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Corporate Presentation

June 2026

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-8100, BHV-1300, BHV-1310, BHV-1400, BHV-1510, BHV-1530 and BHV-1600 development programs), the timing of and the availability of data from our clinical trials, the timing of 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-1955, BHV-8200, BHV-2110, BHV-1490, BHV-1420, BHV-1440, BHV-6500 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; Biohaven’s compliance with applicable U.S. regulatory requirements; the potential commercialization of Biohaven's product candidates; 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.

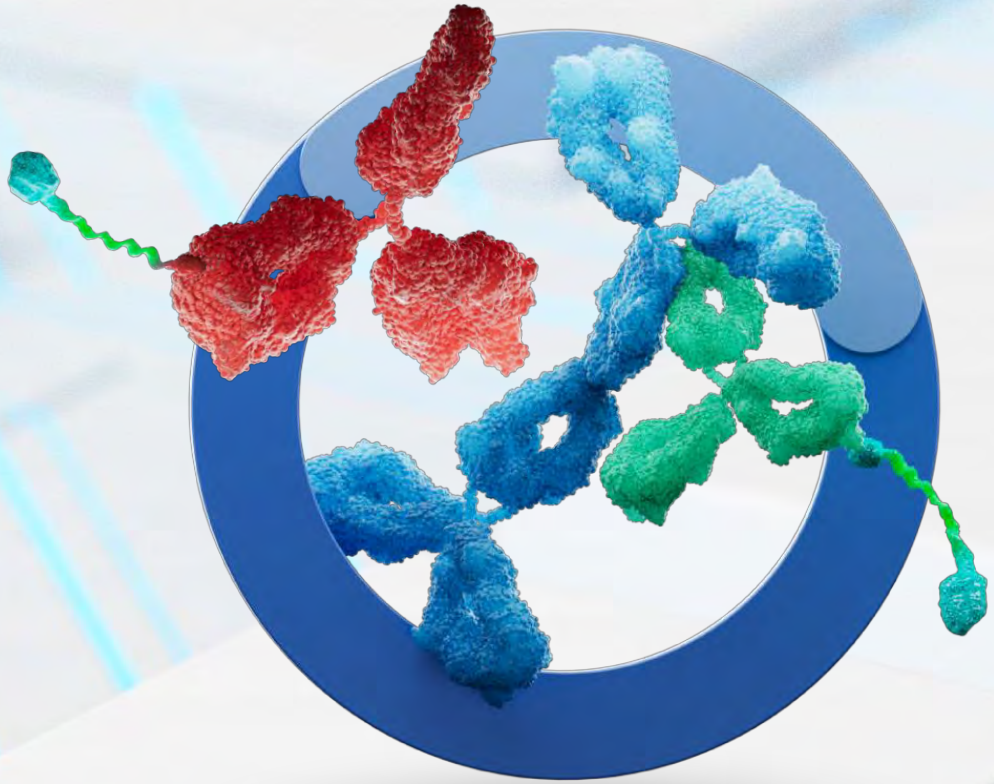
				PRECLINICAL	PHASE 1	PHASE 1B/PHASE 2	PHASE 3	MARKET
INFLAMMATION & IMMUNOLOGY	IgG Degrader	BHV-1300	Common Disease (Graves', RA)					
	Gd-IgA1 Degrader	BHV-1400	IgA Nephropathy					
	β1AR AAb Degrader	BHV-1600	Peripartum Cardiomyopathy					
	TYK2/JAK1 Inhibitor (brain-penetrant)	BHV-8000	Parkinson's Disease					
ION CHANNEL	Kv7 Activator	Opakalim	Focal Epilepsy					
	TRPM3 Antagonist	BHV-2100	Pain Disorders					
MYOSTATIN ACTIVIN	Taldefgrobep Alfa	BHV-2000	Obesity					
ONCOLOGY	Trop2 ADC +/- PD-1	BHV-1510	Advanced or Metastatic Epithelial Tumors					
	FGFR3 ADC	BHV-1530	Urothelial Cancer and Other Tumors					
PRE-CLINICAL DEGRADERS	IgG Degrader	BHV-1310	Rare Disease (Myasthenia Gravis)					
		BHV-1320	Immune-Mediated Disease					
	IgG4 Degrader	BHV-1450	Pemphigus, MuSK MG, LGI-1 Encephalitis					
	PLA2R AAb Degrader	BHV-1420	Membranous Nephropathy					
	TSHR AAb Degrader	BHV-1440	Graves' Disease and TED					
	Proinsulin AAb Degrader	BHV-6500	Type 1 Diabetes					
	IgM Degrader	BHV-1490	IgM Neuropathy, Waldenstroms'					
EMERGING TARGETS	PKM2 Activator	BHV-8100	Neurodegenerative Diseases					
	Intranasal Oxytocin	BHV-1955	Tinnitus					

AAb, Autoantibody.

biohaven®

DEGRADERS:
MoDE™ AND TRAP™

Targeting the Root Cause of Autoimmune Disease

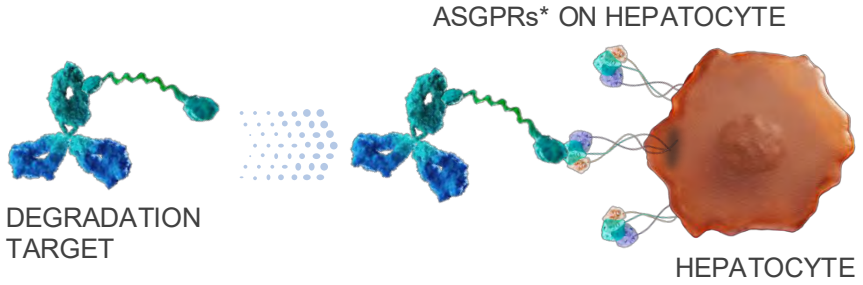


Innovation in Protein Degradation: Harnessing Hepatic ASGPR for Efficient and Safe Removal of Circulating Pathogenic Targets

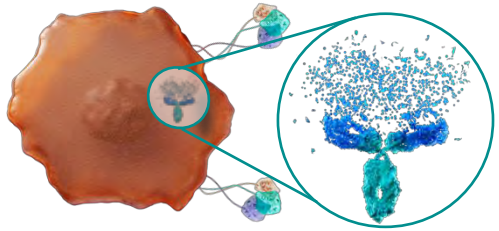
DEGRADERS

BIFUNCTIONAL
MoDE DEGRADERS

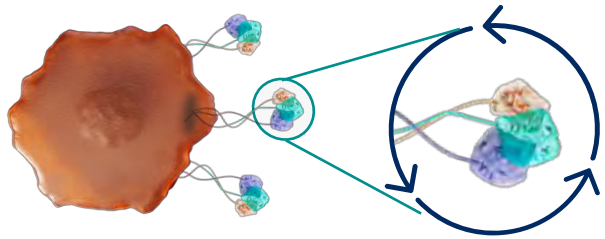
1. MoDE™ and TRAP™ degraders bind circulating target and efficiently deliver to ASGPR on hepatocytes



2. Internalized target is rapidly degraded in hepatic lysosomes



3. ASGPRs are rapidly recycled

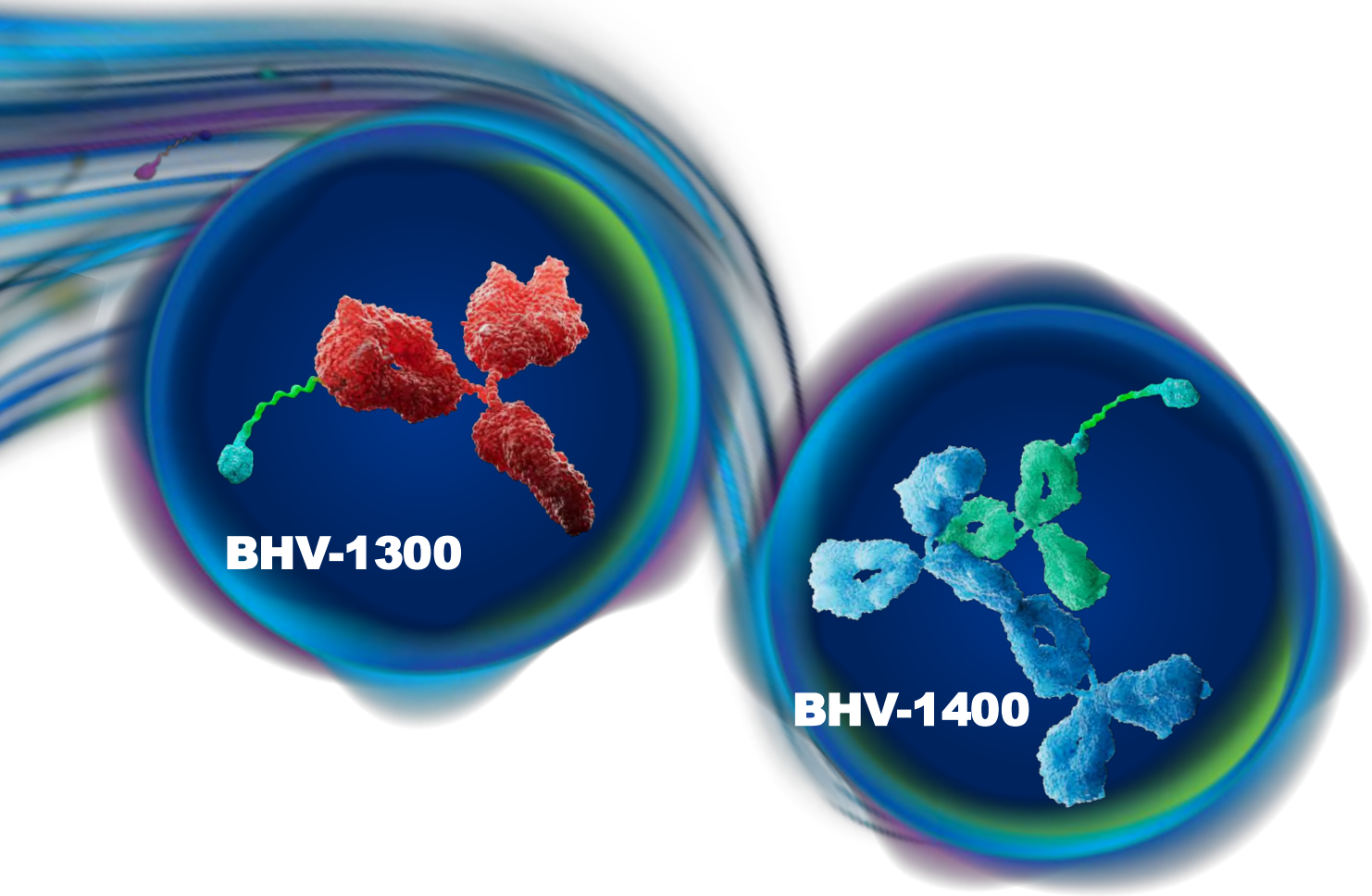


MoDE and TRAP optimized for patient friendly **autoinjector delivery**

*Stylistic representation
ASGPR, asialoglycoprotein receptor

MoDE™ and TRAP™ DEGRADERS

First Extracellular Protein Degraders in the Clinic Demonstrate Rapid and Robust Pharmacodynamic Effects and Compelling Safety in Nearly 200 Individuals Dosed



- Safe
- Well-tolerated
- Highly selective
- Deep and rapid lowering
- Patient outcomes
- Pivotal trials mid-2026

MoDE™ and TRAP™ Degraders

PIPELINE ASSETS

8+

BHV degrader programs

ADDRESSABLE PATIENTS

>2M

across lead programs (US)

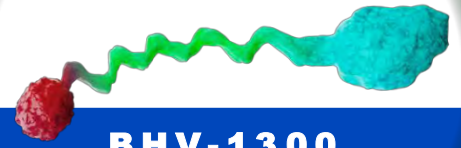
PEAK SALES POTENTIAL

$30B+

combined gross sales

MoDE™

> 1M patients | $7.6B



BHV-1300

IgG degrader

Graves' disease · RA
Sjögren's · Biologic failures

~400K patients | $11.1B

BHV-1310

IgG Degrader

gMG · Systemic sclerosis
AE · ITP

~92K patients | $2.6B

BHV-1450

IgG4 degrader

Pemphigus vulgaris
MuSK MG · IgG4-RD
CIDP

~42K patients | $1.7B

BHV-1490

IgM degrader

Cold agglutinin disease
Anti-MAG
Waldenström's

BHV-1440

TSHR AAb degrader

Graves' disease
Thyroid eye disease

~133K patients | $5.0B

BHV-1400

Gd-IgA1 degrader

IgA nephropathy

TRAP™

~41K patients | $1.3B

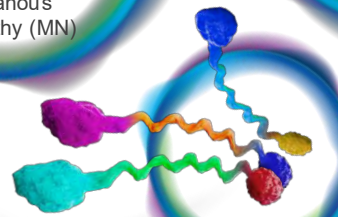
BHV-1420

PLA2R AAb Degrader

Membranous
Nephropathy (MN)

BHV-6500

Pro-Insulin/Insulin
AAB Degrader



Addressable patients for each degrader based on internal assessment of potential patient population. Peak sales potential based on addressable patients for each degrader and internal assessment of gross revenue potential (prior to GTN adjustments or any adjustments associated with clinical risk), adjusted for estimated market share / market penetration.

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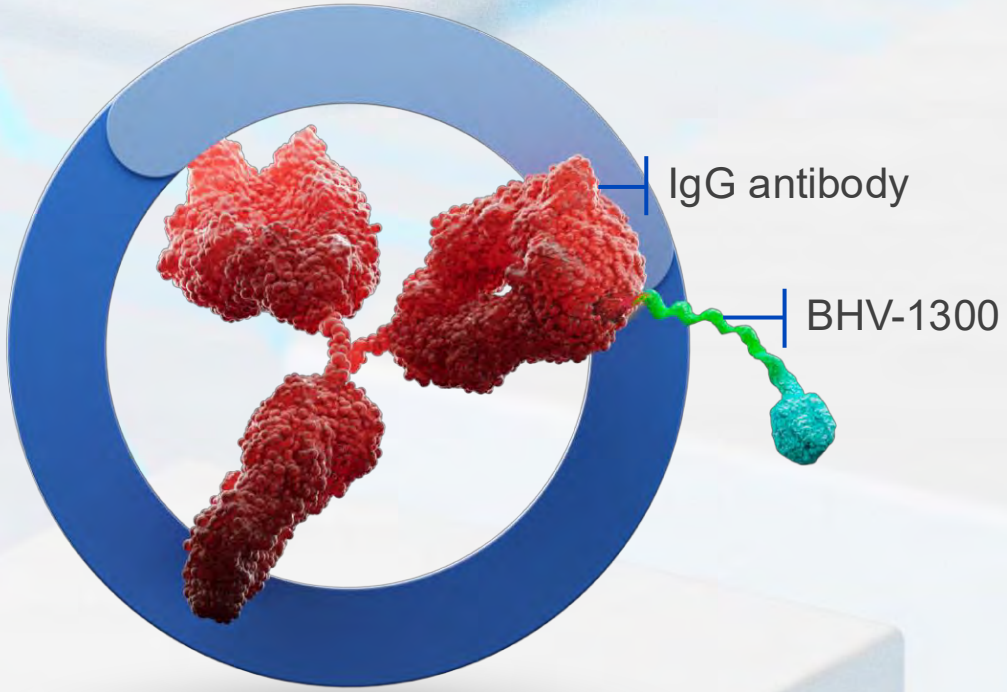
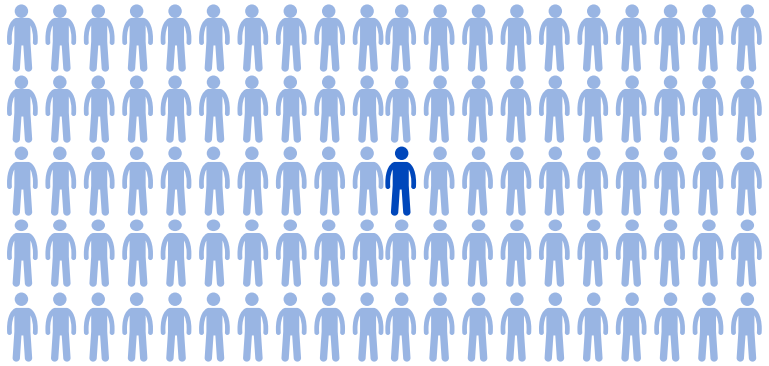
IgG MoDE™ DEGRADER:
BHV-1300

Targeting the Root Cause of Graves' Disease

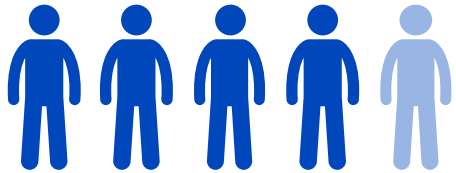


Demographics: Graves' Disease Impacts 1% of the Global Population

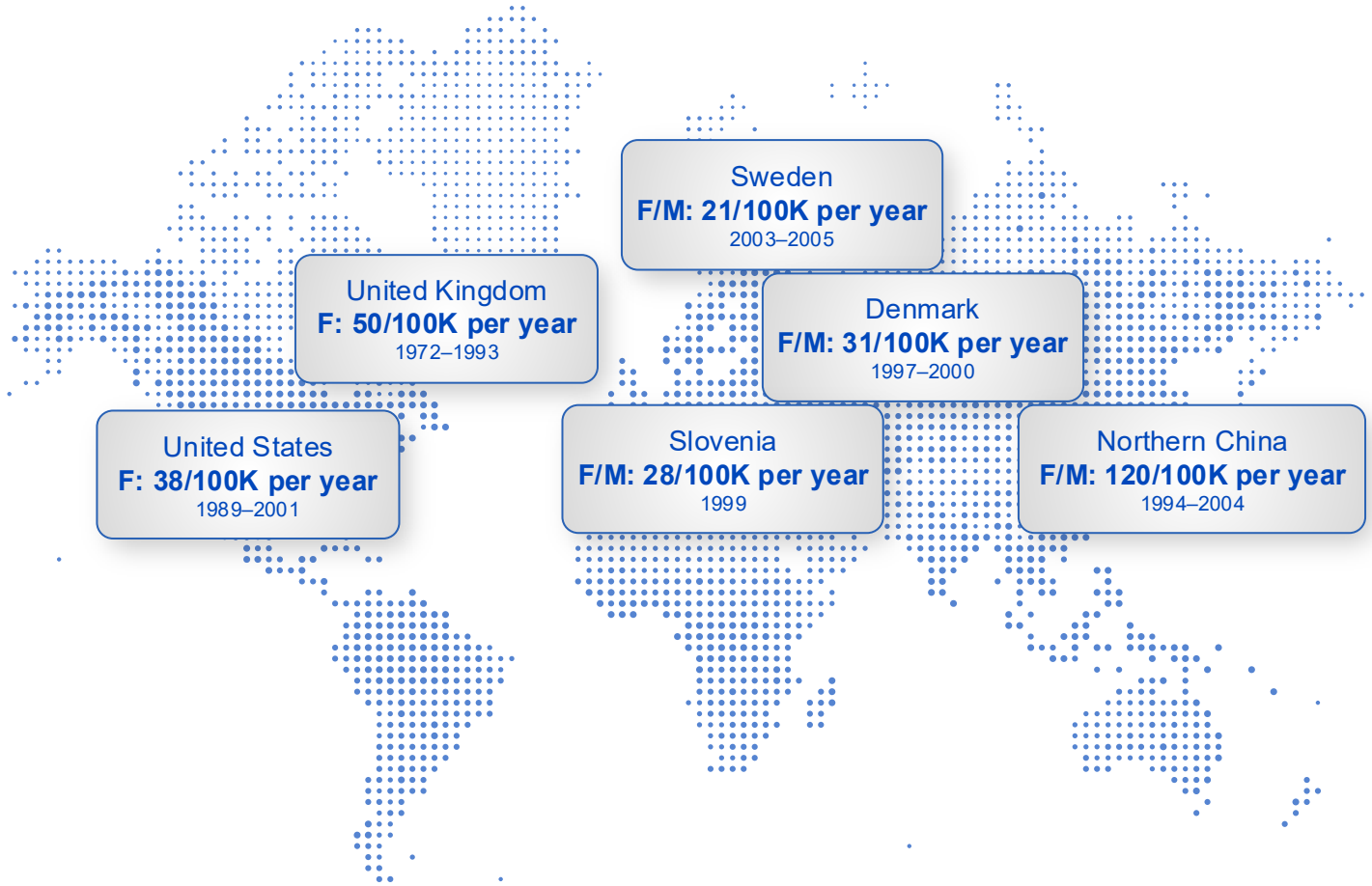
Graves' disease affects nearly 1 in 100 Americans.[1]



About 4 out of 5 cases of hyperthyroidism in the United States are caused by Graves' disease.[1]



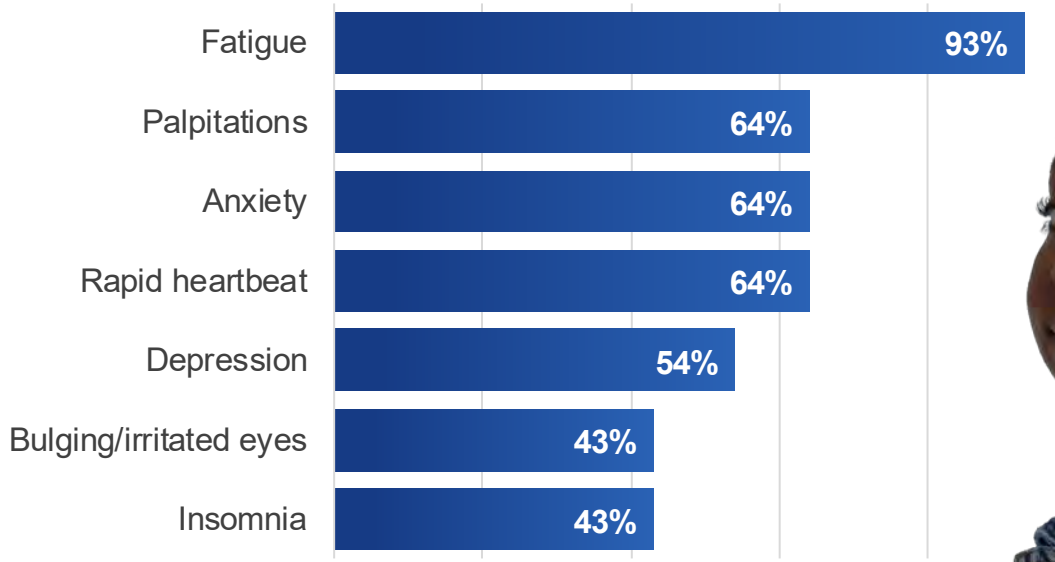
Worldwide incidence of Graves' disease[2]



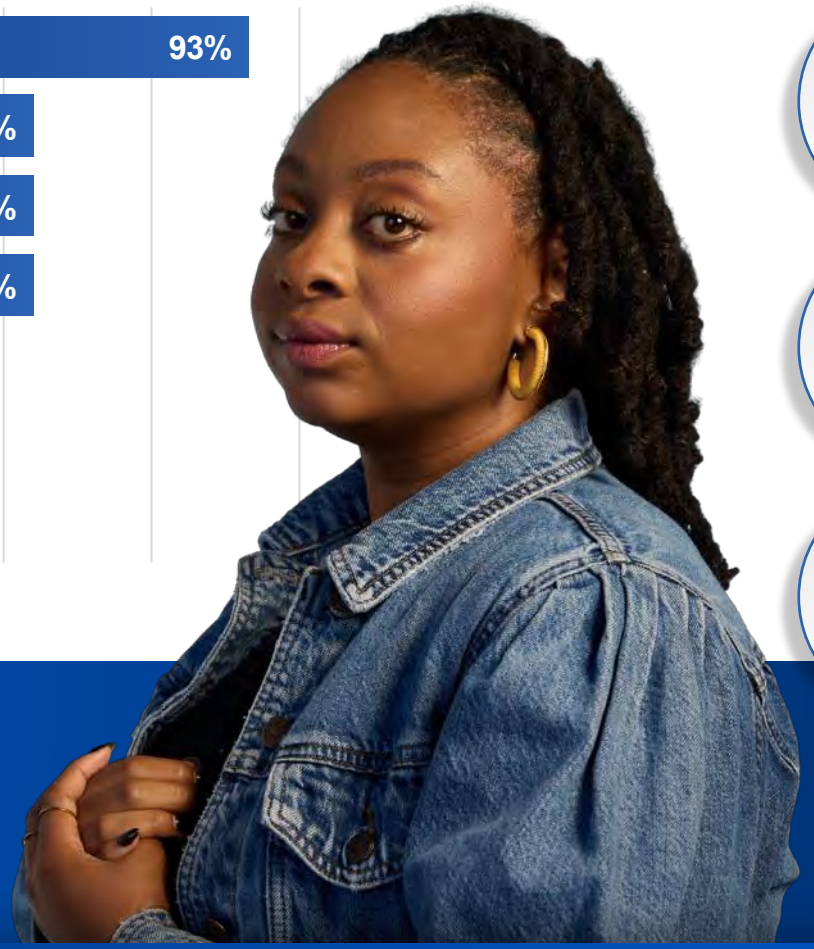
1. Akram. Journal of Surgical Research. 2020. **2.** McLeod. Endocrine, 2012.

