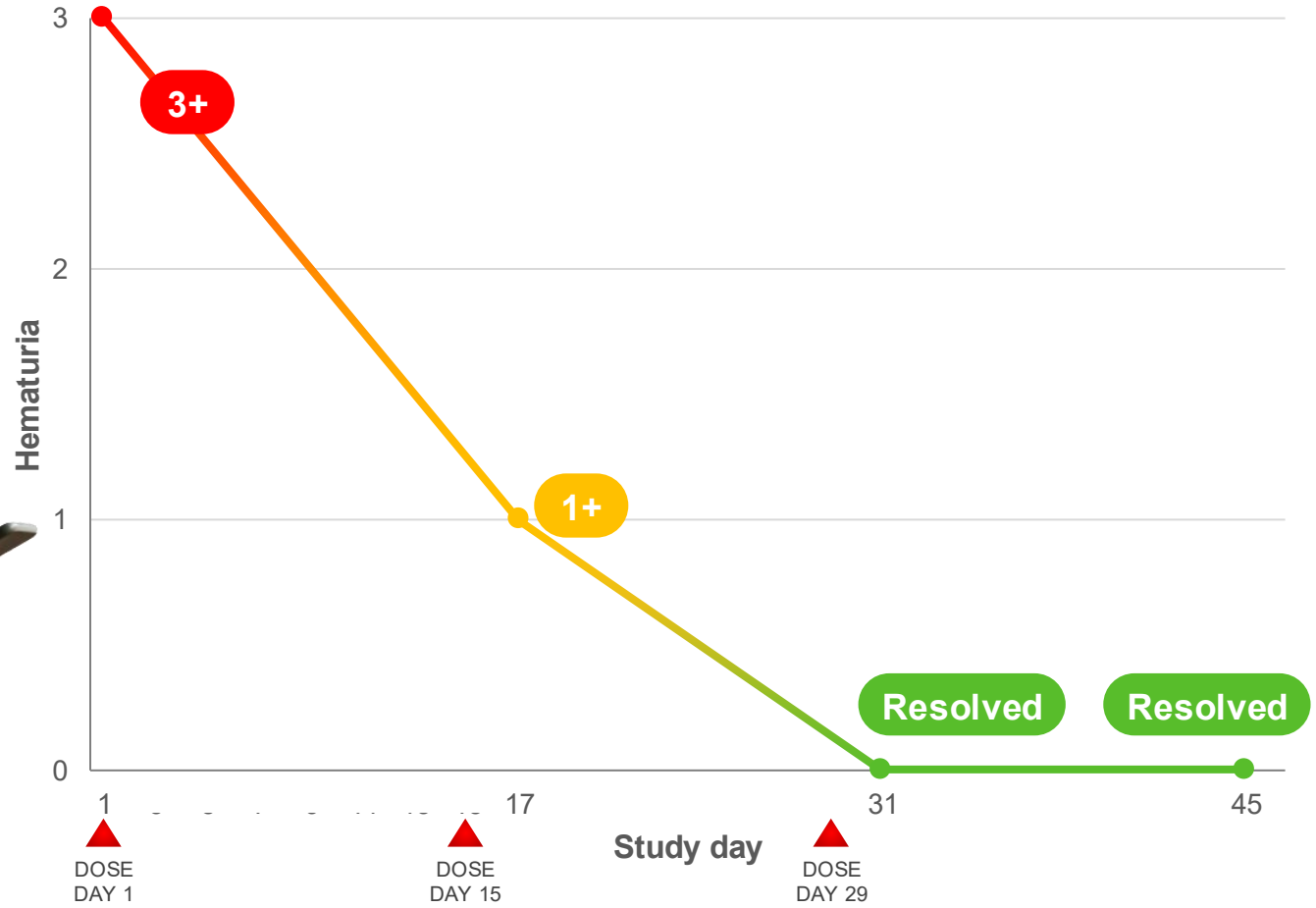


biohaven®

BHV-1400 Early Disease Clinical Experience:
Complete Resolution of Hematuria Within Weeks of Dosing

DEGRADERS





CASE REPORT: Initial IgAN Patient Dosed

- Young female patient
- Normal eGFR
- Chronic hematuria
- Active lifestyle
- Significant fatigue
- Comorbid diabetes

BREAKING NEWS

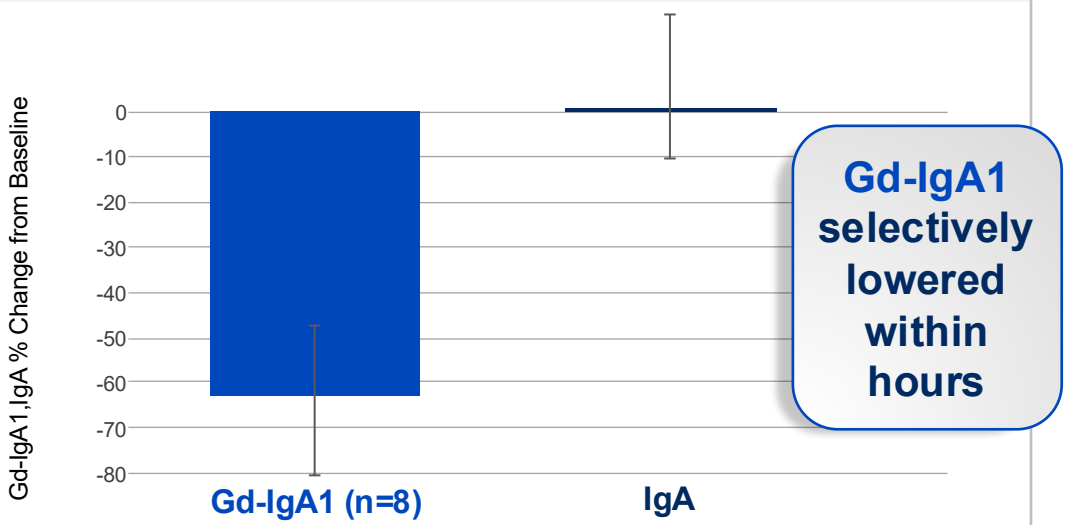
First patient dosed with BHV-1400 experienced complete resolution of hematuria and improvement of fatigue within weeks

BHV-1400 Phase 1: Surpasses B-cell Directed Competition in Speed, Depth and Selectivity

DEGRADERS

BHV-1400: Single dose SC
48 hours
63.3% lowering of Gd-IgA1[1]

SINGLE DOSE



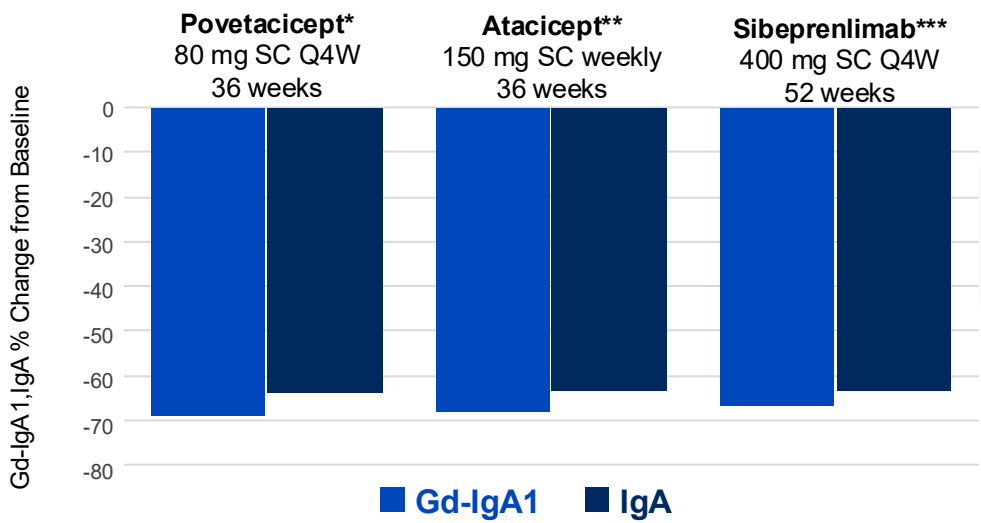
Gd-IgA1 selectively lowered within hours

BHV-1400 effect is rapid, deep and highly selective

*Preliminary data from ongoing study

B-cell targeting therapies:
Multiple dose, 9-12 months[2]

MULTIPLE DOSE



Competitors deplete healthy IgA

Extended dosing lowers Gd-IgA1 but also depletes healthy IgA

* Lafayette. NEJM. 2025. ** Perkovic. NEJM. 2025 ***Sing. National Kidney Foundation Spring Clinical Meeting. 2024

1. IgAN patients and healthy volunteers. **2.** All competitor data presented herein are derived from publicly available sources only. Certain data points have been reconstructed or estimated from published graphical information. No confidential, non-public, or proprietary information was used. This analysis has not been reviewed or validated by the referenced companies.

KEY POINTS

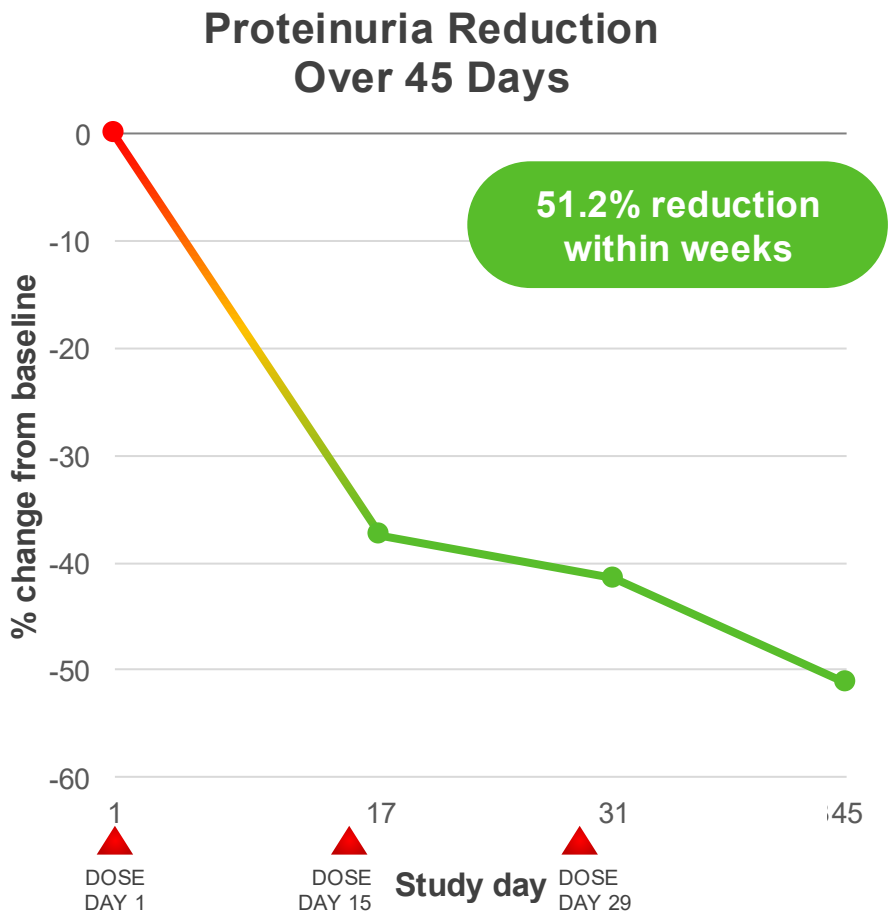
BHV-1400 stands apart as a next-generation IgAN candidate with unmatched speed, depth and selectivity, while sparing IgA, to maintain mucosal defense and reduce infection risk

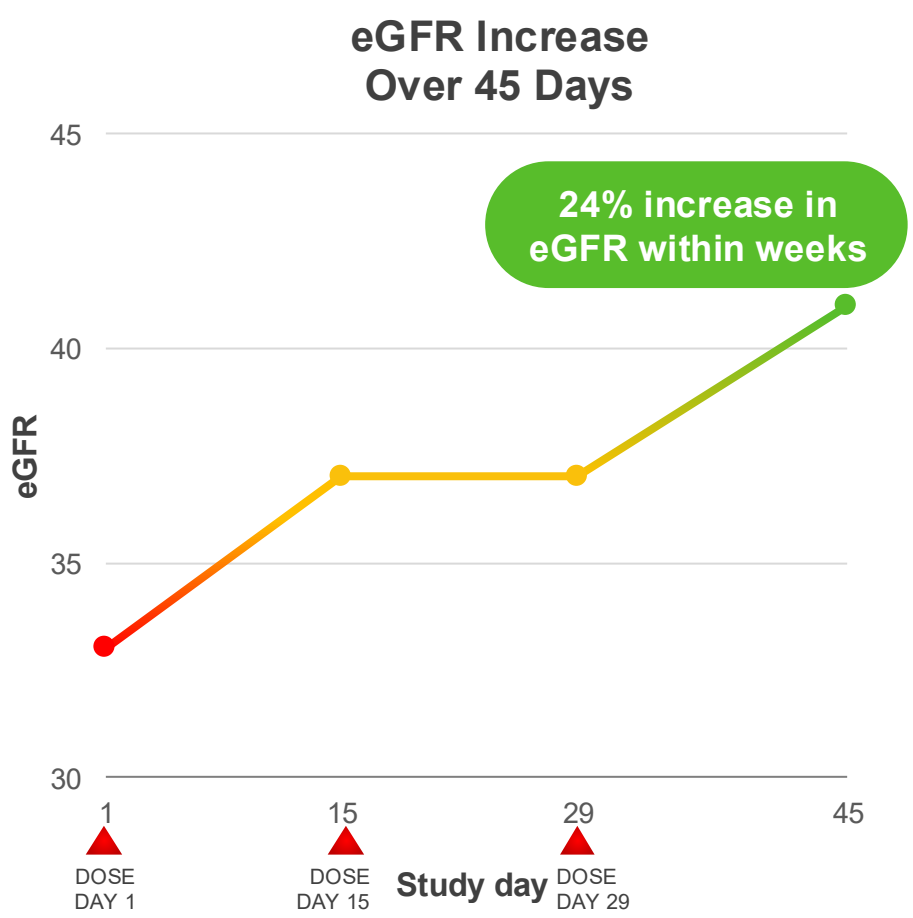
BHV-1400 Advanced Disease Patient: **Rapid Improvement in Proteinuria and Increase in eGFR Within Weeks of Dosing**

DEGRADERS

CASE REPORT: Advanced IgAN Patient Dosed

- Older male patient
- Moderate/severe eGFR at baseline
- Later-stage disease
- Significant proteinuria
- Multiple comorbidities





BREAKING NEWS

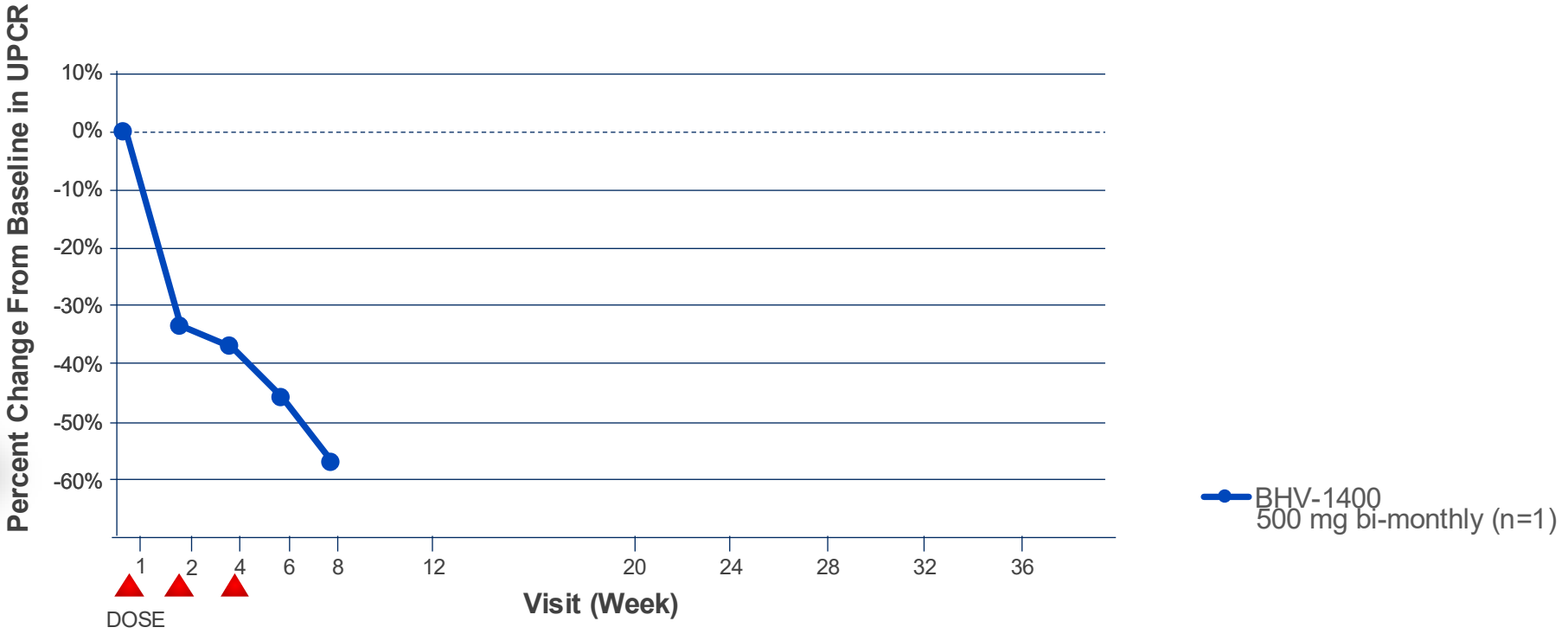
Rapid improvements seen in kidney function and achieved remission within weeks

BHV-1400 Single Patient Data: Rapid and Significant Improvement in Proteinuria

DEGRADERS

64%

reduction of proteinuria within weeks



BREAKING NEWS

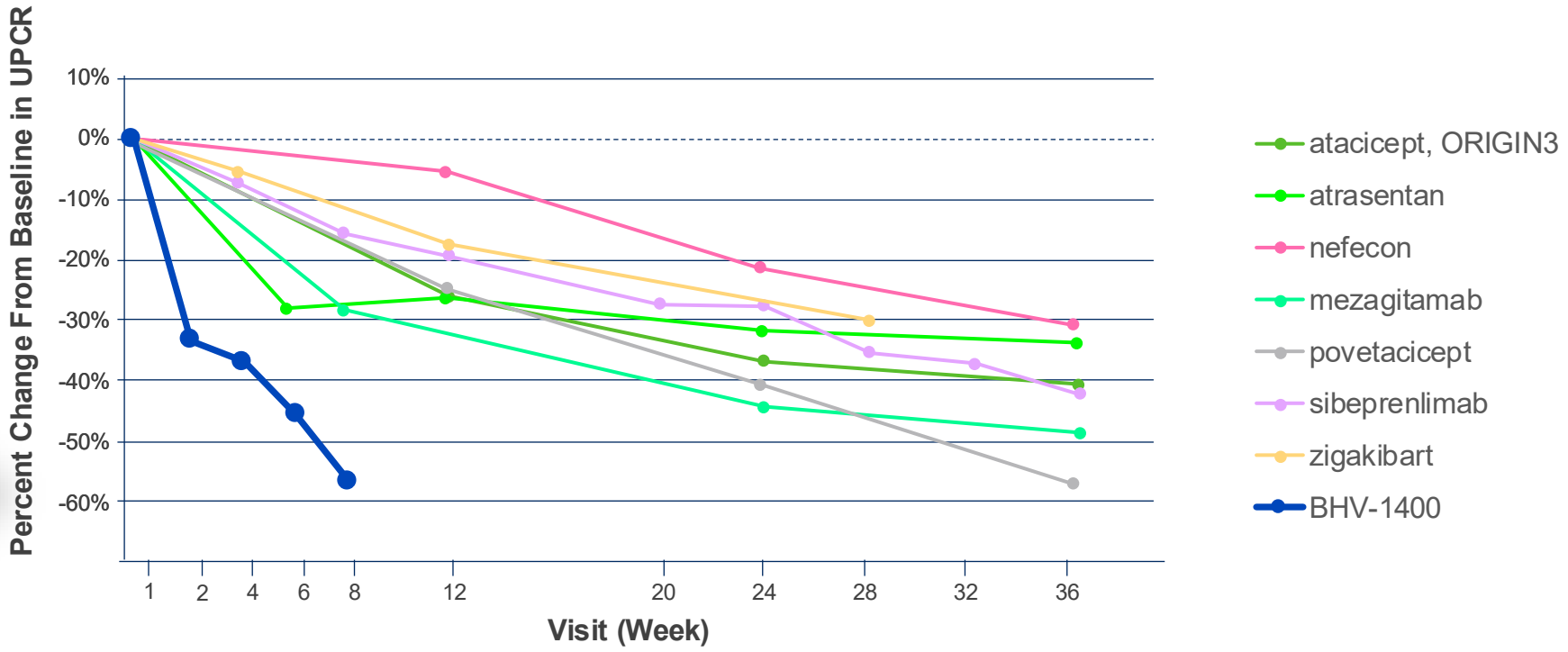
BHV-1400 rapid reductions in Gd-IgA1 translate into faster, deeper reductions in proteinuria as compared to market competitors

BHV-1400 Single Patient Data: Highlights Rapid Proteinuria Reduction Where Competitor Requires Months

DEGRADERS

64%

reduction of proteinuria within weeks



Cross trial competitor data: Barratt. JASN. 2024; Lafayette. NEJM. 2025.; Barratt. MEZA. 2025.; Alpine Immune Sciences. Press Release. 2024.; Perkovic. NEJM. 2025.; Kooienga. Kidney International. 2025.; Lafayette. The Lancet. 2023.; Heerspink 2025.

BREAKING NEWS

Unlike competitors, BHV-1400 acts unprecedented rapidity to reduce proteinuria in days, not months

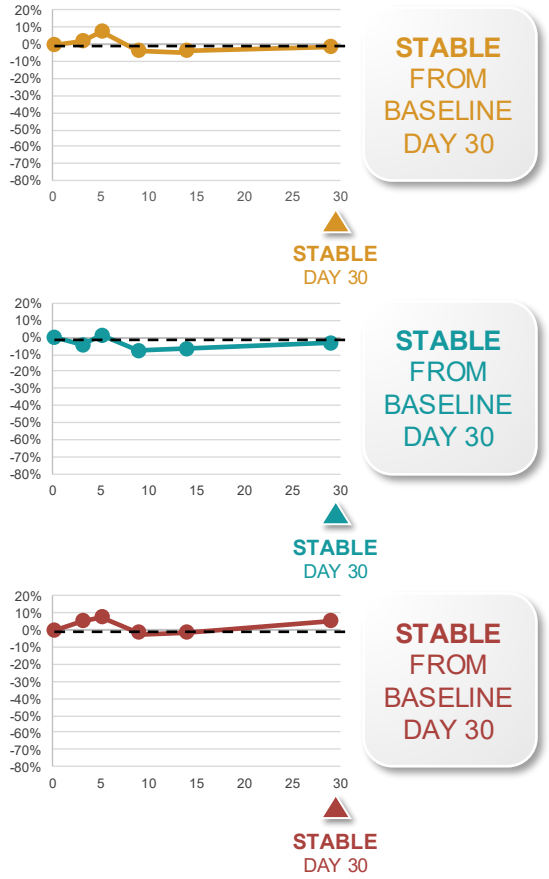
BHV-1400 TRAP™ Degrader Maintains Healthy Immunoglobulins[1,2] While Competitors Show Long-term Immunosuppression

DEGRADERS



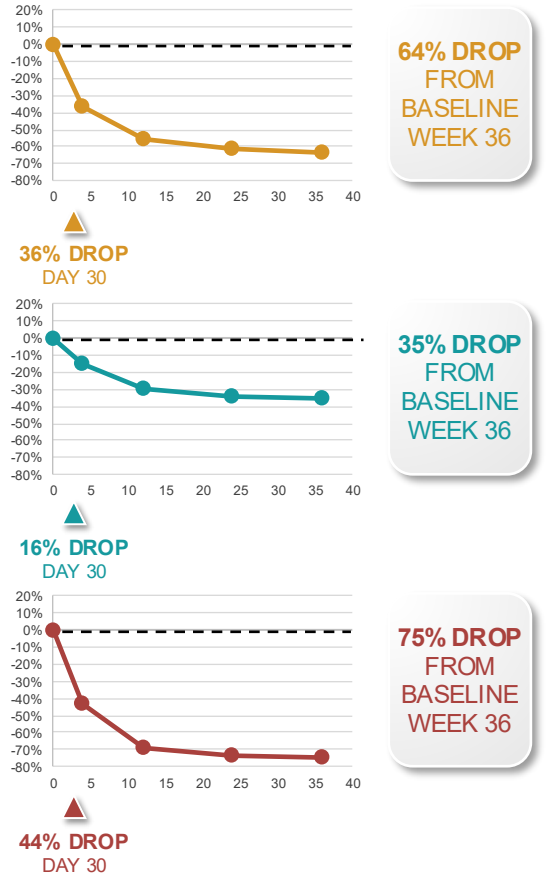
% change from baseline

BHV-1400 Single Dose SC[3]



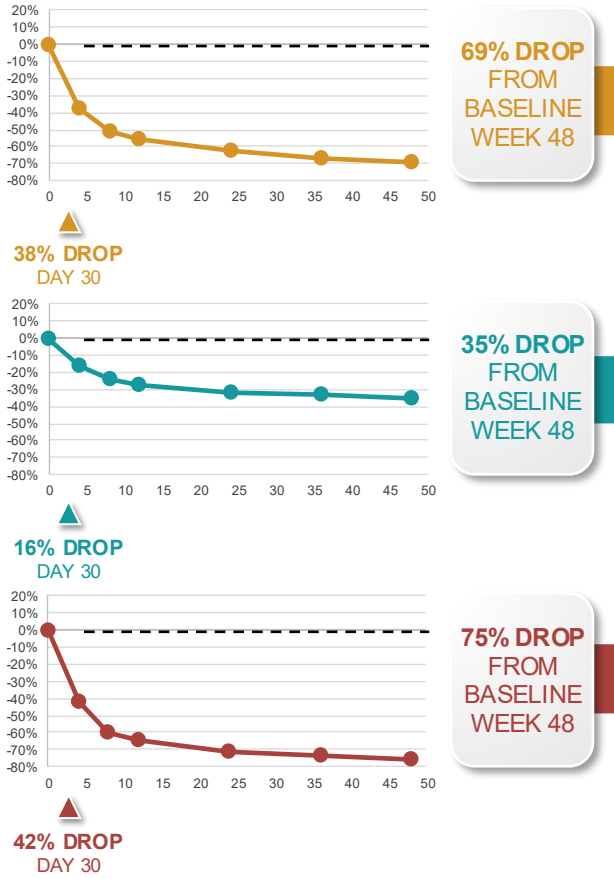
Days

Atacicept 150 mg[2,5]



Weeks

VOYXACT SC[2,4]



Weeks

VOYXACT FDA LABEL[6]

WARNINGS AND PRECAUTIONS

Immunosuppression and Increased Risk of Infections
VOYXACT suppresses the immune system by reducing antibody production, which may increase the risk of infections.