The Under-Recognized Burden of Graves' Disease[1]

DEGRADERS



1. Patient Burden in Graves' Disease: Results From a Mixed Methods Survey



93% report multiple symptoms (≥2)

79% experience 4+ symptoms

72% experience 5+ symptoms

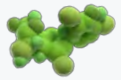
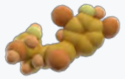
Even among biochemically well-controlled patients,
75% report recurring symptoms —
fatigue, palpitations, anxiety, weight gain

Courtnay
Living with Graves' disease

KEY POINT

Current treatments achieve biochemical control but do not address the underlying antibody-driven disease, antibodies continue to circulate, targeting the thyroid, orbit, brain, and crossing the placenta

TSHR Autoantibodies (TSHR-IgG1) Cause Hyperthyroidism

	Hyperthyroidism (from a toxic nodule)	Graves' disease hyperthyroidism	Autoimmune hypothyroidism (Hashimoto's)
Thyroid stimulating hormone (TSH)	↓	↓	↑
Thyroxine (T4) 	↑	↑	↓
Thyronine (T3) 	↑	↑	↓
Thyroid receptor antibody (TRAb)	−	+	−
Thyroid stimulating immunoglobulin (TSI)	−	+	−
Thyroid peroxidase antibody (TPO Ab)	−	+	+

KEY POINT

TSHR-IgG1 causes Graves' hyperthyroidism, the diagnosis of which is made when a patient has elevated thyroid hormones and TSHR autoantibodies present

Current Treatments for Graves' Disease

CONSERVATIVE

1. **Use of thionamides**
   (ATDs, like methimazole, carbimazole, and propylthiouracil (PTU)
   Aplastic anemia, agranulocytosis, peripheral neuritis, liver issues, secreted in breastmilk
2. **Use of steroids**

ABLATIVE

3. **Surgical removal of the thyroid**
   Lifelong T4 replacement, surgical complications
4. **Use of I-131 radiation treatment**
   Cannot use in pregnancy, TED worsens

BHV-1300 MoDE™ Targets the Root Cause of Graves' Disease and TSHR Autoantibody-Driven Diseases

DEGRADERS

EXCESS HORMONES

T3 T4

TSHR AUTOANTIBODY DRIVEN DISEASES

- Thyroid eye disease
- Graves' embryopathy
- Thyroid dermopathy
- Graves' disease

STABILIZED HORMONES

T3 T4

BHV-1300

BHV-1300 degrades disease-causing TSHR-IgG1 antibodies

Not an FcRn Inhibitor: Biohaven IgG MoDE™ Degrader Differentiates as a Novel MOA, Potential Paradigm Shifting Therapy

DEGRADERS

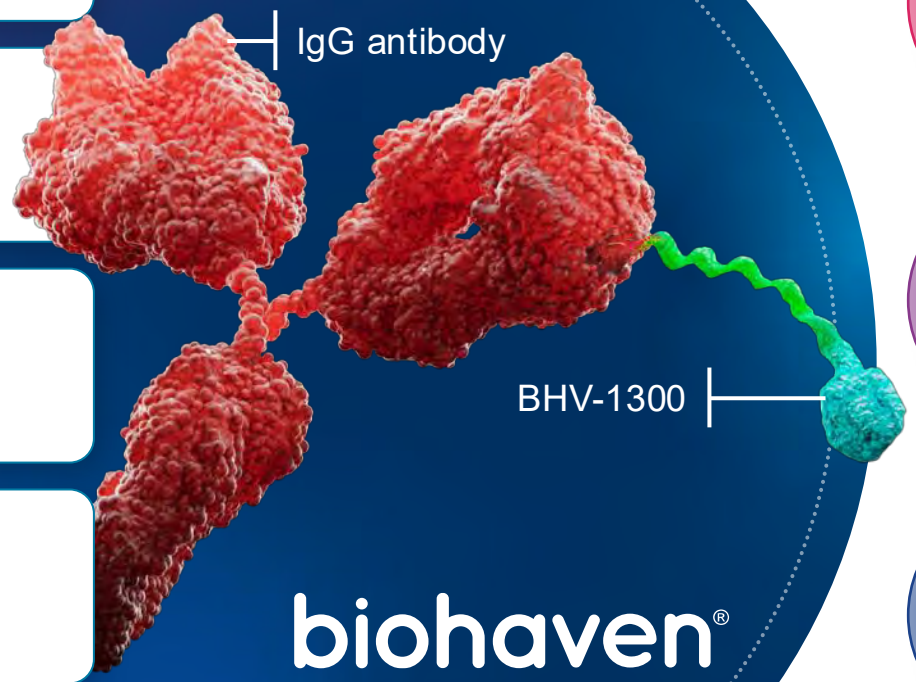
83% IgG LOWERING
BY DAY 18

DID NOT INCREASE
HEADACHES

DID NOT INCREASE
CHOLESTEROL

SMALL MOLECULE

AUTOINJECTOR
ADMINISTRATION IN PIVOTAL TRIALS



IMAAVY™ J&J

- 74.6% IgG lowering after load, 68.8% in maintenance in Vivacity MG-3[1,2]
- **IV infusion**
- Increased cholesterol (24%), muscle spasms (12%), edema (12%)

Vyvgart® argenx

- Approximately 61% IgG lowering at week 4 (VYVGART Hytrulo® in MG trial)[3] (average 75% in MAD)[4]
- Prefilled syringe
- Cyclical dosing can lead to symptom rebound

Rystiggo® UCB

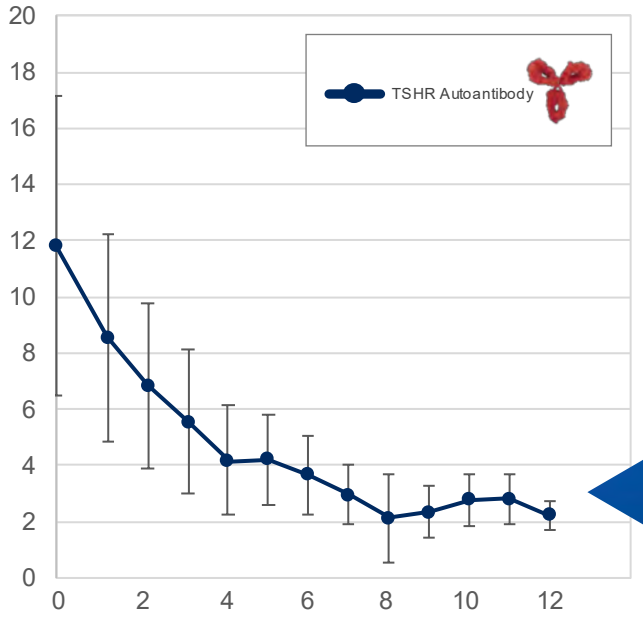
- Approximately 76% IgG lowering in the MycarinG study[5]
- Healthcare administered SC infusion
- **44% headaches**
- Cyclical dosing can lead to symptom rebound

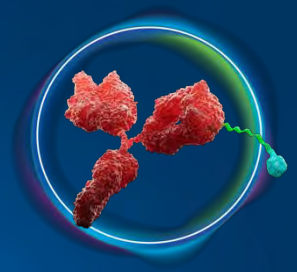
1. Median of the maximal total IgG % change from baseline **2.** 84% IgG lowering (twice the labeled frequency) in Phase 1; Ling. Clin Pharmacol Ther. 2019. **3.** Howard, Jr. ADAPT (SC) Data – 2024; **4.** Ulrichts. J. Clin. Invest. 2018. **5.** MAD data unavailable. MG Data from Bril, Lancet Neurology. 2023 – MyCarinG study.

BHV-1300 MoDE™ Deeply Removes Disease-Driving TSHR Autoantibody and Normalizes Thyroid Hormones

DEGRADERS

TRAb Change from Baseline

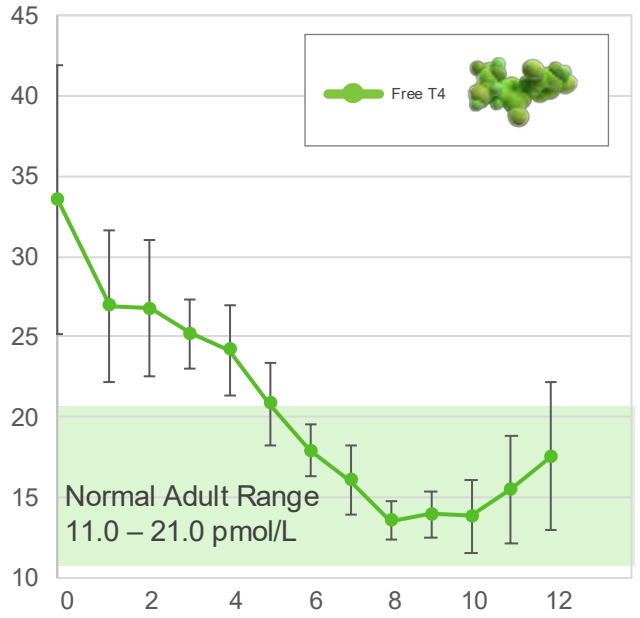




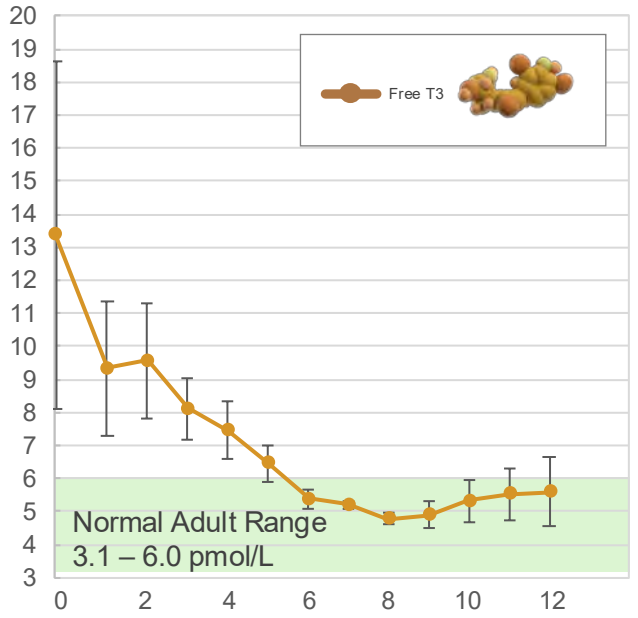
BHV-1300 RAPIDLY REMOVES >80% OF TSHR AUTOANTIBODIES
the root cause of Graves' disease

1,000 mg SC weekly dosing

Change from Baseline Free T4



Change from Baseline Free T3



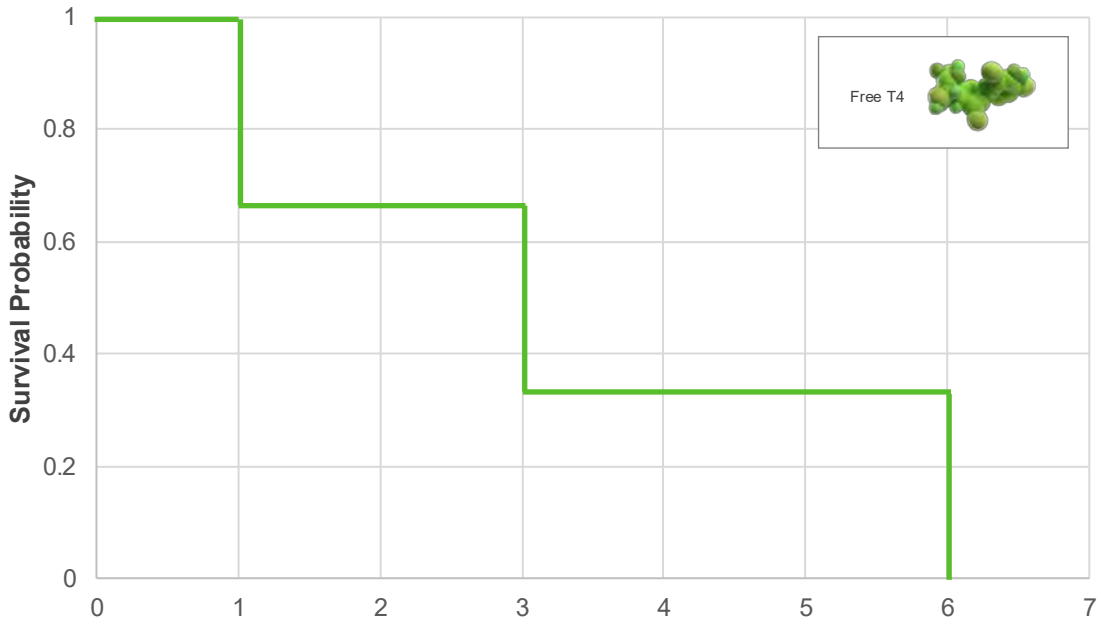
Weeks Post-Dose 1

Preliminary data from ongoing study, analysis conducted May 22, 2026. Data represents mean and standard error concentration of TRAb, Free T4, and Free T3 in the three consecutive overt hyperthyroid participants with Graves' disease. Participants were dosed with BHV-1300 SC 1000 mg weekly for 12 weeks (n=2) or BHV-1300 1000 mg SC weekly for 4 weeks followed by 500 mg SC weekly for 8 weeks. Values of TRAb below the limit of quantification (LOQ) were set to LOQ/2.

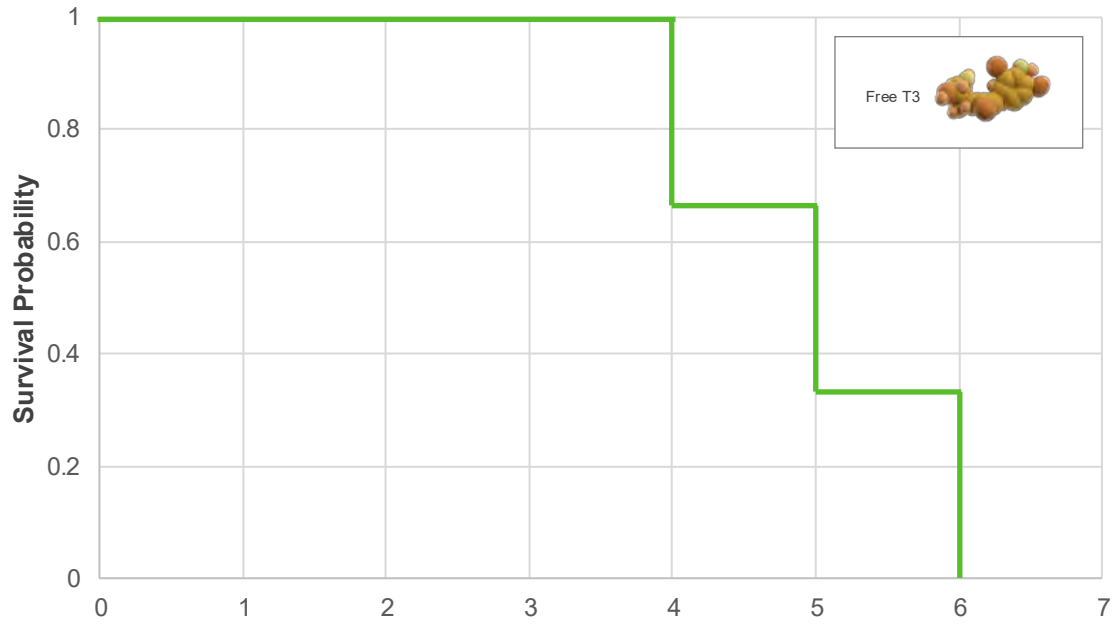
BHV-1300 Rapidly Normalizes Thyroid Hormones in Patients With Graves' Hyperthyroidism

DEGRADERS

Kaplan-Meier Curve of Time to Normalization of Free T4



Kaplan-Meier Curve of Time to Normalization of Free T3



Weeks Post-Dose 1

Preliminary data from ongoing study, analysis conducted April 8, 2026. Graphs represent time to normalization of Free T3 and Free T4 in participants (n=3) with Graves' disease and overt hyperthyroidism at baseline administered BHV-1300 1,000 mg SC weekly for 12 weeks (n=2) or BHV-1300 1,000 mg SC weekly for 4 weeks followed by 500 mg SC weekly for 8 weeks (n=1).

KEY POINT

In hyperthyroid patients receiving BHV-1300 1,000 mg SC weekly, Free T4 normalized within an average of 3 weeks and Free T3 normalized within an average of 5 weeks

Lead MoDE™ Degrader, BHV-1300, Enters Phase 3

DEGRADERS

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

BHV-1300 pivotal trial in Graves' disease commencing mid-2026

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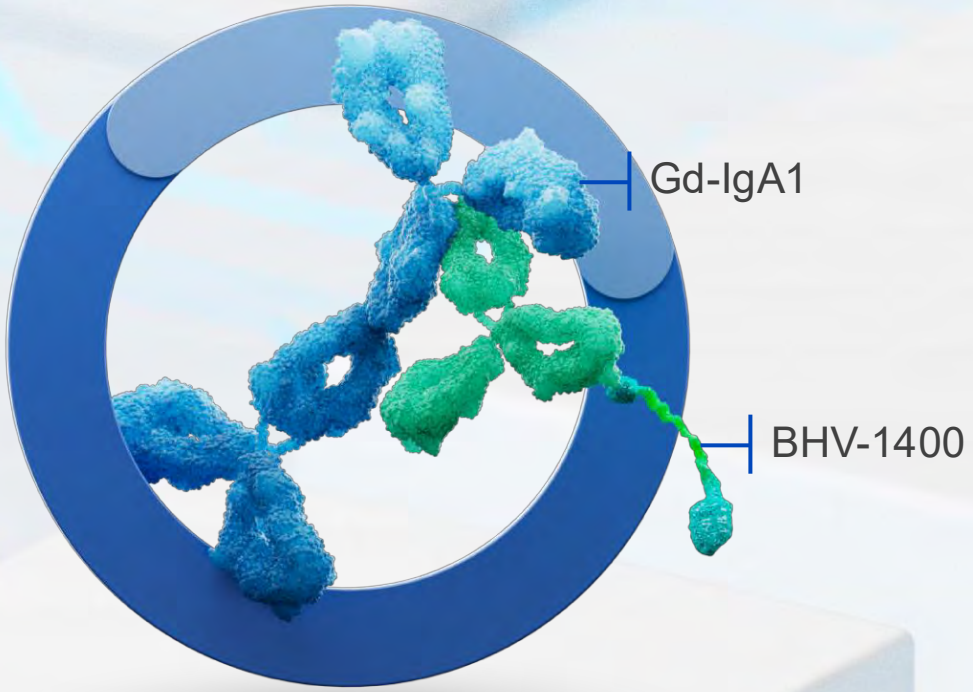
Gd-IgA1 TRAP™ DEGRADER: BHV-1400

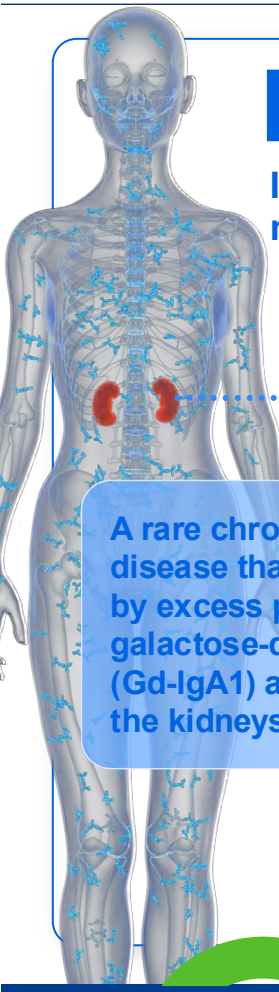
Targeting the Root Cause of IgA Nephropathy



UNIVERSITY OF LEICESTER

By the Time IgAN Is Diagnosed, Kidney Function Is Already Significantly Compromised

DEGRADERS



IgAN

IgA nephropathy

A rare chronic kidney disease that is caused by excess production of galactose-deficient IgA1 (Gd-IgA1) antibodies in the kidneys

IgAN predominantly affects people in their **MOST PRODUCTIVE YEARS OF LIFE**

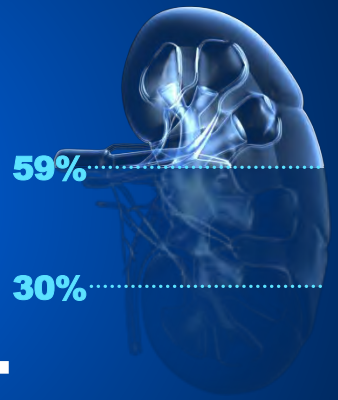
20s	**30s**
MEDIAN AGE AT DIAGNOSIS[1]	MOST COMMON AGE RANGE[1]

CKD STAGE 3

MOST COMMON AT DIAGNOSIS

At diagnosis, kidney function is already reduced to

30–59% OF NORMAL



Camille

Diagnosed with IgAN in 2020 at age 25

IgAN demands rapid intervention and a therapy safe enough to last a lifetime

1. Pitcher. Kidney Int. 2023.

TREATMENT GOAL

Every month matters — rapid intervention to preserve kidney function and limited remaining nephrons

Guidelines Focus on Removal of Pathogenic Gd-IgA1

KDIGO

DEGRADERS

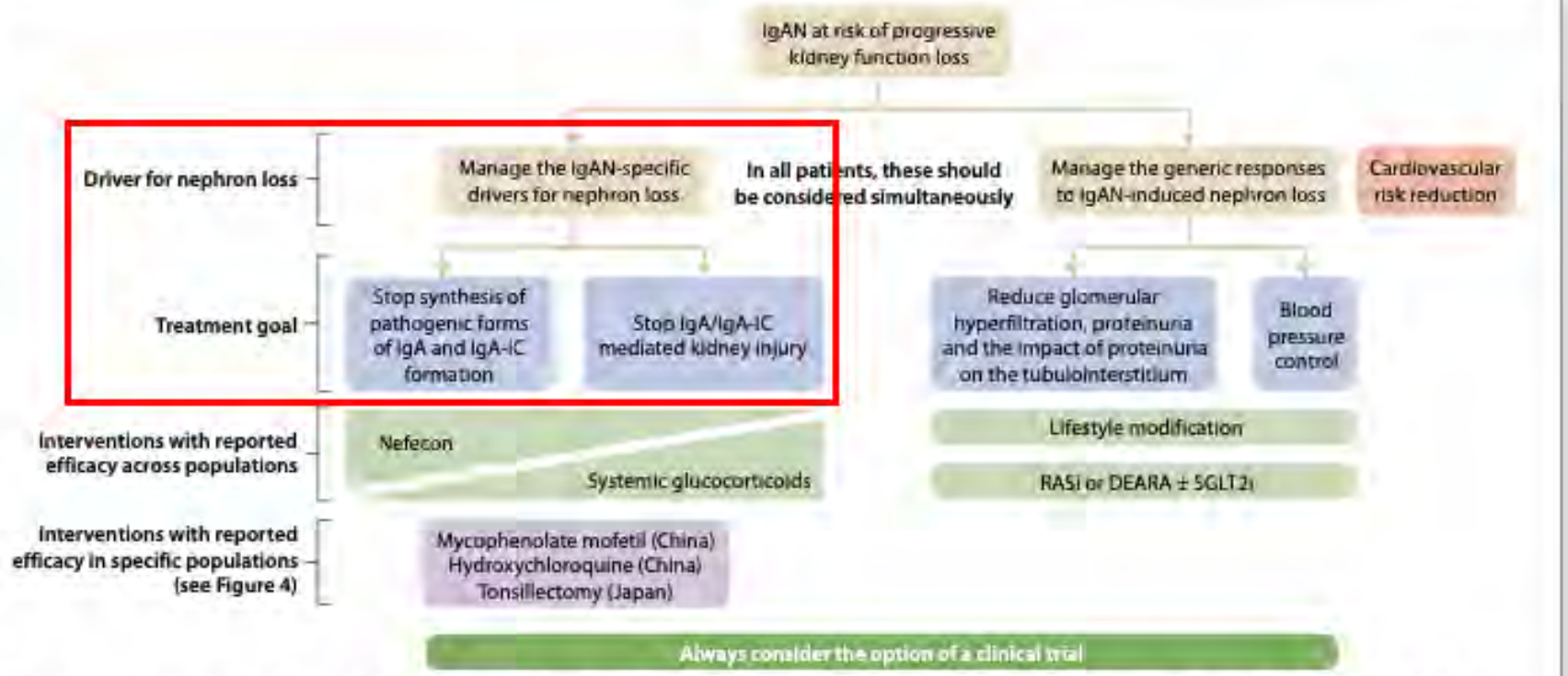
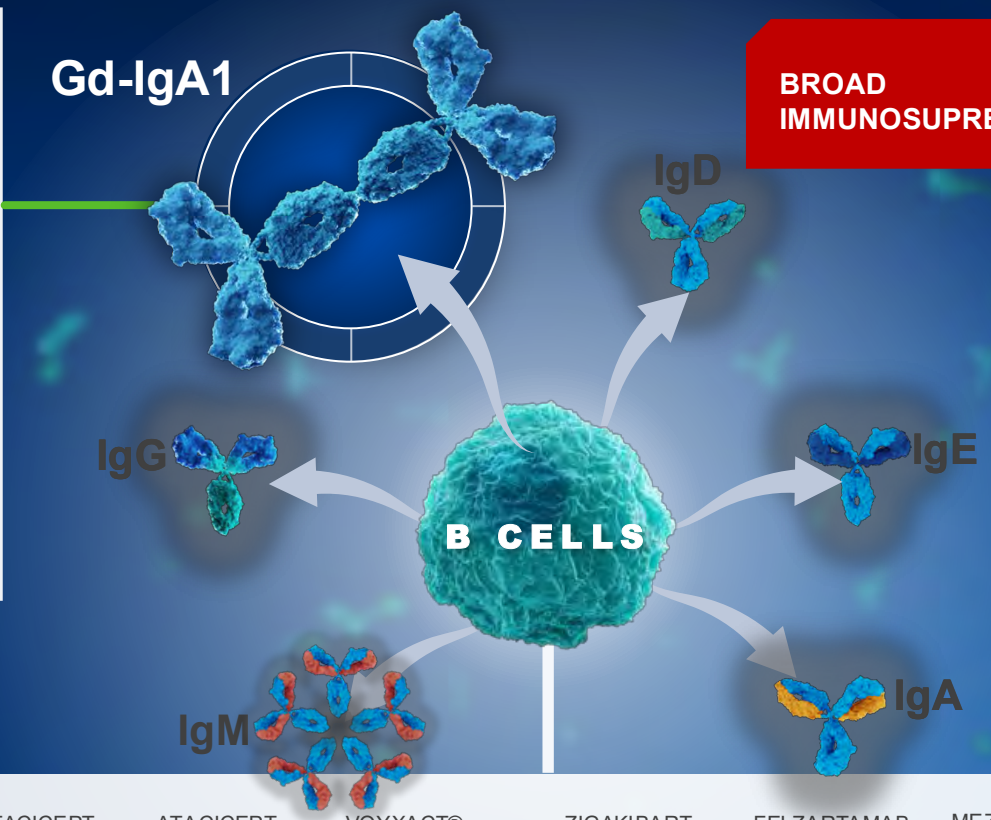


Figure 3 | Treatment targets in immunoglobulin A nephropathy (IgAN) and the positioning of drugs included in this guideline. Reflecting current understanding, Nefecon is shown as having a predominant effect on the production of pathogenic forms of IgA and IgA-containing immune complexes (IgA-ICs), with an undetermined direct effect of systemically absorbed budesonide on the kidneys. Systemic glucocorticoids have a well-documented anti-inflammatory effect within the kidneys and an undetermined direct effect on the production of pathogenic forms of IgA and IgA-ICs. Strategies to manage the generic response to IgAN-induced nephron loss may also include the use of 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase inhibitors in selected patients. DEARA, dual endothelin angiotensin receptor antagonist; RASi, renin-angiotensin system inhibitor; SGLT2i, sodium-glucose cotransporter-2 inhibitor.

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

DEGRADERS

TRAP™ Degrader
BHV-1400 SELECTIVELY DEGRADES ONLY Gd-IgA1
Targeting the pathogenesis of disease without immunosuppression



BROAD IMMUNOSUPRESSION
TARPEVO®
calliditas THERAPEUTICS

INHIBIT COMPLEMENT SYSTEM
WITH BROAD IMMUNOSUPPRESSION
FABHALTA® NOVARTIS
SEFAXERSEN IONIS Roche
ULTOMIRIS® CHINOOK THERAPEUTICS NOVARTIS

TARGET B CELLS
WITH GLOBAL IMMUNOGLOBULIN SUPPRESSION
POVETACICEPT ALPINE IMMUNOSCIENCES VERTEX
ATACICEPT vera therapeutics
VOYXACT® Otsuka
ZIGAKIBART CHINOOK THERAPEUTICS NOVARTIS
FELZARTAMAB Biogen
MEZAGITAMAB Takeda

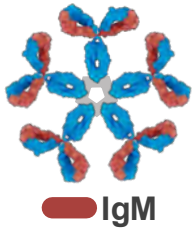
TARGET ENDOTHELIAN RECEPTOR
FILSPARI® TRAVERE THERAPEUTICS
VANRAFIA® CHINOOK THERAPEUTICS NOVARTIS

KEY POINT

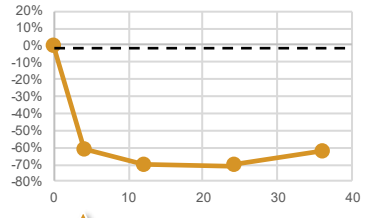
BHV-1400 is the only therapy designed to remove pathogenic Gd-IgA1, the root cause of IgAN, while preserving healthy immune function

Competitors Show Long-Term Immunosuppression[1,2]

DEGRADERS

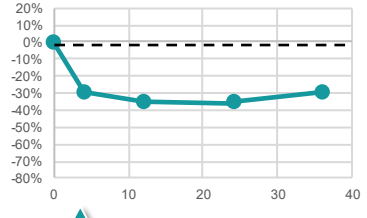


Mezagitamab 600 mg SC[2,4]



61% DROP
MONTH 4

62% DROP FROM BASELINE MONTH 36

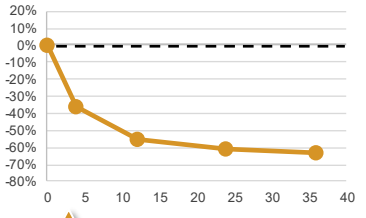


30% DROP
MONTH 4

30% DROP FROM BASELINE MONTH 36

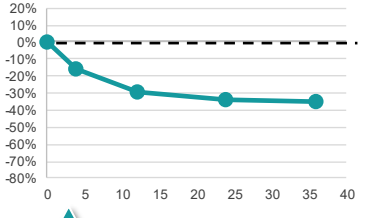
IgM Data not Reported

Atacicept 150 mg[2,5]



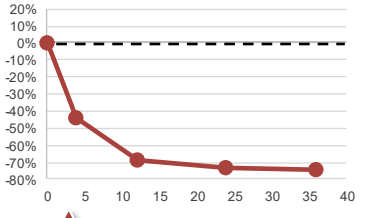
36% DROP
DAY 30

64% DROP FROM BASELINE WEEK 36



16% DROP
DAY 30

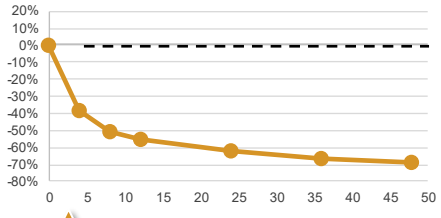
35% DROP FROM BASELINE WEEK 36



44% DROP
DAY 30

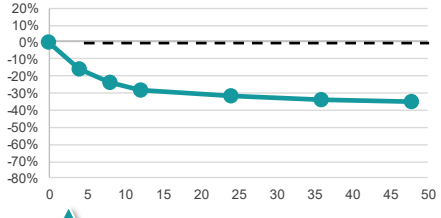
75% DROP FROM BASELINE WEEK 36

VOYXACT SC[2,6]



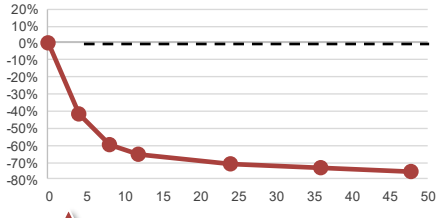
38% DROP
DAY 30

69% DROP FROM BASELINE WEEK 48



16% DROP
DAY 30

35% DROP FROM BASELINE WEEK 48



42% DROP
DAY 30

75% DROP FROM BASELINE WEEK 48

VOYXACT FDA LABEL[7]

WARNINGS AND PRECAUTIONS

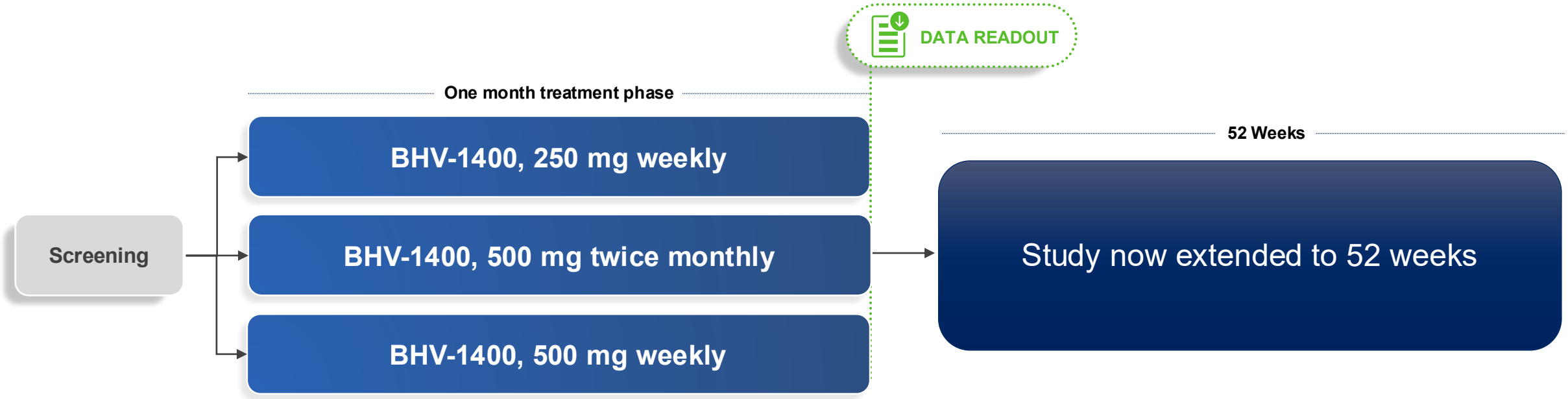
Immunosuppression and Increased Risk of Infections
VOYXACT suppresses the immune system by reducing antibody production, which may increase the risk of infections.

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.

Months **Weeks** **Weeks**

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. Barratt. American Society of Nephrology Kidney Week 2025. Poster FP-PO0808. **5.** Lafayette. Kidney International. 2024 **6**. Lafayette. NEJM. 2025. **7**. https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/761434s000lbl.pdf

IgAN Patient Expansion Cohort Study Design



KEY STUDY DETAILS

Study Design: Open-label, n=10, potential to increase

Population: Male and female adults with biopsy proven IgAN, UPCR ≥ 0.5 g/d, eGFR ≥ 30

Endpoints: Primary safety, pharmacodynamic measures

Demographic and Clinical Characteristics of the Patients Receiving BHV-1400 at Baseline

DEGRADERS

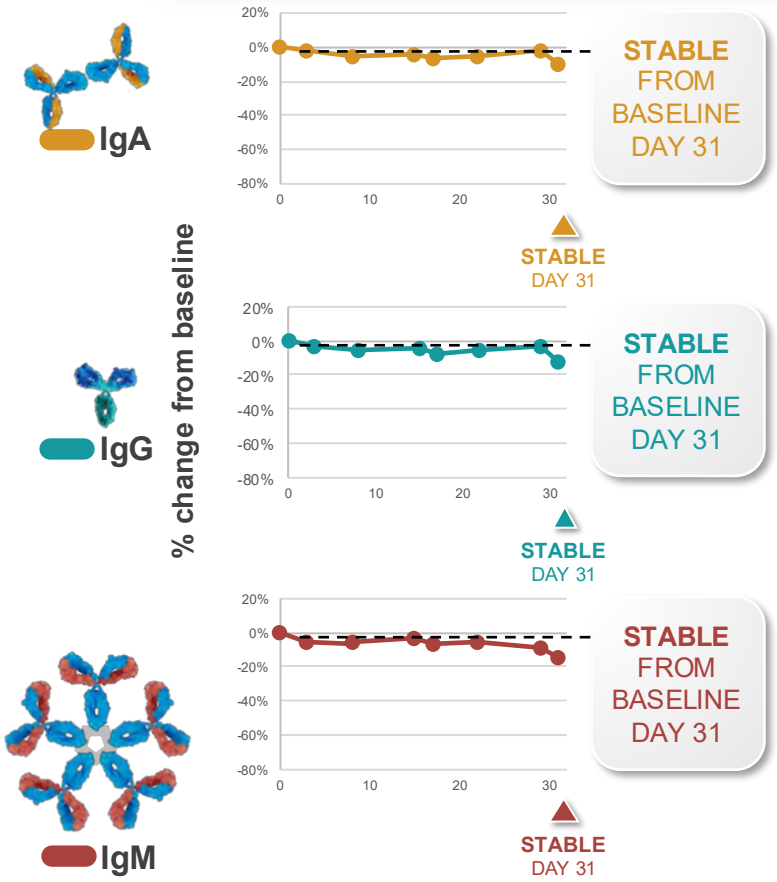
Characteristics	Overall (n=10)
Age (yrs) Mean [Min, Max]	43.6 [27, 65]
Sex n (%) Male Female	 6 (60%) 4 (40%)
Race n (%) Asian White Unknown	 3 (30%) 6 (60%) 1 (10%)
Spot UPCR* (mg/g) Mean [Min, Max]	766.4 [213–2144]
eGFR** (ml/min/1.73 m^2) Mean [Min, Max]	67 [33–124]
Hematuria (1+, 2+, or 3+) n (%)	2 (20%)
Time from Biopsy (yrs) Mean [Min, Max]	3.03 [0.3, 8.9]

UPCR, urinary protein-to-creatinine ratio. **eGFR:** estimated glomerular filtration rate.

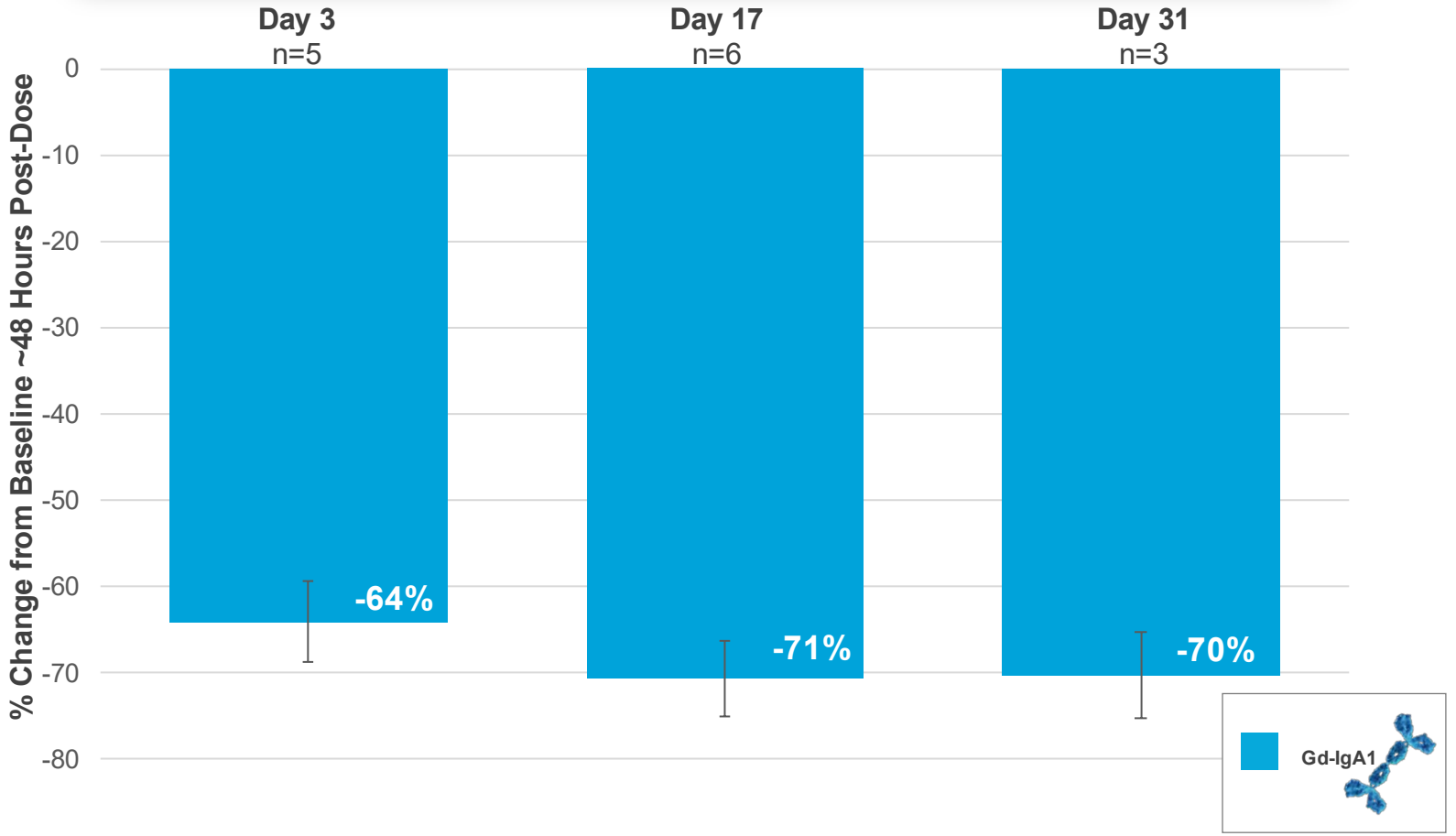
BHV-1400 500 mg SC Bi-Monthly Deeply and Selectively Removes Gd-IgA1 Without Suppression of Normal Healthy IgA in Patients With IgAN

DEGRADERS

Healthy Immunoglobulins



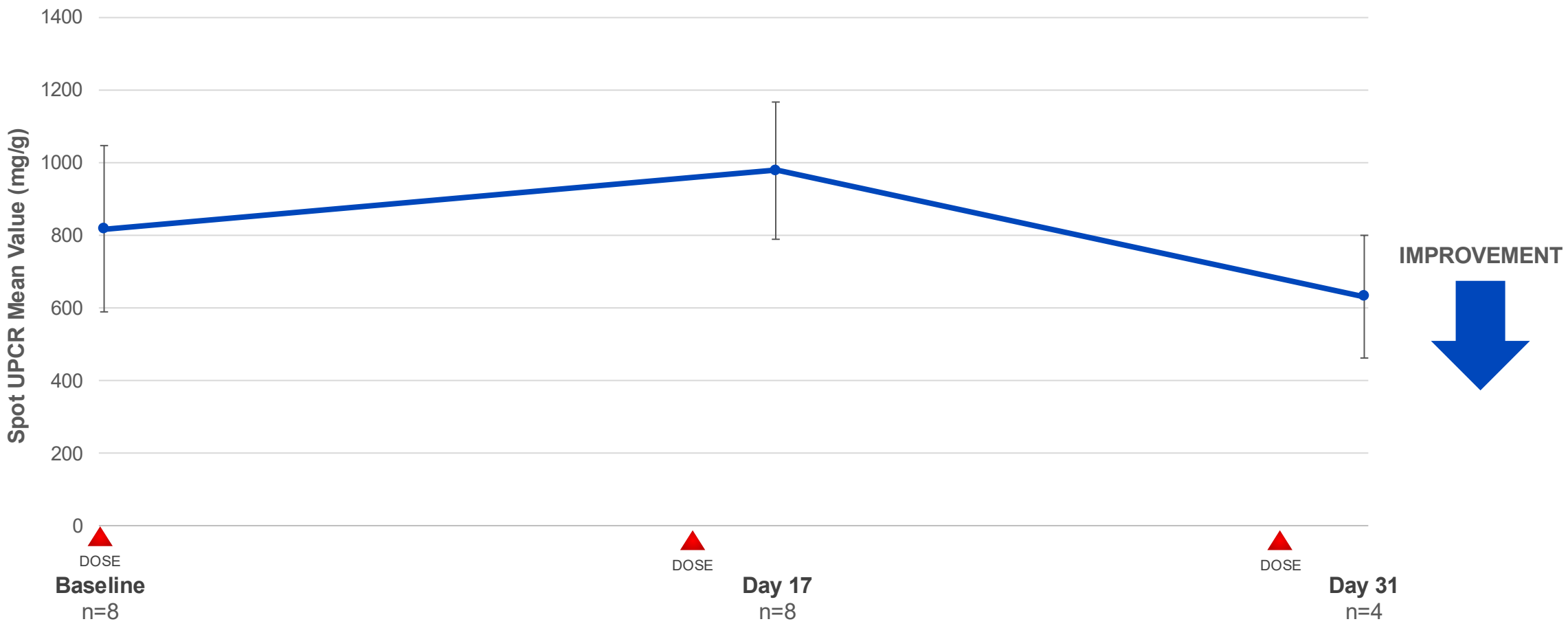
Gd-IgA1



Preliminary data from ongoing study. Data represents mean % change in immunoglobulins in patients with IgAN administered one month of BHV-1400 500 mg every two weeks. Error bars represent standard error.

UPCR Change With One Month of Dosing BHV-1400 SC

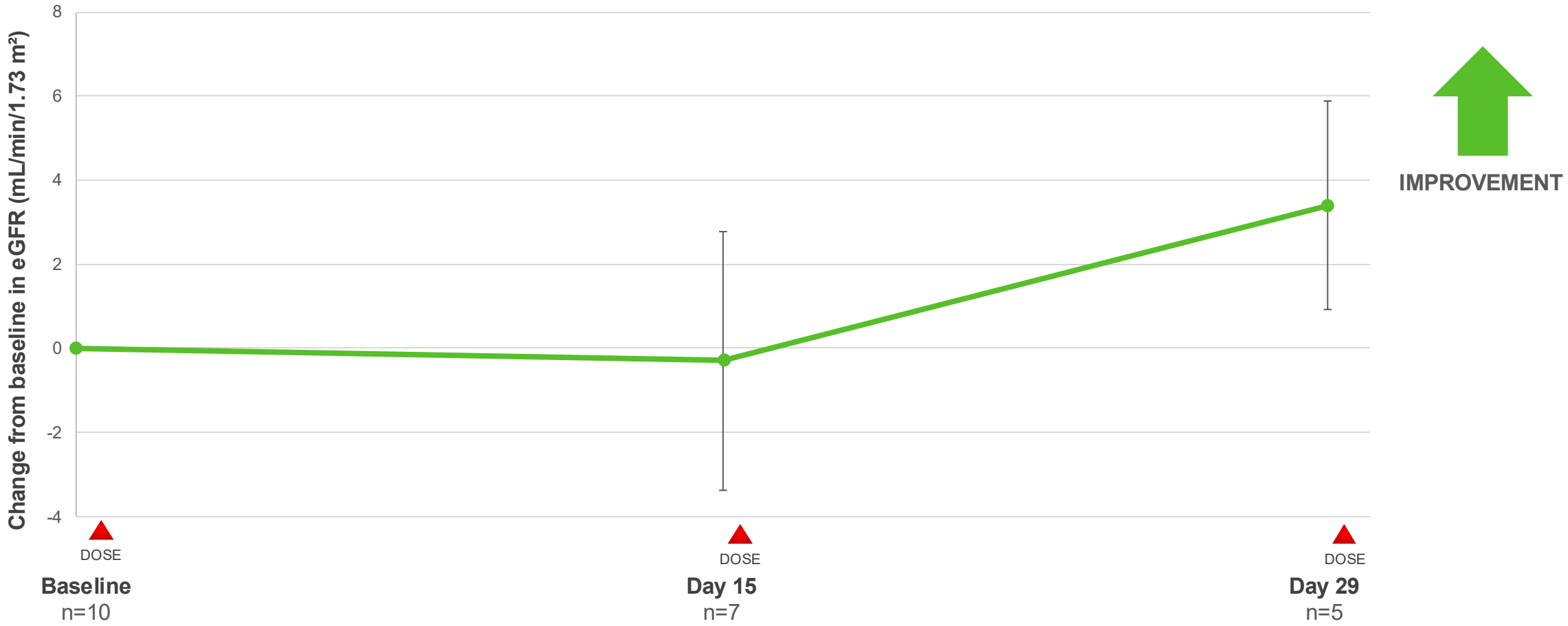
DEGRADERS



Preliminary data from ongoing study. Graph represent mean and standard error of spot UPCR in participants with IgAN (n=8) receiving BHV-1400 SC for one month of dosing. Data from Day 17 or Day 31 not yet available for all participants.

eGFR Change With One Month of Dosing BHV-1400 SC

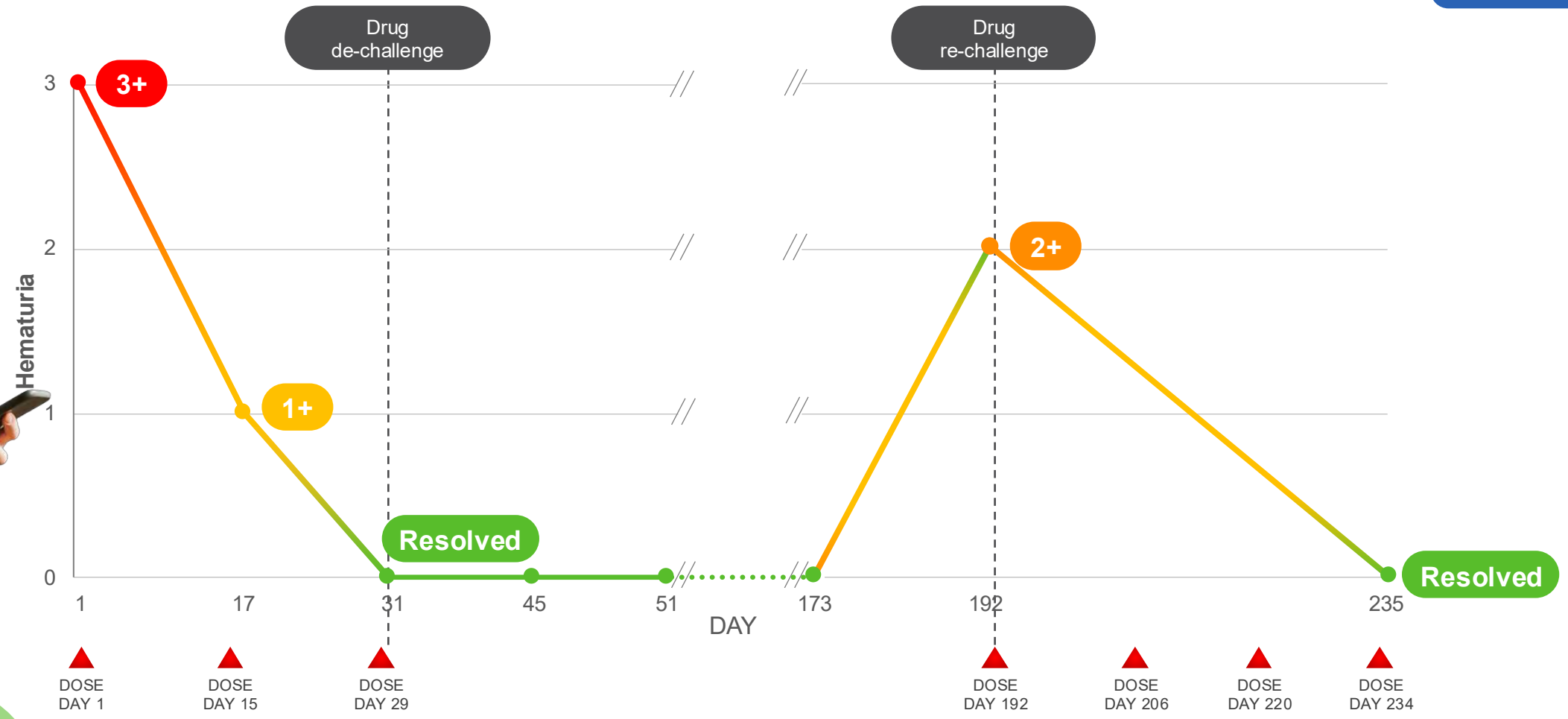
DEGRADERS



Preliminary data from ongoing study. Graph represents mean and standard error of eGFR in participants with IgAN (n=10) receiving BHV-1400 SC for one month of dosing. Data from Day 15 or Day 29 not yet available for all participants.

BHV-1400 Hematuria Case Report: De-Challenge Re-Challenge Recaptures Complete Hematuria Resolution

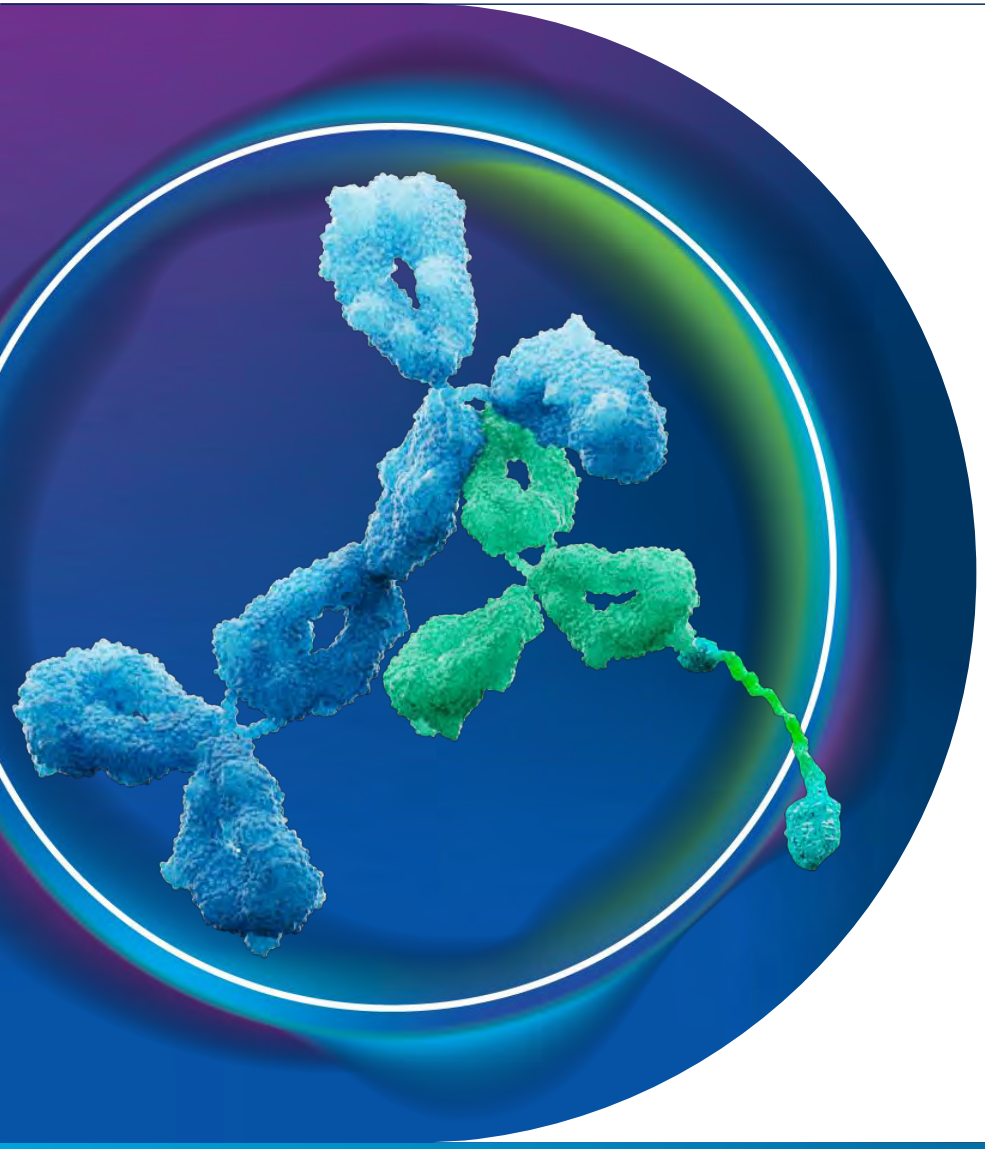
DEGRADERS



KEY POINTS

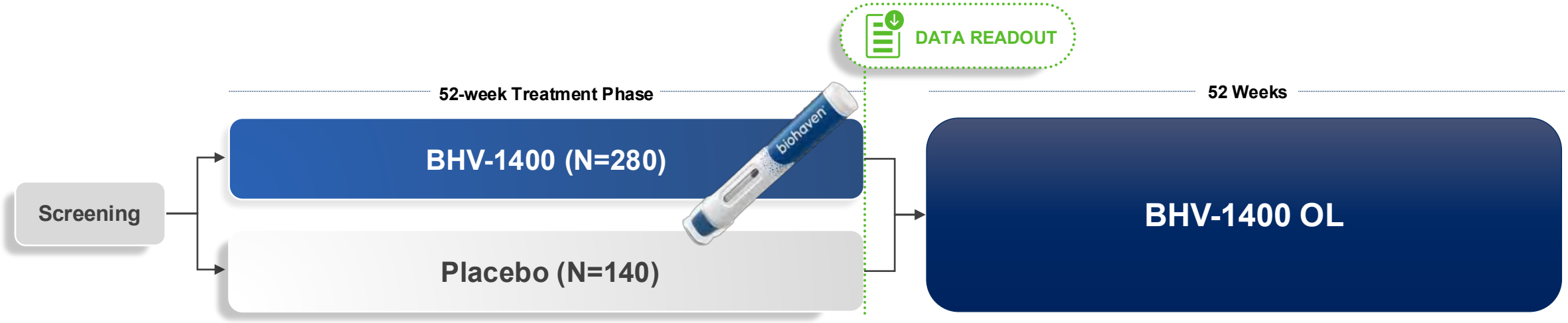
- Re-treatment with BHV-1400 500 mg every two weeks initiated upon disease recurrence after extended off-treatment period
- Complete resolution of hematuria re-achieved within weeks of reinitiation

BHV-1400 Phase 1b IgA Nephropathy Patient Cohort — Safety Summary



- 10 participants with IgA nephropathy
- Most AEs were mild and self-resolving
- No treatment discontinuations for adverse events
- No clinically significant trends in vitals, ECGs, or labs (including AST/ALT/Tbili)
- No clinical evidence of cardiovascular, renal, hepatic, or hematologic toxicity
- Preservation of IgA, IgG, IgM, IgE
- No SAEs, severe AEs, or AEs resulting in discontinuation of therapy