Immunosuppression and Immunization Risks
Because of its mechanism of action, VOYXACT may interfere with the immune responses to vaccines and increase the risk of infection from live vaccines.

1. Competitors did not report IgE. **2.** All competitor data presented herein are derived from publicly available sources only. Certain data points have been reconstructed or estimated from published graphical information. No confidential, non-public, or proprietary information was used. This analysis has not been reviewed or validated by the referenced companies. **3.** Solid dots represent the mean of the maximal total IgG % change from baseline **4.** Lafayette. NEJM. 2025 **5.** Lafayette. Kidney International. 2024 6. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/761434s000lbl.pdf

First Clinical Experience With BHV-1400 Shows Paradigm Shifting Potential

DEGRADERS



EARLY DISEASE

LOWERED
Gd-IgA1

IgG IgE IgA IgD IgM

MAINTAINED
HEALTHY IMMUNOGLOBULINS

LOWERED
HEMATURIA

LOWERED
PROTEINURIA

STABILIZED or IMPROVED
eGFR



ADVANCED DISEASE

BHV-1400: Selective Removal of Disease-Causing Gd-IgA1 Without Immunosuppression Compared to Competitors

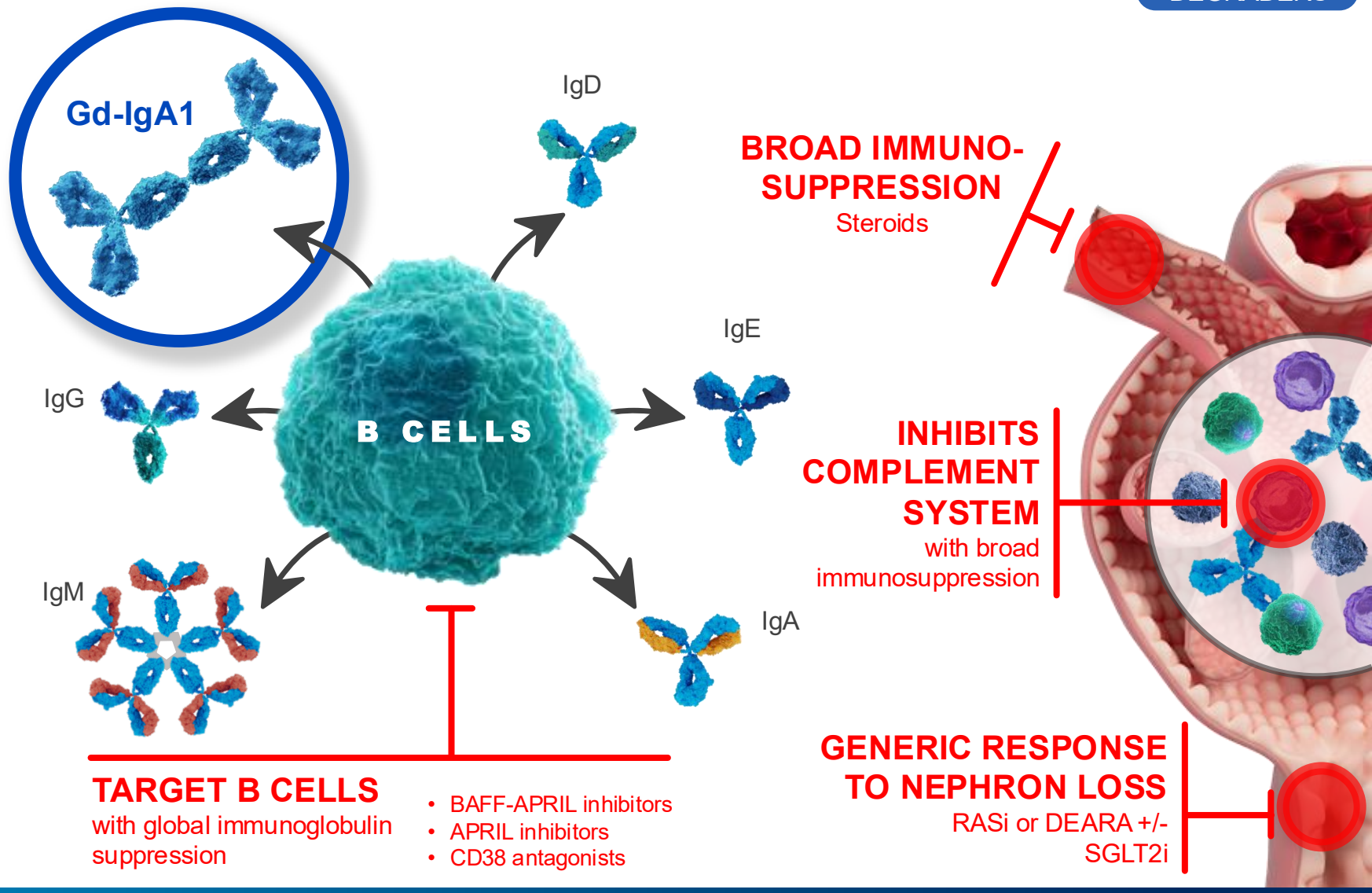
DEGRADERS

PRECISION TARGETING

biohaven®
TRAP™ Degrader

BHV-1400 SELECTIVELY DEGRADES Gd-IgA1

Targeting the pathogenesis of disease without immunosuppression



IgAN Represents a Multi-$B US Opportunity

IgAN US population	**112K–199K US IgAN patients**[2]
IgAN patients with proteinuria ≥0.5g/day for which KDIGO guidelines[3] recommend treatment with DMTs that reduce pathogenic Gd-IgA1	**85K–151K patients**[4] **eligible for BHV-1400**
US annualized pricing of Tarpeyo — US WAC pricing of Voyxact[5]	**$180K–390K per patient per year**

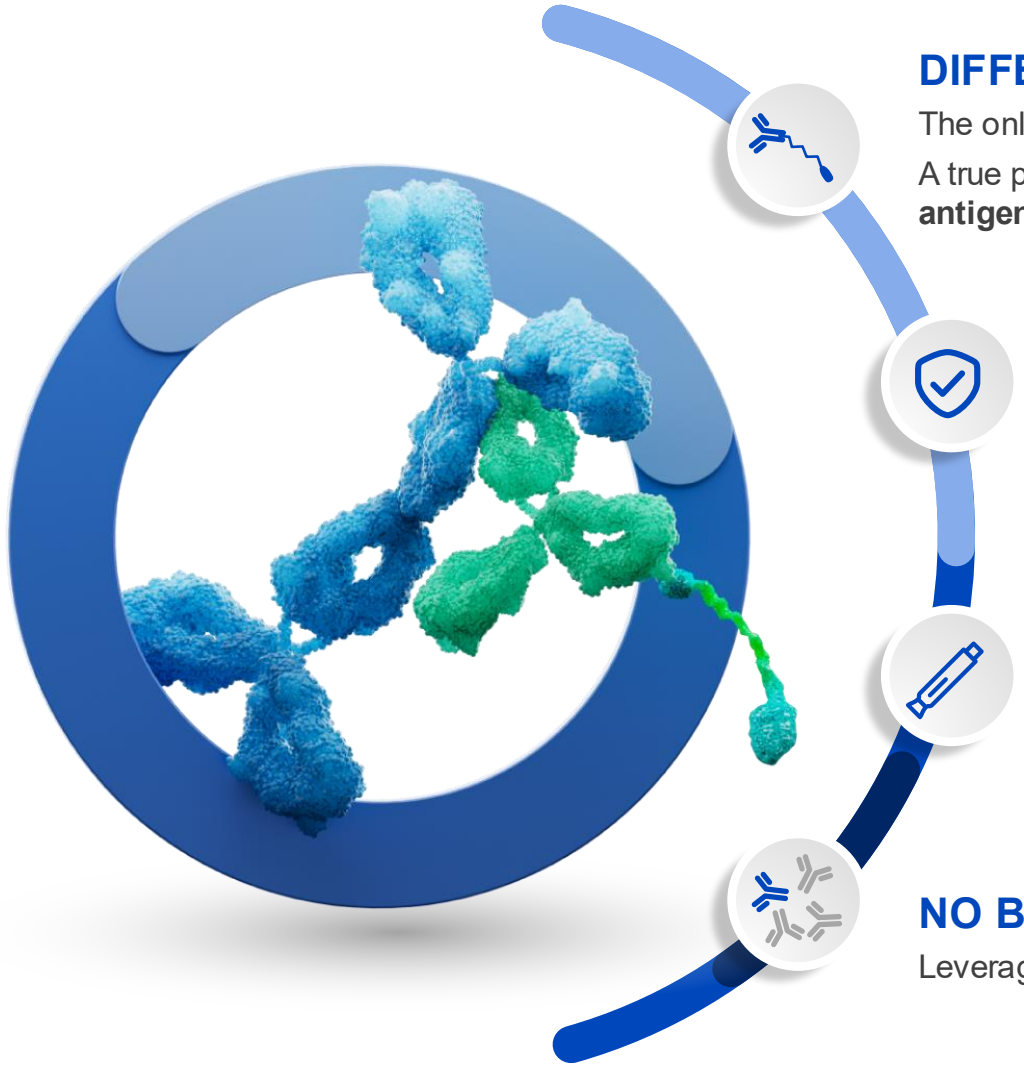
$20B+[1,5]

Potential US market size

DMT, Disease Modifying Therapy.

1. Goldman Sachs August 11, 2025 IgAN report (assumes $150K annualized net pricing). **2.** Cantor Fitzgerald & Co US Equity Research March 18, 2025 and Nov 25, 2025. **3.** KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV). **4.** Pitcher. CJASN. 2023. **5.** Based on December 2025 announced Voyxact WAC of $30K per vial, Q4W dosing, $390K annualized. IgAN market size could be twice as large with Voyxact pricing.

BHV-1400: A Singular Asset Combining a First-in-Class Mechanism, Compelling Human Data and a Clear Commercial Path



DIFFERENTIATED SCIENCE

The only therapy that directly **targets and eliminates** the root **pathogenic driver** of IgAN

A true paradigm shift from production inhibition or downstream damage control to **precision antigen removal**

DE-RISKED BIOLOGY

Human proof-of-concept achieved with **rapid (>70%),** deep and durable **target engagement**

The fundamental mechanism is **validated**, shifting focus to **clinical execution**

MASSIVE OPPORTUNITY

Positioned to become a **foundational therapy** by addressing the **core unmet need** in a **multibillion-dollar market**

A clear, **regulator-endorsed clinical path** to approval and commercialization

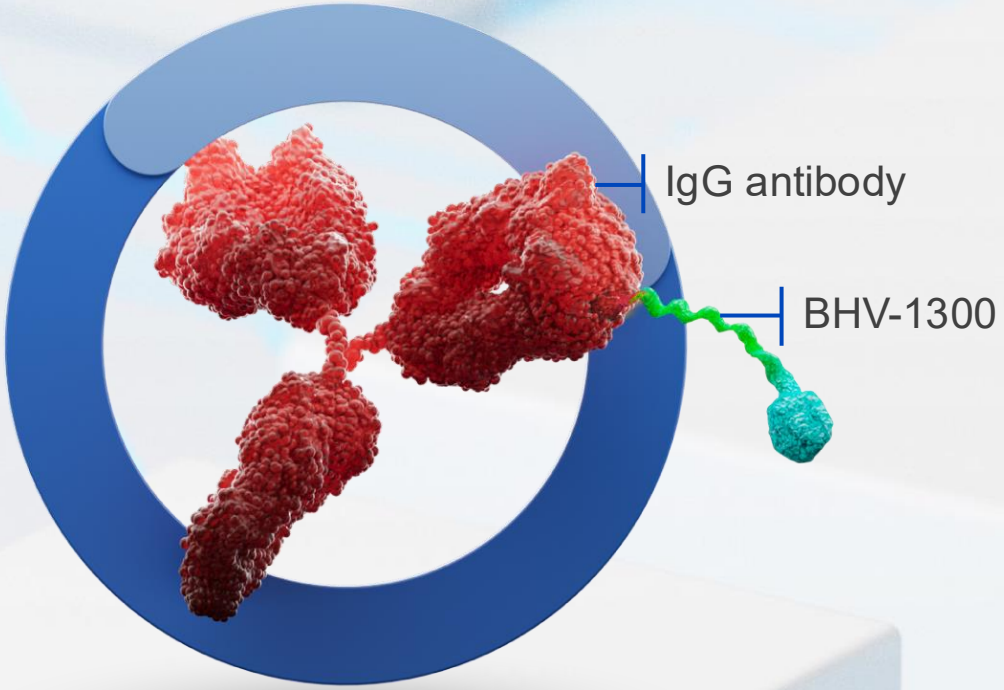
NO BROAD IMMUNOSUPPRESSION

Leverages a **natural clearance pathway** rather than inhibiting immune cell function

biohaven®

DEGRADERS:
BHV-1300 MoDE™

Graves' Disease



BHV-1300: Potential to Transform Care Across Multiple IgG Mediated Diseases

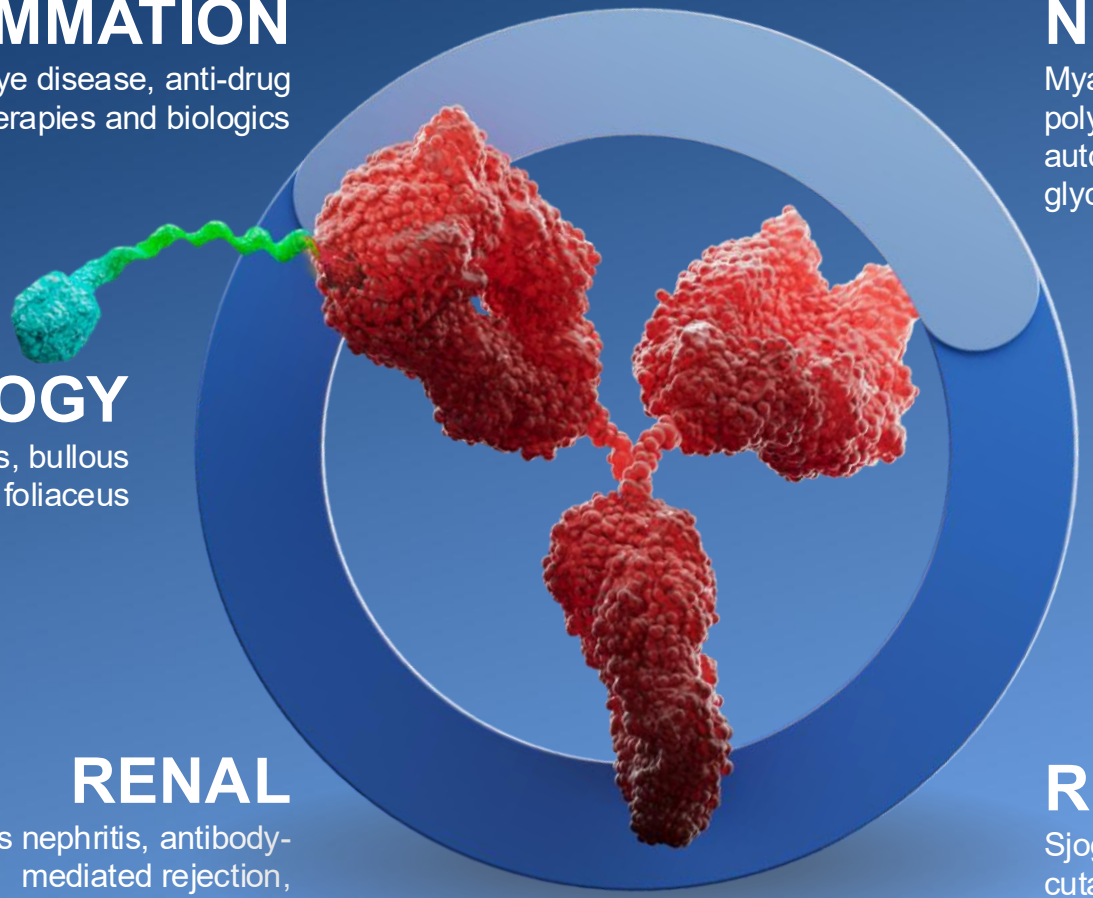
DEGRADERS

IMMUNOLOGY AND INFLAMMATION

Graves' diseases, thyroid eye disease, anti-drug antibodies for gene therapies and biologics

NEUROLOGY AND NEUROMUSCULAR

Myasthenia gravis, chronic inflammatory demyelinating polyneuropathy, neuromyelitis optica spectrum disorder, autoimmune encephalitis, myelin oligodendrocyte glycoprotein antibody disease



DERMATOLOGY

Pemphigus vulgaris, bullous pemphigoid, pemphigus foliaceus

HEMATOLOGY

Immune thrombocytopenia, autoimmune hemolytic anemia

RENAL

Lupus nephritis, antibody-mediated rejection, membranous nephropathy

RHEUMATOLOGY

Sjogren's, systemic sclerosis, rheumatoid arthritis, cutaneous lupus erythematosus, myositis, fibromyalgia

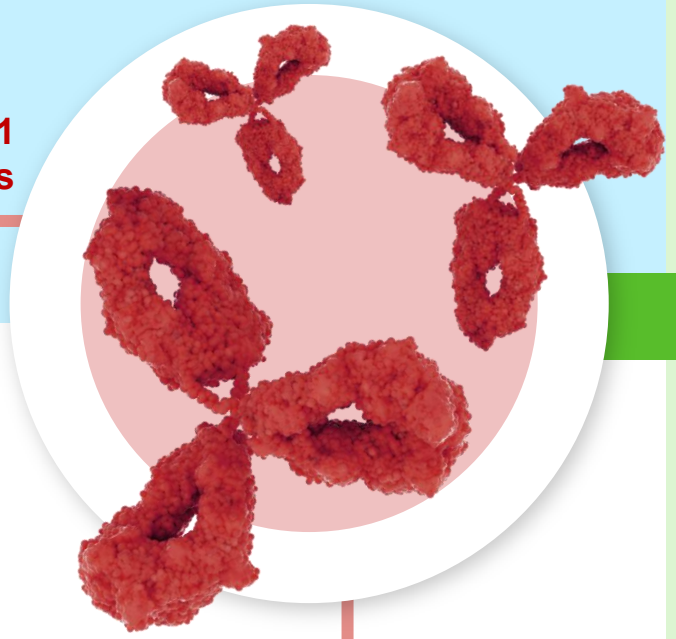
Biohaven IgG Degrader Targets the Root Cause of a Broad Autoimmune Disease to Treat and Prevent Multi-Organ Complications

DEGRADERS

1 PRODUCTION

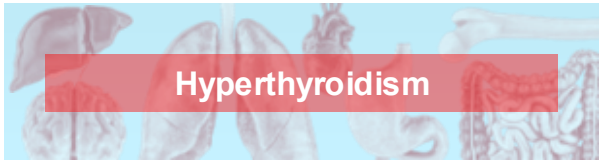


TSHR IgG1 autoantibodies



2 EFFECTS

TSHR autoantibodies induce excess secretion of hormones causing **hyperthyroidism**



TSHR autoantibodies also bind outside the thyroid and results in:







3 INTERVENTION

IgG1, IgG2, IgG4



Biohaven MoDE™ redirects disease-causing target to liver for removal

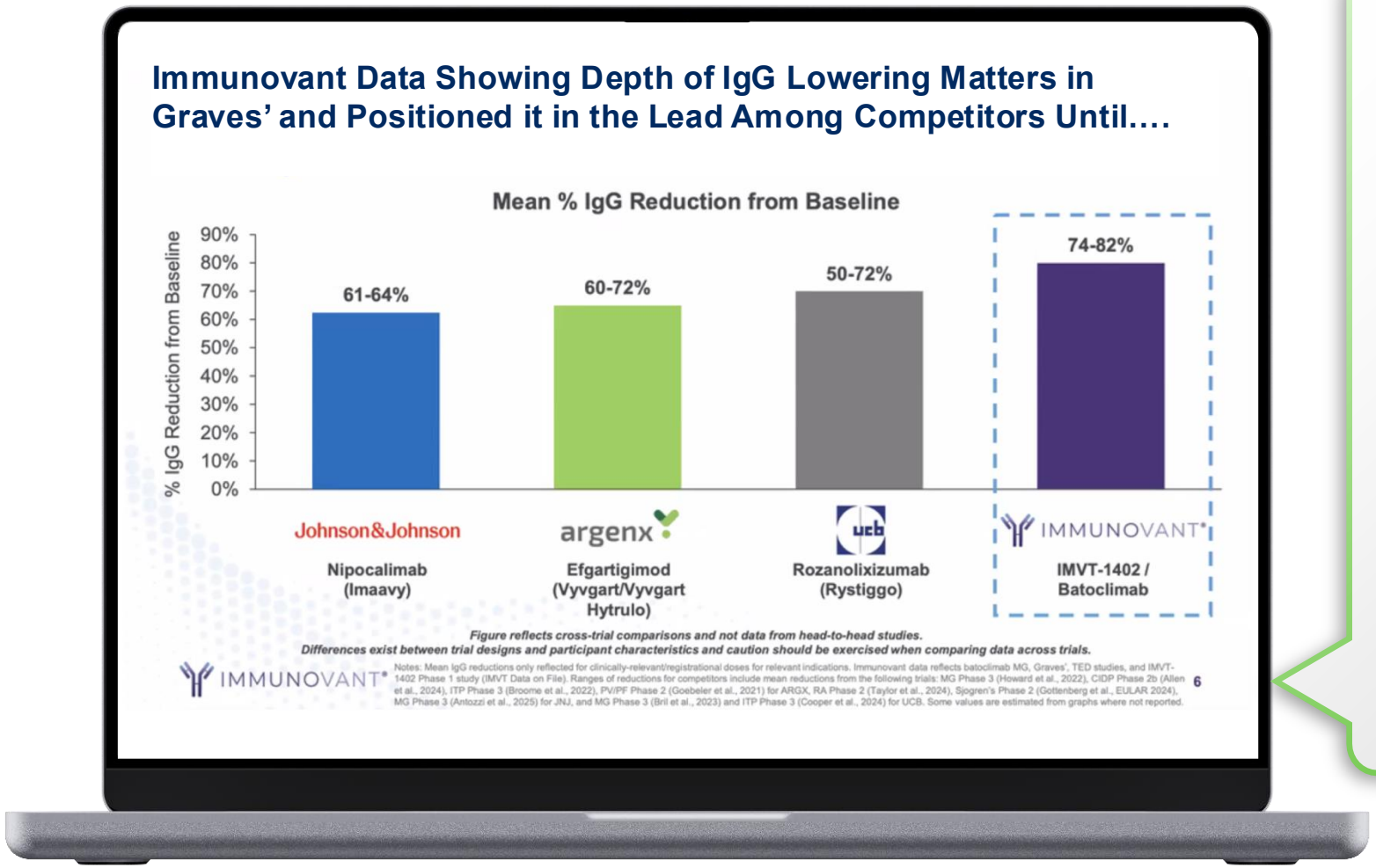


Biohaven's degrader removes IgG to eliminate the disease driver of Graves'

Source: Graves' disease. Nat Rev Dis Primers. 2020.

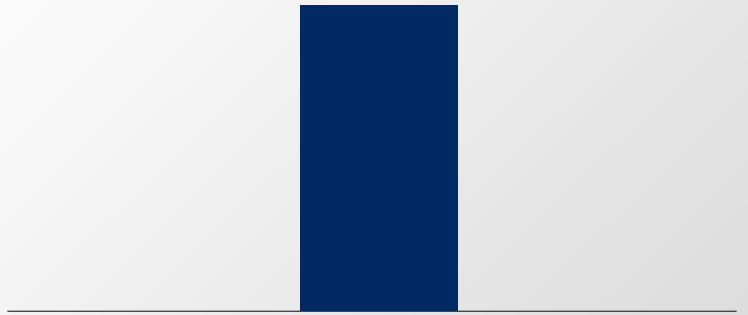
BHV-1300 MoDE, a Class of its Own, Sets New Benchmark for IgG Reduction Versus Leading Therapies

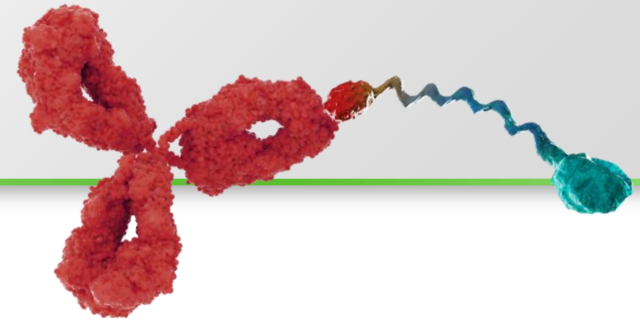
DEGRADERS



Subcutaneous BHV-1300 achieved mean % IgG reductions >80% **with maximal lowering up to**

87%[1]





1. Data on file, 4-week MAD. Mean % reduction calculated as mean maximum reductions in high-dose mad group after 3 doses.