Lead TRAP Degrader BHV-1400 Enters Phase 3



KEY STUDY DETAILS

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

Population: Male and female adults with biopsy proven IgAN

Dose: 500 mg bimonthly, at home administration

Endpoints: Δ UPCR, Δ in eGFR, Δ in Gd-IgA1 at week 52

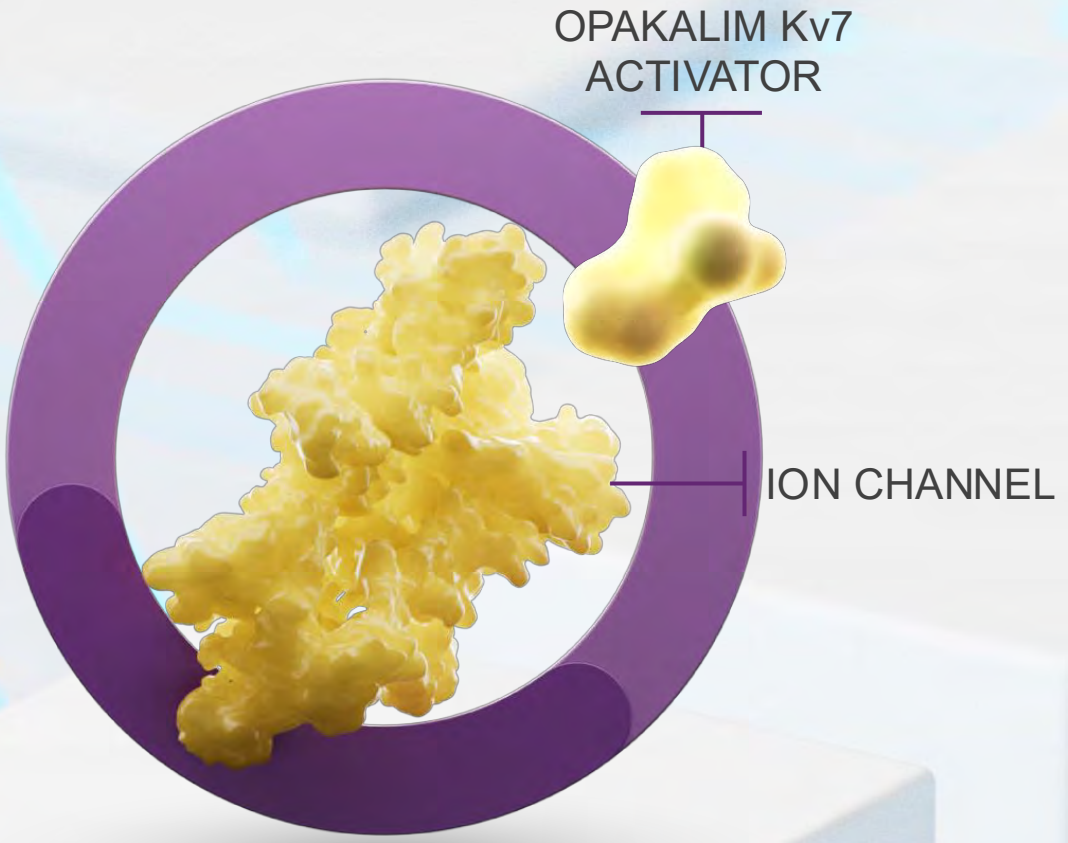
KEY POINT

BHV-1400 pivotal trial in IgA nephropathy commencing mid-2026

biohaven®

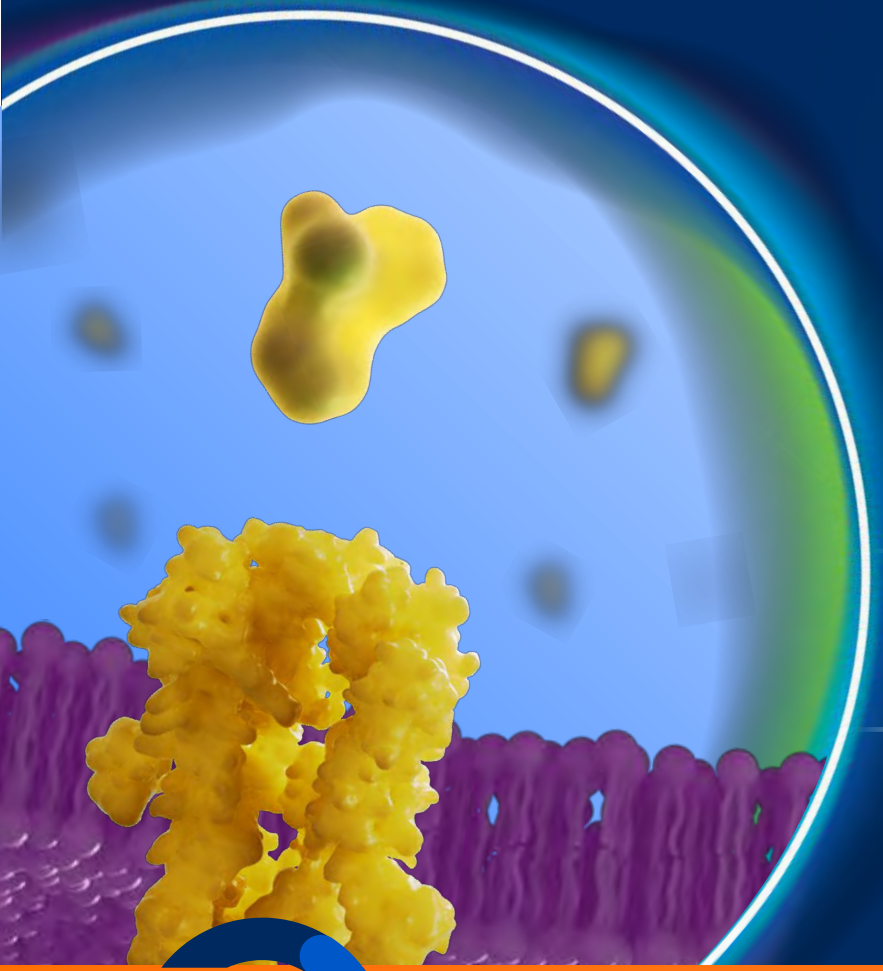
SELECTIVE Kv7 ACTIVATOR: OPAKALIM

Revolutionizing Epilepsy Treatment With a Modern Kv7 Activator



An Epilepsy Treatment Designed With Patients and Physicians in Mind

Kv7



Opakalim offers potential for easy-to-use, once-daily treatment with no titration to control seizures without the burdensome side effects frequently reported with approved ASMs and those in development

Selectively activates Kv7.2/7.3 channels—a validated MOA for treating epilepsy—without impacting GABA receptors

Exhibits preliminary efficacy signals in focal epilepsy OLE, KCNQ2-DEE and now idiopathic generalized epilepsy

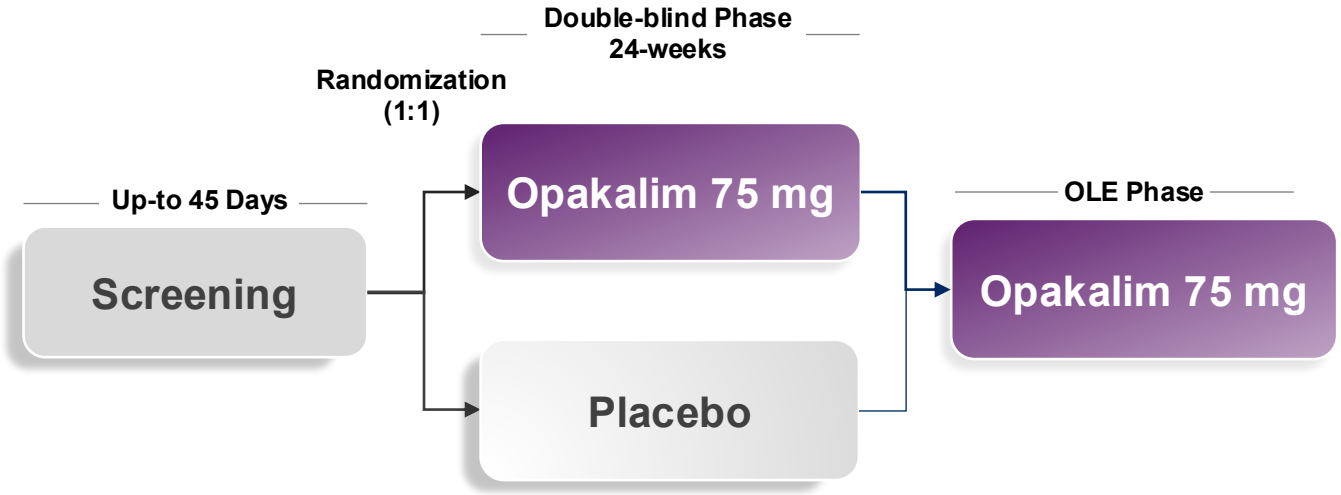
Demonstrates exceptional safety profile with low rates of CNS adverse events across all trials (1000+ subjects)

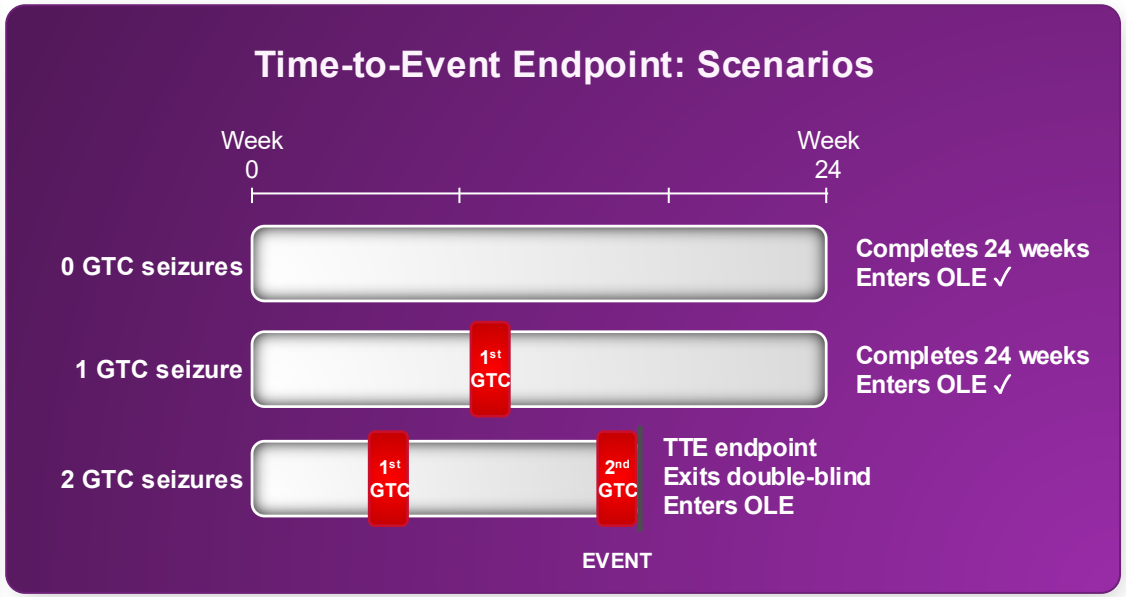
BREAKING NEWS

Recent clinical data updates reinforce efficacy and differentiated tolerability

Idiopathic Generalized Epilepsy Time-to-Event Study Design

Kv7





KEY STUDY DETAILS

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

Endpoint: Primary - Time-to-event (Event = 2nd day with GTC seizure), **Secondary -** GTC seizure freedom

Population: Subjects 18–75 with IGE and intractable GTC seizures

Key Entry Criteria: 3 GTC seizures within the historic 16-week seizure assessment period

Study terminated early due to enrollment and strategic portfolio prioritization; GTC: generalized tonic-clonic; IGE: idiopathic generalized epilepsy; TTE: time-to-event

Demographics and Baseline Disease Characteristics

Kv7

	Opakalim 75 mg n=15	Placebo n=12*
Age (mean)	**37**	43
Sex (% female)	**73%**	83%
Region (% US)	**47%**	25%
BMI (mean)	**27**	28
Number of epilepsy treatments at screening		
1 to 2	**60%**	67%
3 to 4	**40%**	33%
Number of previous and current ASMs		
≤ 6	**11 (73%)**	10 (83%)
> 6	**4 (27%)**	2 (17%)
Age at IGE diagnosis (mean)	**14**	13
Years since IGE diagnosis (mean)	**23**	30

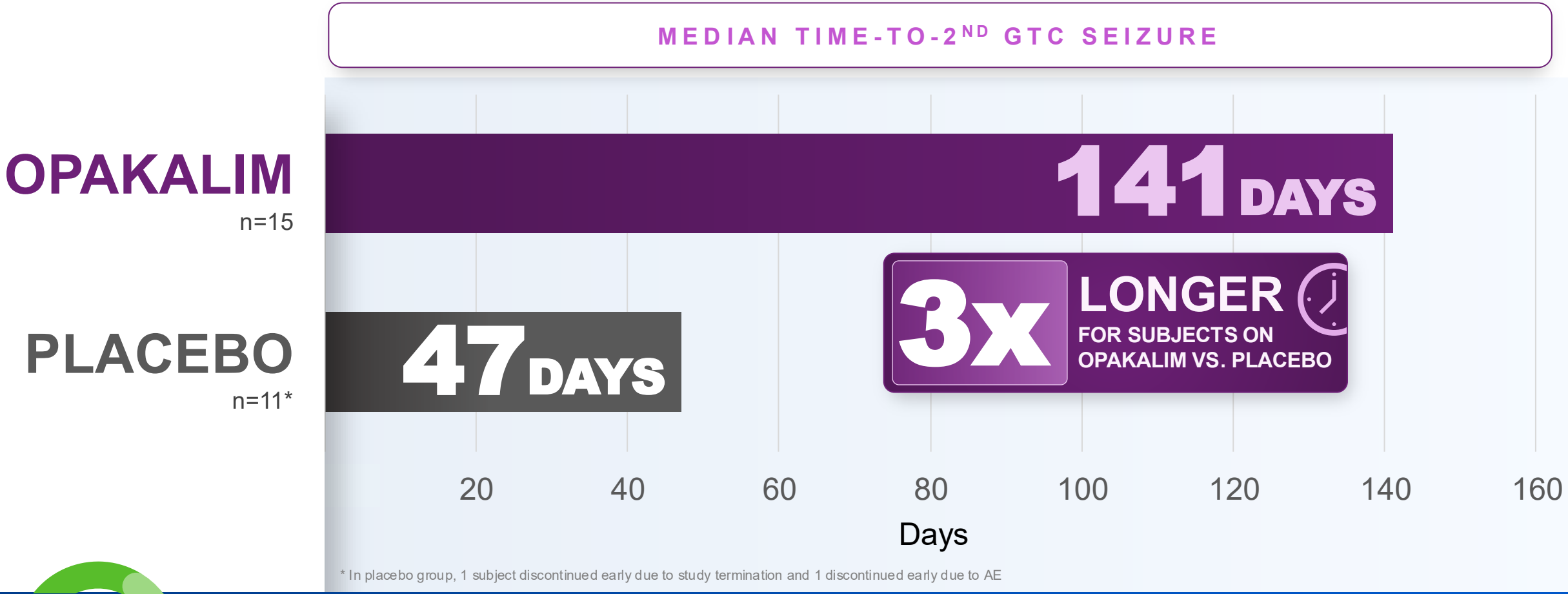
* In placebo group, 1 subject did not have post-dose efficacy data

KEY POINT

Highly treatment-resistant idiopathic generalized epilepsy population

Opakalim Prolongs Time-to-2nd GTC Seizure

Kv7



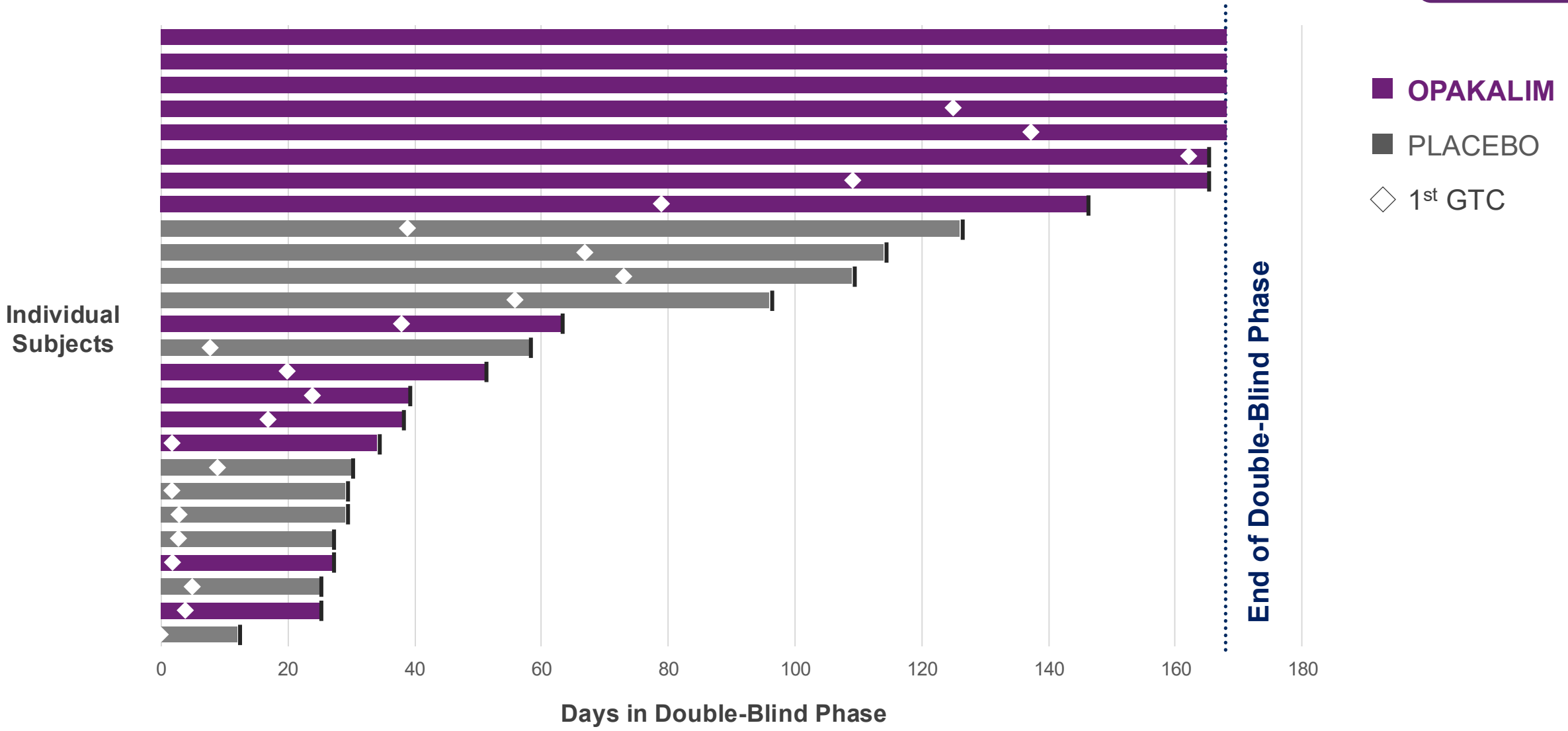
* In placebo group, 1 subject discontinued early due to study termination and 1 discontinued early due to AE

KEY POINT

Efficacy signal observed in idiopathic generalized epilepsy population

Opakalim Prolongs Time-to-2nd GTC Seizure and Time in Double-Blind

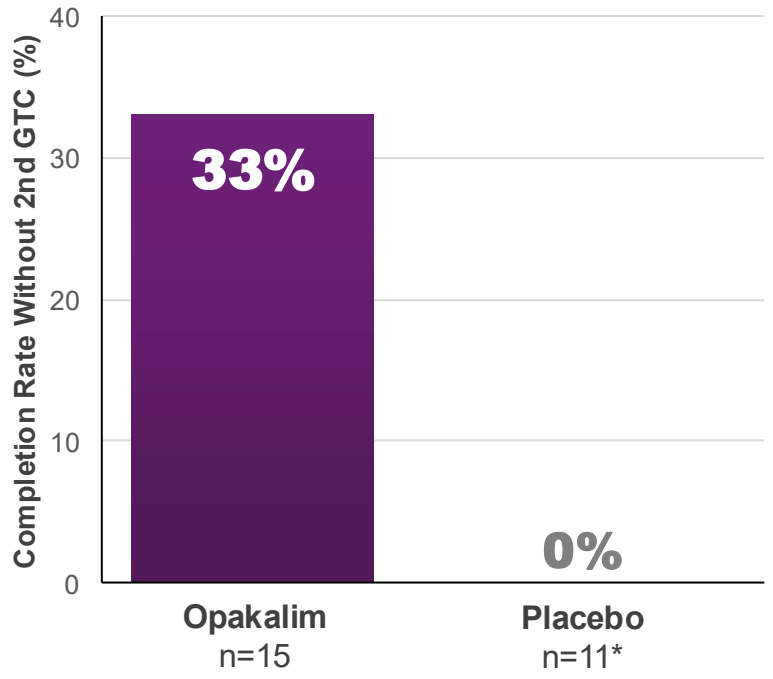
Kv7



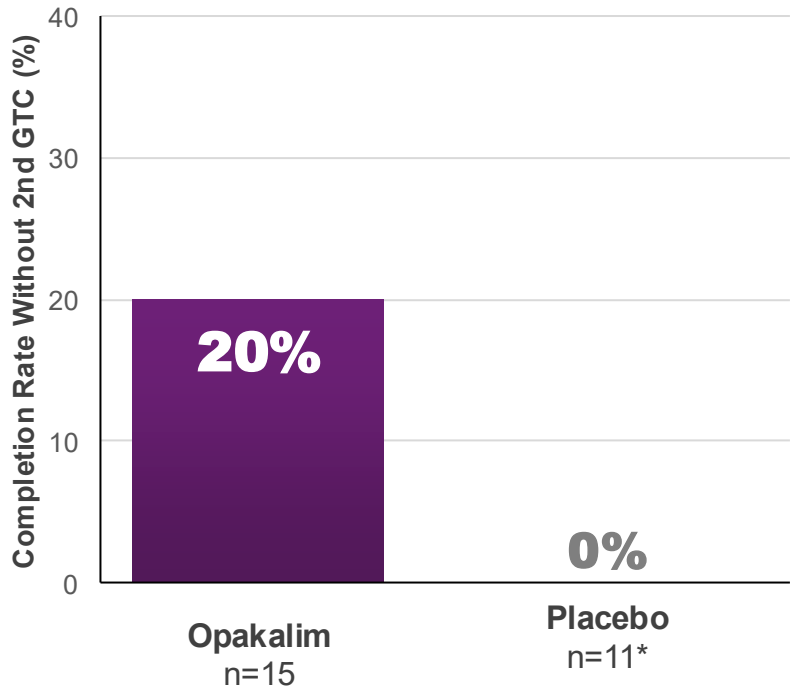
Opakalim Prolongs Time in Double-Blind and Enhances Seizure Control

Kv7

33% of Opakalim-Treated Subjects Completed Six-Month Double-Blind Phase Without 2nd GTC



20% of Opakalim-Treated Subjects Completed Double-Blind Phase Seizure Free

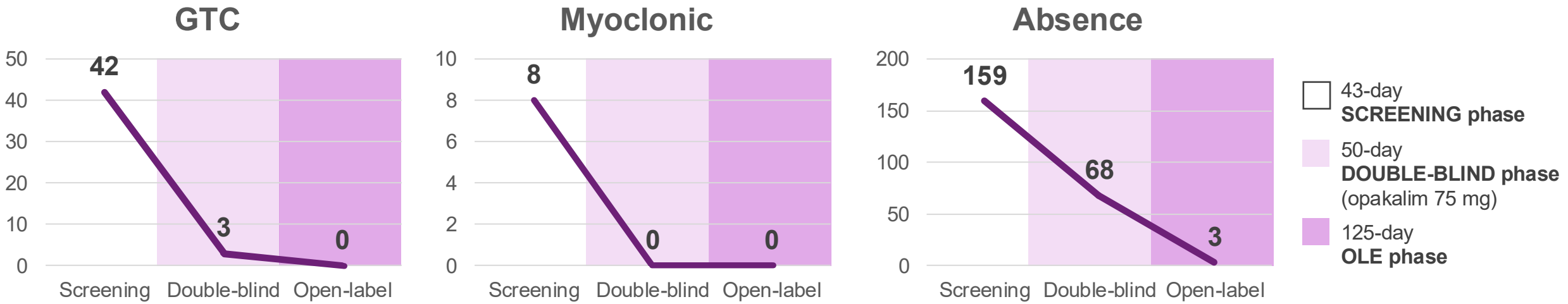


* In placebo group, 1 subject discontinued early due to study termination and 1 discontinued early due to AE

Promising Case of Broad-Spectrum Generalized Seizure Efficacy

Kv7

Our patient has very difficult-to-treat IGE, since entering the opakalim trial she has experienced reduction in all seizure types.



In terms of tolerability, she continues to do very well, with no adverse events.

Opakalim IGE Double-Blind Phase topline data, 1H 2026

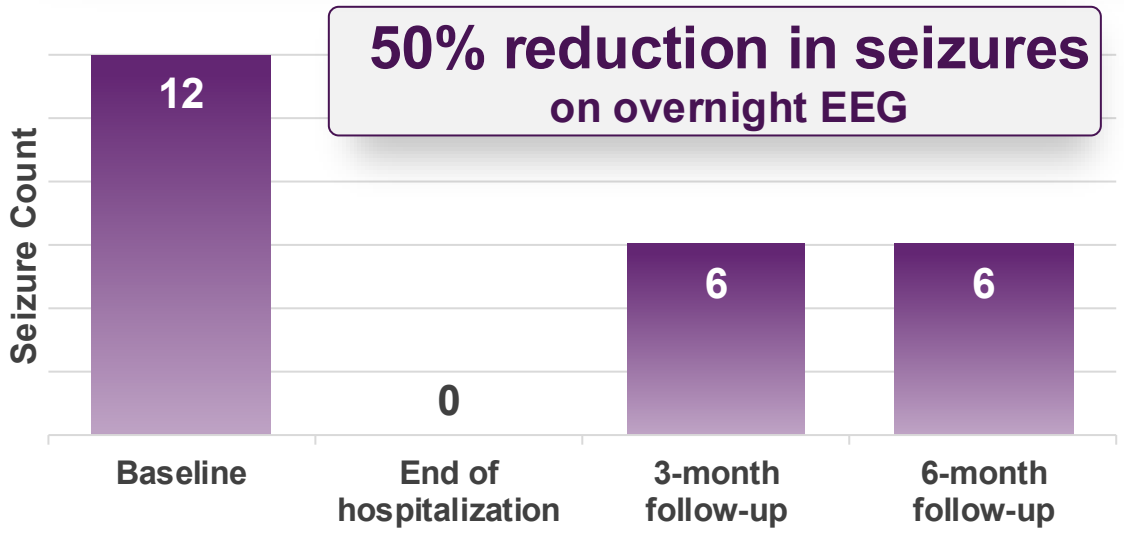
KEY POINTS

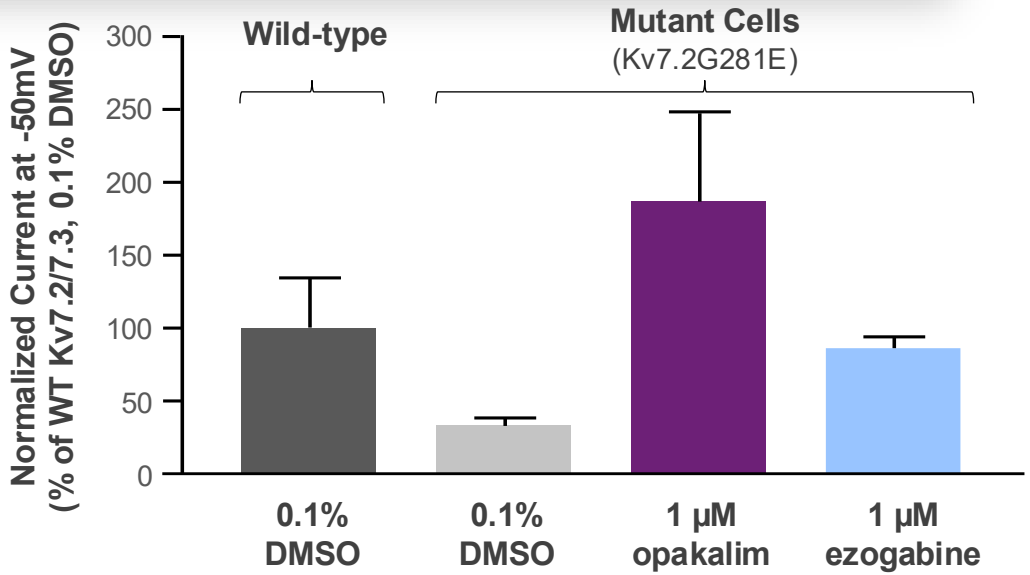
- 50% of subjects with myoclonic seizures became myoclonic seizure free on opakalim
- 33% of subjects with absence seizures became absence seizure free on opakalim

Efficacy Signal Demonstrated in KCNQ2-DEE

9-year-old boy with
- **Refractory KCNQ2-DEE**
- **Kv7 activation-dependence**

- Heterozygous for Kv7.2 G281E mutation
- Daily tonic seizures at baseline despite 3 ASMs including 1st gen Kv7 activator
- Prior attempts to taper 1st gen Kv7 activator resulted in **status epilepticus, ICU admission and developmental regression**





Olson. AAN 2026. Poster #P10 11-002; Equivalent exposures to 75 mg dose in pivotal focal epilepsy studies

KEY POINT

Successfully transitioned from 1st gen Kv7 to opakalim and stable for 6+ months

Efficacy Signal 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

>54% OF PATIENTS SHOWED

50% RESPONSE RATE

OVER ANY CONSECUTIVE 6-MONTH PERIOD IN OLE (n>100)

* RISE 2 Part B: opakalim 75 mg

Opakalim ongoing focal epilepsy preliminary data 1H 2026 for open-label 6-month completers; French. Epilepsia Open. 2025; Indirect comparisons between compounds based on publicly available data.