The Devastating Consequences of Graves' Disease (GD)

DEGRADERS

Thyroid eye disease (TED)

- Between 25–50 percent have eye abnormalities, which are known as Graves' ophthalmopathy[1]
- Up to 5% progress to severe TED[1]

Thyroid cancer

2X risk[3]

Metabolism

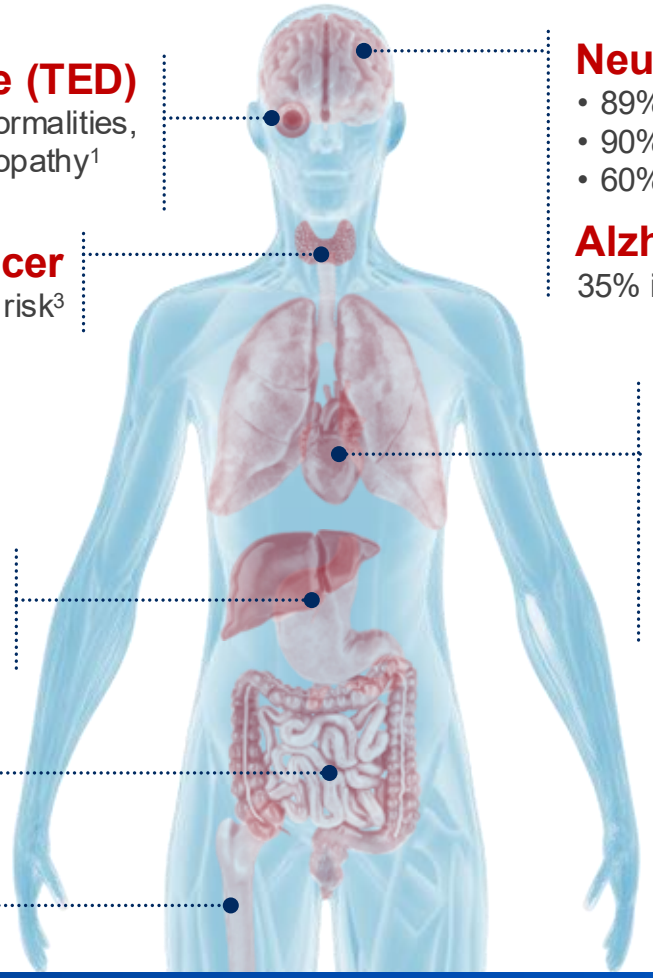
20% higher risk of diabetes mellitus[8]

Reproductive health

- 50% of women experience infertility[9]
- Higher risk of preeclampsia

Bone health

30% increased risk of fractures[10]



Neuropsych

- 89% of GD patients experience cognitive dysfunction[1]
- 90% suffer anxiety[2]
- 60% Depression[2]

Alzheimer's disease

35% increased risk[4]

50% higher risk of cardiovascular associated mortality[9]

- 15% have AFIB[5]
- 8% develop heart failure[6]
- 15% higher risk of myocardial infarction[7]
- 12% higher risk of stroke[7]

1. Sahli. J Opthalmol. 2017. **2.** Bathla. IJEM. 2016. **3.** Johansson. IJEM. 2023. **4.** Adams. JAMA Intern Med. 2023. **5.** Kostopoulos. G. Eur. Thyroid J. 2024. **6.** Fan SWD. touchREV Endocrinol. 2024. **7.** HJ Kim. Thyroid 2020. **8.** Biondi. Endocr Rev. 2019. **9.** Brandt. Thyroid. 2013. **10.** Blum. JAMA. 2015.

KEY POINT

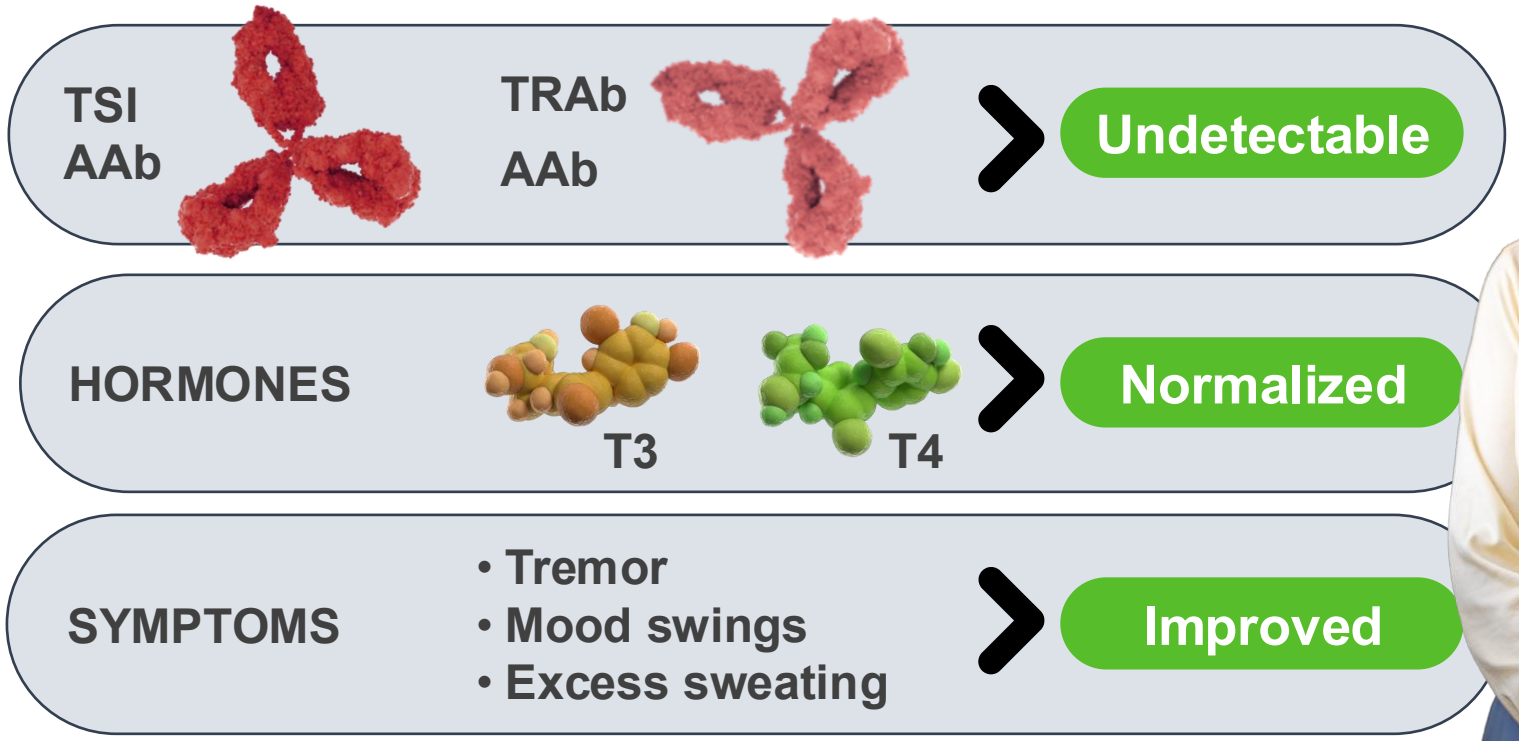
Graves' disease high morbidity and mortality despite antithyroid drug (ATD) treatment remains a public health burden

BHV-1300: First Graves' Patient Dosed — Pathogenic Antibody Levels Undetectable and Thyroid Hormones Normalized Within First Month

CASE REPORT: Initial Graves' Patient Dosed

- Middle-aged female
- Newly diagnosed with Graves' disease
- Hyperthyroid at baseline
- Tremor, mood swings and excess sweating at baseline

After 4 Weeks of Treatment…





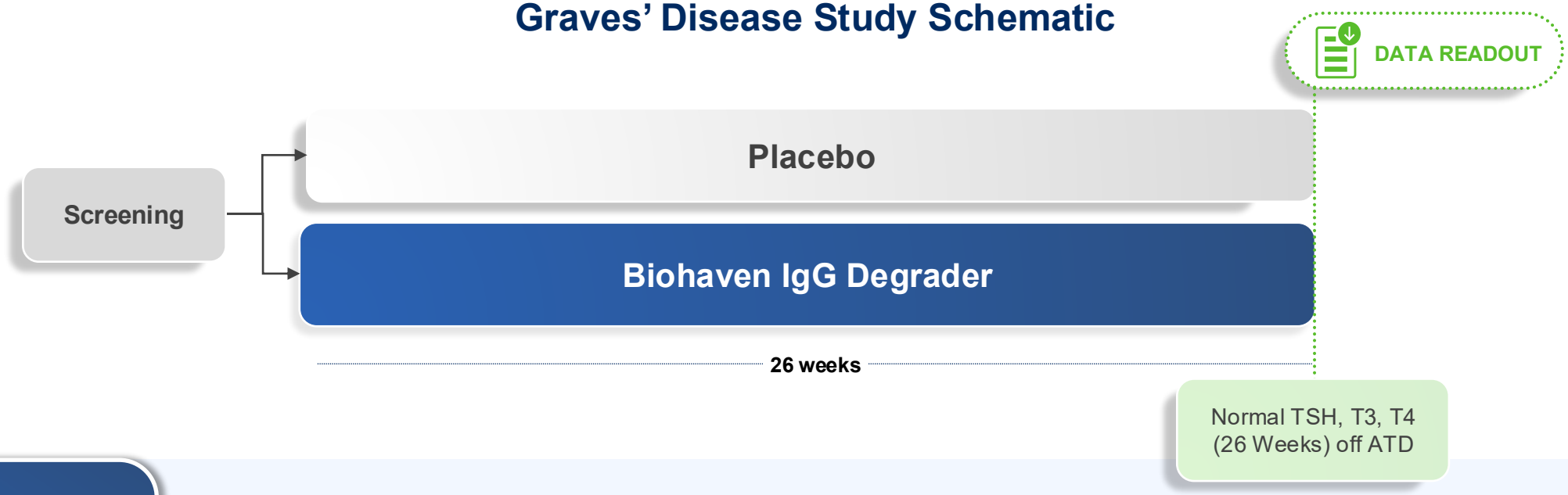
TSI, Thyroid Stimulating Immunoglobulin; **TRAb**, Thyrotropin Receptor Antibodies; **AAb**, Autoantibody.

KEY POINT

Within one month, pathogenic antibodies became undetectable, thyroid hormones normalized and patient reported improved mood, sweating and tremor

Graves' Disease Pivotal Trial

Graves' Disease Study Schematic



KEY STUDY DETAILS

Study Design: Randomized, double-blind, placebo-controlled trial

Population: Male and female adults with Graves' disease

Endpoints: Normal T3, T4 and TSH off ATD at week 26

ATD, Antithyroid Drugs.

KEY POINT

Deep IgG lowering and early Graves' patient data derisk 2026 registrational clinical trial with biomarker endpoint

Graves' Disease: Significant Patient Opportunity

DEGRADERS



$15B+

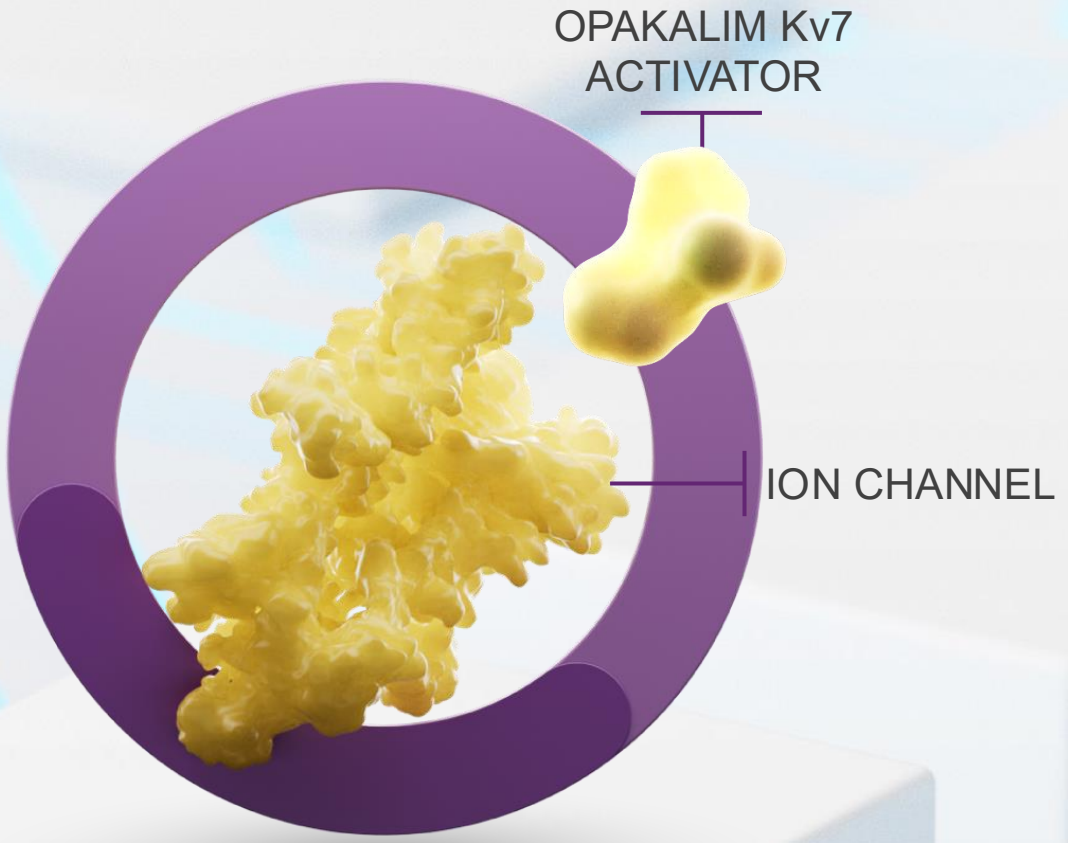
Potential US market size[5]

1. 1% adult population; NIDDK, AAFP, Cleveland Clinic, American Thyroid Association, MedlinePlus, Yale Medicine. **2.** 50% diagnosed; Yashkin. Clin Diabetes Endocrinol. 2024. **3.** Forian Database analysis 6/1/16-9-23-24 E050,E0501,E0500 were the primary Dx lookup codes. **4.** 35-43%; Azizi. J Endocrinol Invest. 2022. Kubota. Thyroid 2008. **5.** Biohaven internal analysis

biohaven®

ION CHANNEL: OPAKALIM SELECTIVE Kv7 ACTIVATOR

Revolutionizing Epilepsy Treatment With a Modern Kv7 Activator



Kv7 Activation Is a Clinically Validated Mechanism of Action for the Treatment of Epilepsy

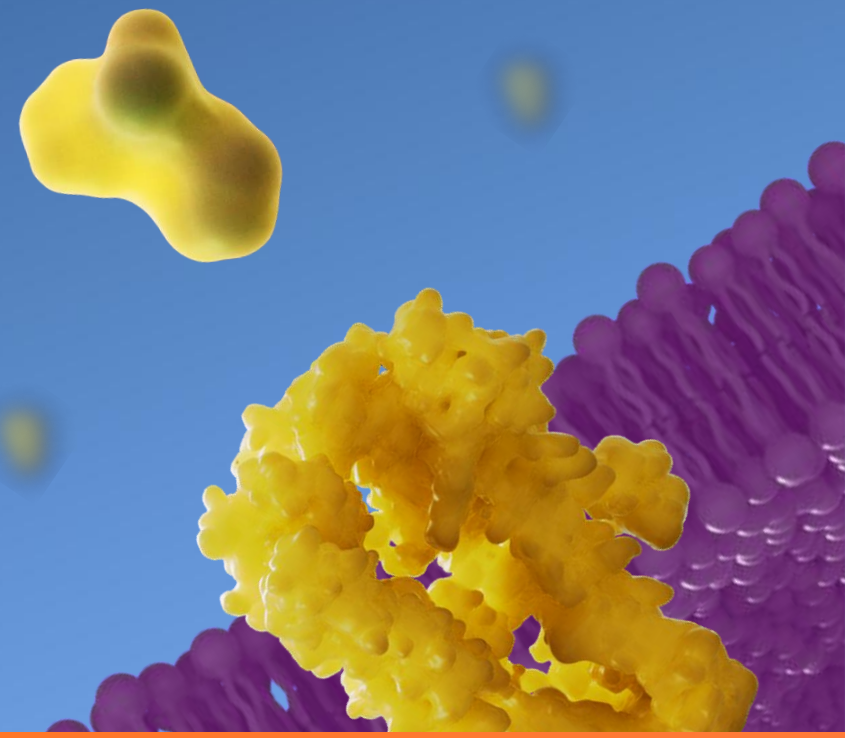
Kv7

Opakalim, a selective Kv7 activator, offers paradigm-shifting potential to control seizures without the burdensome side effects frequently reported with approved antiseizure medicines and those in development

DESIGNED TO SELECTIVELY ACTIVATE Kv7.2/7.3 CHANNELS WITHOUT IMPACTING GABA RECEPTORS

DEMONSTRATES EXCEPTIONAL SAFETY PROFILE WITH LOW RATES OF CNS ADVERSE EVENTS ACROSS STUDIES

DELIVERING FOCAL EPILEPSY TOPLINE DATA IN 2H 2026



BREAKING NEWS

Efficacy signals observed in open-label epilepsy data, comparable to competitor data, with favorable tolerability

High Unmet Need for Novel, Well-tolerated and Effective Antiseizure Medicines

Kv7



EMILY, Living with Epilepsy

PREVALENCE[1]

3.5 MILLION

people with epilepsy (PWE) in the US

REFRACTORY[2]

UP TO 40%

of PWE continue to have seizures despite treatment

HEALTHCARE SPENDING[3]

$24.5 BILLION

annual spending in the US (direct costs)

1. www.cdc.gov/mmwr/volumes/66/wr/mm6631a1.htm. **2.** French. Epilepsia. 2007. **3.** www.cdc.gov/epilepsy/data-research/facts-stats/index.html#:~:text=Health%20care%20spending,3.

High Unmet Need for Novel, Well-tolerated and Effective Antiseizure Medicines

Kv7

ADVERSE EVENTS[1]

80%

of PWE report AEs after starting an ASM

ADHERENCE[2]

UP TO 60%

of PWE report non-adherence to ASMs

COMORBIDITIES[3]

50%

of PWE report at least 1 comorbidity

QUALITY OF LIFE[4]

50%

of PWE report lower QoL



VENIKA, Living with Epilepsy

1. Baker. Epilepsia. 1997. **2.** Donahue. Neurol Clin Pract. 2025. **3.** Bosak. Epilepsy & Behavior. 2025. **4.** Strzelczyk. Epilepsy & Behavior. 2023.

Opakalim Overcomes the Challenges of Approved Epilepsy Therapies

Kv7

APPROVED THERAPIES	OPAKALIM
Burdensome • Dosing multiple times per day • Months long titration schedules	**Easy-to-use** • Once-daily tablet • No need to titrate
Poor CNS tolerability • High rates of CNS AEs • Somnolence, dizziness, cognitive problems	**Minimal CNS side effects** Very low rates of CNS AEs
Make comorbidities worse 50% of patients report comorbidities i.e., mood	**Does not make comorbidities worse**
Limited seizure control Up to 40% of patients are refractory to treatment	**Improved seizure control** Clinically validated MoA complementary to existing ASMs allowing rational combinations

ASMs, Antiseizure Medications.

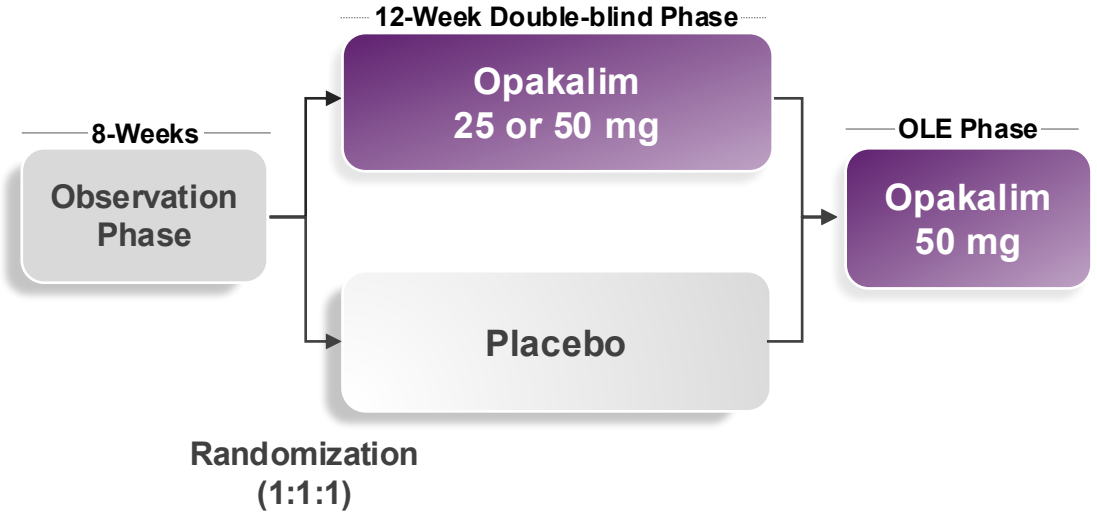
KEY POINT

Opakalim offers compelling ASM profile for patients with potential to drive enhanced clinical outcomes and superior quality of life

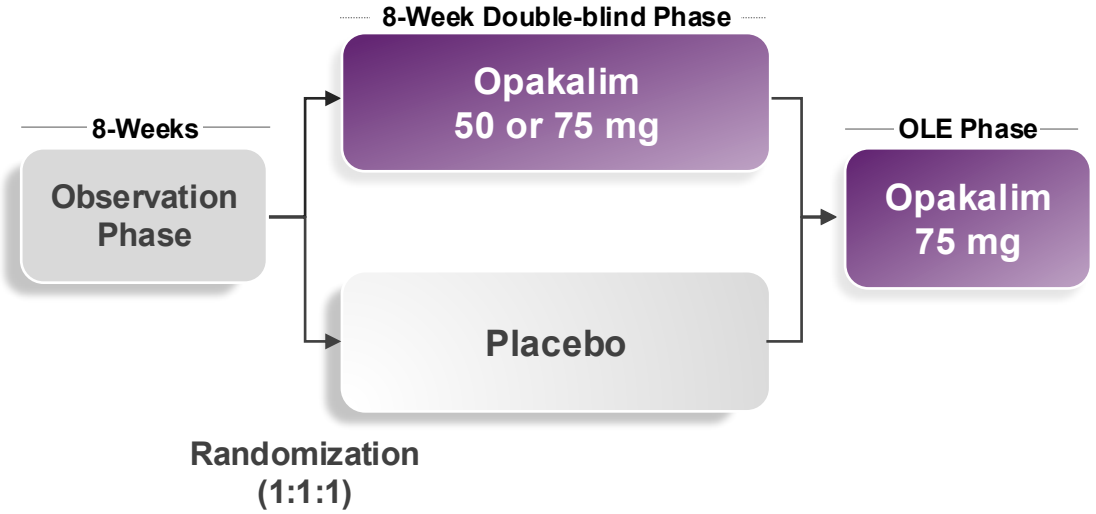
Two Ongoing Studies in Focal Epilepsy With Opakalim

Kv7

Study RISE 2



Study RISE 3



KEY STUDY DETAILS

Study Design: Randomized, double-blind, placebo-controlled trials

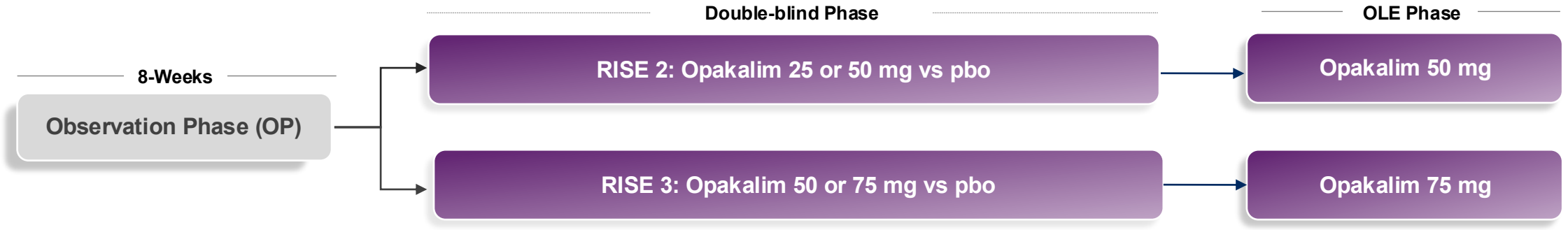
Population: Subjects 18–75 years of age with refractory focal epilepsy

Key Entry Criteria: Average of ≥4 observable focal seizures per 28 days; taking 1 to 3 antiseizure medications

Endpoints: Change in seizure frequency, 50% responder rate

Efficacy Signals Observed in Focal Epilepsy Open-Label Data

Kv7



SEIZURE FREQUENCY
Pretreatment Baseline in OP

VS

SEIZURE FREQUENCY
On Treatment with Opakalim 75 mg in OLE (n=144)

>50% OF PATIENTS SHOWED

50% RESPONSE RATE

OVER THE FIRST 6 MONTHS OF OLE

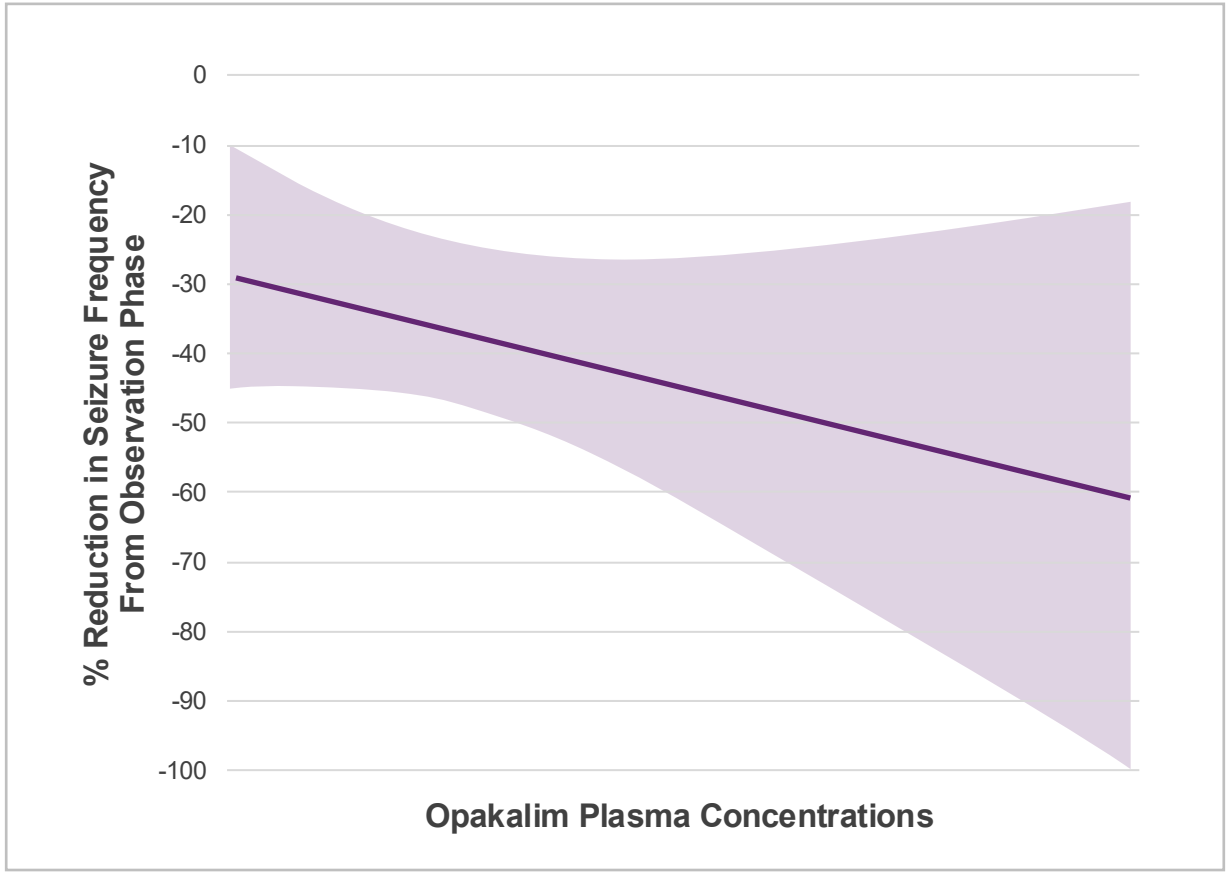
Preliminary data as of 4Q 2025 for 6-month completers.