KEY POINT

50% RR for opakalim (54%) comparable to azetukalner (56%)

Exceptional Tolerability Observed in Focal Epilepsy Open-Label Data

Kv7

Preferred Term	Opakalim 50 mg	Opakalim 75 mg	Opakalim Pooled
Headache	4.5%	6.4%	5.7%
Nasopharyngitis	4.5%	6.4%	5.7%
Seizure	5.3%	3.7%	4.3%
Dizziness	3.0%	5.0%	4.3%
Fatigue	3.0%	4.1%	3.7%
Fall	2.3%	4.6%	3.7%
Upper respiratory tract infection	3.0%	4.1%	3.7%
Back Pain	3.8%	3.2%	3.4%
Insomnia	5.3%	2.3%	3.4%
Nausea	3.8%	2.8%	3.1%
Diarrhea	6.0%	1.4%	3.1%

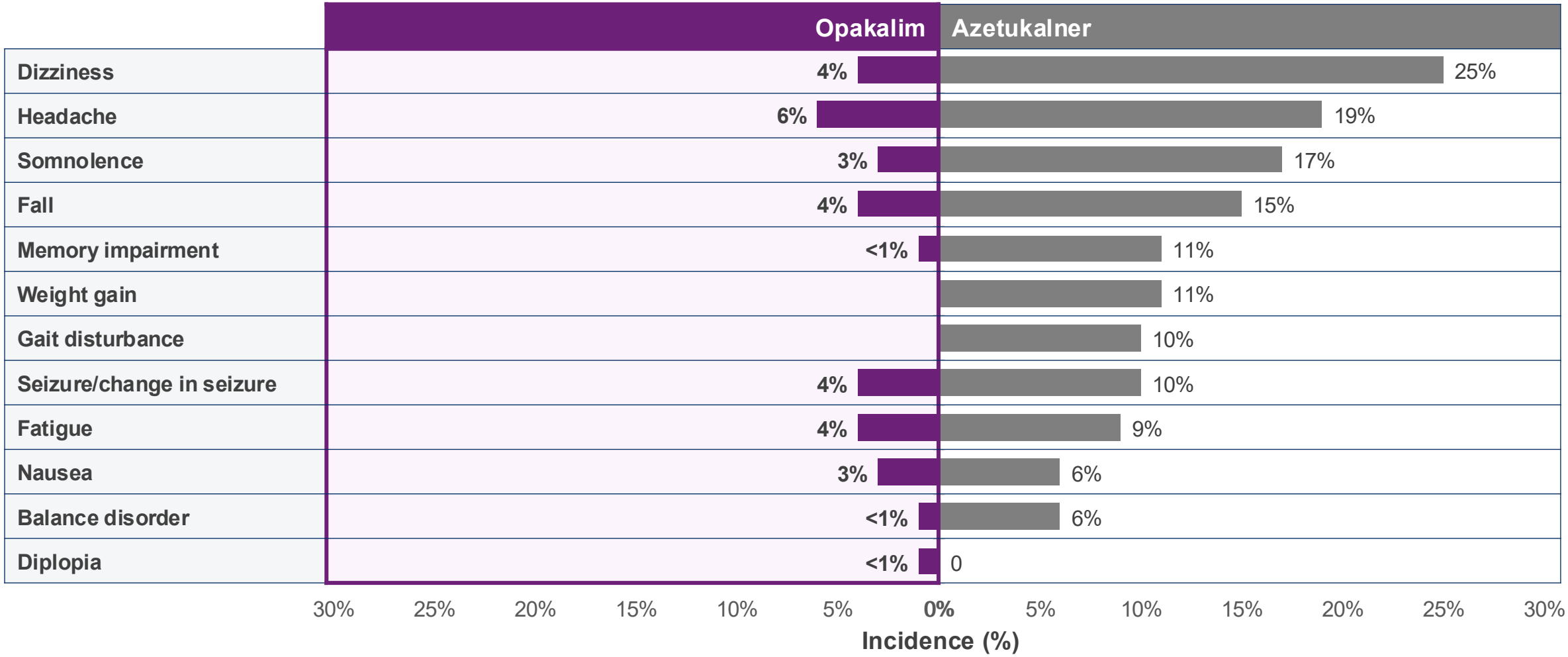
Adverse events reported in ≥3% of pooled participants, opakalim ongoing focal epilepsy open-label preliminary data 1H 2026

KEY POINT

Low incidence, majority mild and spontaneously resolved

Opakalim Demonstrates Favorable Tolerability vs. Azetukalner in Focal Epilepsy Open-Label Trials

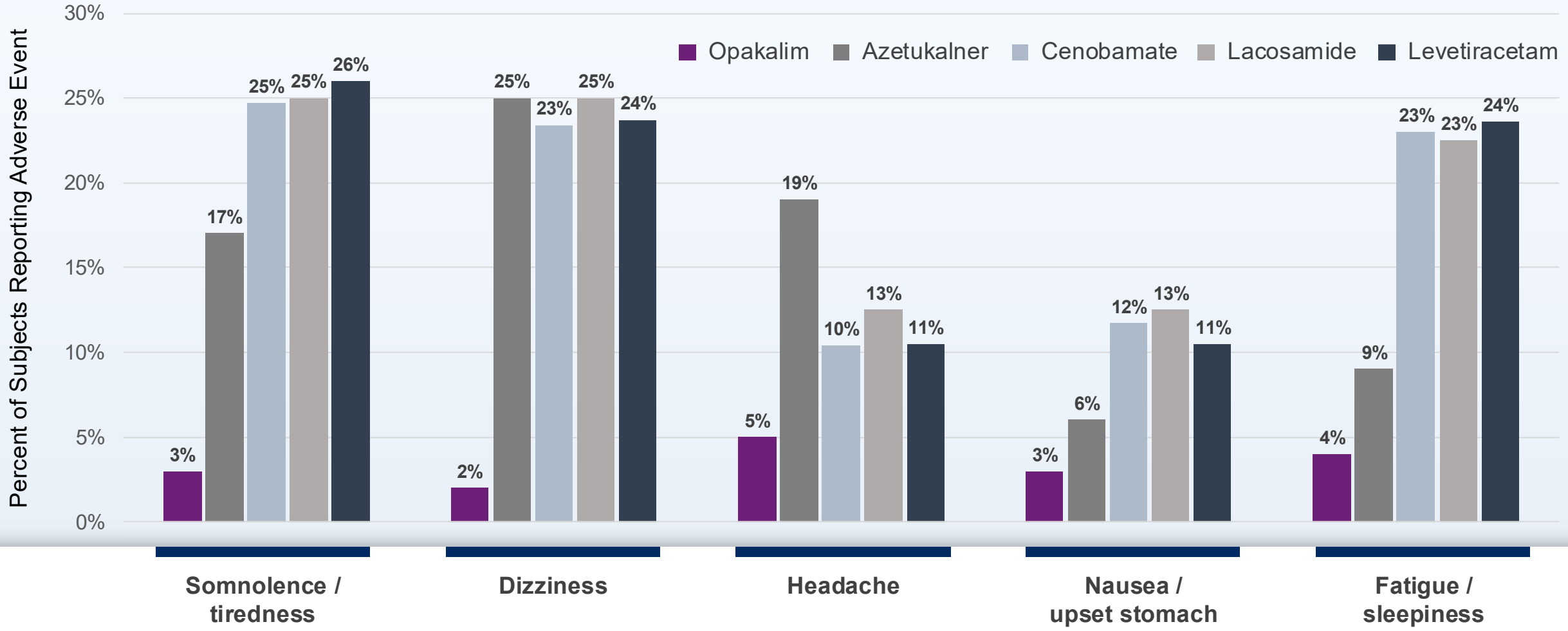
Kv7



Opakalim ongoing focal epilepsy open-label preliminary data 1H 2026; percentages rounded to the nearest whole percent; Azetukalner focal epilepsy data from Open Label Study – French. AES 2025. Poster #3.356.

Opakalim Demonstrates Favorable Tolerability vs. Approved and Investigational ASMs

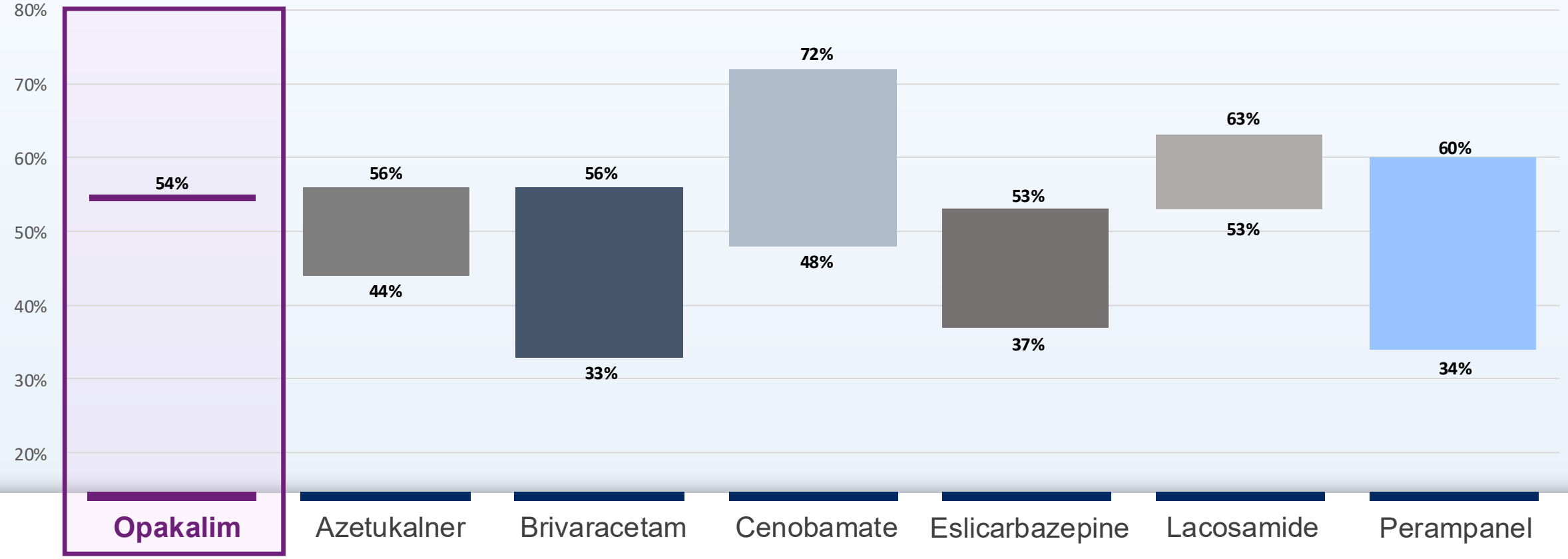
Kv7



Opakalim ongoing focal epilepsy open-label preliminary data 1H 2026; Azetukalner data from Open Label Study – French. AES 2025. Poster #3.356; Cenobamate, lacosamide and levetiracetam data from Winter. CNS Drugs. 2024.

50% Responder Rate in OLE Trials of Several Approved and Investigational ASMs

Kv7



Opakalim ongoing focal epilepsy open-label preliminary data 1H 2026; Hufnagel. Epilepsy Res. 2013; Halász. Epilepsia. 2010; Strzelczyk. Epilepsia. 2021; Ben-Menachem. Epilepsy Res. 2021; O'Brien. Epilepsia. 2020; Klein. Neurology. 2022; Strzelczyk. Expert Rev Clin Pharmacol. 2015; Husain. Epilepsia. 2012; French, J. Epilepsia Open. 2025; Rektor. Epilepsia. 2020.

KEY POINT

Opakalim OLE preliminary efficacy outcomes fall within reported range of responder rates for other ASMs in OLE trials

Opakalim Is Easy-to-Use With a Projected Favorable Tolerability Compared to Approved ASMs

Kv7

		No titration	Favorable CNS tolerability	Low neuropsychiatric AEs	Low metabolic / electrolyte AEs	Low SJS or DRESS risk
L1	Lamotrigine	✗✗	✓	✓	✓	✗✗
	Levetiracetam	✓	✓	✗	✓	~
	Oxcarbazepine	✗	✗	✓	✗	✗
L2	Lacosamide	✗	✓	✓	✓	✓
	Eslicarbazepine	✗	✗	✓	✗	~
	Brivaracetam	~	✗	✗	✓	✓
L3	Zonisamide	✗	✗	✗	✗	✓
	Cenobamate	✗✗	✗	✓	✓	✗✗
	Topiramate	✗	✗	✗	✗	✓
	Opakalim (Kv7)	✓	✓	✓	✓	✓

✓ Favorable ~ Variable ✗ Unfavorable ✗✗ Very Unfavorable

AE, Adverse Event. **SJS,** Stevens-Johnson Syndrome. **DRESS,** Drug Reaction with Eosinophilia and Systemic Symptoms

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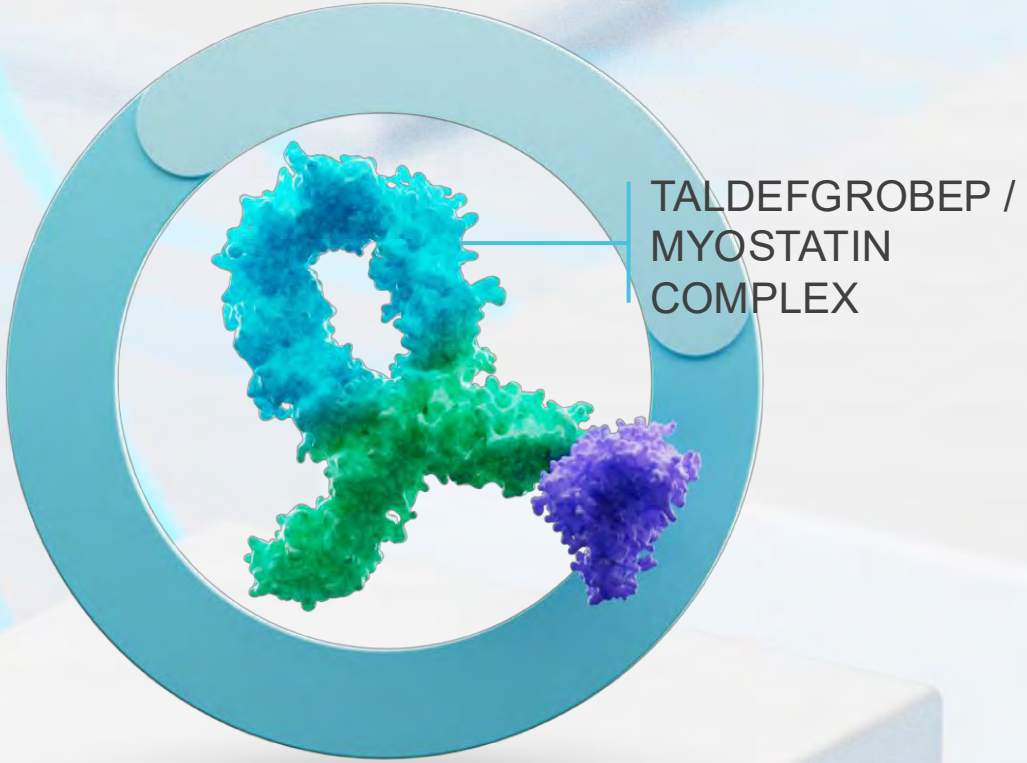
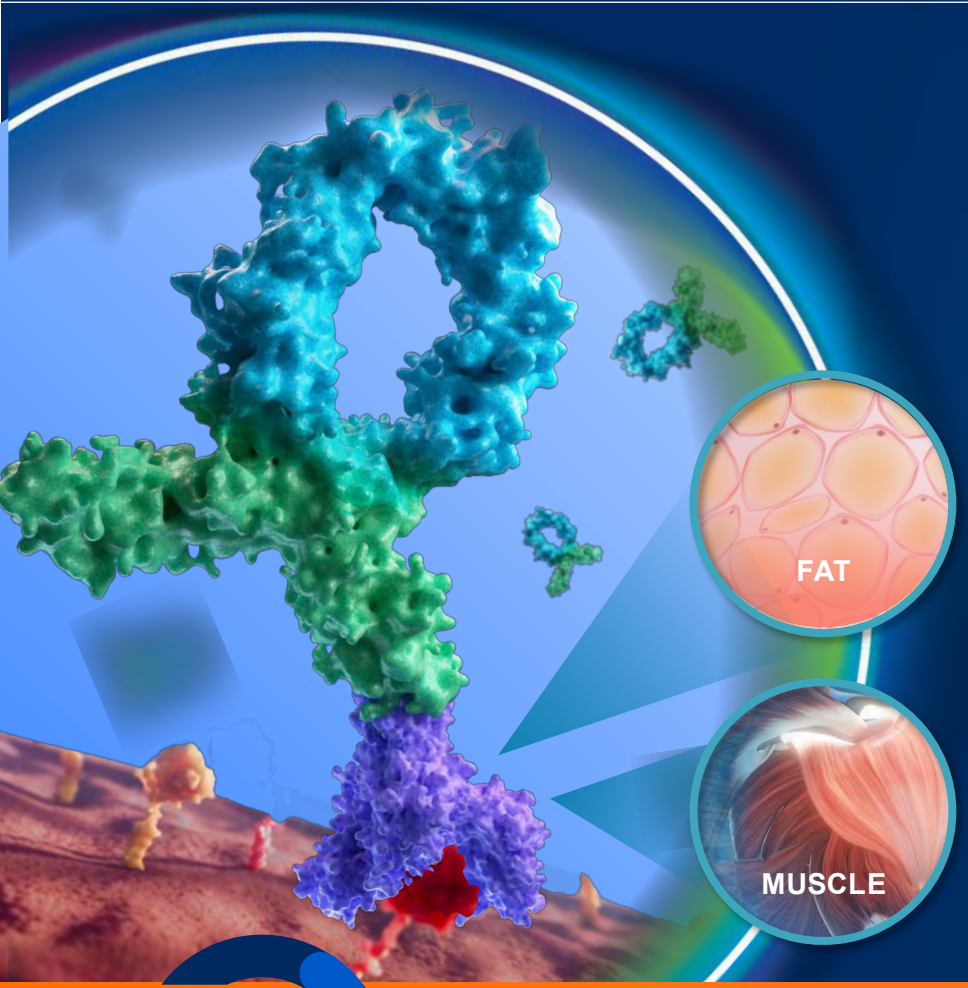
MYOSTATIN ACTIVIN PATHWAY INHIBITOR:
TALDEFGROBEP ALFA



Targeting High-Quality Weight Loss in Obesity

Targeting High-Quality Weight Loss With Myostatin-Activin Inhibition

TALDEFGROBEP



Taldefgrobep directly targets fat and muscle while avoiding intolerable adverse effects

Novel myostatin-activin MOA for healthy weight loss
Inhibits ActRII signaling in muscle and adipose tissue

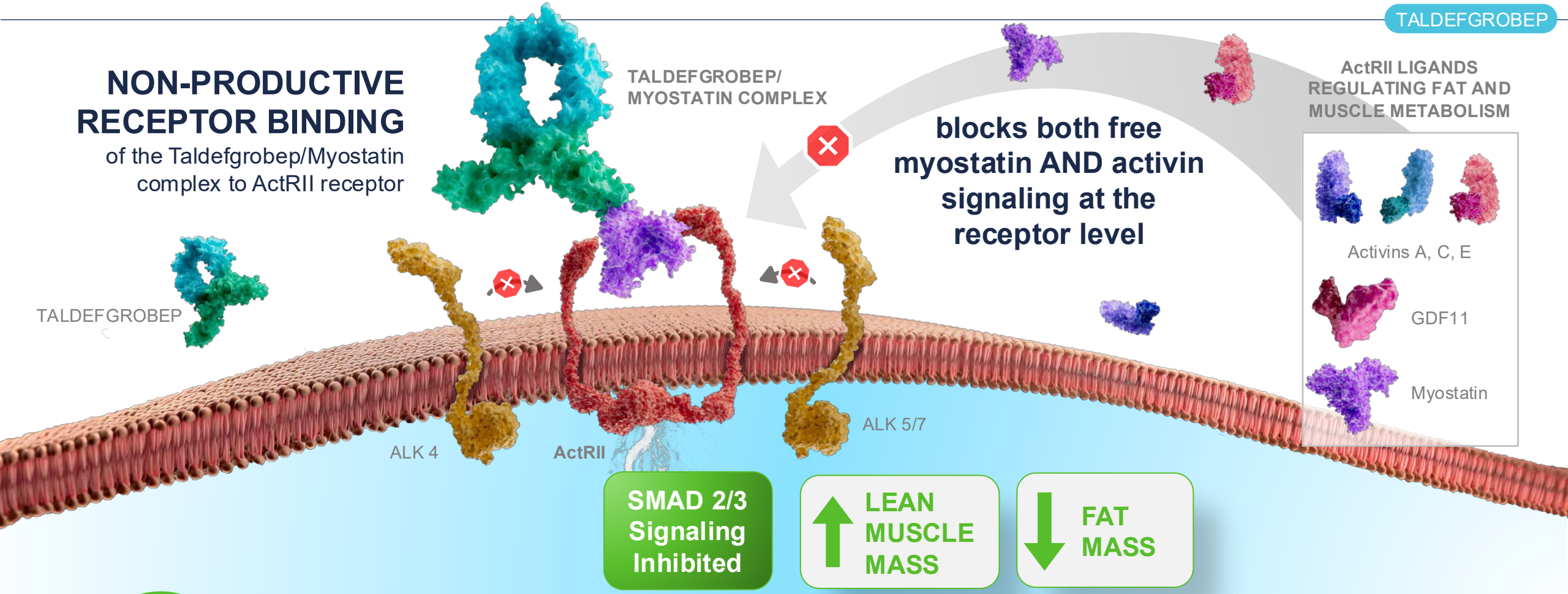
Favorable safety profile established in >700 treated to date
Low rates of muscle- and GI-related AEs

Convenient dosing
Administration by subcutaneous autoinjector

BREAKING NEWS

Phase 2 proof-of-concept study topline expected 2H 2026

Taldefgrobep Is a Novel Competitive Inhibitor of ActRII Signaling



ActRII, activin type II receptors. **ALK 4/5/7,** activin (type I) receptor-like kinases. **GDF11,** growth and differentiation factor 11

KEY POINT

Taldefgrobep binds ligands and competitively inhibits receptor activation in tissues where myostatin and multiple activins (e.g., A, C, E) are active

Unmet Needs Remain in Obesity Management Despite New Therapies

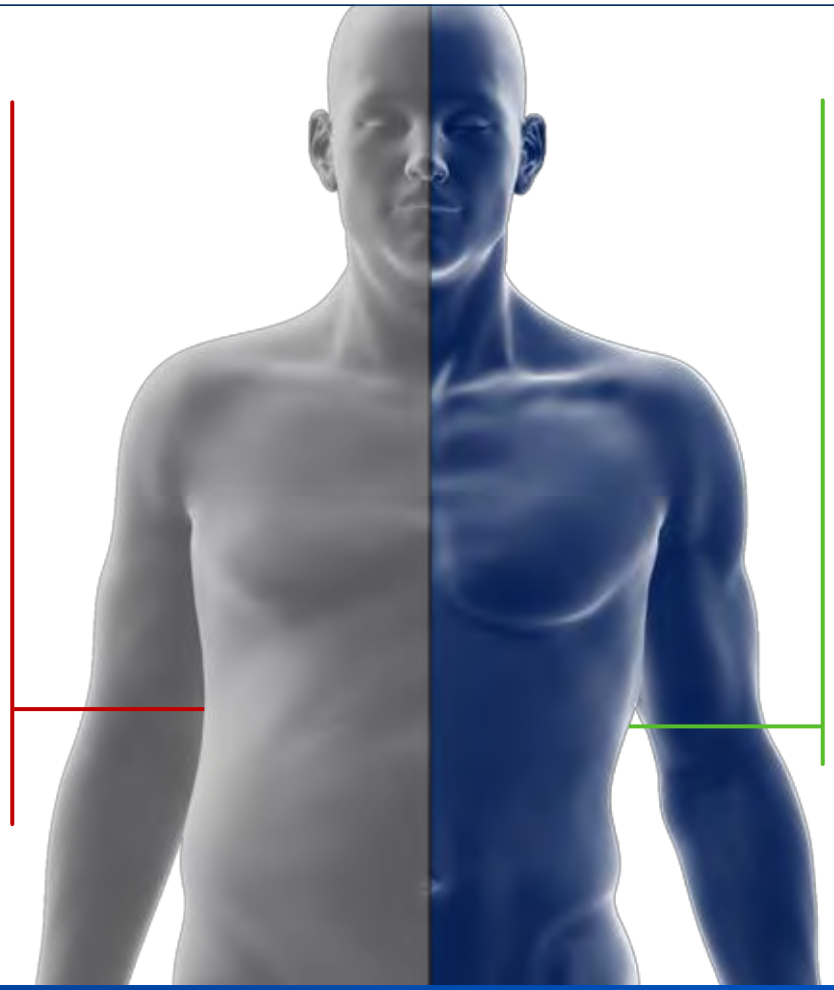
TALDEFGROBEP

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

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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%

- 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

VAT, Visceral Adipose Tissue.

KEY POINT

Taldefgrobep targets bimagrumab-like efficacy

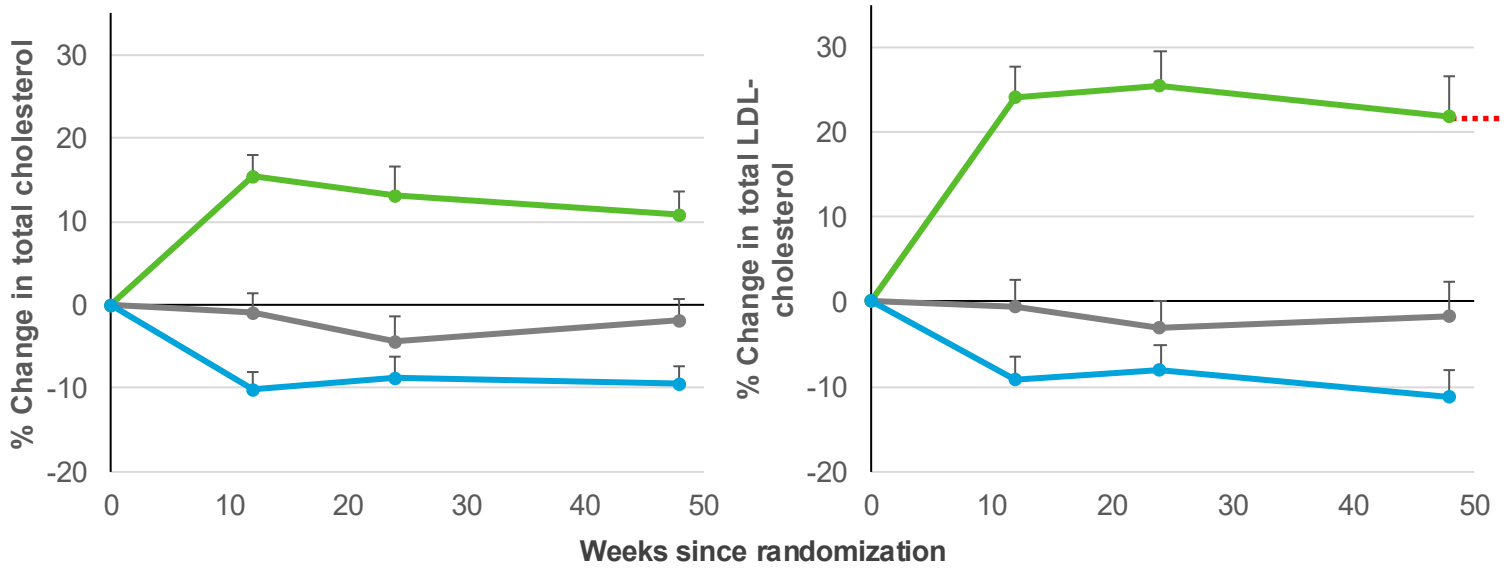
Bimagrumab Provides Reason To "**Believe**" With Compelling Efficacy but Limited by Safety/Tolerability

TALDEFGROBEP

	Placebo	Bimagrumab (30 mg IV)	Semaglutide (2.4 mg SC)
Muscle Spasms	5.5%	**73.7%**	8.9%
Diarrhea	5.5%	**49.1%**	35.7%
Acne	3.6%	**43.9%**	8.9%

Poor tolerability due to irreversible ActRIIB binding

Percent Change in Cholesterol From Baseline at 48 Weeks



Elevation in lipids secondary to high peak exposures following IV administration

Bimagrumab 30 mg/kg
Semaglutide 2.4 mg
Placebo

Heymsfield. Nature Medicine. 2026.

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

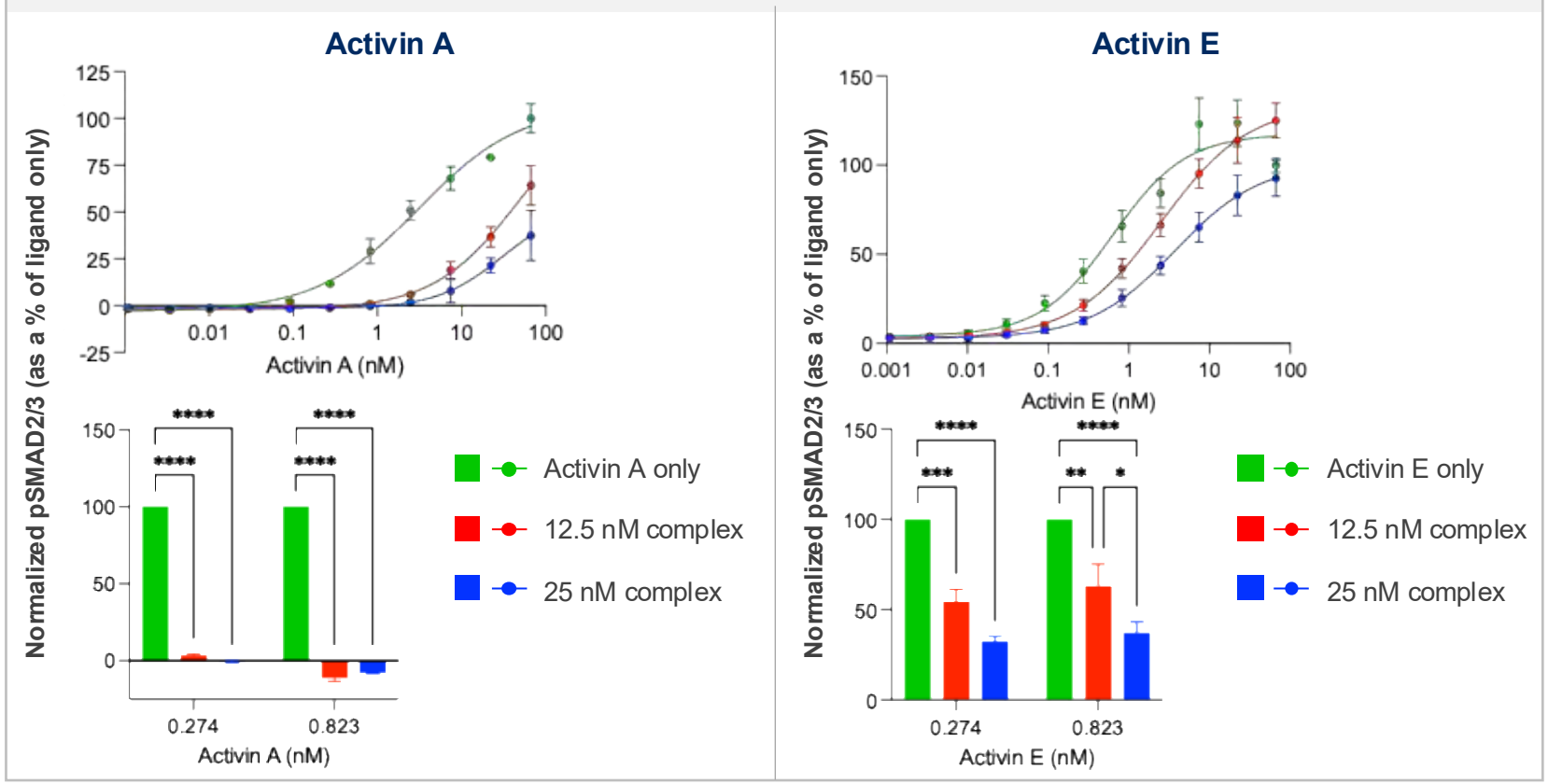
1. Study CN001001 conducted in healthy adults receiving taldefgrobep (15-180 mg QW x 1 month). **2.** Garito. Diabetes Obes Metab. 2018. **3.** Heymsfield. JAMA Network Open. 2021.

KEY POINT

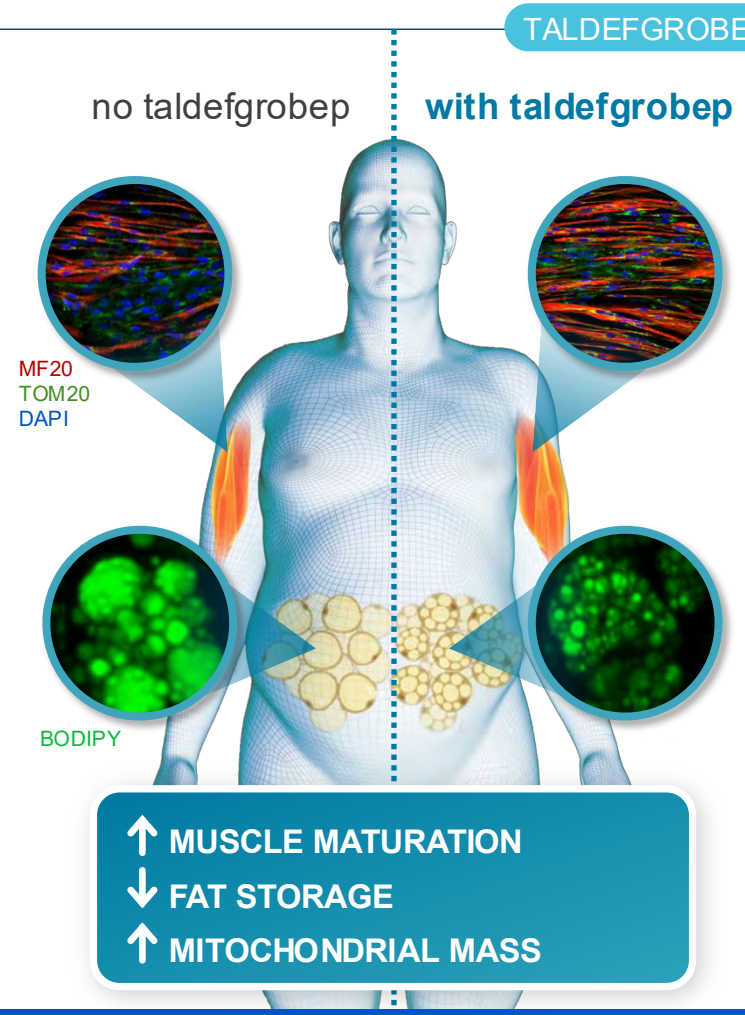
Favorable safety profile established in >700 participants across diverse clinical populations

Taldefgrobep/Myostatin Complex Competes With Activin Signaling To Improve Muscle Differentiation and Fat Storage

T/M Complex Inhibits Activin-Induced Signaling Through ActRII



*p<0.05, **p<0.01, ***p<0.001, ****p<0.0001



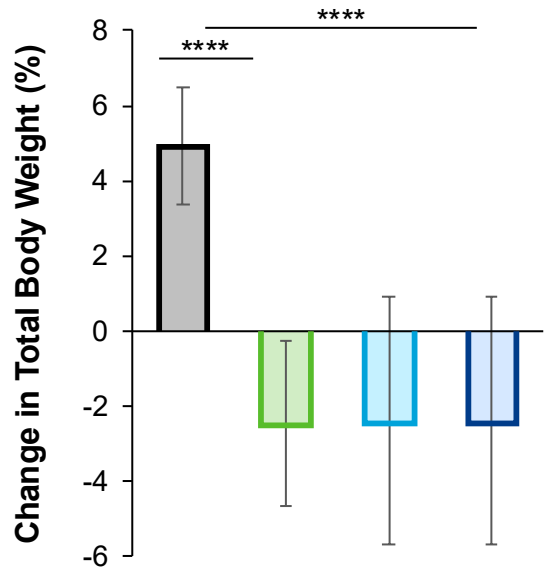
KEY POINT

The T/M complex, at clinically relevant exposures, inhibits activin A- and activin E-induced ActRII signaling

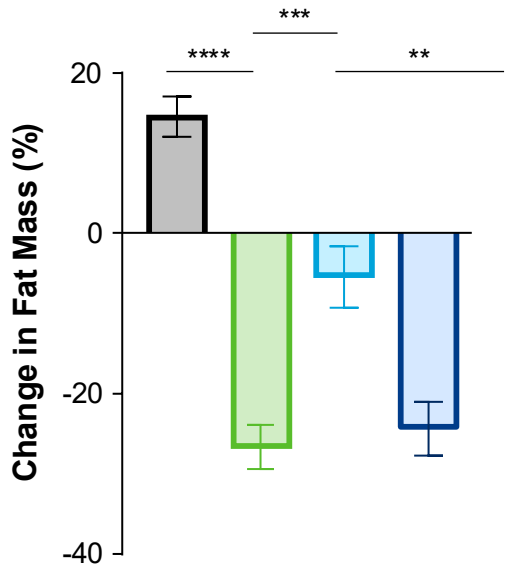
In an Obese Mouse Model, Taldefgrobep Demonstrated Significant Reductions in Fat Mass While Increasing Lean Mass

TALDEFGROBEP

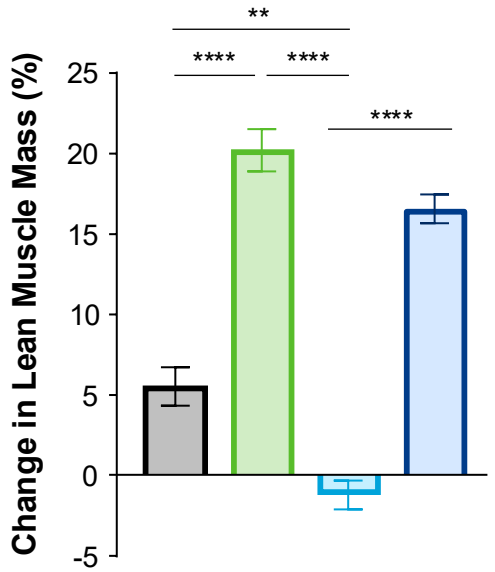
Change in Total Body Weight, From Baseline at 8 Weeks



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

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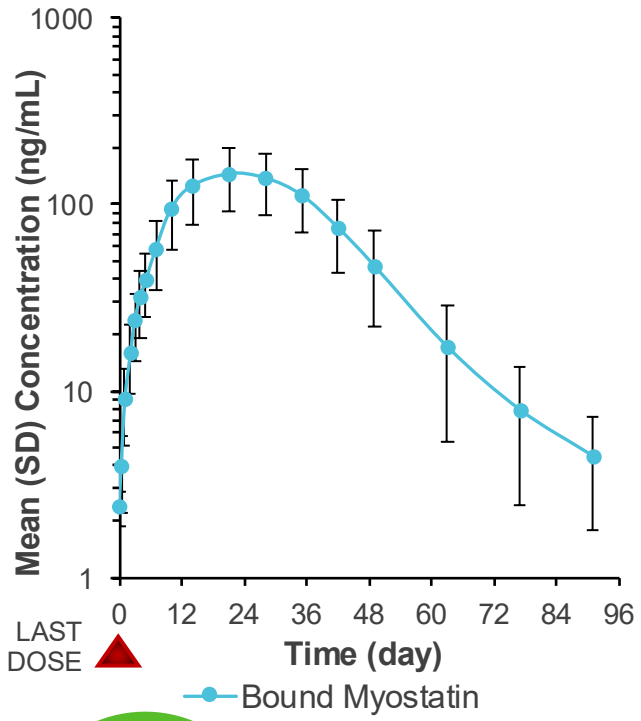
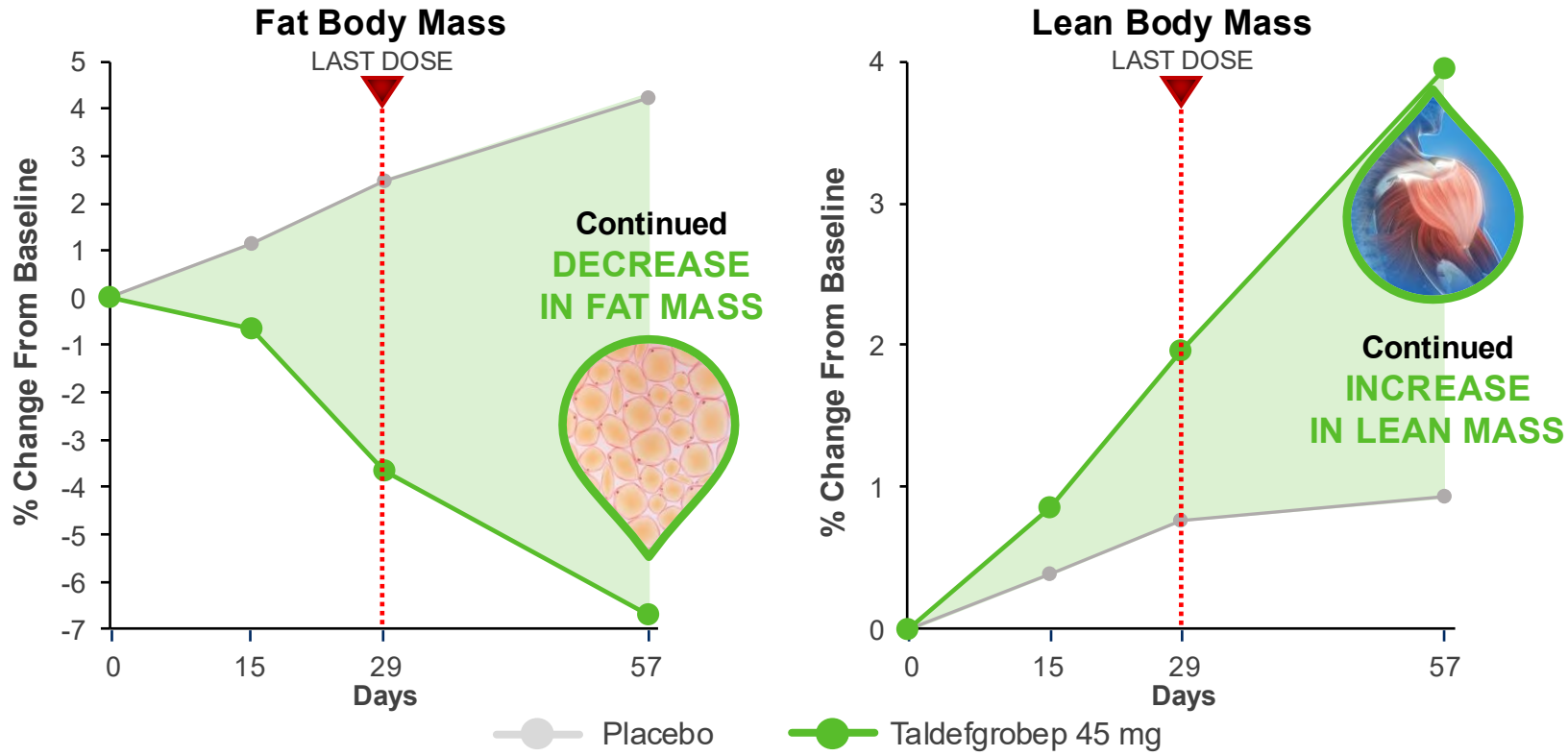
Peak Plasma Concentrations of **Taldefgrobep-Myostatin Complex** Occurs 20-30 Days Post Dose



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 Has an Established, Favorable Safety Profile

Safety database includes **more than 700** treated trial participants

Assessed across a **wide dose range** (4 mg to 180 mg SC QW) and **broad demography**

Data from repeat dosing up to **192 consecutive weeks**

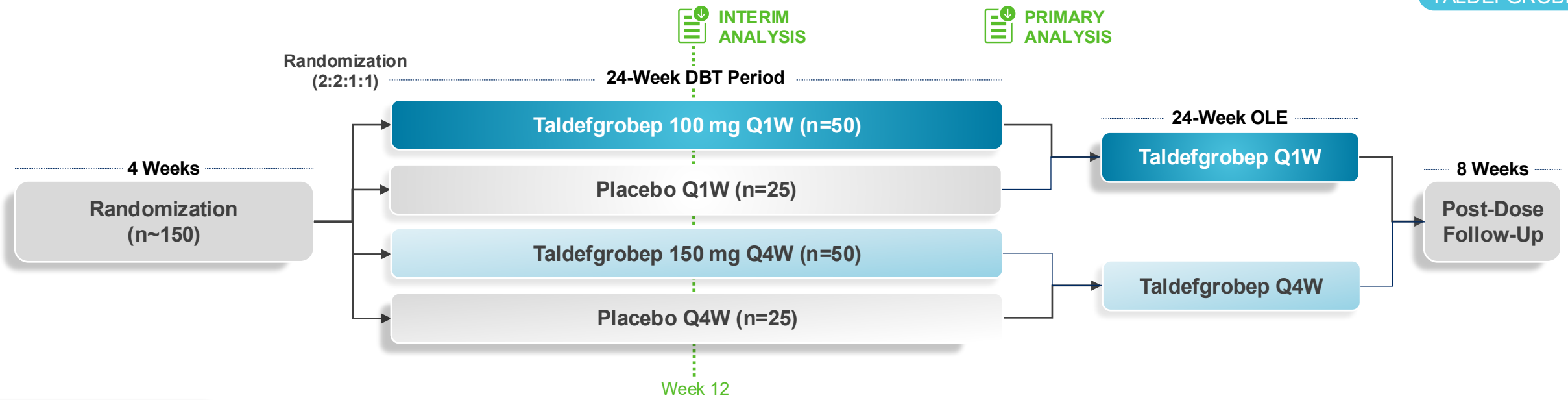
- **LOW RATES** of SAEs and AEs leading to discontinuation
- **LOW RATES** of GI- and muscle-related AEs commonly reported with other myostatin-activin pathway inhibitors
- **No identified serious signature clinical safety events**

KEY POINT

Safety profile well-suited for indication in chronic weight management

Ongoing Phase 2 Monotherapy Dose-Ranging (Q1W + Q4W) Study

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

Blinded Interim Analysis (when 30% complete Week 12)**:** Taldefgrobep PK/PD (free myostatin and T/M complex concentrations) and plasma lipids by treatment assignment

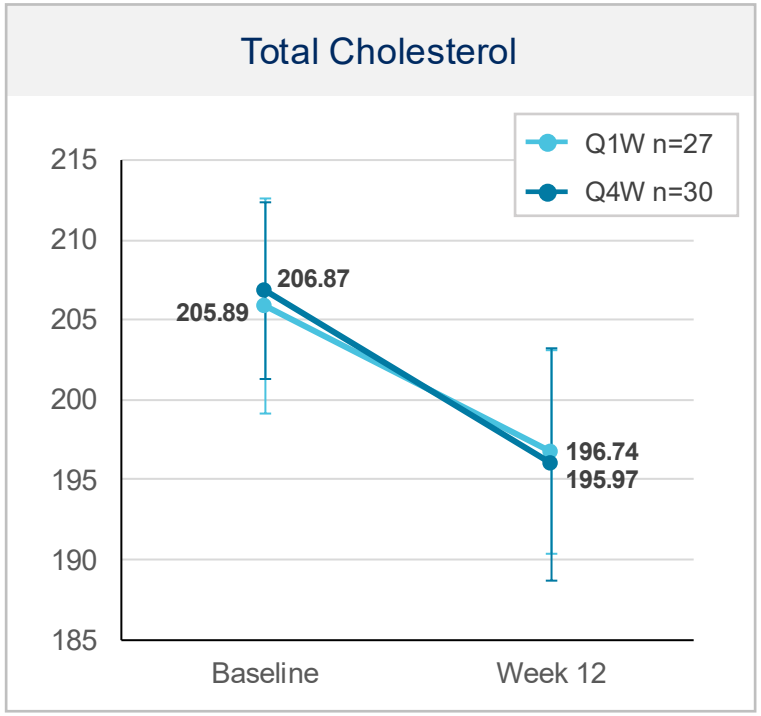
Endpoints: % change in total body weight, fat mass and lean mass at Week 24. Whole-body MRI in subset

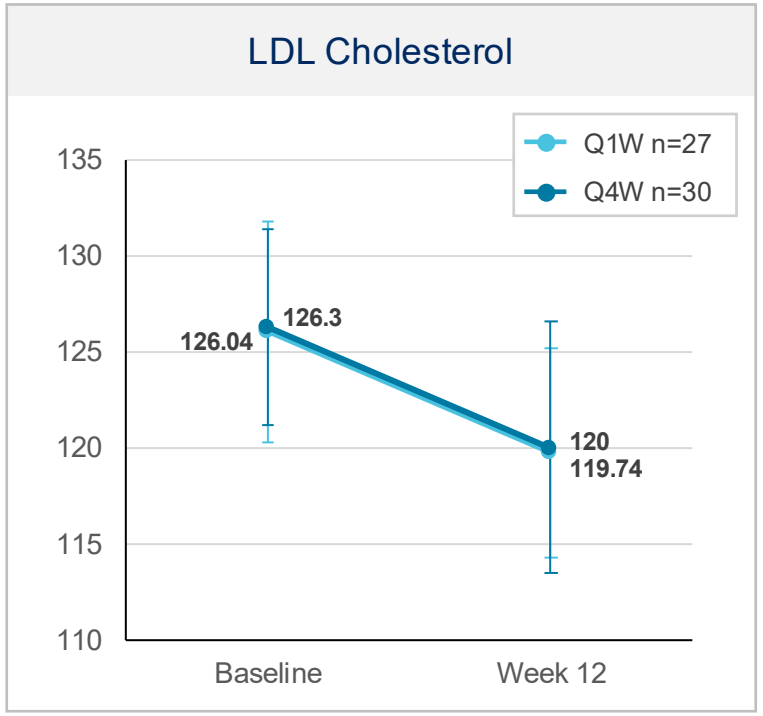
KEY POINTS

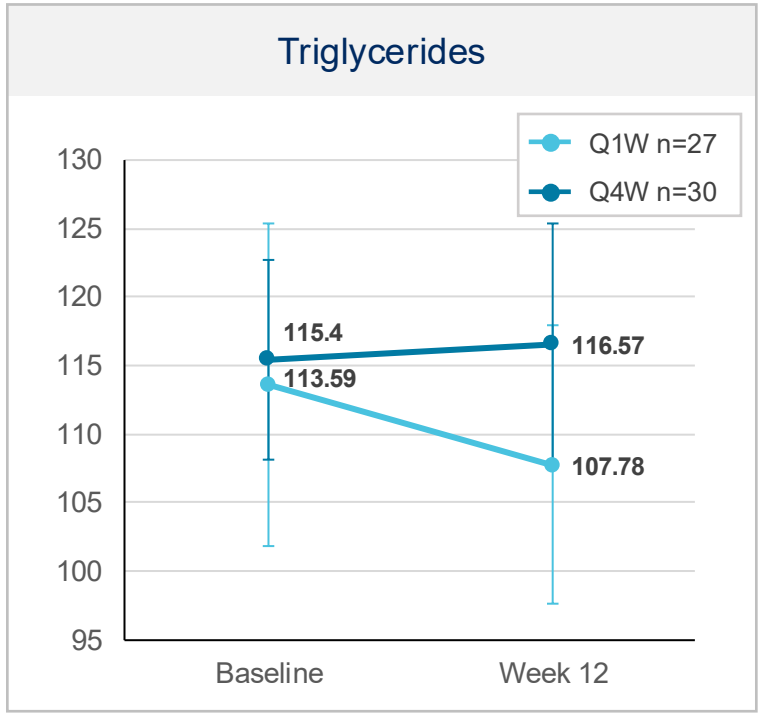
- PK/PD interim analysis completed
- Topline primary analysis expected 2H 2026

Blinded Interim Data by Q1W vs. Q4W, Taldefgrobep + Placebo
No Adverse Effects on LIPIDS at Week 12

TALDEFGROBEP







- No treatment-emergent G2-4 elevations in total cholesterol or triglyceride values
- 1 participant with emergent G3 LDL elevation; had G2 elevation at baseline

Error bars represent standard error

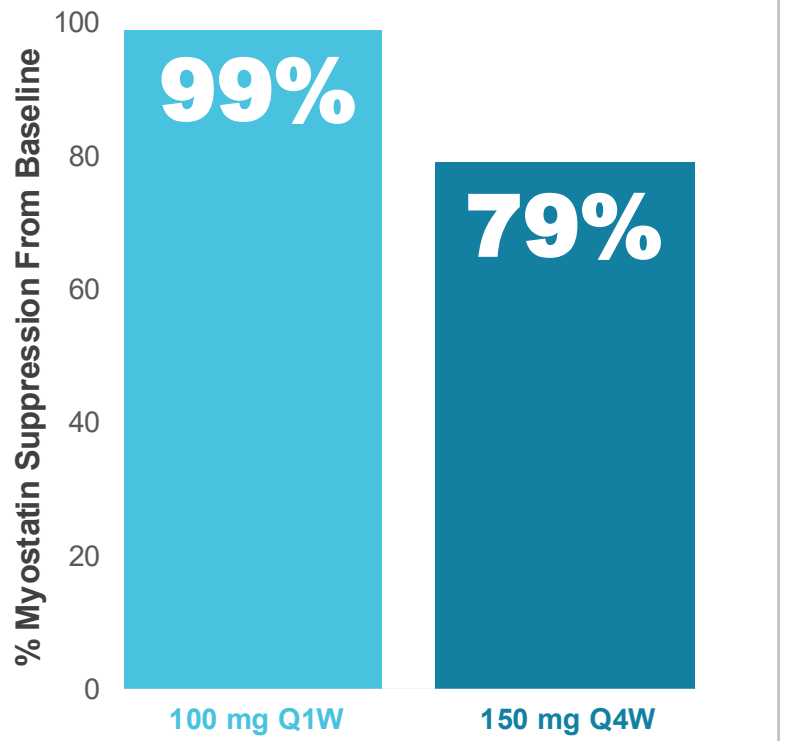
KEY POINT

No identified adverse trends in lipid parameters demonstrating differentiated safety from other myostatin-activin pathway inhibitors

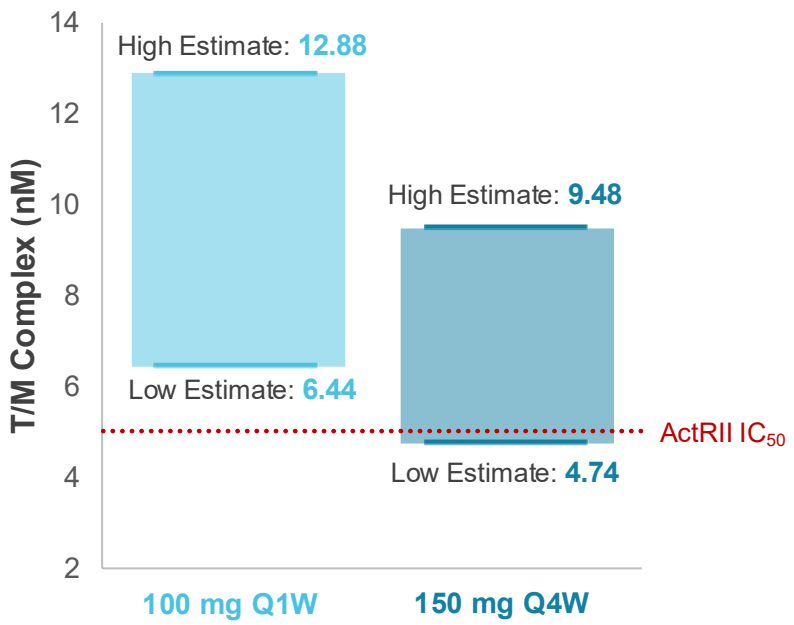
Interim Phase 2 PK/PD Data Supports Weekly and Monthly Dosing in Obesity