BREAKING NEWS

Efficacy signals observed in open-label epilepsy data, comparable to competitor data

Antiseizure Efficacy Correlates With Opakalim Concentration in Focal Epilepsy Open-Label Data

Kv7



Preliminary exposure-response analysis for pooled participants in focal epilepsy open-label study

BREAKING NEWS

Exposure-response analysis shows clear reductions in seizure frequency with increasing opakalim concentrations

Excellent Tolerability Observed in Focal Epilepsy Open-Label Data

Kv7

Preferred Term	50 mg	75 mg	Pooled
Headache	2.4%	6.9%	4.9%
Fatigue	3.2%	4.2%	3.7%
Upper respiratory tract infection	3.2%	4.2%	3.7%
Nasopharyngitis	2.4%	4.2%	3.4%
Back pain	3.2%	2.8%	3.0%
Fall	1.6%	4.2%	3.0%
Somnolence	**1.6%**	**4.2%**	**3.0%**
Pain in extremity	2.4%	2.8%	2.6%
Nausea	4.0%	1.4%	2.6%
Seizure	2.4%	2.8%	2.6%
Irritability	2.4%	2.1%	2.2%
Diarrhea	3.2%	1.4%	2.2%
Dizziness	**2.4%**	**2.1%**	**2.2%**

Adverse events reported in ≥2% of pooled participants, data as of 4Q 2025.

BREAKING NEWS

Low rates of adverse events reported with opakalim in open-label epilepsy data

Opakalim Offers Potential Best-in-Clinic Kv7-Activator Profile

Kv7

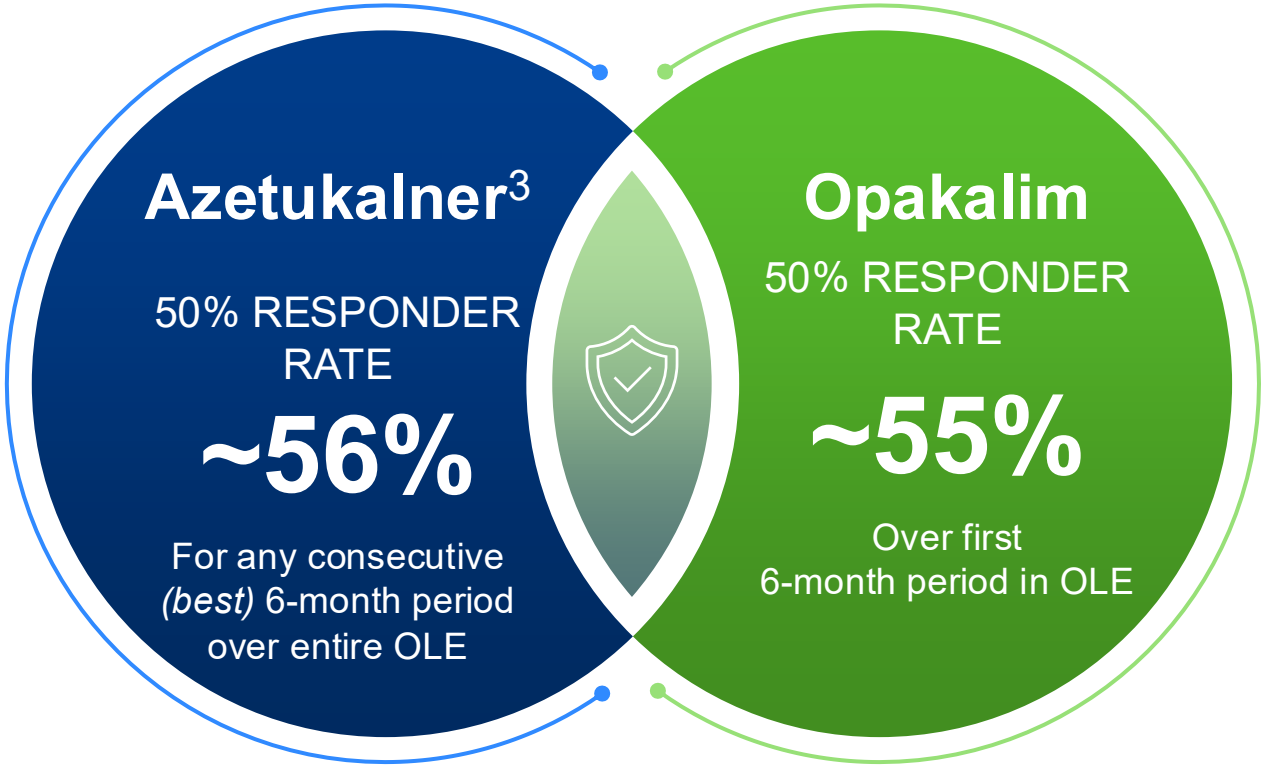
Lower rates of CNS AEs

with opakalim compared to azetukalner in ongoing OLE studies*

AE	Azetukalner[1]	Opakalim[2]
Dizziness	25%	2%
Headache	19%	5%
Somnolence	17%	3%
Fall	15%	3%
Memory impairment	11%	<1%
Weight gain	11%	-
Gait disturbance	10%	-
Seizure/change in seizure	10%	3%
Fatigue	9%	4%
Nausea	6%	3%
Balance disorder	6%	<1%
Diplopia	-	<1%

Antiseizure response rates

between opakalim and azetukalner in ongoing OLE studies*



Opakalim and azetukalner study participants have similar baseline characteristics including baseline seizure frequencies of ~13/month

1. French AES Poster #3.356 2025. **2.** Pooled patients, data as of 4Q 2025. **3.** French. Epilepsia Open. 2025. *Indirect comparisons between compounds based on publicly available data.

Opakalim Represents a Potential Paradigm-Shift for Patients With Epilepsy Based on its Exceptional Tolerability Profile

Kv7

	Cenobamate[1]	Perampanel[2]	Brivaracetam[3]	Azetukalner[4]	Opakalim[5]
Somnolence	28%	19%	10%	17%	3%
Dizziness	28%	42%	13%	25%	2%
Fatigue	17%	9%	8%	9%	4%
Headache	15%	12%	14%	19%	5%
Diplopia	9%	-	-	-	<1%
Nausea	7%	-	-	6%	3%
Balance disorder	6%	-	-	6%	<1%
Gait disturbance	6%	-	-	10%	-
Fall	5%	-	6%	15%	3%
Seizure/change in seizure	5%	-	11%	10%	3%
Memory impairment	-	-	-	11%	<1%
Weight gain	-	-	-	11%	-

1. Sander. Epilepsia. 2021. **2.** Rektor. Epilepsia. 2020. **3.** Toledo. Epilepsy Behav. 2021. **4.** French AES Poster #3.356 2025. **5.** Pooled patients from ongoing focal epilepsy open label study, data as of 4Q 2025.

KEY POINT

Opakalim exhibits much lower rates of commonly reported AEs, when comparing open-label data to that from 3 recently approved ASMs and 1 in development

Efficacy Signal Demonstrated in Child With Refractory KCNQ2-DEE

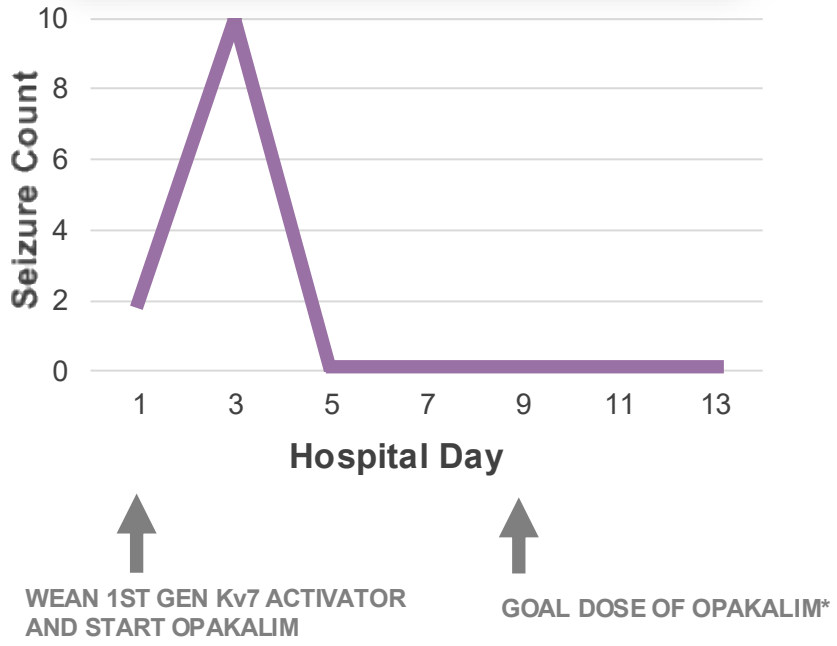
Kv7

9-YEAR-OLD BOY WITH KCNQ2-DEE HIGHLY DEPENDENT ON Kv7 ACTIVATION

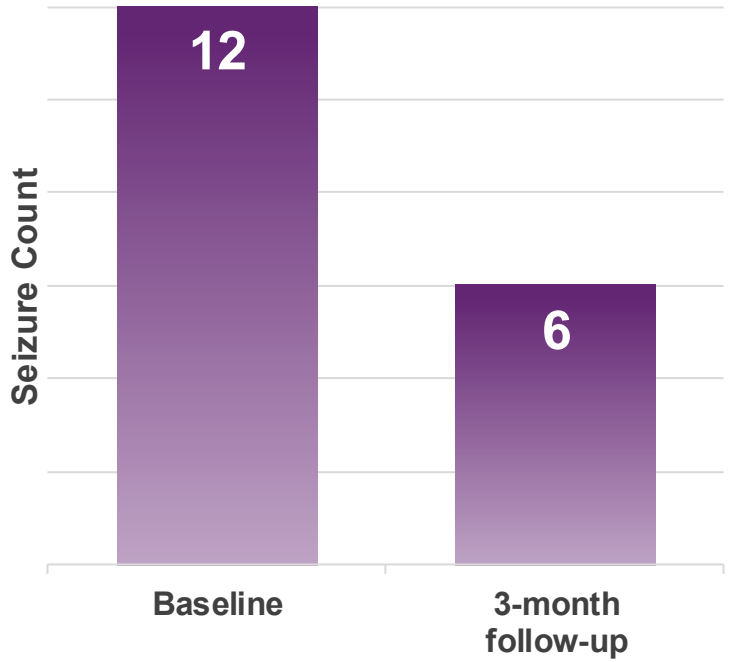
Daily seizures at baseline despite treatment with 3 ASMs including a 1st generation Kv7 activator

Multiple prior attempts to taper 1st gen Kv7 activator resulted in status epilepticus

12 days seizure free
after starting opakalim



3-month follow-up EEG showed
50% reduction in seizures



Olson AES Poster #3.469 2025. * Equivalent exposures to 75 mg dose in pivotal focal epilepsy studies.

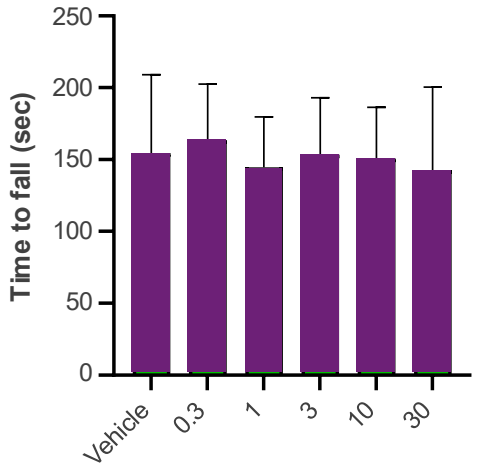
KEY POINT

- Successfully transitioned from 1st generation Kv7 activator to opakalim
- Well-tolerated with no treatment emergent adverse effects and behavior and alertness maintained

Opakalim Consistently Demonstrates Exceptional Tolerability Across All Stages of Development

Kv7

PRECLINICAL
No effects on motor performance on rotarod at effective doses



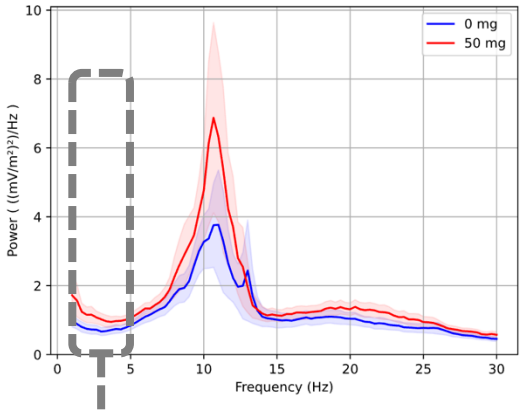
Data on file.

PHASE 1
Not associated with CNS AEs typical of other ASMs

CNS AEs	Opakalim Pooled MAD
Headache	11%
Dizziness	6%
Dysgeusia	2%
Hypoesthesia	2%
Paresthesia	2%
Presyncope	2%

Awsare AAN Poster #P9-011 2025.

EEG BIOMARKER STUDY
Minimal impact on spectral power in slower frequencies (delta) consistent with lack of somnolence



No increase in delta power

Lerner AES Poster #2.510 2023.

PH2/3 FOCAL OLE
Low rates of AEs including CNS AEs typical of other ASMs

AEs	Opakalim Pooled
Headache	5%
Fatigue	4%
URI	4%
Nasopharyngitis	3%
Back pain	3%
Fall	3%
Somnolence	**3%**
Pain in extremity	3%
Nausea	3%
Seizure	3%
Irritability	2%
Diarrhea	2%
Dizziness	**2%**

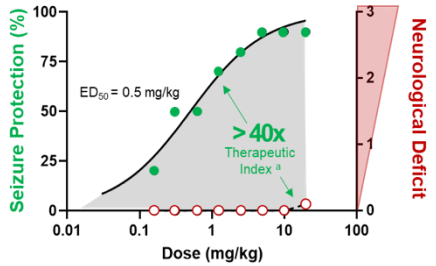
Preliminary data as of 4Q 2025.

AE, adverse event; **ASM**, antiseizure medicine; **CNS**, central nervous system; **OLE**, open-label extension.

Opakalim Consistently Demonstrates Potential for Efficacy in Epilepsy Across All Stages of Development

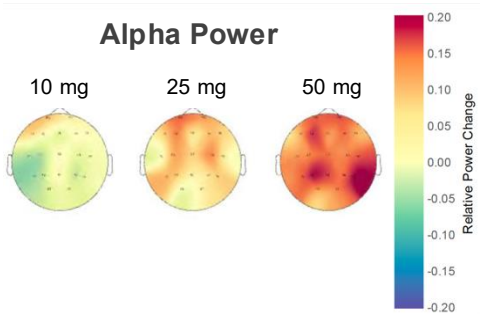
Kv7

PRECLINICAL
Seizure protection with a wide therapeutic index



Dworetzky ASENT Poster 2023.

EEG BIOMARKER STUDY
Dose-related pharmacodynamic effects in human brain



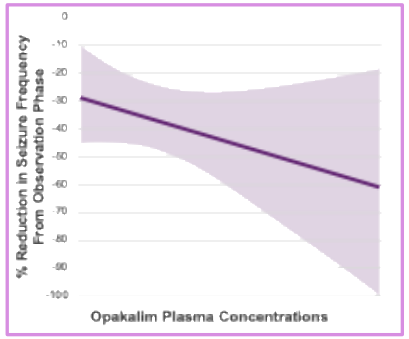
Lerner AES Poster #2.510 2023.

PH2/3 FOCAL OLE
50% responder rate comparable to other ASMs & clear exposure-response for antiseizure efficacy

50% RESPONDER RATE

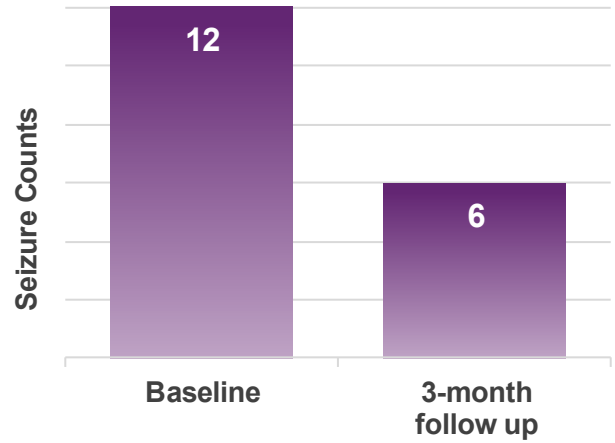
>50%

OVER THE FIRST 6 MONTHS OF OLE



Preliminary data as of 4Q 2025.

KCNQ2-DEE PATIENT
Successfully transitioned from 1st generation Kv7 activator to opakalim



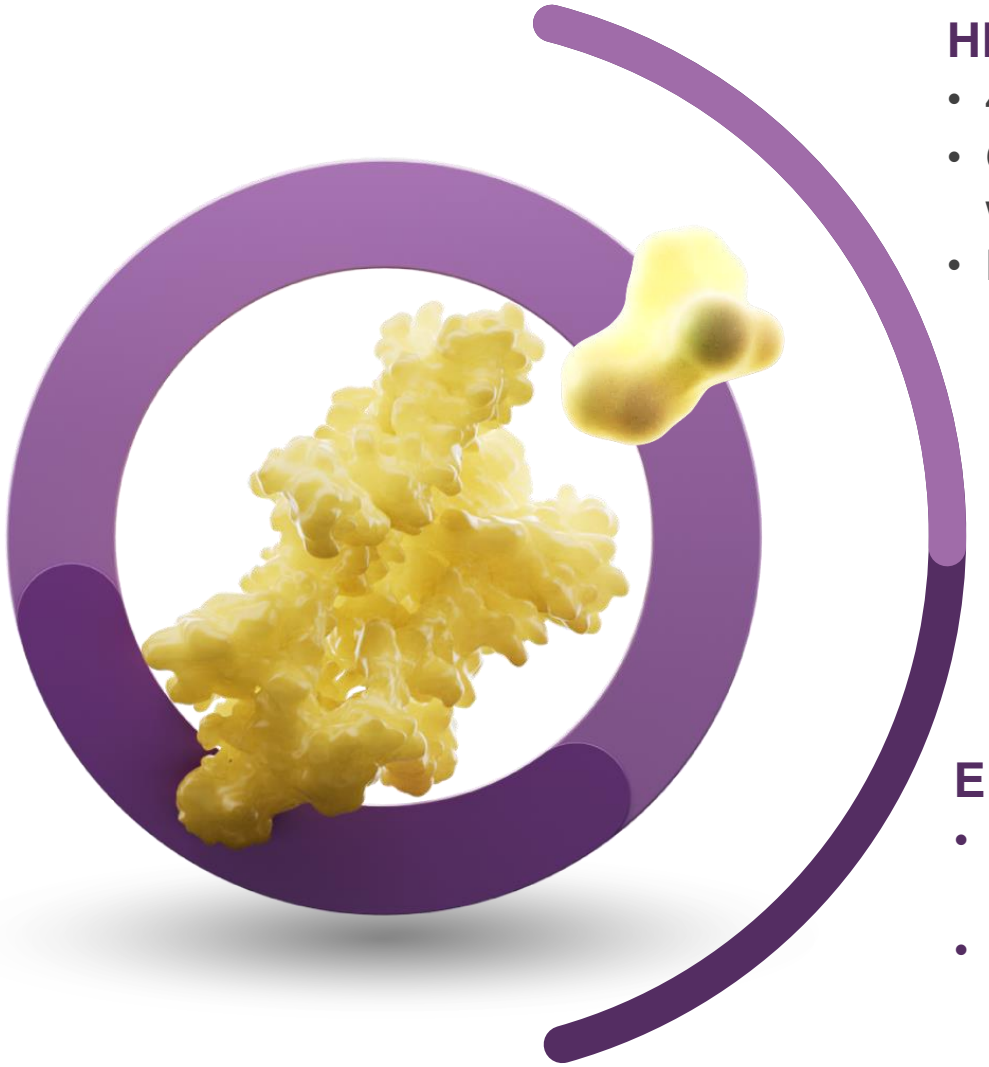
Seizure counts obtained during overnight video EEG at both time periods

Olson AES Poster #3.469 2025.