TALDEFGROBEP

% Myostatin Suppression at Week 12 in Overweight/Obese Adults



Taldefgrobep-Myostatin Complex at Week 12 in Overweight/Obese Adults



AT WEEK 12:

- Observed mean taldefgrobep concentrations are consistent with model-predicted
- Robust suppression of free myostatin throughout Q1W and Q4W dosing intervals
- T/M complex concentrations comparable between dosing regimens and at/above target ActRII IC_{50}

KEY POINT

Interim data suggest monthly dosing can achieve robust myostatin suppression and formation of T/M complex levels at/above targeted ActRII IC_{50} levels

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

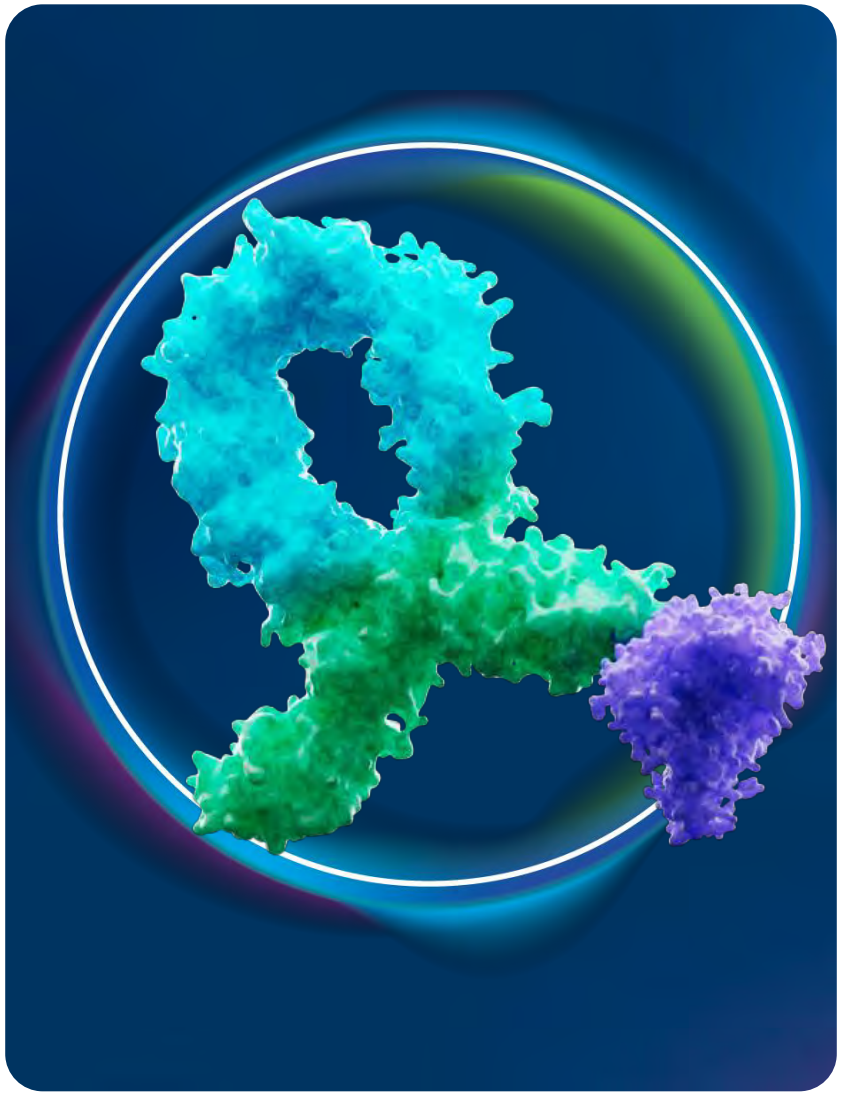
TALDEFGROBEP

Total body weight loss meeting current regulatory standards

Benefit as monotherapy and in combination with GLP-1 therapies

Convenient subcutaneous autoinjector with potential for monthly dosing

Favorable safety and tolerability



Fat mass loss comparable to GLP-1 therapies

Visceral adipose tissue loss favorable to GLP-1 therapies

Increase in lean muscle mass highly differentiated from GLP-1 therapies

Increase in bone density favorable to GLP-1 therapy

Taldefgrobep Can Benefit a Broad Spectrum of People Living With Obesity

TALDEFGROBEP

6.6M–10M
Older patients at risk of sarcopenia

5–8M
People intolerant or refractory to GLP-1

5–7M
People living with BMI ≥40

5–10M
People unable to tolerate high dose GLP-1

MONOTHERAPY

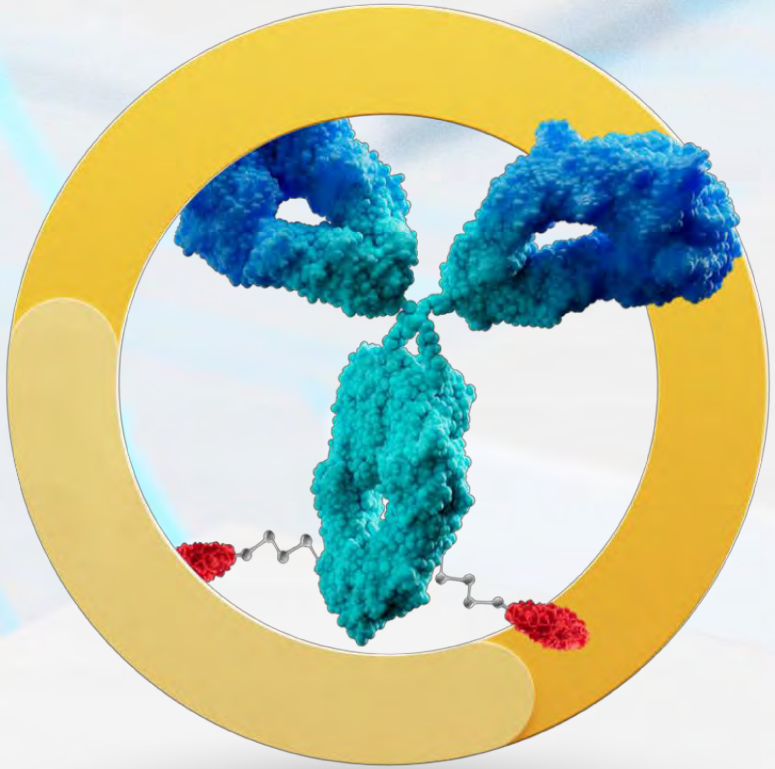
GLP-1 ADJUNCT

Source: BHVN Market Research and Analytics Data on file

KEY POINT

Meaningful reductions in fat mass, preservation of lean muscle mass, once-monthly dosing and high tolerability drive market potential

biohaven®

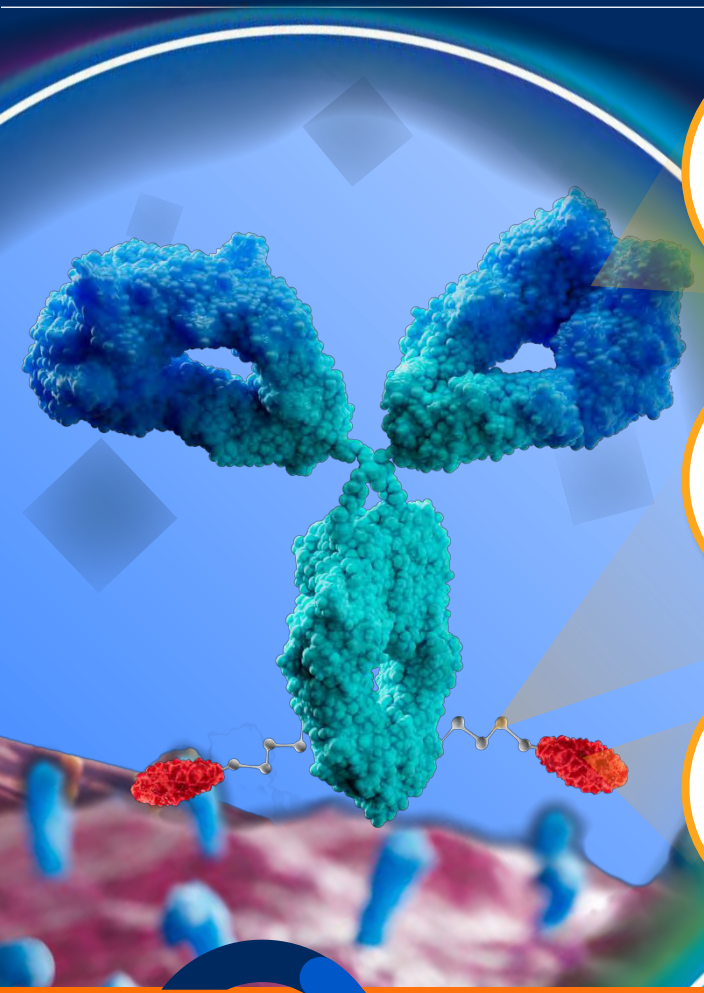


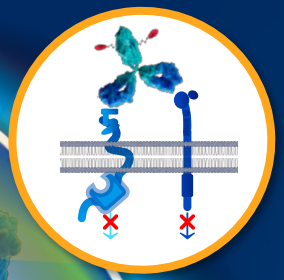
ONCOLOGY

Modular Next-Generation ADC Technologies: Optimizing for Clinical Performance

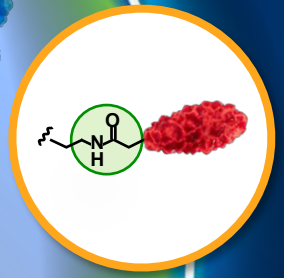
Innovative Biohaven Technologies Enabling Differentiated Next-Generation ADCs

ONCOLOGY

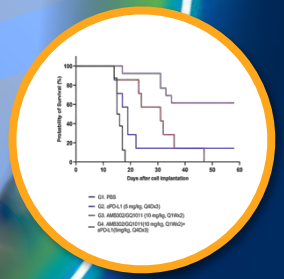




OPTIMIZED MONO AND BISPECIFIC MABS



NEXT-GEN MODULAR ADC CONJUGATION TECHNOLOGIES



PROPRIETARY TOPOIX AND MULTI-CLASS PAYLOADS

Clinical Proof-of-Principle Establishing the Power and Flexibility of the Platform to Optimize Across Broad Range of ADC Designs and Combination Strategies Including CPI

First clinical demonstration (BHV-1510 Trop2)
Highly stable, differentiated PK and safety, compelling activity with CPI combination

Pioneering FGFR3 ADC (BHV-1530)
Novel mAb, potential to address both FGFR3 alterations and overexpression

Platform tech enables multi-class payload loading
Clinically differentiated TopoIx payload, foundational for CPI and other potentially synergistic MOA combinations

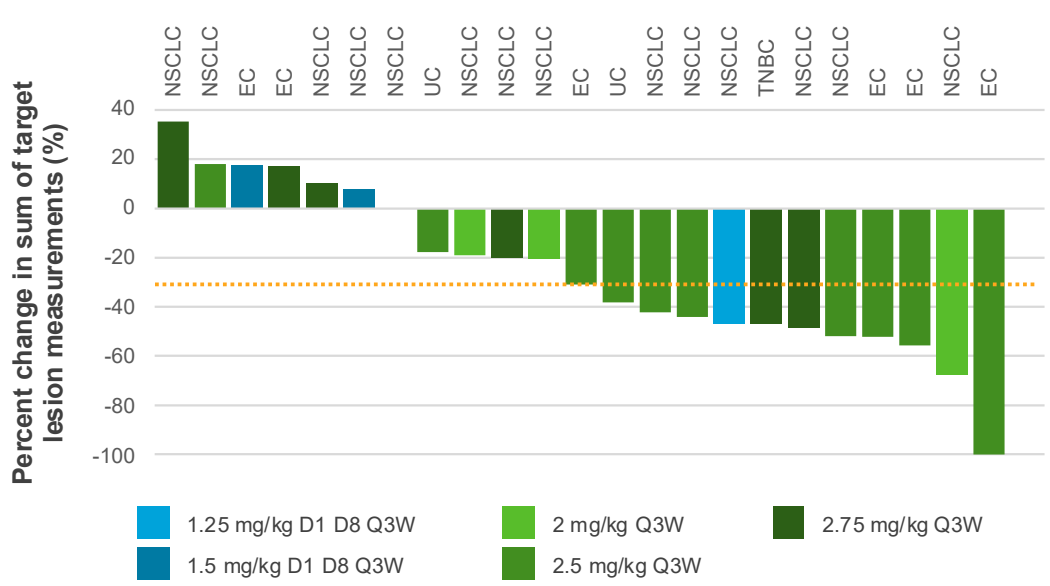
BREAKING NEWS

- **BHV-1510:** Robust enrollment in endometrial + cemiplimab combination expansion cohort
- **BHV-1530:** First activity observed, including pretreated FGFR3-altered urothelial cancer patient
- **Discovery:** Preclinical data supporting next-wave bispecific mAb, multi-payload formats

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

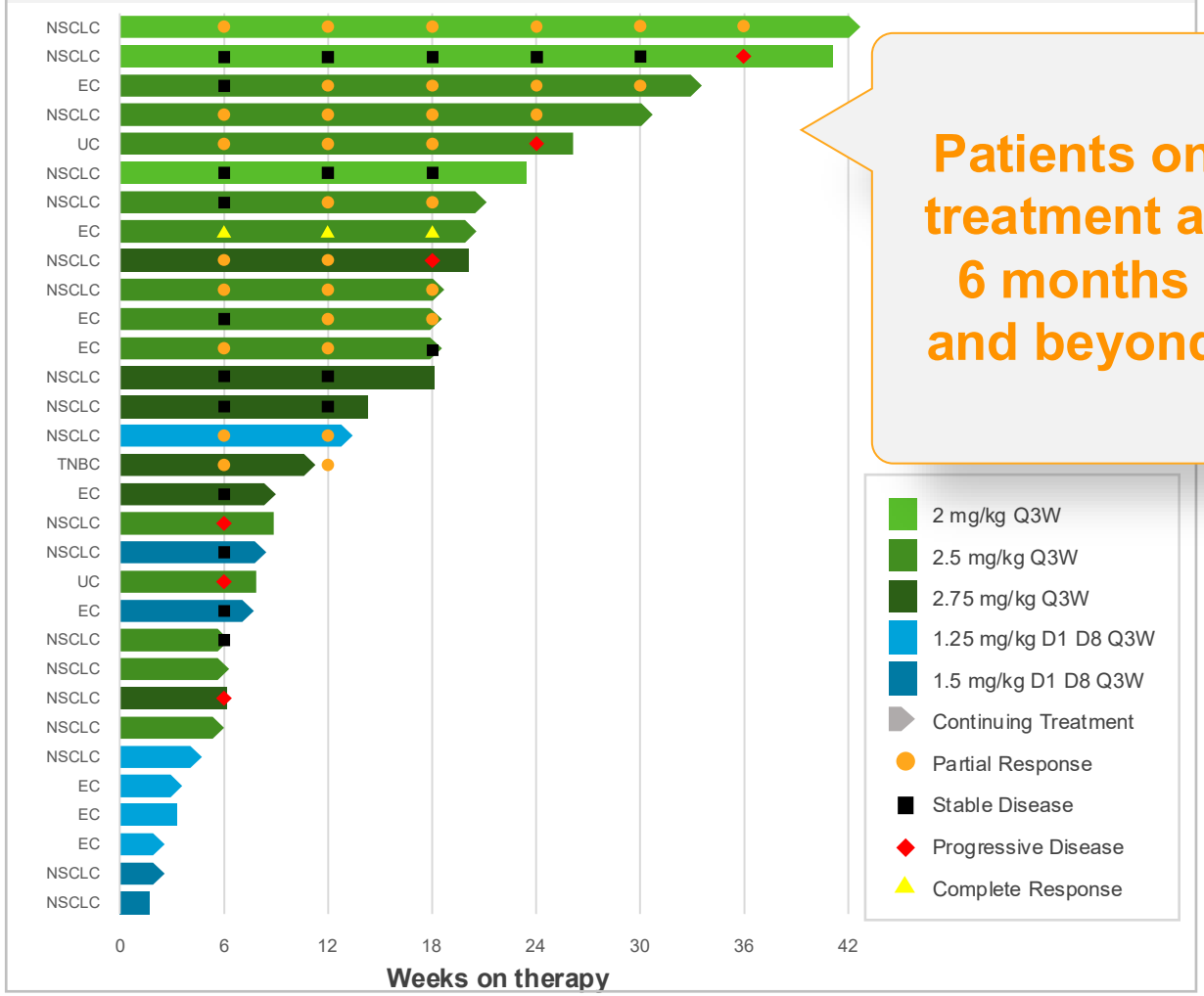
ONCOLOGY

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



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

BHV-1510 + Cemiplimab: Treatment Duration



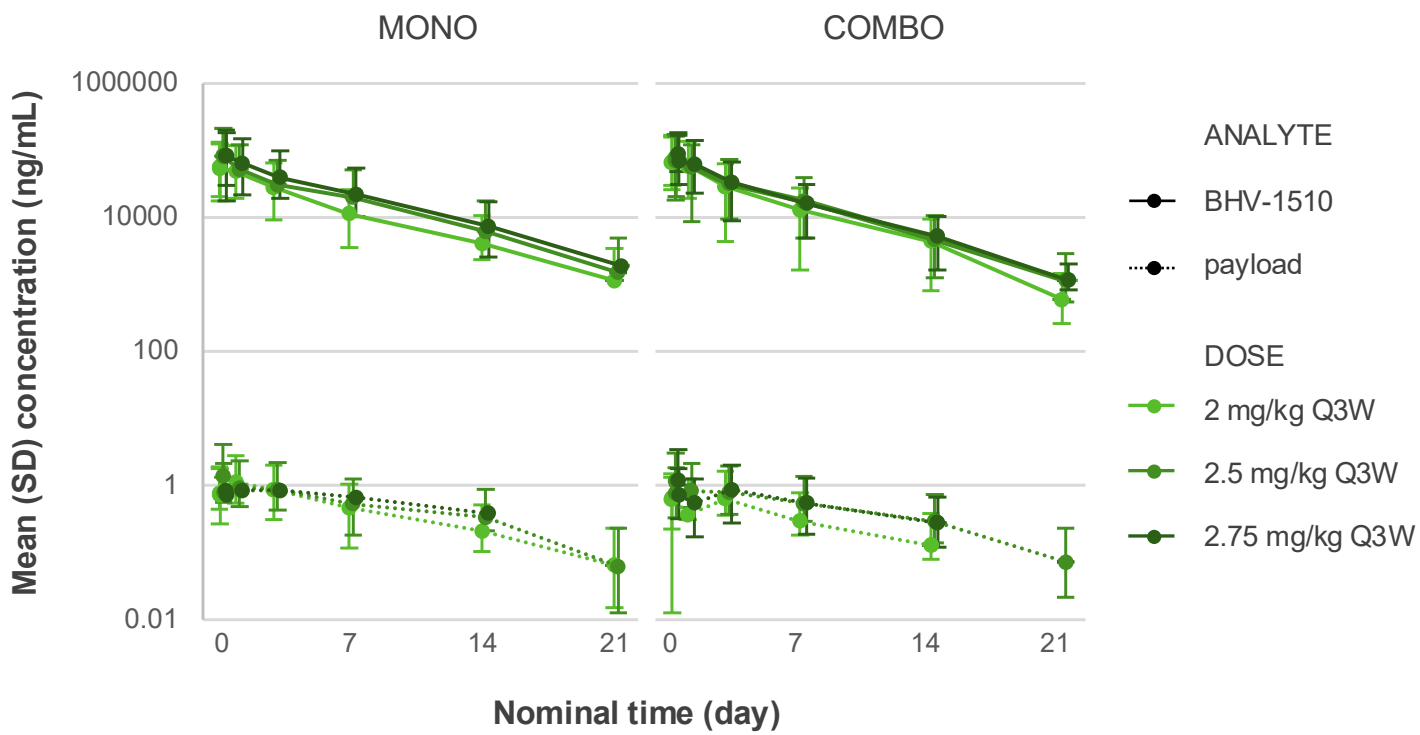
Patients on treatment at 6 months and beyond

*Cemiplimab (anti-PD1) provided through a supply agreement with Regeneron; dose of cemiplimab 350 mg Q3W

Source: Micaily. ESMO Immuno-Oncology Congress 2025. Poster 252P.

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

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



Source: Micaily. ESMO Immuno-Oncology Congress 2025. Poster 252P.

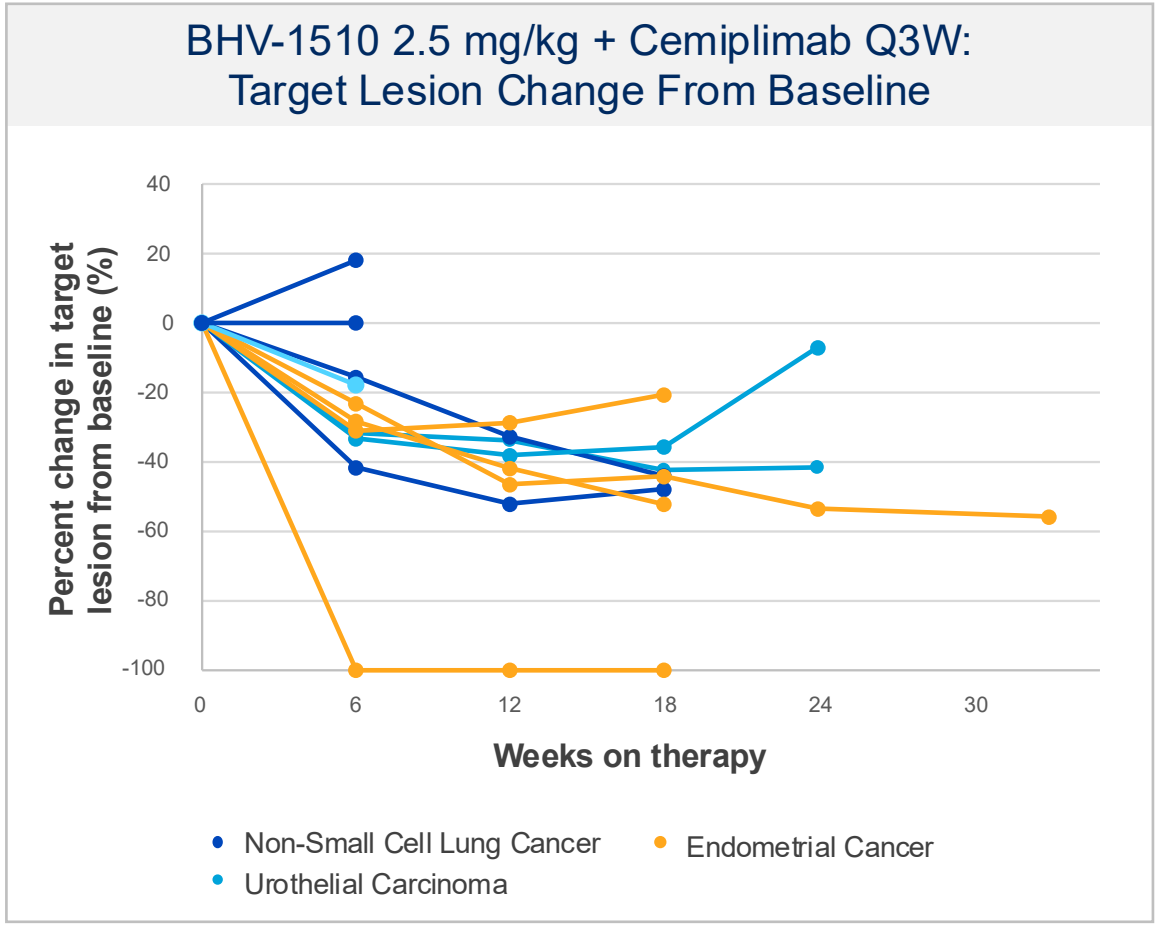
KEY POINT

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

Encouraging Early Clinical Activity and High Response Rates of BHV-1510 + Cemiplimab Enable First Tumor-Specific Expansions

ONCOLOGY

- **Dose escalation complete:** Compelling efficacy in difficult-to-treat patients
  - Responses in heavily pretreated patients, including with brain metastases; majority with prior anti-PD(L)1 exposure
- **Rapid onset of benefit:** Tumor shrinkage / PRs at 1st scan
- **Differentiated safety profile:** Low rates of hematological toxicities and diarrhea; no ILD observed*
- **Favorable PK profile:** Highly Stable ADC



* By independent adjudication

Source: Micaily. ESMO Immuno-Oncology Congress 2025. Poster 252P.