AE, adverse event; **ASM**, antiseizure medicine; **CNS**, central nervous system; **OLE**, open-label extension

Revolutionizing Epilepsy Treatment With Opakalim, a Modern Kv7 Activator

Kv7



HIGH UNMET NEED FOR NOVEL ANTISEIZURE MEDICINES

- 40% of the 3.5M PWE have seizures despite available treatments
- Over 50% of PWE have treatment-related adverse events, difficulty with adherence, exacerbation of comorbidities and lower quality of life
- Epilepsy costs over $24.5B annually

PROMISING PROFILE IN EARLY DEVELOPMENT

- **Preclinical:** Showed seizure protection with a wide therapeutic index
- **Phase 1:** Not associated with CNS AEs typical of other ASMs
- **EEG biomarker study:** Opakalim demonstrated target engagement measured by changes in EEG spectral power with minimal impact on slow wave frequencies (associated with somnolence)

EFFICACY SIGNALS WITH EXCEPTIONAL TOLERABILITY

- **Focal epilepsy Phase 2/3 OLE:** Data shows 50% responder rate comparable with other ASMs, along with low rates of AEs
- **KCNQ2-DEE compassionate use:** Child with refractory seizures successfully transitioned from 1st generation Kv7 activator to opakalim

AE, adverse event; **ASM**, antiseizure medicine; **CNS**, central nervous system; **PWE**, people with epilepsy.

biohaven®

MYOSTATIN ACTIVIN INHIBITOR: TALDEFGROBEP ALFA

Targeting High-Quality Weight Loss



Targeting High-Quality Weight Loss With Myostatin Activin Mechanism

TALDEFGROBEP

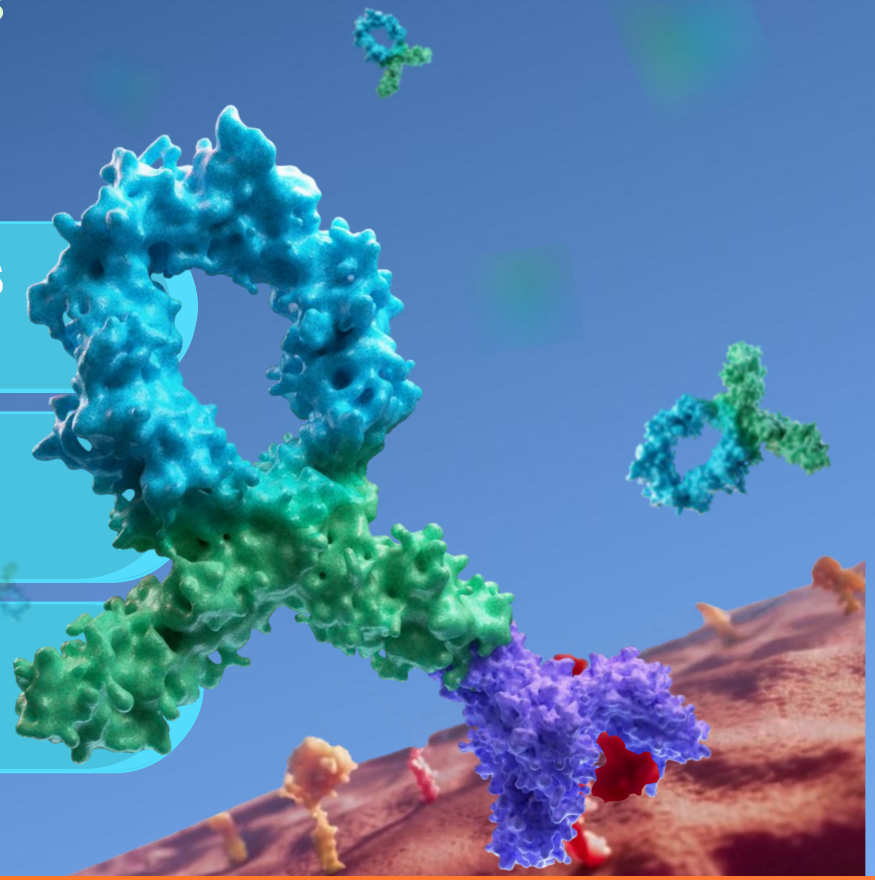
Taldefgrobep directly targets fat, builds muscle and increases bone density while avoiding intolerable adverse effects



MYOSTATIN ACTIVIN MOA FOR HEALTHY WEIGHT LOSS
Targeting myostatin, activin A and activin E/ALK7 signaling

SAFETY DATABASE IN >700 TREATED TO DATE
Differentiated safety profile

CONVENIENT DOSING
Potential for once monthly self-administration



BREAKING NEWS

Initiated Phase 2 monotherapy study with topline expected 2026

Unmet Needs Remain in Obesity Management Despite New Therapies

TALDEFGROBEP

Unprecedented reductions in total body weight are limited by:

Significant muscle loss
Up to 40% of GLP-1-induced weight loss is lean mass[1,2]

Increased Risk for Fractures
Pelvic fractures were 4–5x more common on Wegovy vs PBO[3]

Intolerable adverse events
Two-thirds stop GLP-1 therapy within 1 year[4]



Opportunity in obesity management:

Increase muscle mass and bone density while reducing fat mass
Including metabolically-active VAT

Minimal or no clinically meaningful adverse events
Suitable for chronic administration

Support convenient dosing
Potential for monthly dosing in an easy-to-use autoinjector for better adherence

VAT, Visceral Adipose Tissue.

1. Wilding. N Engl J Med. 2021. **2.** McCrimmon. Diabetologia. 2020. **3.** Wegovy USPI. Update NOV 2025. Accessed 29-DEC-2025. **4.** Prillman. Scientific American. 2024.

KEY POINT

Taldefgrobep targets the limitations of current therapies, offering a novel and differentiated solution for patients living with obesity

Clinical Data Has Validated Myostatin Activin Inhibitor Class Uncovering Optimal Profile for Efficacy and Safety in Obesity

TALDEFGROBEP

	Fat Reduction	Weight Loss	Favorable Safety Profile	Potential SC Monthly Dosing	MOA Directly Targeting: Adipose Tissue	MOA Directly Targeting: Skeletal Muscle
Taldefgrobep (Biohaven)	✓	✓	✓	✓	✓	✓
Bimagrumab Activin Receptor Blocker (Lilly)	✓	✓	✗	✗	✓	✓
Apitegromab*, SRK439 Anti-Myostatin (Scholar Rock)	✗	✗	✓	✗	✗	✓
Trevogrumab* Anti-Myostatin (Regeneron)	✗	✗	✓	✗	✗	✓
Trevogrumab + Garetosmab* Anti-Myostatin + Anti-Activin A (Regeneron)	✓	✓	✗	✗	✗	✓
WVE-007, ARO-INHBE, ARO-ALK7 Activin E or ALK7 siRNA (Wave, Arrowhead)	✓	✓	?	✓	✓	✗

*Apitegromab, Trevogrumab and Trevogrumab + Garetosmab Phase 2 studies were conducted in combination with a GLP-1 agent.

KEY POINT

Taldefgrobep offers optimal mechanistic profile for efficacy and safety

Myostatin Activin Pathway Inhibition Demonstrates Benefits in Total Body Weight, Fat and Muscle

TALDEFGROBEP

BELIEVE: 1-Year Trial of Bimagrumab in Adults Living with Overweight or Obesity

	Placebo	Bimagrumab (30 mg iv)	Semaglutide (2.4 mg sc)	Combination (bima + sema)
EFFICACY				
Total body weight (W48)	**-2.5%**	**-9.7%**	-14.3%	-20.2%
Total fat mass (W48)	-5.2%	**-25.3%**	**-24.8%**	-42.2%
Total lean mass (W72)	-0.5%	**2.5%**	-7.4%	-2.9%
Visceral Adipose tissue (W48)	-2.1%	**-40.2%**	-29.5%	-54.8%
SAFETY				
Muscle Spasms	5.5%	**73.7%**	8.9%	63.6%
Diarrhea	5.5%	**49.1%**	35.7%	49.1%
Acne	3.6%	**43.9%**	8.9%	52.7%

- Exceeds 5% differential guideline set by the FDA
- Change comparable to semaglutide
- Preservation/improvement in lean mass
- Greater reduction in metabolically-active VAT vs semaglutide

Poor tolerability due to irreversible ActRIIB binding

VAT, Visceral Adipose Tissue.

KEY POINT

Taldefgrobep targets bimagrumab-like efficacy with a favorable safety/tolerability profile

Taldefgrobep Avoids GI- and Muscle-Related AEs Commonly Reported in Bimagrumab Clinical Trials

TALDEFGROBEP

Muscle-/GI-Related AEs	**Taldefgrobep** SAD/MAD Pooled[1] 15-180 mg n=103	**Bimagrumab** 30 mg/kg[2] Single Dose Study n=10	**Bimagrumab** 10 mg/kg[3] Q4W Multi-dose Study n=37
Acne	**0%**	**30%**	3%
Muscle spasm	**3%**	**30%**	**41%**
Musculoskeletal stiffness	**0%**	**30%**	NA
Myalgia	**1%**	**30%**	NA
Muscle weakness	**1%**	**10%**	NA
Diarrhea	**2%**	**10%**	**41%**
Nausea	**1%**	NA	**11%**
Lipase level increased	**0%**	0%	**11%**

NA = data not available

1. Study CN001001 conducted in healthy adults receiving taldefgrobep (15-180 mg QW x 1 month). **2.** Garito. Diabetes Obes Metab. 2018. **3.** Heymsfield. JAMA Network Open. 2021.

KEY POINT

Favorable safety profile established in >700 participants across diverse clinical populations

Taldefgrobep Has Direct Effects on Muscle and Fat — Optimizing Quality of Weight Loss

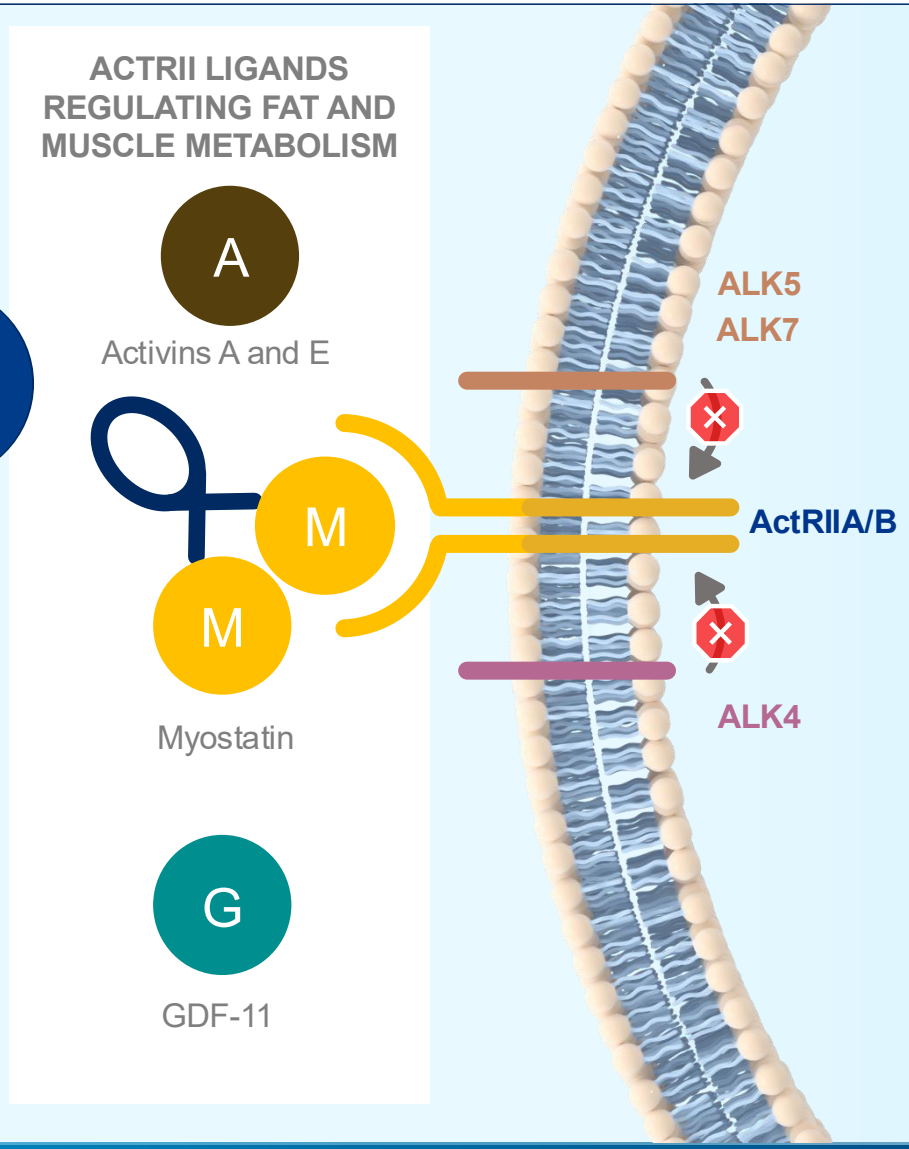
TALDEFGROBEP

Taldefgrobep

BINDS
active myostatin, GDF-11

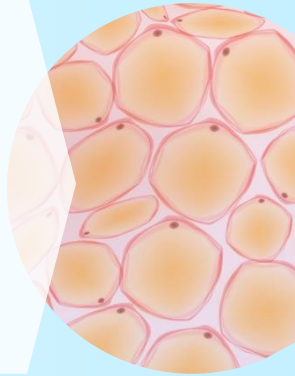
FORMS COMPLEX
that attaches to ActRIIA/B
prevents ActRII binding (ALK4,5,7)

INHIBITS
ActRIIA/B signaling by key ligands including Activin A and Activin E



Direct effect on adipocytes leads to **lipolysis**

Increased mitochondrial content and reduced fat storage

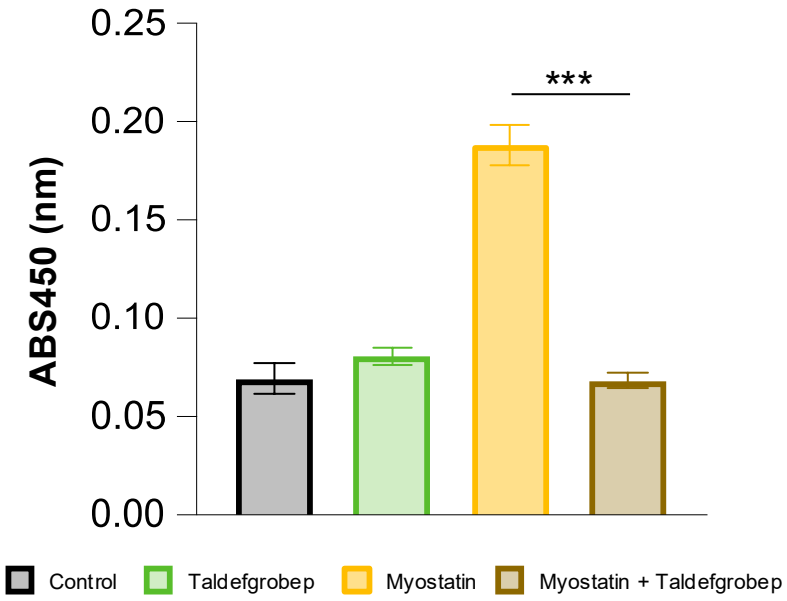


Inhibiting signal transduction in skeletal muscle leads to **increased muscle mass**

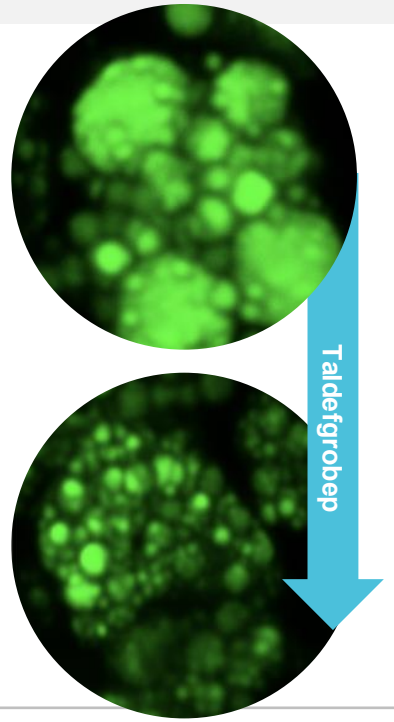


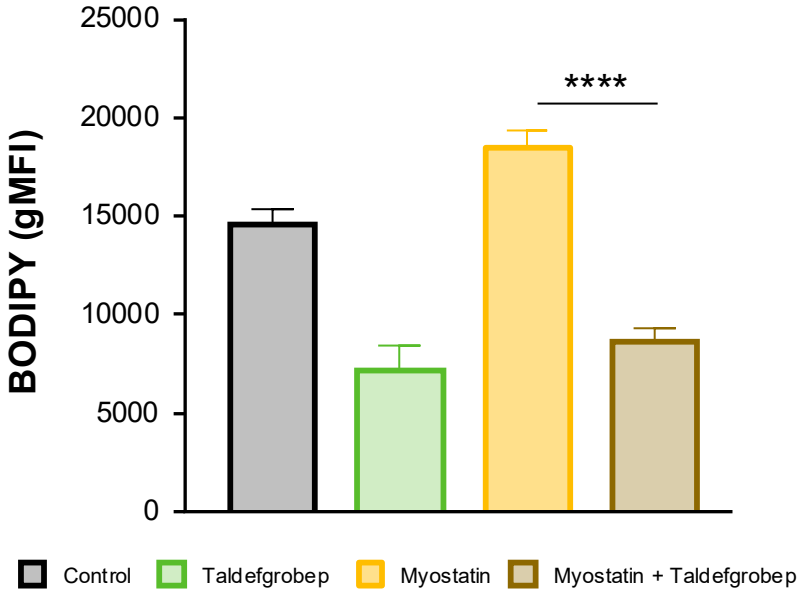
Taldefgrobep Inhibits ActRII / ALK7 Signaling to Reduce Fat Storage

Taldefgrobep Decreases Phospho-SMAD2/3 Signaling in Adipocytes



Taldefgrobep Reduces Fat Storage in Adipocytes[1]





Significance tested with two-tailed t test. ***P<0.001; ****P<0.0001.
gMFI, geographic mean fluorescence intensity.
1. Ma E et al. ObesityWeek 2025.

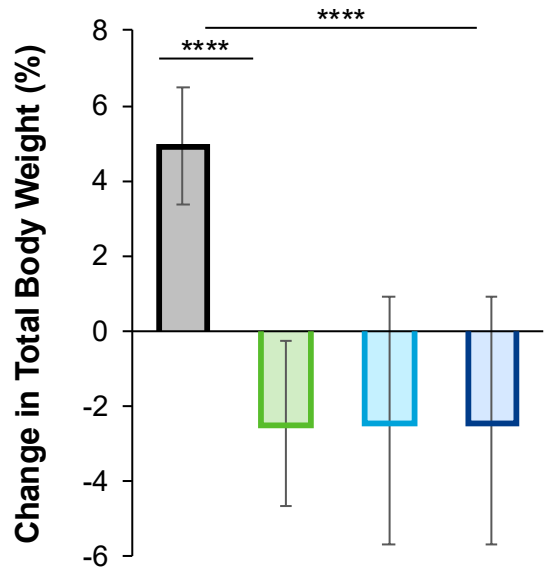
KEY POINT

Taldefgrobep directly reduces adipose tissue storage of fat

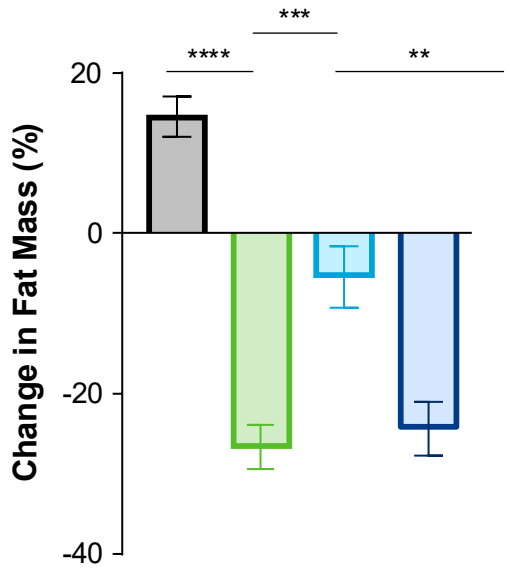
In an Obese Mouse Model, Taldefgrobep Demonstrated Significant Reductions in Fat Mass While Increasing Lean Mass

TALDEFGROBEP

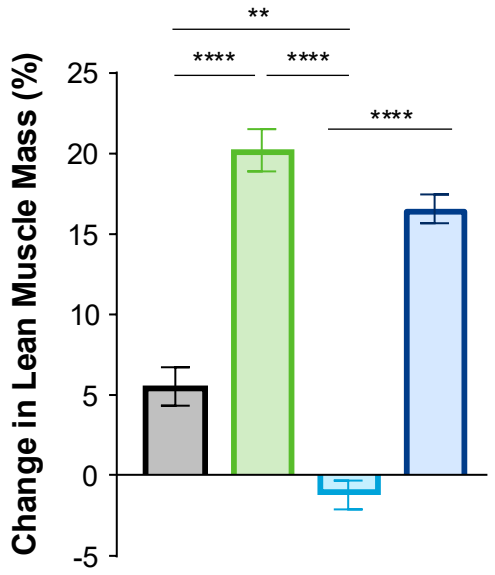
Change in Total Body Weight, From Baseline at 8 Weeks



Change in Fat Mass From Baseline at 8 Weeks



Change in Lean Muscle Mass From Baseline at 8 Weeks



Vehicle | Taldefgrobep | Semaglutide (40 µg/kg QD) | Taldefgrobep + Semaglutide (40 µg/kg QD)

n = 15 for vehicle; n = 16 for all other groups. Error bars represent standard error of the mean. Significance evaluated using Tukey's multiple comparisons test. **P < 0.01; ***P < 0.001; ****P < 0.0001; **QD,** once daily; taldefgrobep alfa. Bechtold. ObesityWeek 2024. Poster 350.

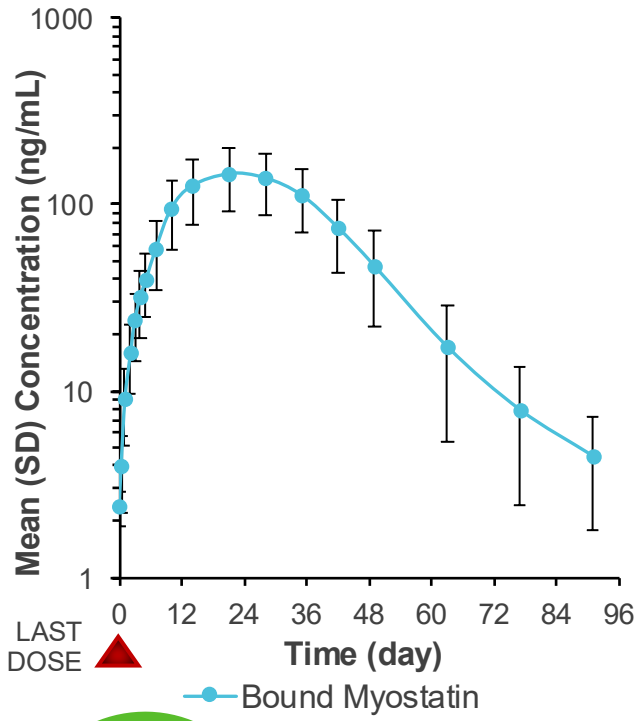
KEY POINT

Taldefgrobep demonstrated reductions in fat mass and increases in lean mass as monotherapy and in combination with a GLP-1 receptor agonist

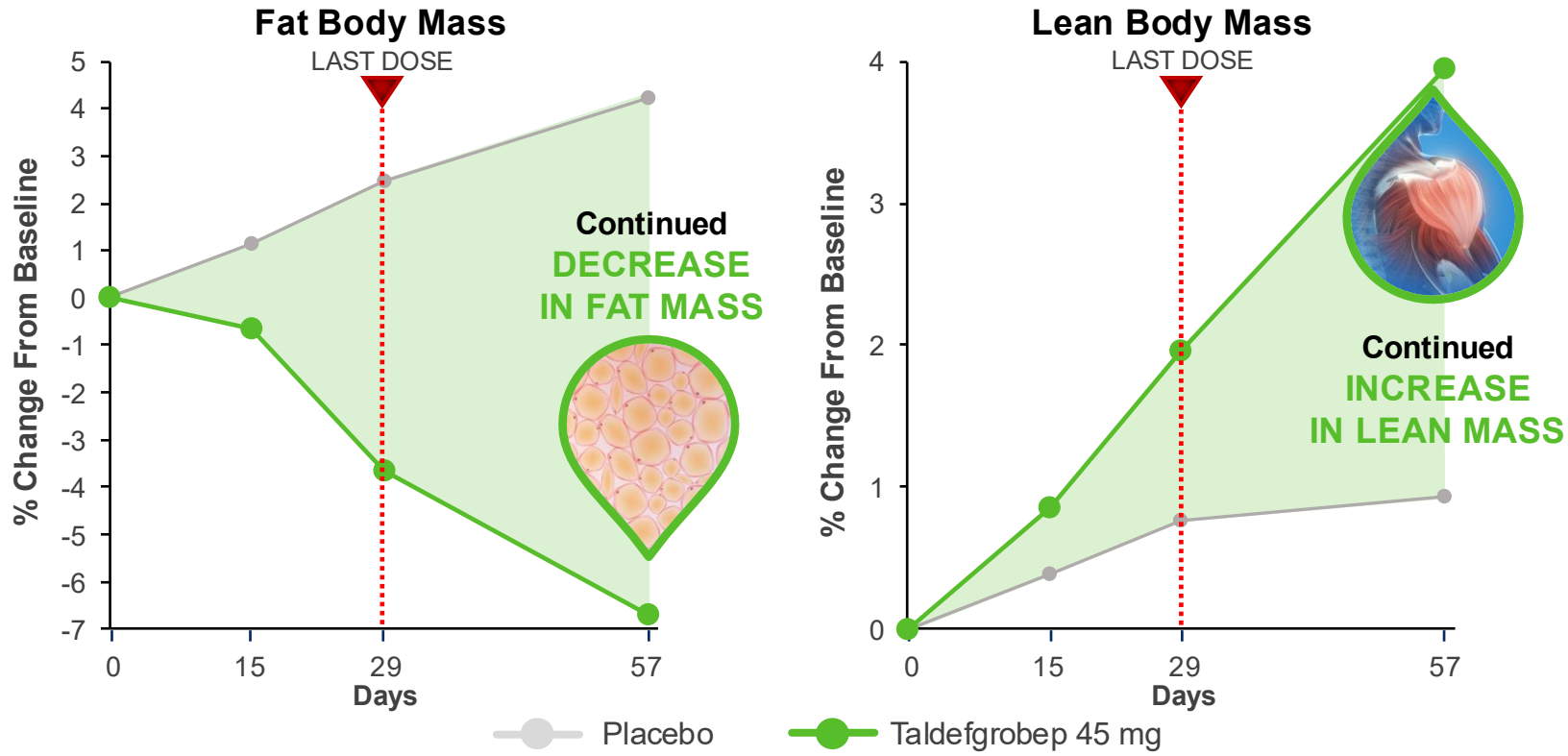
Taldefgrobep Improves Body Composition in Non-Obese Adults with Potential for Monthly Dosing in Obesity

TALDEFGROBEP

Peak Plasma Concentrations of **Taldefgrobep-Myostatin Complex** Occurs 20-30 Days Post Dose



In Healthy Adults at One Month Post Dosing, **Taldefgrobep** Demonstrates:

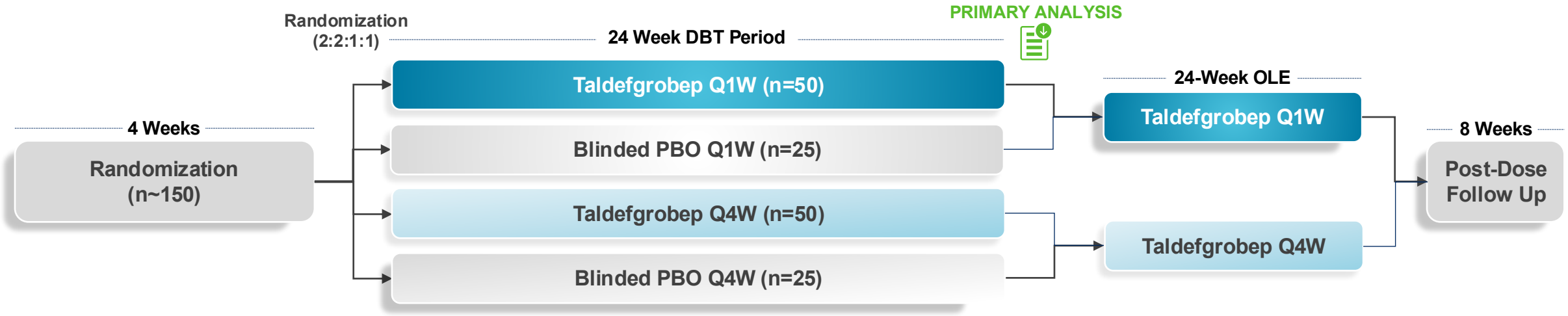


KEY POINT

Taldefgrobep-myostatin complex increases lean mass and also directly inhibits Activin E / ALK7 signaling in adipose tissue

Taldefgrobep Monotherapy Dose-Ranging (Q1W and Q4W) Study Initiated

TALDEFGROBEP



KEY STUDY DETAILS

Study Design: Phase 2, randomized, double-blind, placebo-controlled dose-ranging study

Population: Male and female adults (18 to 65 years-old) with overweight or obesity

Endpoints: % change in total body weight, fat mass and lean mass at Week 24

KEY POINT

FPFV achieved 4Q 2025 with topline results expected in 2026

Taldefgrobep Offers a Novel Approach to Address the Needs for People Living With Obesity

TALDEFGROBEP



Fat mass loss comparable to GLP-1 therapies

Total body weight loss meeting current regulatory standards

Visceral adipose tissue loss favorable to GLP-1 therapies

Increase in lean muscle mass

Increase in bone density

Benefit as monotherapy and in combination with GLP-1 therapies

Favorable safety and tolerability

Convenient subcutaneous autoinjector with potential for monthly dosing

biohaven®



ONCOLOGY

Next-Generation ADC Technologies: Built to Deliver Improved Efficacy, Safety and Scalability

Developing Highly Differentiated Next-Generation ADCs

ONCOLOGY

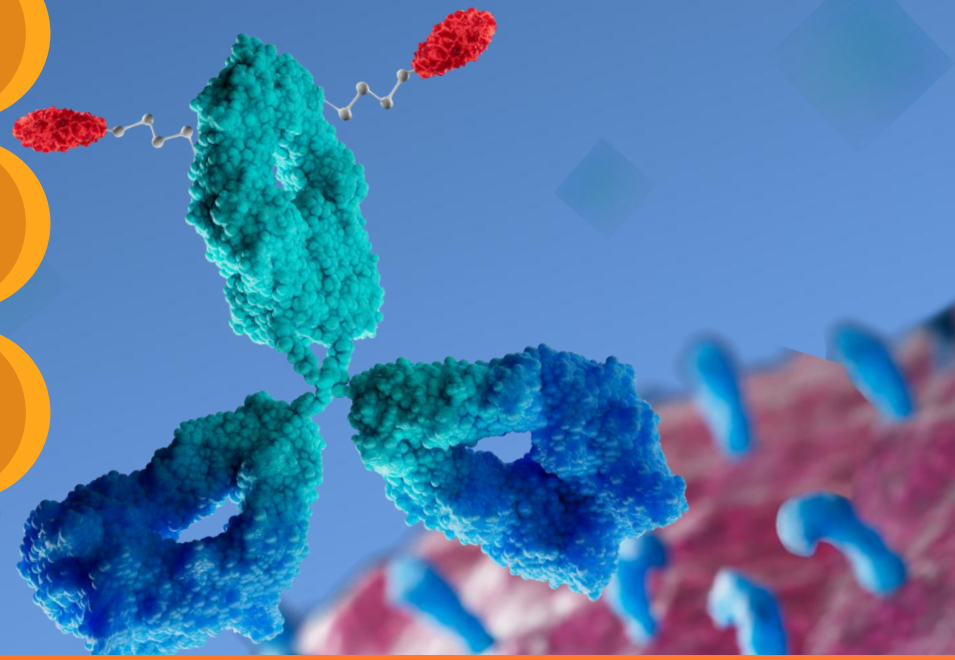
Proprietary platform solving for superior efficacy, safety, stability, and scalability

FIRST CLINICAL DEMONSTRATION WITH BHV-1510 TROP2 ADC
- Compelling activity with checkpoint combination
- Highly stable, differentiated PK and safety

FIRST-IN-HUMAN FGFR3 ADC (BHV-1530)
- Early dose escalation with no DLTs
- First-in-class potential in FGFR3 driven tumors

CLINICALLY DIFFERENTIATED TOPOIX PAYLOAD

Expanding high-value targets including bispecifics



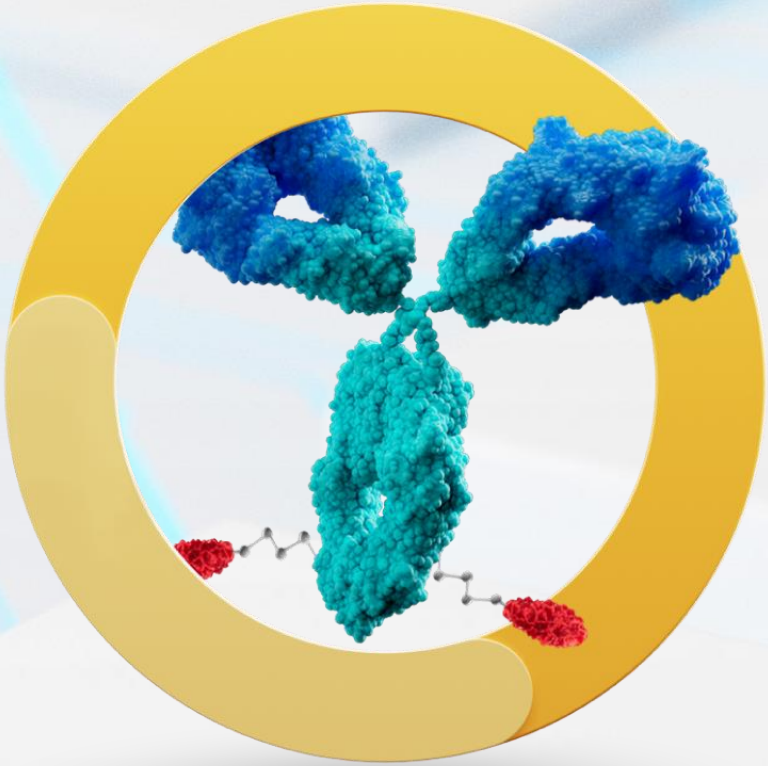
BREAKING NEWS

First patients dosed with novel subcutaneous ADC (BHV-1510)

biohaven®

BHV-1510

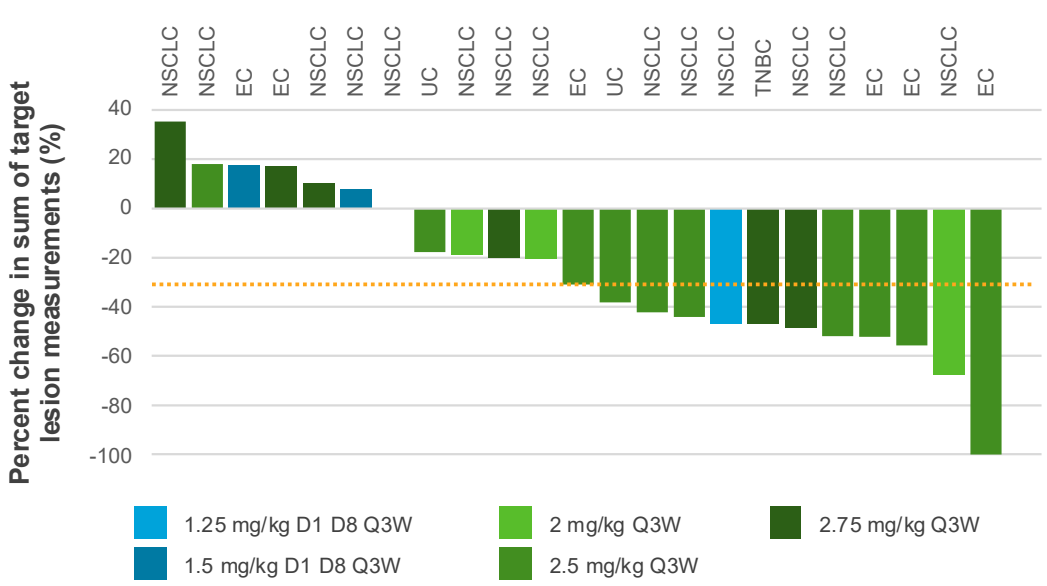
Trop2 ADC



BHV-1510 + Cemiplimab Leads to Rapid, Deep and Durable Responses in Heavily Pretreated Patients, Majority With Prior Anti-PD(L)1

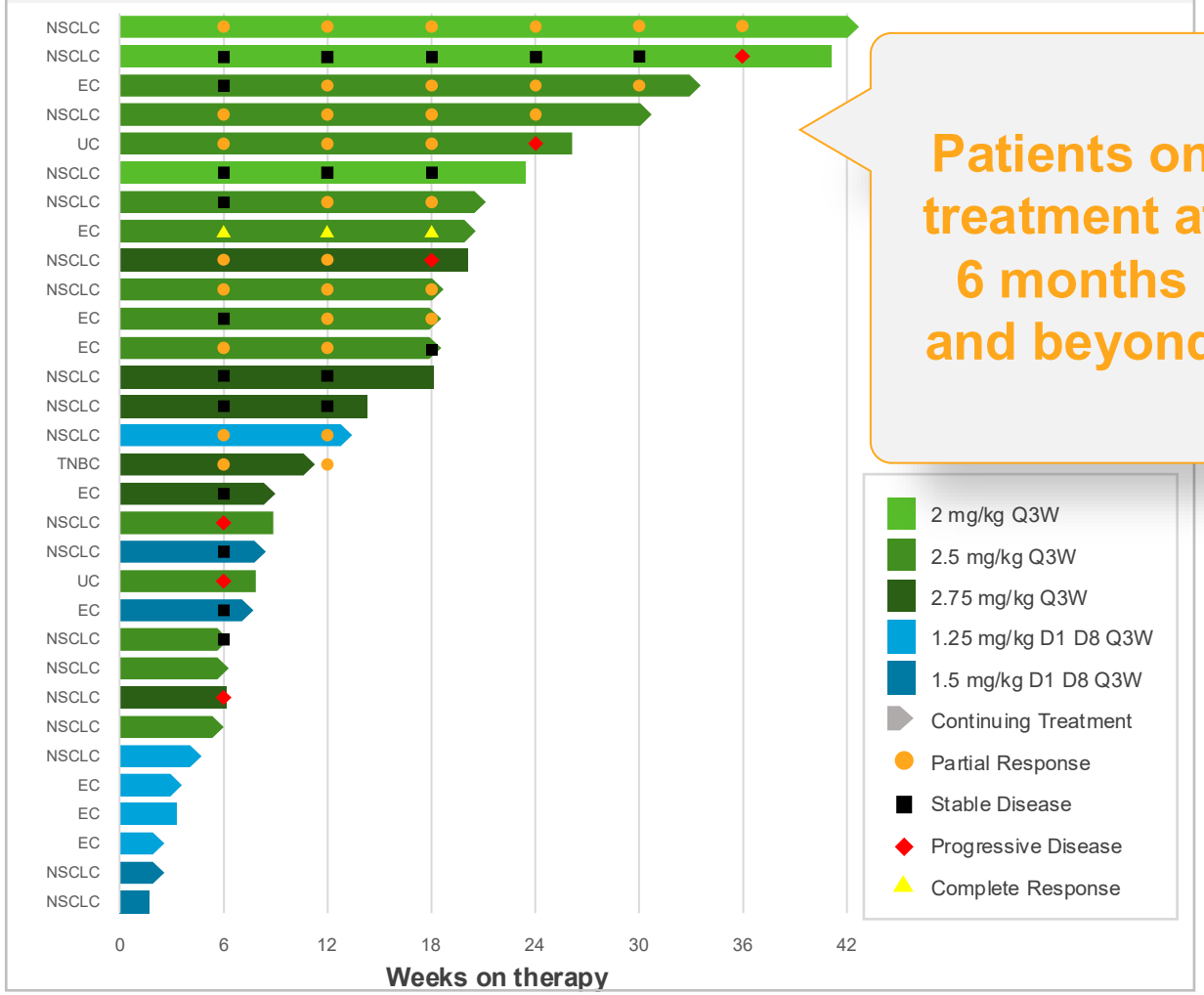
ONCOLOGY

BHV-1510 + Cemiplimab*: Percent Change in Target Lesions



Majority (87.1%) had prior anti-PD(L)1 exposure

BHV-1510 + Cemiplimab: Treatment Duration



Patients on treatment at 6 months and beyond

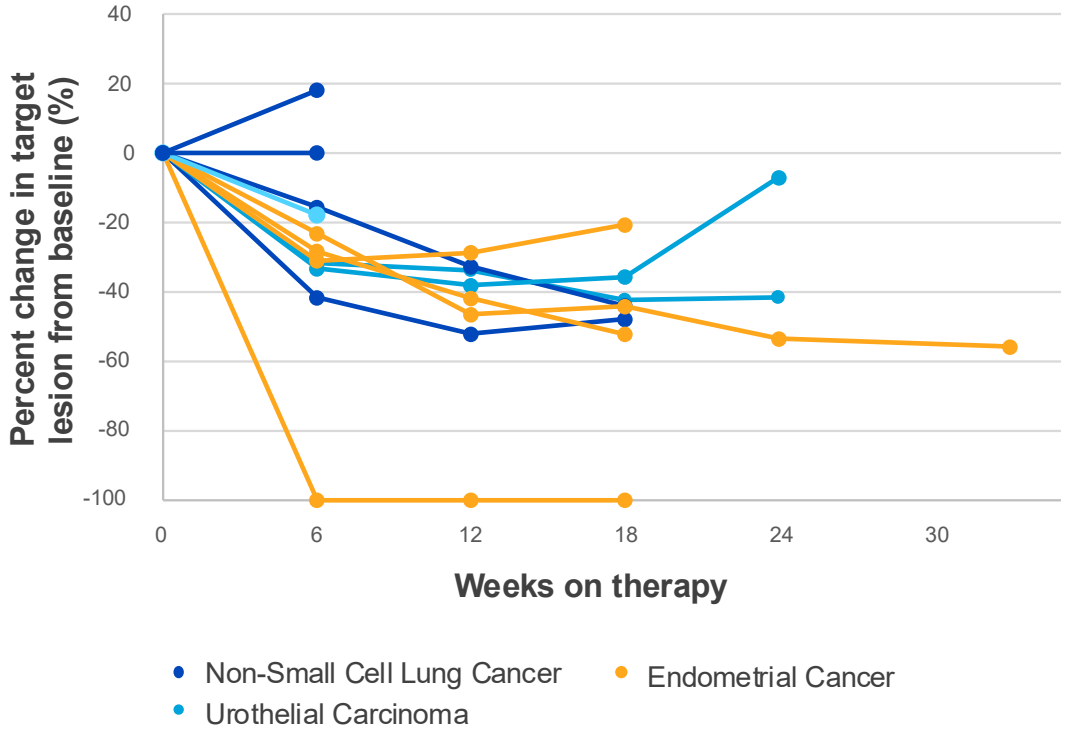
*Cemiplimab (anti-PD1) provided through collaboration agreement with Regeneron; dose of cemiplimab 350 mg Q3W
Source: Biohaven poster presented at 2025 ESMO Immuno-Oncology Congress.

Encouraging Early Clinical Activity and High Response Rates of BHV-1510 + Cemiplimab

ONCOLOGY

- **Compelling efficacy at 2.5 mg/kg Q3W with confirmed ORR 73% (8/11)**
  - NSCLC 60% (3/5)
  - Endometrial cancer 100% (4/4)
  - Urothelial cancer 50% (1/2)
- **Activity in difficult-to-treat patients:** Responses observed in patients with brain metastases; heavily pretreated, majority with prior anti-PD(L)1
- **Rapid onset of benefit:** Tumor shrinkage / PRs at 1st scan
- **Differentiated safety profile:** Low rates of hematological toxicities and diarrhea; no ILD observed

BHV-1510 2.5 mg/kg + Cemiplimab Q3W: Target Lesion Change from Baseline



KEY POINT

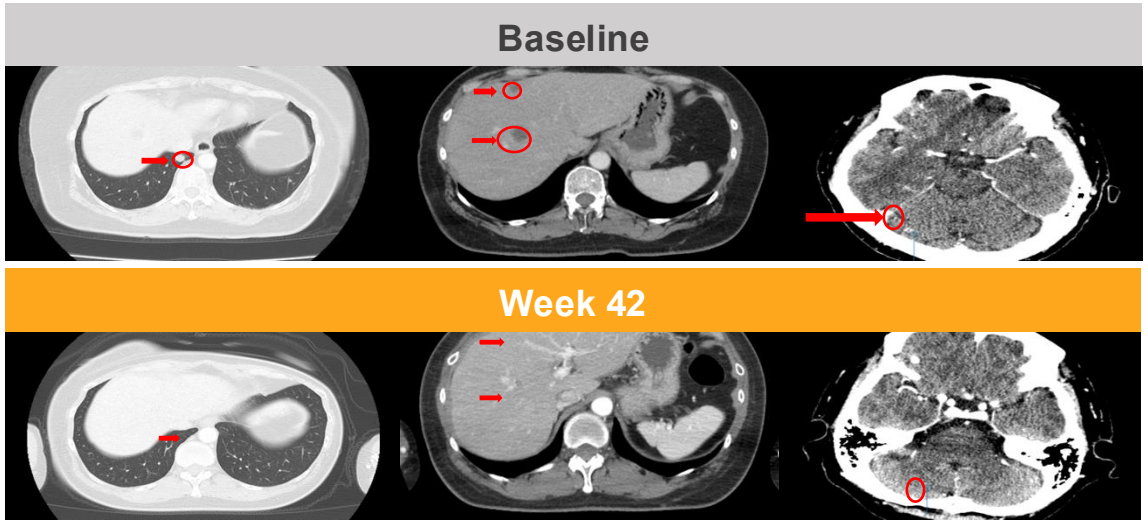
- Early data suggests synergy with anti-PD1 and potential to move into earlier lines
- Expansion cohort initiated in endometrial cancer

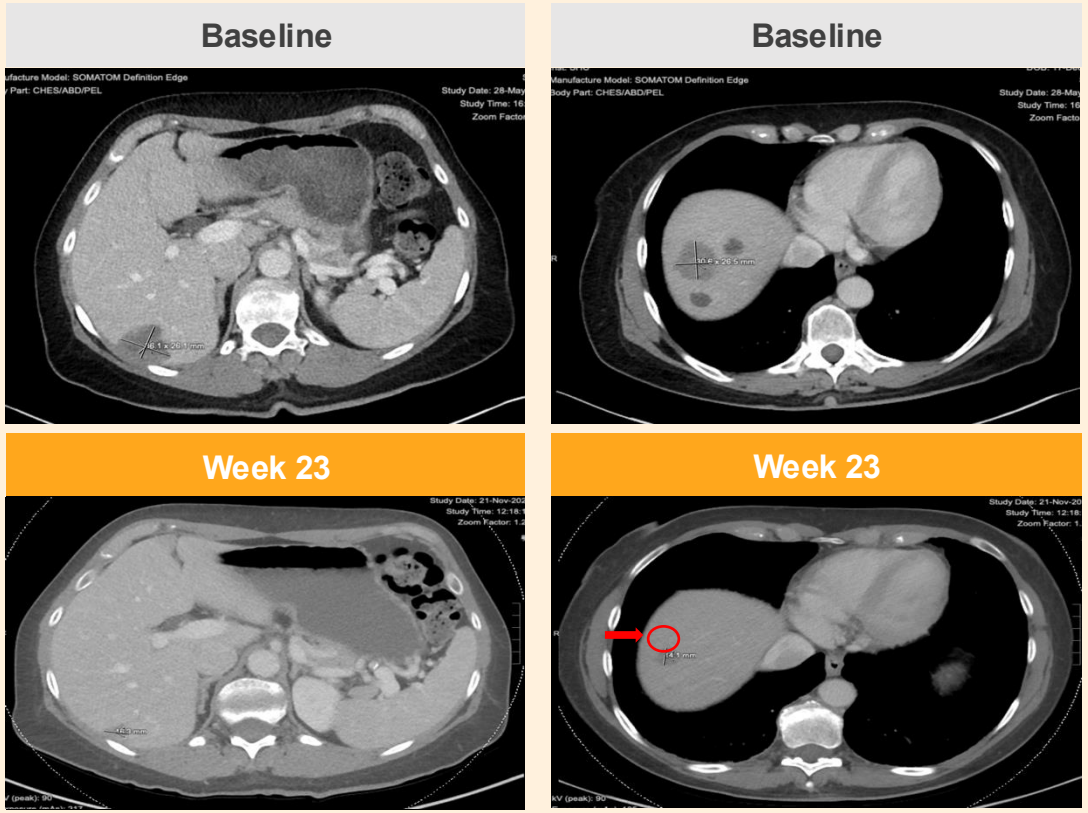
Objective Responses in Patients Failing SOC Therapy Including Targeted Therapy and Immunotherapy

ONCOLOGY

Case Study #1

- A 57-year-old female with EGFRmt NSCLC with confirmed partial response (67% reduction) continuing on therapy at 42 weeks with deepening response
- Prior therapies included multiagent chemotherapy and two EGFR targeting agents





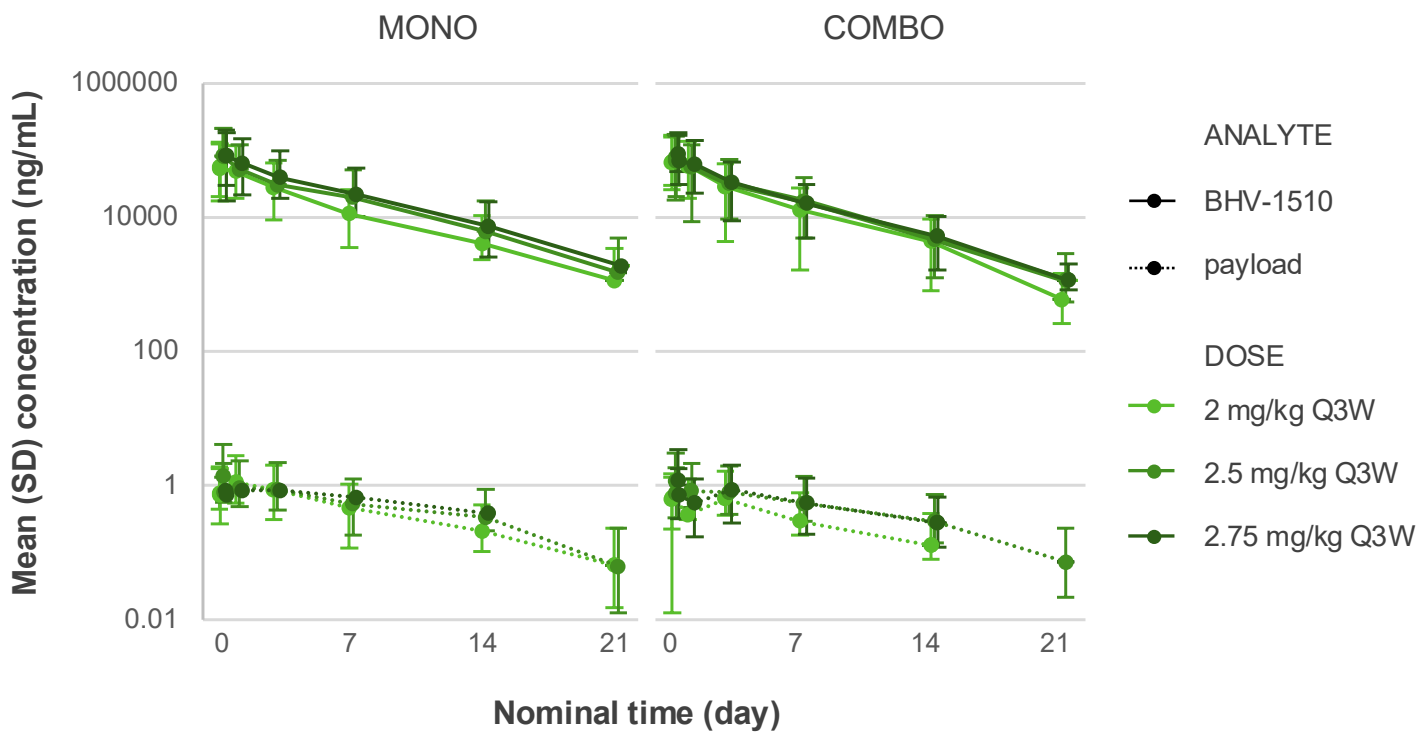
Case Study #2

- A 63-year-old female with endometrial carcinoma with a confirmed partial response (53% reduction)
- Prior therapies included chemotherapy and anti-PD-1 therapy

BHV-1510 Demonstrates a Favorable PK Profile With Highly Stable ADC

ONCOLOGY

BHV-1510 (ADC) and Payload Concentration-Time Plots
for Cycle 1 (with or without Cemiplimab)



KEY POINT

The unconjugated payload concentration was low with a payload-to-ADC molar ratio <1%, indicating that ADC was highly stable in the circulation

First and Only Trop2 ADC in Clinic With Subcutaneous Delivery

ONCOLOGY

Initial patients dosed with SC BHV-1510

- Target PK profile achieved
- Early data shows optimized potential with no dose limiting toxicities, no stomatitis in cycle 1
- First patient dosed with 25% reduction in target lesions after single SC administration

SC delivery offers several key advantages

- **Patient friendly delivery:** No IV access needed; minutes versus hours with competitors
- **Reduced site-of-care burden:** Less infusion-suite demand; supports community clinics
- **Optimized PK:** Potential for improved tolerability and efficacy

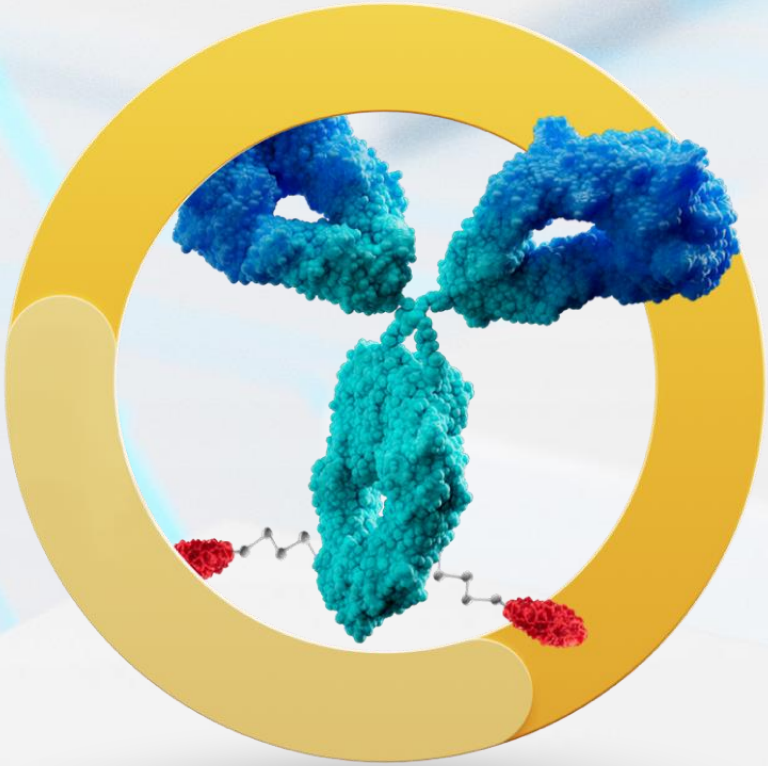
KEY POINT

Advancing SC to optimize safety and efficacy; potentially differentiating Biohaven's ADC platform from competitors

biohaven®

BHV-1530

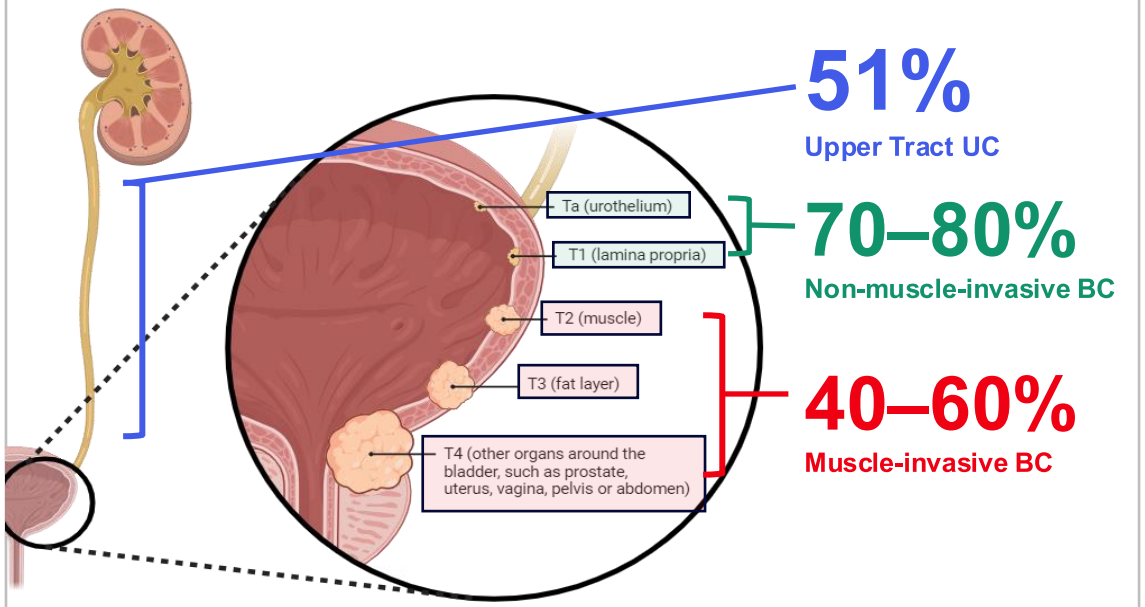
FGFR3 ADC



FGFR3 Is a Promising Therapeutic Target for Urothelial Cancer (UC) and Other Tumors With High Unmet Need

ONCOLOGY

FGFR3 Overexpression and Alteration (%) in Urothelial Carcinoma



~15% of HNSCC have FGFR3 mutations with overexpression noted in nearly half of oral and oropharyngeal cancers

38% of lung cancers with FGFR3 expression suggests substantial overexpression

5% of endometrial cancers have FGFR3 alterations, potentially indicating more widespread overexpression

4% of cervical cancers exhibit the FGFR3-TACC3 fusion, which is associated with higher FGFR3 expression

~ 8% of glioblastomas show a FGFR3-TACC3 fusion, commonly linked to increased levels of FGFR3 protein

HN, Head and neck squamous cell carcinomas; **SCLC**, Small cell lung cancer; **UC**, Urothelial carcinoma.

KEY POINTS

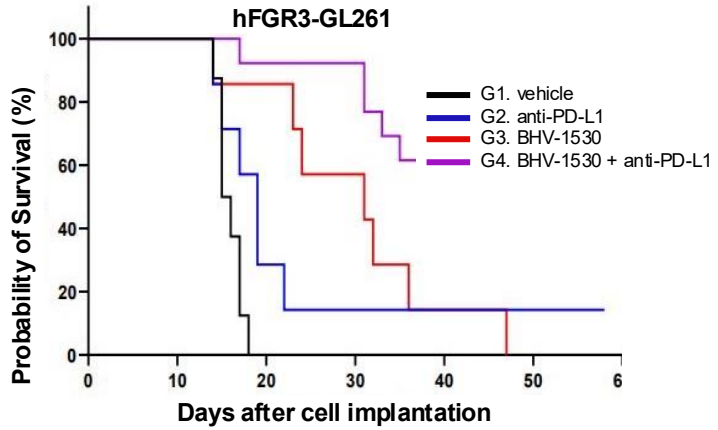
BHV-1530 first-in-class opportunity to treat FGFR3-altered *and* overexpressed patient across multiple tumors

BHV-1530: First-in-Class ADC With Proprietary TopoIx Payload Synergistic With Anti-PD(L)1

ONCOLOGY

- **Compelling preclinical efficacy across FGFR3-altered and FGFR3-overexpressing tumor models:** Demonstrated as monotherapy and in combination with CPI
- **Clinical progress:** First patient dosed 2Q 2025; no DLTs or emerging safety signals to date
- **Early signs of activity:** Early tumor reduction observed in a patient with an FGFR3 mutation
- **PK profile consistent with next-generation ADC platform:** Highly stable ADC conjugate

BHV-1530 shows **synergistic activity** *in vivo* with anti-PD-L1 combination



Group	% Increased Life Span (ILS)	Median Survival (days)
G1	-	15
G2	27%	19
G3	107%	31
G4	>300%	>63

KEY POINT

FIH study ongoing with no DLTs to date, dosing in the anticipated efficacious range

biohaven®

ION CHANNEL: BHV-2100 TRPM3 ANTAGONIST

Targeting Pain at its Source

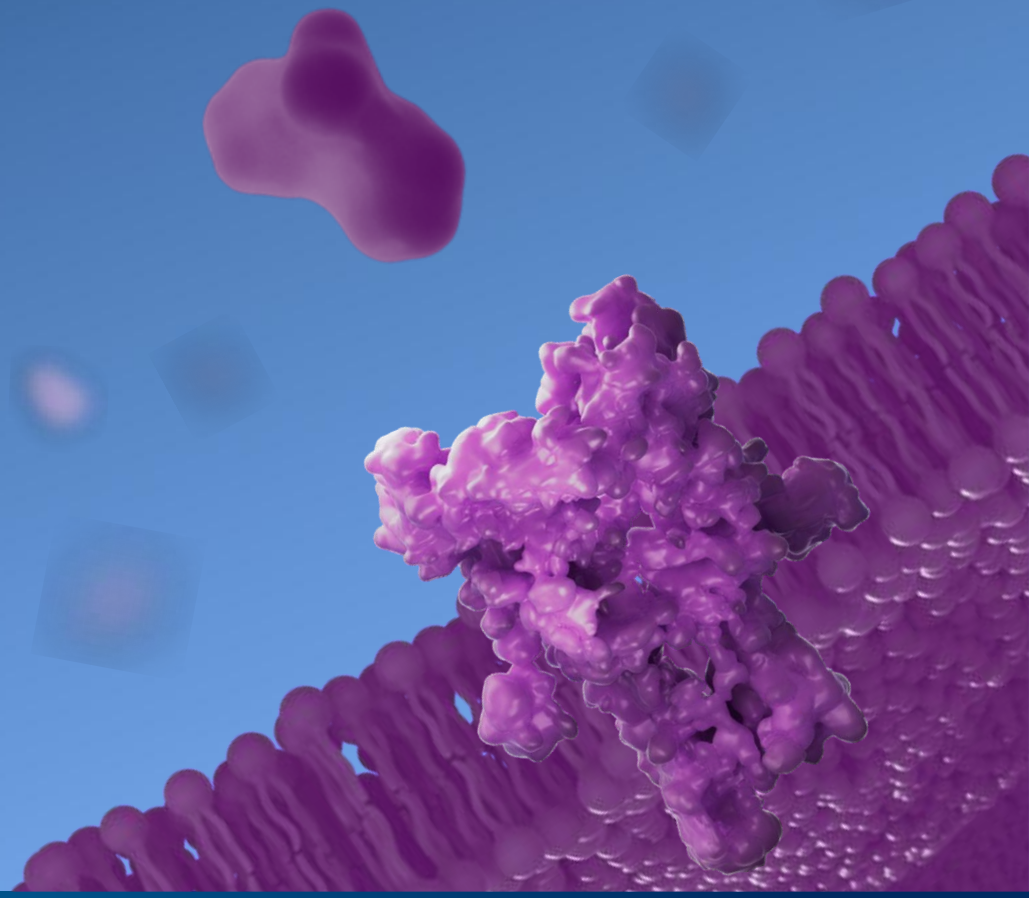


BHV-2100: Phase 2 First-in-Clinic TRPM3 Antagonist for Pain

TRPM3

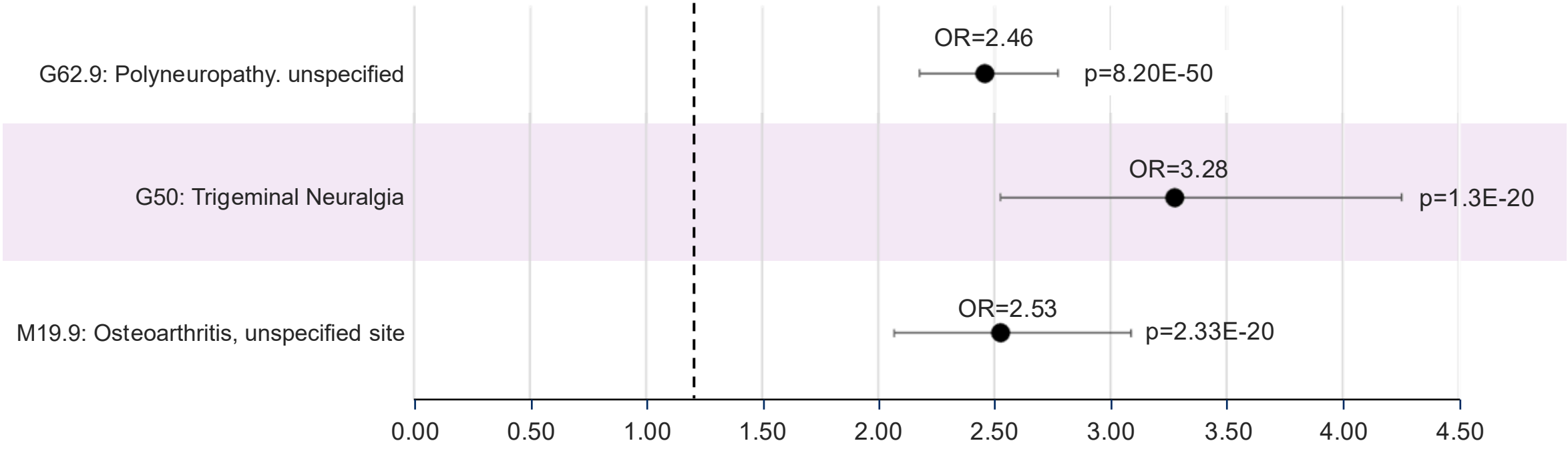
Oral, peripherally acting small molecule that targets pain at its source—peripheral nociceptors—and avoids the liabilities of other TRP channel modulators

- **NOVEL MOA WITH HUMAN GENETIC VALIDATION**
- **ROBUST EFFICACY IN NONCLINICAL PAIN MODELS**
- **FAVORABLE CLINICAL PHARMACOLOGY**
- **LOW POTENTIAL FOR CNS SIDE EFFECTS**
- **NO TEMPERATURE-RELATED LIABILITY IN HUMANS**



UK Biobank Analysis Provides Human Genetic Validation for TRPM3

TRPM3



OR: Odds Ratio.
p value claculated using the Benjamini-Hoschberg procedure for False Discovery Rate; error bars represent 95% confidence intervals

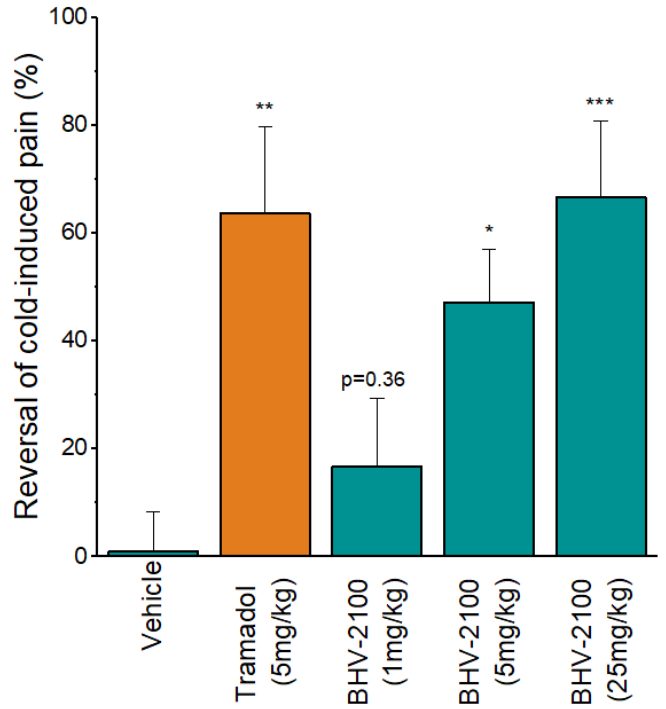
KEY POINT

TRPM3 variants increase the risk for neuropathic pain, trigeminal neuralgia and osteoarthritis, further corroborating with preclinical experiments

BHV-2100: Peripheral TRPM3 Antagonism Reverses Pain in Multiple Neuropathic Pain Models

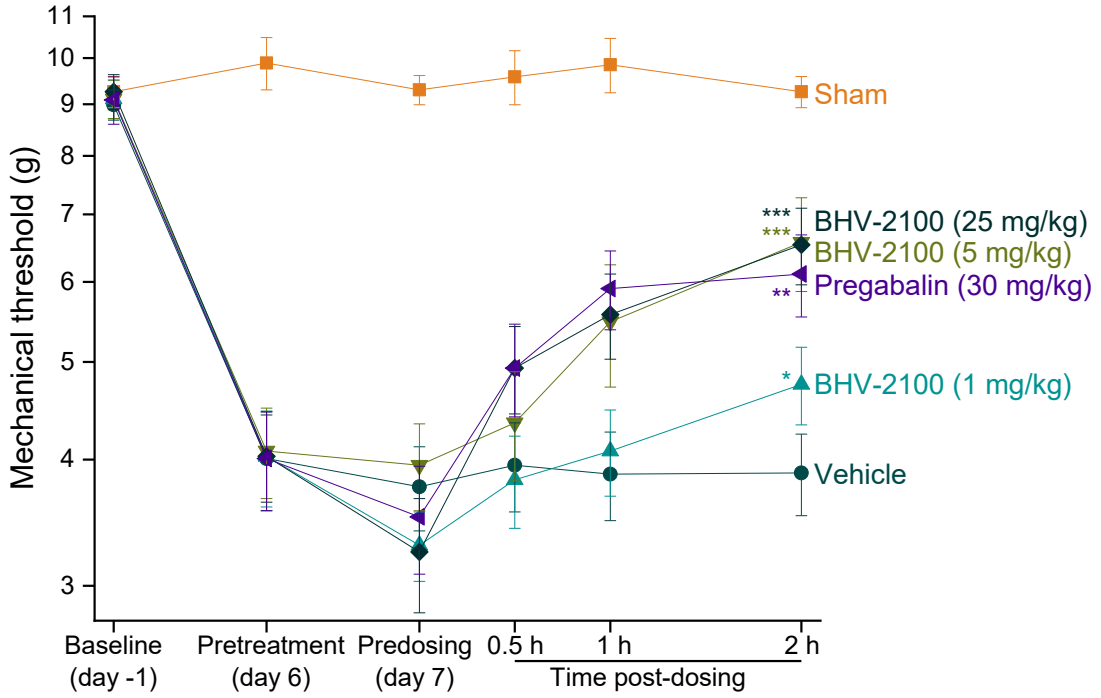
TRPM3

Chemotherapy-Induced Neuropathic Pain Model[1]



Mouse oxaliplatin model

Diabetic Neuropathy Model[1]



Rat streptozotocin (STZ) model

* P<0.05. ** P<0.01. *** P<0.001

1. Vriens. IASP 2024. Poster WE727

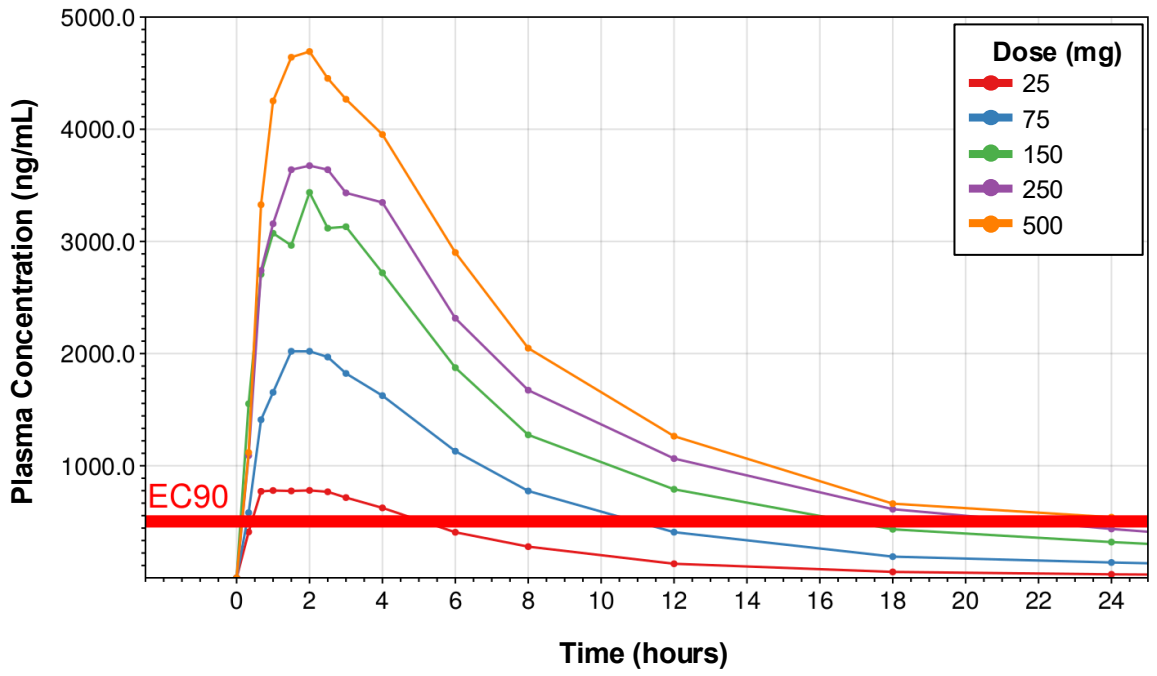
KEY POINT

Comparable analgesic efficacy as tramadol or pregabalin without sedative effects

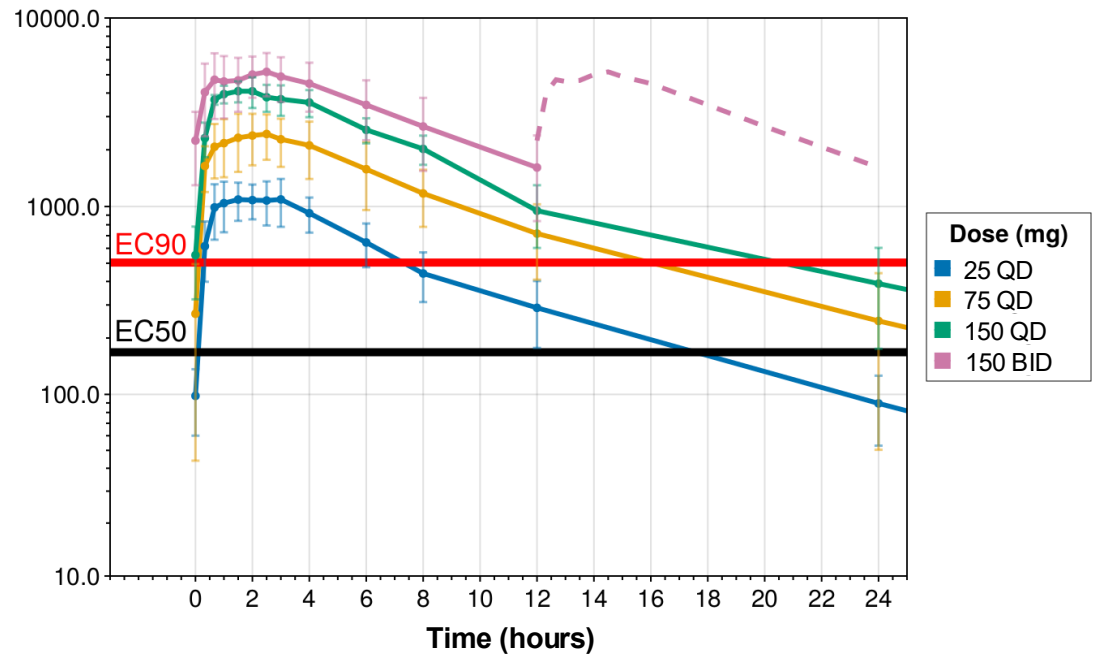
Ideal Pharmacokinetic Profile for Acute and Chronic Pain

TRPM3

Single Dose Exceeds EC90 by 20 Minutes



Multiple Doses Provide 24-Hour Coverage Over EC50 and EC90



EC90 represents the estimated plasma concentration threshold based on a preclinical model.

KEY POINT

Phase 1 data in healthy subjects shows plasma PK exceeds EC90 by 20 minutes at all doses tested and is sustained above EC50 for 24 hours at doses > 25 mg QD

BHV-2100: Safety and Tolerability

TRPM3

- No dose limiting toxicities
- No related SAEs or severe TEAEs
- Most TEAEs mild and resolved spontaneously
- Low rates of CNS AEs
- No clinically significant trends in vital signs (including body temperature), laboratory values or ECGs

>500 clinical trial participants dosed

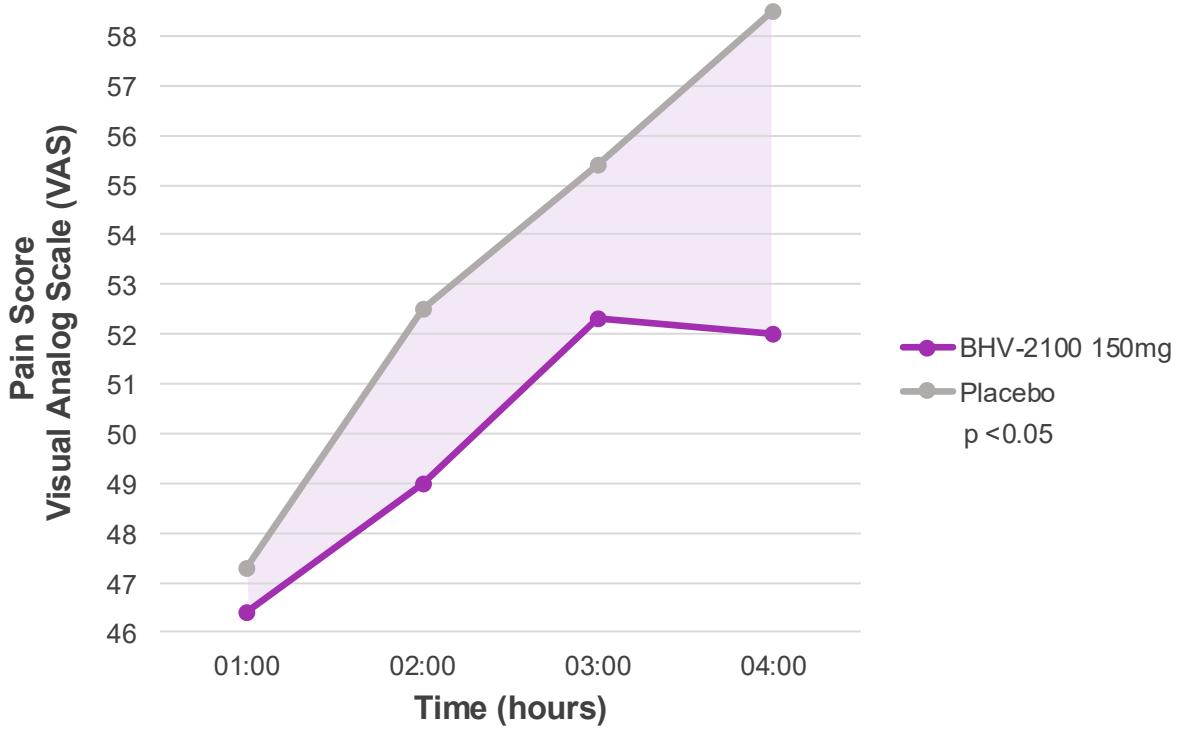
KEY POINT

BHV-2100 demonstrated excellent safety and tolerability in all studies

BHV-2100: Academic Laser-Induced Pain PoC Study Demonstrates Anti-Nociceptive and Anti-Hyperalgesic Effects

TRPM3

Improvements in Self-Reported VAS Pain Score



No Temperature Related Liabilities



- No effects observed on core temperature
- No change on heat pain threshold

Differentiated from Other TRP Channel Modulators

Preliminary Data up to Tmax; p-value out to 8-hour test period

KEY POINT

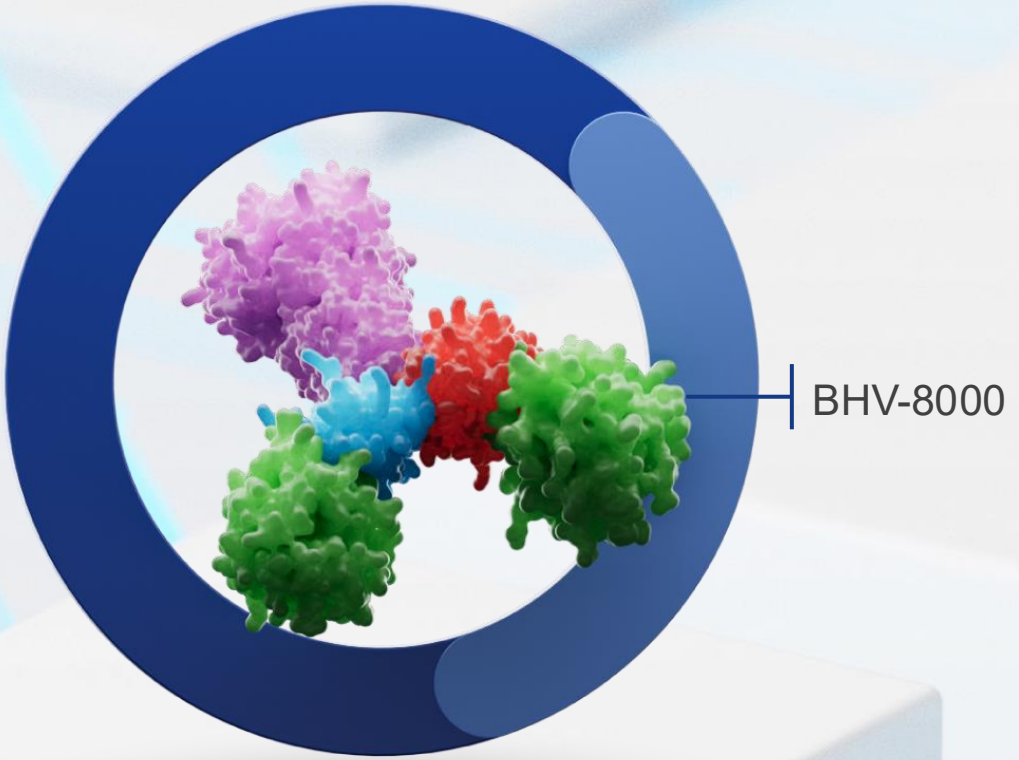
Novel non-opioid peripherally acting TRPM3 inhibitor reduces inflammatory pain without temperature related liabilities in human POC study

biohaven®

I&I: BHV-8000

BRAIN-PENETRANT TYK2/JAK1 INHIBITOR

Leveraging Validated Immune Targets To Stop Neuroinflammation



Brain-Penetrant TYK2/JAK1 Inhibitor To Treat Parkinson's Disease

TYK2/JAK1

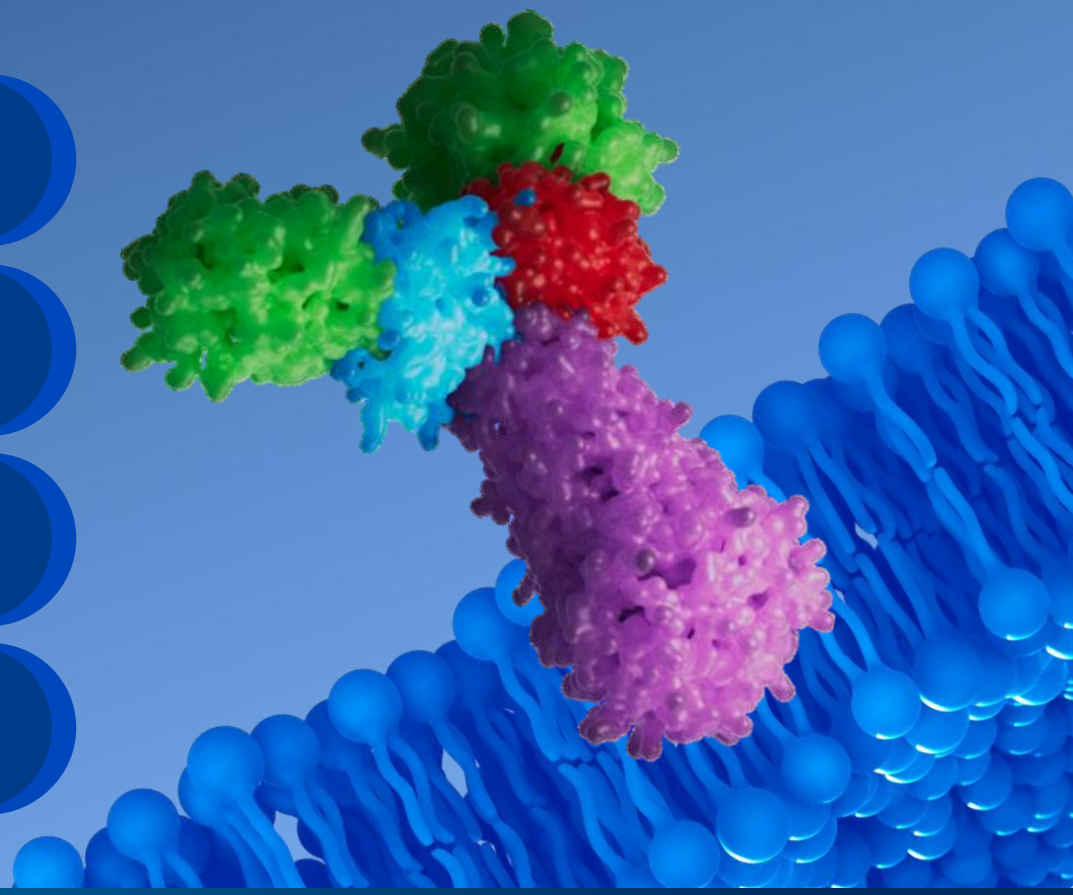
BHV-8000 addresses the widespread immune dysregulation that drives the onset and progression of neurodegenerative disorders

SUPPRESSES INNATE AND ADAPTIVE IMMUNE CELL ACTIVATION AND INFILTRATION

HIGH SELECTIVITY FOR TYK2/JAK1 AVOIDS JAK2/3 SAFETY LIABILITIES

DEMONSTRATED TARGET ENGAGEMENT, ROBUST BRAIN PENETRATION AND A PROMISING SAFETY PROFILE

PHASE 2/3 TRIAL IN EARLY PD ONGOING



Widespread Immune Dysregulation Is a Hallmark of PD

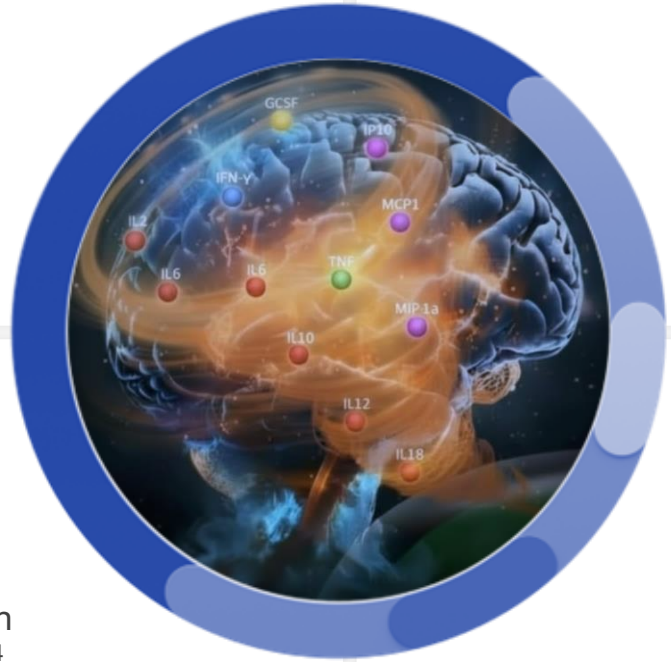
TYK2/JAK1

PD meets criteria for autoimmunity based on pathophysiology and genetics

- Misidentification of self-proteins (α-synuclein) as foreign antigen triggers immune response[1]
- GWAS studies link PD risk to HLA gene variants involved in antigen presentation[2]

Epidemiology reveals increased risk of PD in individuals with other autoimmune diseases[3]

- Epidemiological studies suggest immune dysfunction and inflammation are key to the development of PD[4]
- Reduction in rates of PD have been seen when this population is exposed to immune-modulating therapies[5]



PD animal models demonstrate immune dysregulation drives neurodegeneration[6]

- In mouse models, T cells specific to α-synuclein peptides can induce dopaminergic neuronal loss[7]
- Manipulation of immune components (T cells) affect α-synuclein-induced neurodegeneration[8]

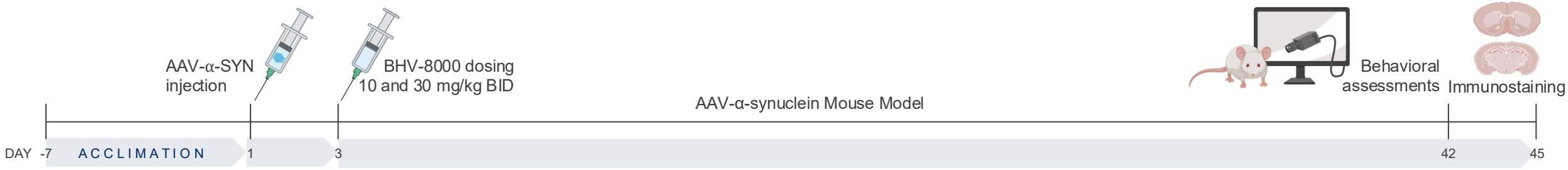
PD patient samples and imaging exhibit characteristic proinflammatory signatures[9,10]

- Proinflammatory cytokines (e.g., IL-6, TNF-α, IFNɣ) are found in CSF and blood of PD patients[11]
- PD brains express high levels of HLA-DR+ reactive microglia[12]

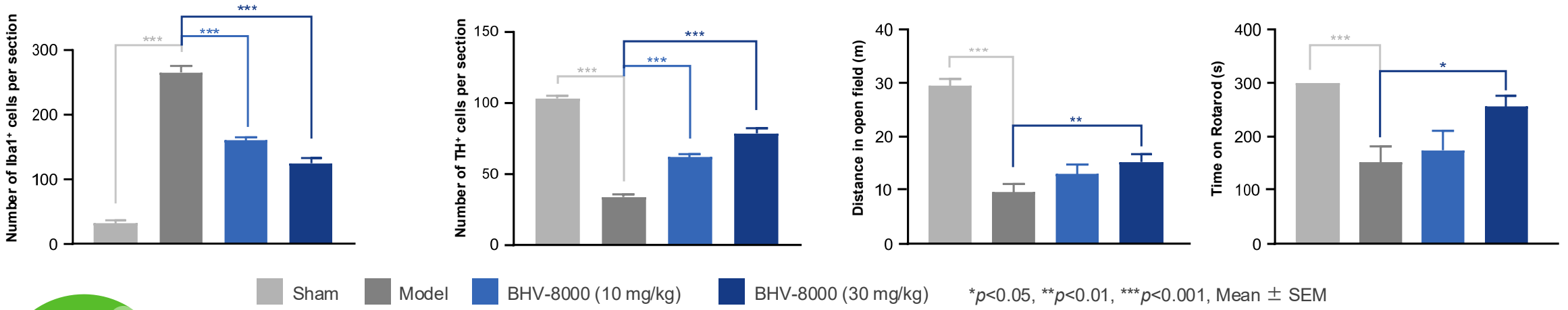
1. Sulzer. Nature. 2017. **2.** Wissemann. Am J Hum Genet. 2013. **3.** Li. Front Immunol. 2023. **4.** Tansey. Nat Rev Immunol. 2022. **5.** Potashman. Parkinsonism Relat Disord. 2025. **6.** Roodveldt. Brain. 2024. **7.** Karikari. Brain Behav Immun. 2022. **8.** Williams. Brain. 2021. **9.** Yacoubian. Mov Disord. 2023. **10.** Pajares. Cells. 2020. **11.** Qu. Nature. 2023. **12.** McGeer. Neurology. 1988.

BHV-8000 Reduces Glial Activation, Increases Neuronal Survival, and Improves Function in AAV-α-syn Mouse Model of Parkinson's Disease

TYK2/JAK1



Reduced Inflammation	Increased Neuronal Survival	Improved Function	
Reduced the Number of Activated Glial Cells (Iba1+) in the Substantia Nigra:	Increased the Survival of TH+ Neurons in the Substantia Nigra:	Increased Movement Distance in an Open Field by:	Increased Time on Rotarod by:



KEY POINT

BHV-8000 reduces inflammation and improves neurological function

BHV-8000 Demonstrates Target Engagement, Robust Brain Penetration and a Promising Safety Profile in Phase 1 Studies

TYK2/JAK1

Phase 1 program completed

- Includes SAD/MAD study in healthy adults

Well-tolerated

- No SAEs or severe AEs
- No adverse laboratory trends related to study drug

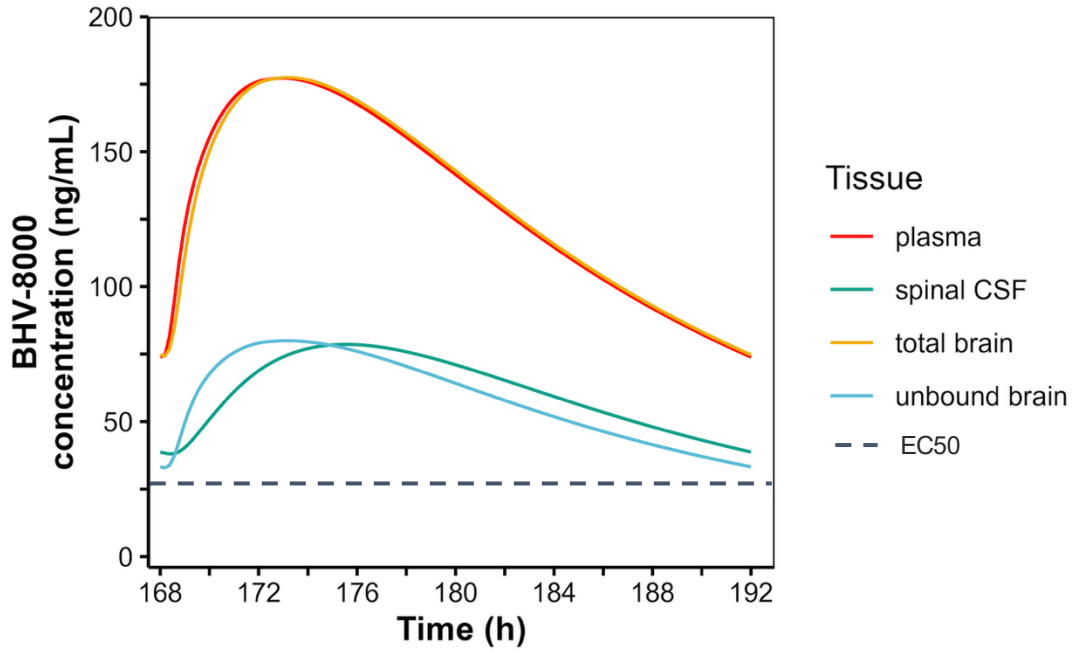
Evidence of target engagement

- Reduced plasma inflammatory cytokines downstream of TYK2 (IP-10, IFNβ) and JAK1 (hsCRP, IFNβ)

Robust brain penetration

- Exposures in CNS approximately 50% of plasma concentrations

BHV-8000 Brain Exposures at 20 mg QD



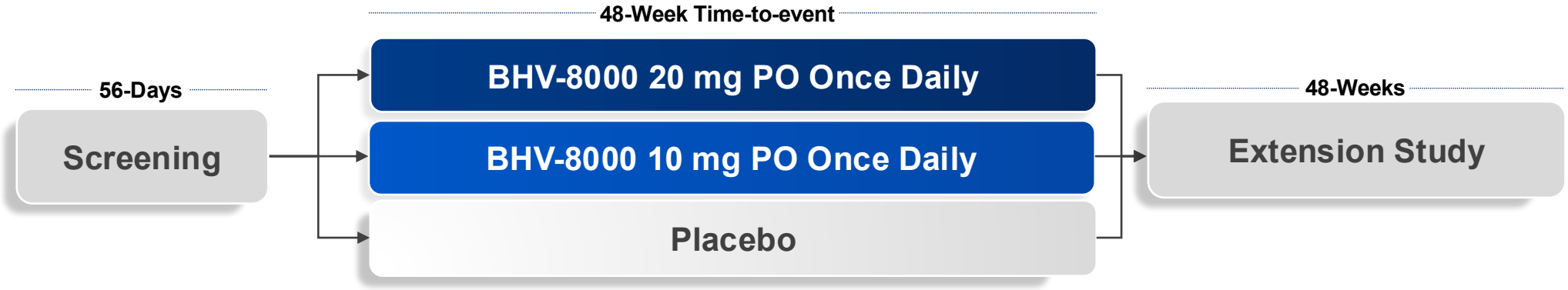
Modelling data

KEY POINT

Brain exposure sustained above target EC50s for 24 hours at clinical doses

BHV-8000: Phase 2/3 Study in Early Parkinson's Disease Ongoing

TYK2/JAK1



KEY STUDY DETAILS

Study Design: Phase 2/3, randomized 1:1:1, double-blind study

Population: Early untreated PD

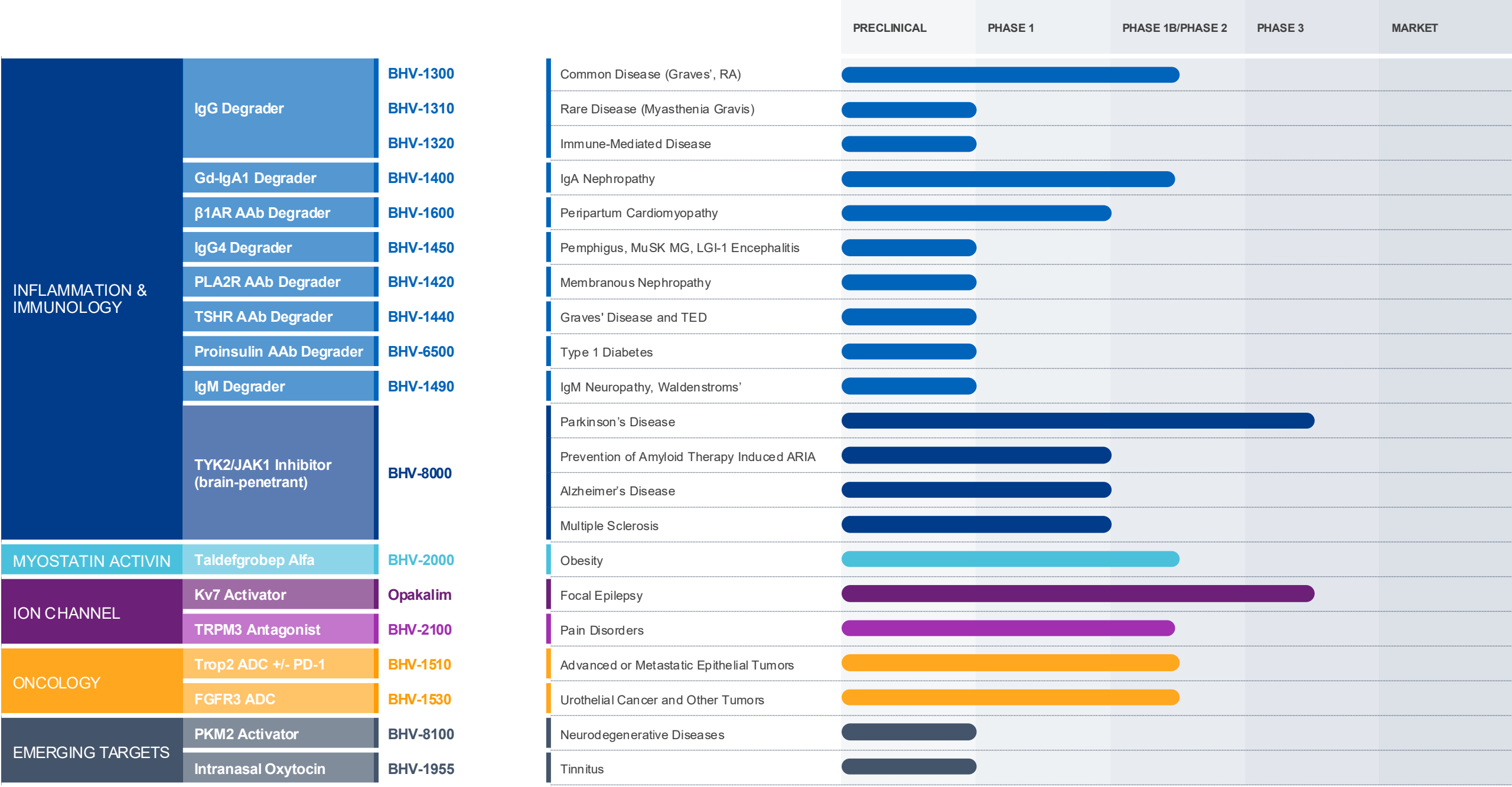
Endpoints: Primary: Time to qualifying worsening event on MDS-UPDRS Part II

Secondary: Change in MDS-UPDRS Part III, CGI-S, DaT-SPECT, PARCOMS composite scale

MDS-UPDRS, Movement Disorder Society – Unified Parkinson's Disease Rating Scale; **CGI-S**, Clinician Global Impression of Severity scale; **PARCOMS**, Parkinson's composite scale that includes most sensitive items from MDS-UPDRS and PDQ-39.

KEY POINT

Time-to-event design enables efficient trial



ARIA, Amyloid-related imaging abnormalities; **AAb**, Autoantibody.

Next-Generation Pipeline...



biohaven®

DAYS MATTER™

Financial Update

DAYS MATTER™

$264M[3]

3Q 2025 Cash on hand

+$312M

Capital raise since last Q[4]

145.1M[2]

CGRP



1. Cap reached if aggregate annual US net sales of rimegepant and zavegepant amount to $8.15B. Royalty payments would be in respect of years ended on or before 12/31/40. **2.** As of November 13, 2025; includes 26.8M shares issued in November 2025 equity offering inclusive of 3.5M share overallotment and pro forma for the Jan 2026 sale of 12.5 mill shares. **3.** As of September 30, 2025; **4.** Net proceeds from November 2025 equity offering, including greenshoe and January 2026 sale of shares to Janus Henderson investors.

Key Milestones Anticipated in 2026

			1H 2026	2H 2026
INFLAMMATION & IMMUNOLOGY	**Gd-IgA1 Degrader | BHV-1400**	IgA Nephropathy	Initiate Pivotal IgAN	
	IgG Degrader | BHV-1300	Common Disease (Graves', RA)		Initiate Pivotal Graves'
	TYK2/JAK1 Inhibitor | BHV-8000 (brain-penetrant)	Parkinson's Disease	Ongoing Phase 2/3 Trial	
MYOSTATIN ACTIVIN	**Taldefgrobep Alfa | BHV-2000**	Obesity		Phase 2 Topline
ION CHANNEL	**Kv7 Activator | Opakalim**	Focal Epilepsy		Pivotal Topline
ONCOLOGY	**Trop2 ADC +/- PD-1 | BHV-1510**	Advanced or Metastatic Epithelial Tumors	Initiate expansion cohort in endometrial cancer	
	FGFR3 ADC | BHV-1530	Urothelial Cancer and Other Tumors	Phase 1 in urothelial cancer	



biohaven®

DAYS MATTER™