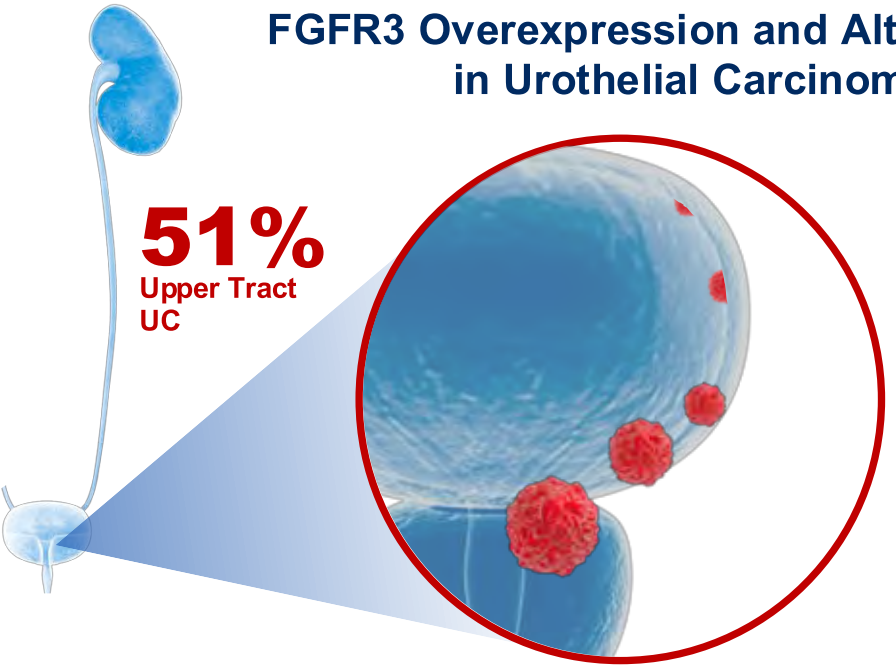
KEY POINTS

- Early data suggests synergy with anti-PD-1 and potential to move into earlier lines
- Endometrial cancer expansion cohort with combination enrolling robustly — data to inform pivotal path

BHV-1530: Favorable Early Clinical Profile — No FGFR-Class Toxicities and Early Signs of Antitumor Activity

ONCOLOGY

FGFR3 Overexpression and Alteration (%) in Urothelial Carcinoma



51%
Upper Tract UC

70–80%
Non-muscle-invasive BC

40–60%
Muscle-invasive BC

~50%
Metastatic UC

FGFR3

~50% of HNSCC

~ 40% of NSCLC

4–8% alterations in other cancers

As of April 2026. Data from ongoing study

Compelling preclinical efficacy across FGFR3-altered and FGFR3-overexpressing tumor models: Demonstrated as monotherapy and in combination with CPI

Clinical progress: First patient dosed April 2025: initial cohorts successfully completed:

- No dose limiting toxicities
- No treatment related SAEs
- TRAEs predominantly mild (Grade 1–2)
- No hyperphosphatemia, nail disorders, central serous retinopathy

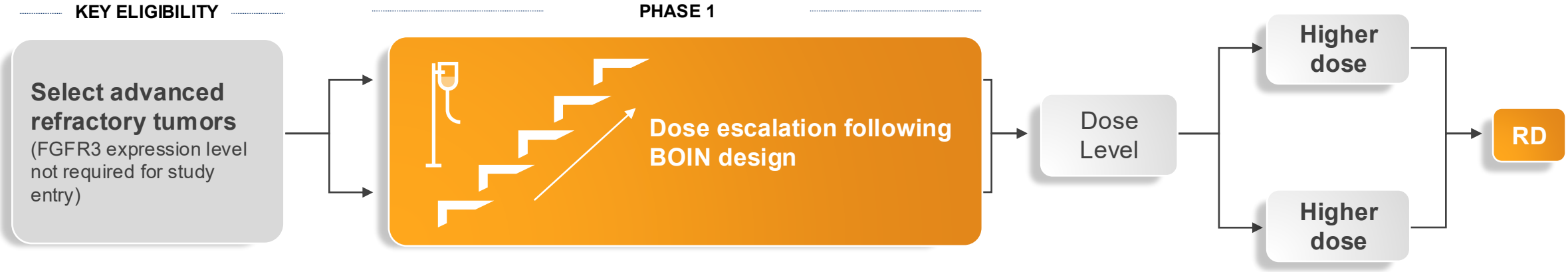
Early signs of activity: Early tumor reduction in patients with FGFR3 alterations and wild-type overexpression, as dosing in the predicted efficacious range

KEY POINTS

- Early tumor reductions in FGFR3-altered and WT overexpressing patients, including urothelial cancer
- Dose escalation continuing with no DLTs to date

BHV-1530: Phase 1 Study in Advanced Tumors

ONCOLOGY



KEY STUDY DETAILS

Study Design: Open label, dose escalation (Ph1)

Population: Advanced UC, HNSCC, NSCLC having failed SOC therapy

Treatment Duration: Until disease progression or toxicity

Endpoints: Safety and tolerability, ORR, PFS, PK and ADA

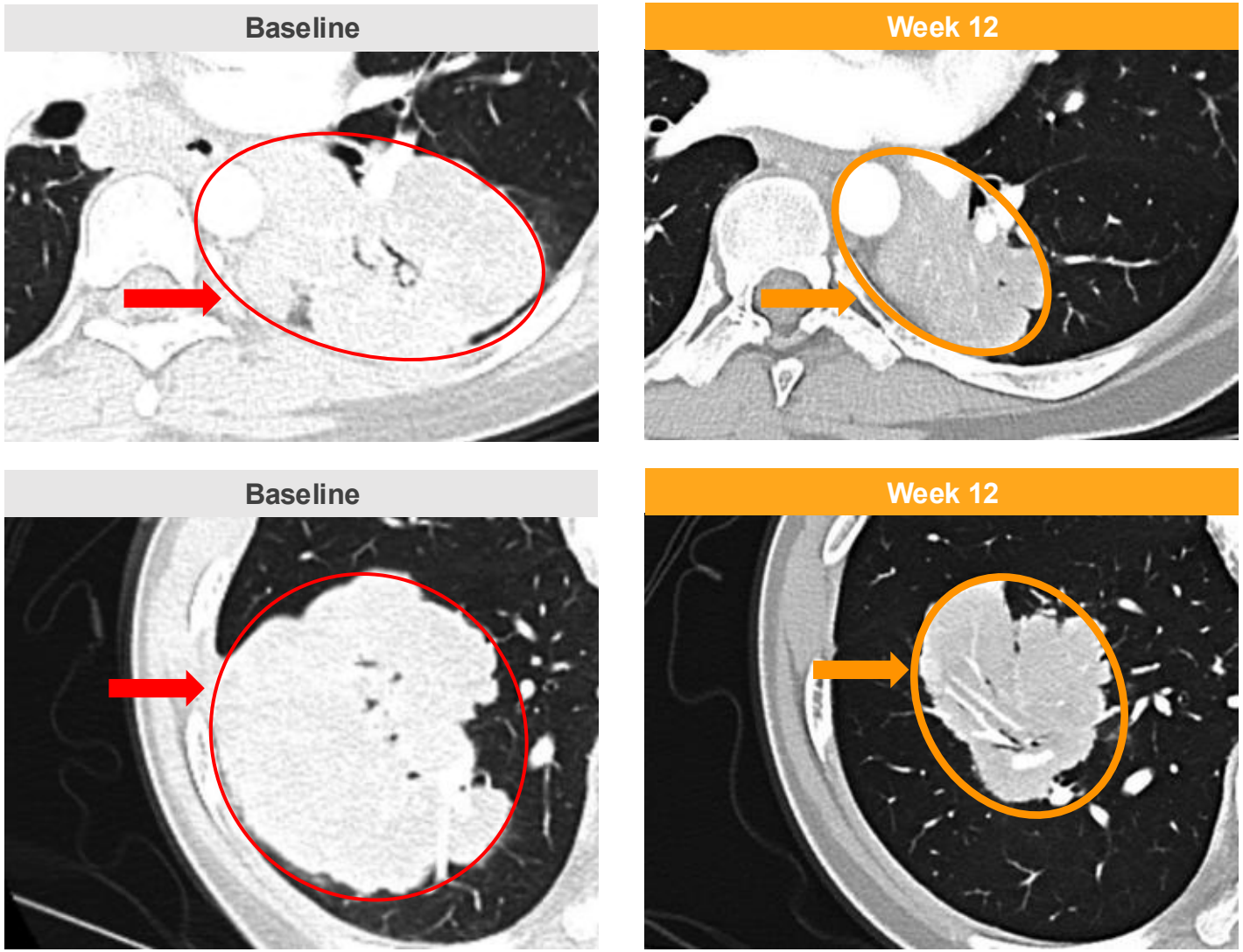
ORR, Overall Response Rate; **PFS,** Progression Free Survival; **ADA,** Antidrug Antibody; **BOIN**, Bayesian Optimal Interval; **RD**, Recommended Dose.

KEY POINT

Early tumor reduction in patients as dosing in the predicted efficacious range

Case Narrative: Metastatic Urothelial Cancer

- 28 y/o female with urothelial cancer with a FGFR3::TACC3 fusion, with metastasis to lungs and thoracic lymph node
- 4 prior lines of therapy including
  - Padcev and Keytruda and 2 FGFR targeting small molecules-Balversa and LOXO-435
- Tolerating treatment with mild nausea and no FGFR related toxicity



BHV-1530: Potential First FGFR3 ADC — Broader Reach, No Class Toxicities, Clear Development Path

BHV-1530 ADC vs. Small-Molecule FGFR3 Inhibitor

	Small Molecule Inhibitor	BHV-1530
Target Population	FGFR3-altered only ~limited addressable population	**Altered AND overexpressing — full FGFR3+ population**
Class Toxicities	Hyperphosphatemia nail disorders, retinopathy	**None observed — no FGFR inhibitor-class toxicities**
Mechanism	Kinase inhibition resistance develops readily	**ADC: targeted delivery of cytotoxic payload**
CPI Combination	Limited — overlapping toxicity concerns	**Engineered for CPI synergy** **TopoIx → ICD →**

3x larger TAM in mUC (15-20% are FGFR3 altered and up to 50% are altered/or overexpressed)

DEVELOPMENT ROADMAP

PH1 DOSE ESCALATION
NOW • ACTIVE
- No DLTs; no FGFR-class toxicities
- Early tumor reduction in altered + WT overexpressing

DOSE OPTIMIZATION+POC
- Identify recommended Ph2 dose
- UC expansion POC
- Confirm activity across FGFR3 subtypes

COMBO EXPANSION
- Expansion cohort in UC with CPI combo
- Pivotal design conversations begin

1L mUC - PIVOTAL PATH
- 1L mUC combination with CPI
- Monotherapy in 2L+-selected pts

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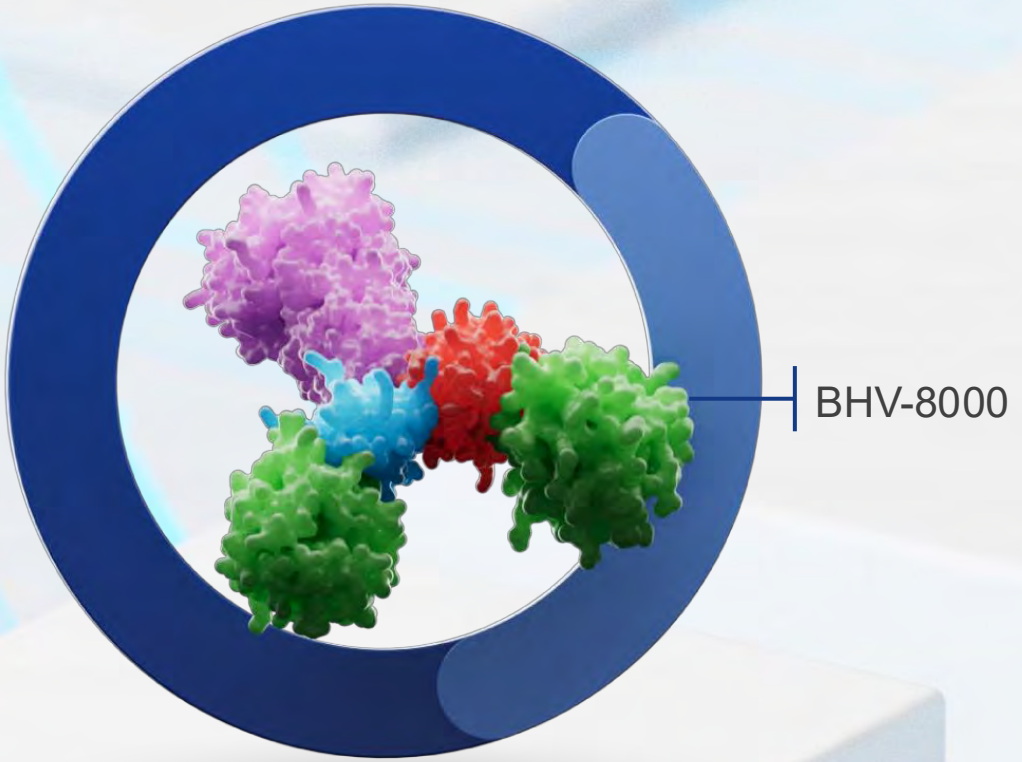
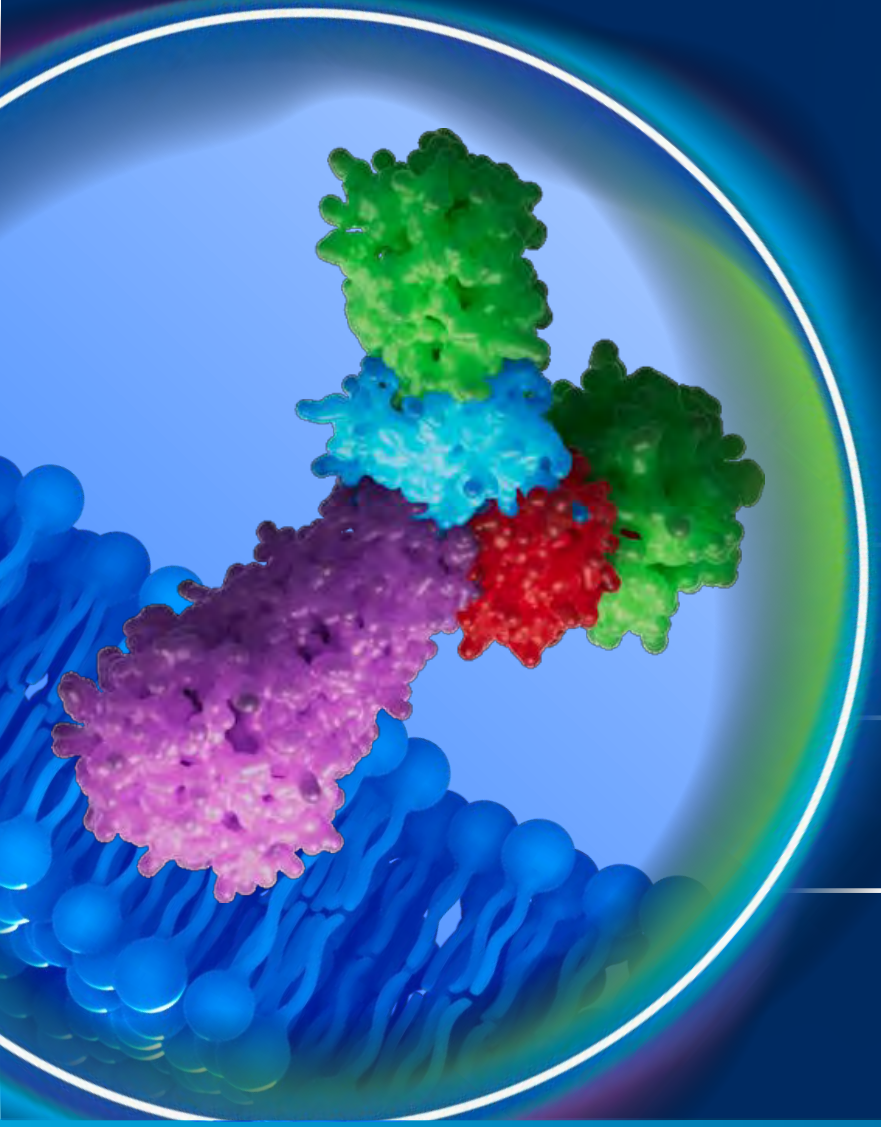
BRAIN-PENETRANT TYK2/JAK1 INHIBITOR: BHV-8000



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

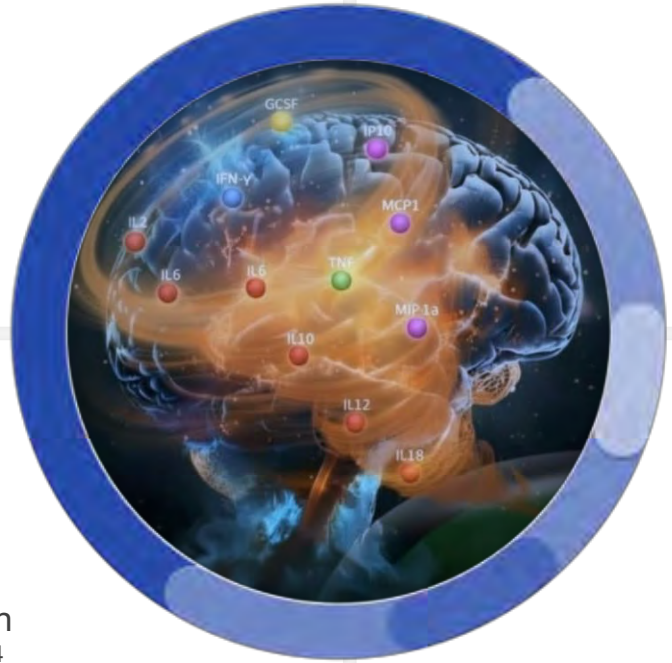
Phase 2/3 trial in early PD ongoing

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]



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]

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]

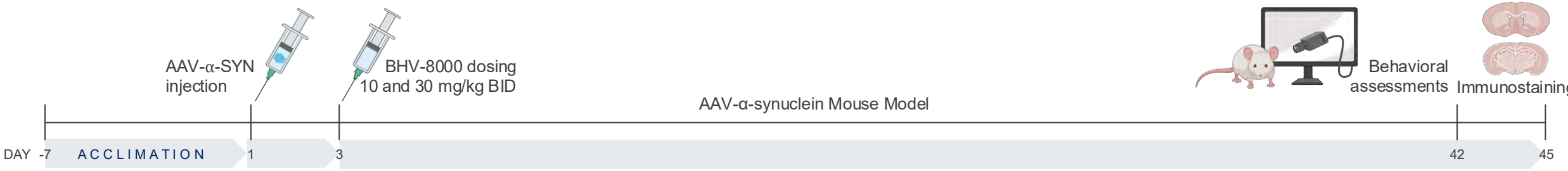
PD patient samples and imaging exhibit characteristic proinflammatory signatures[9,10]

- 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]

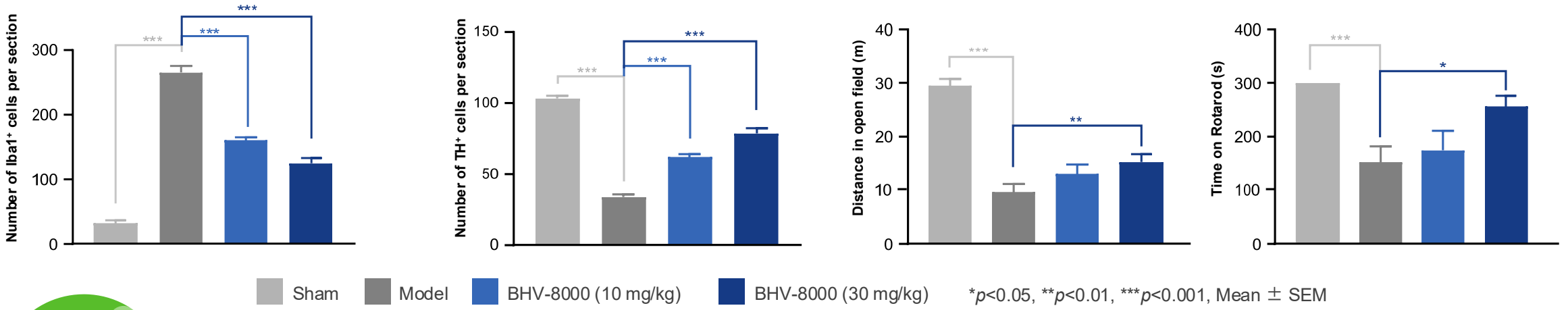
1. Sulzer. Nature. 2017. **2.** Wissemann. Am J Hum Genet. 2013. **3.** Li. Front Immunol. 2023. **4.** Tansey. Nat Rev Immunol. 2022. **5.** Potashman. Parkinsonism Relat Disord. 2025. **6.** Roodveldt. Brain. 2024. **7.** Karikari. Brain Behav Immun. 2022. **8.** Williams. Brain. 2021. **9.** Yacoubian. 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

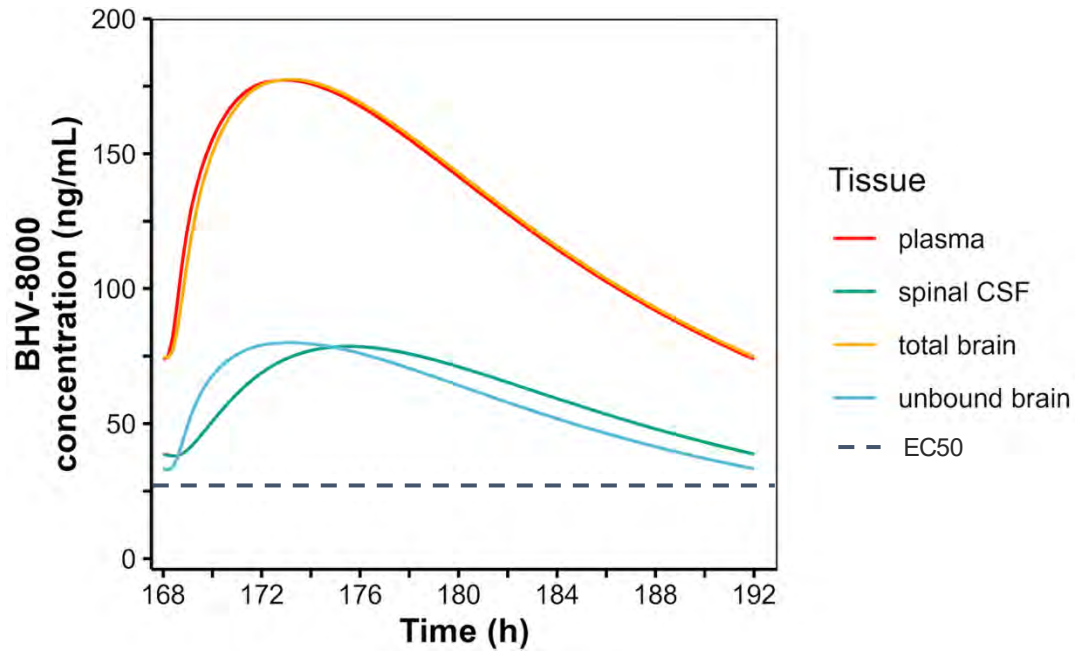
Evidence of target engagement

- 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



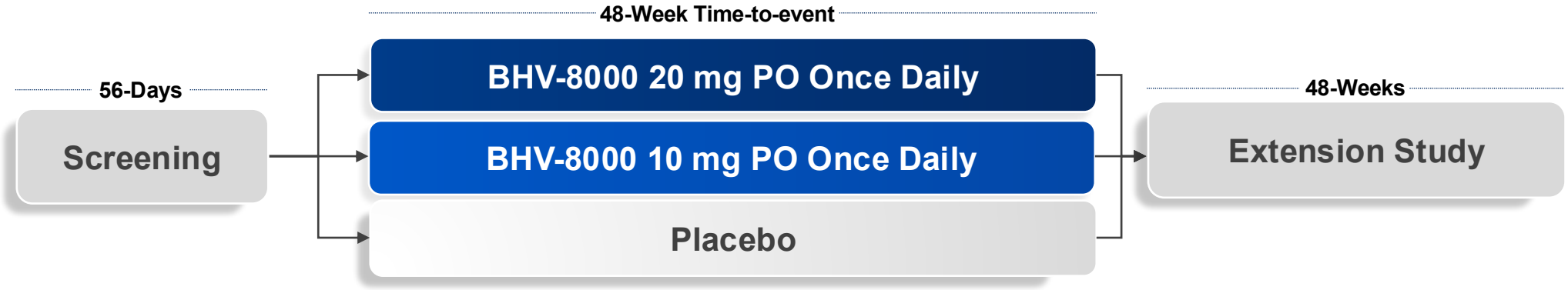
Modelling data

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

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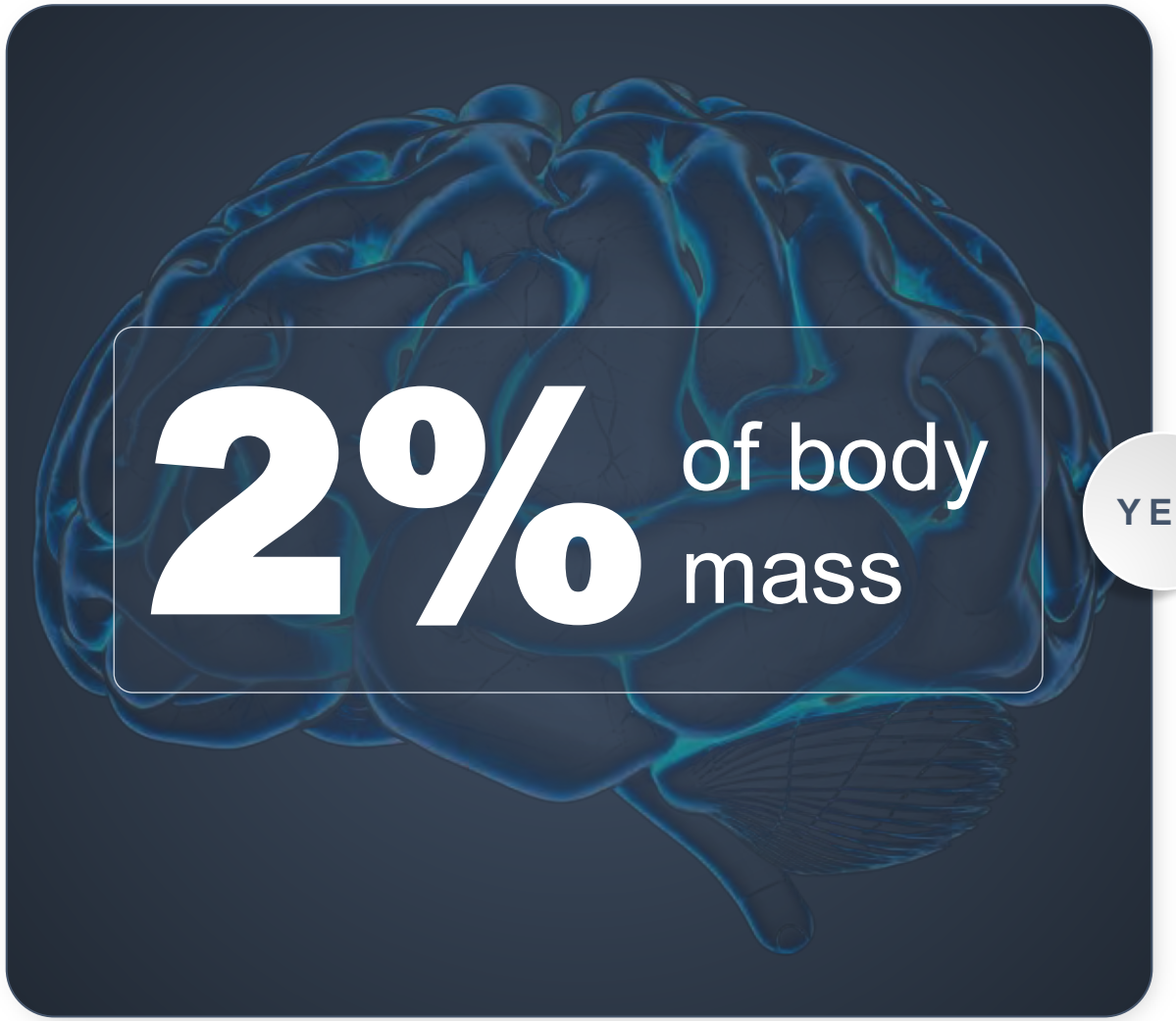
BRAIN-PENETRANT PKM2 ACTIVATOR:
BHV-8100

DAYS MATTER™

Targeting Brain Hypometabolism to Treat Neurodegenerative Diseases

The Brain Has Considerable Energetic Requirements

DISCOVERY



YET

15% of **cardiac output**

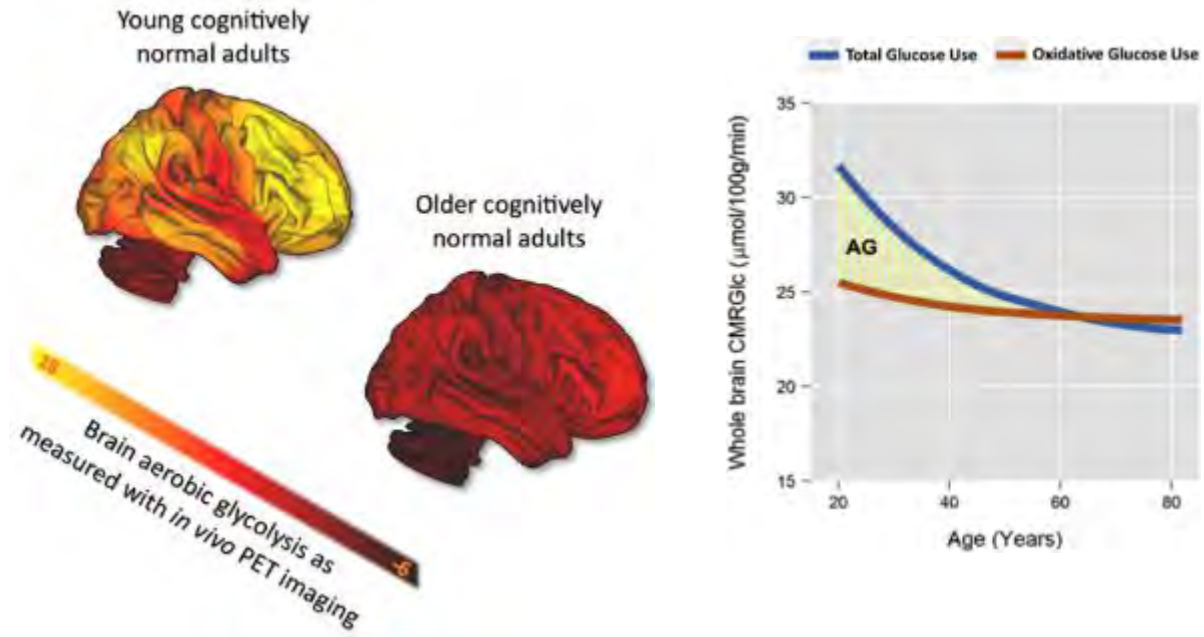
25% of whole-body **glucose utilization**

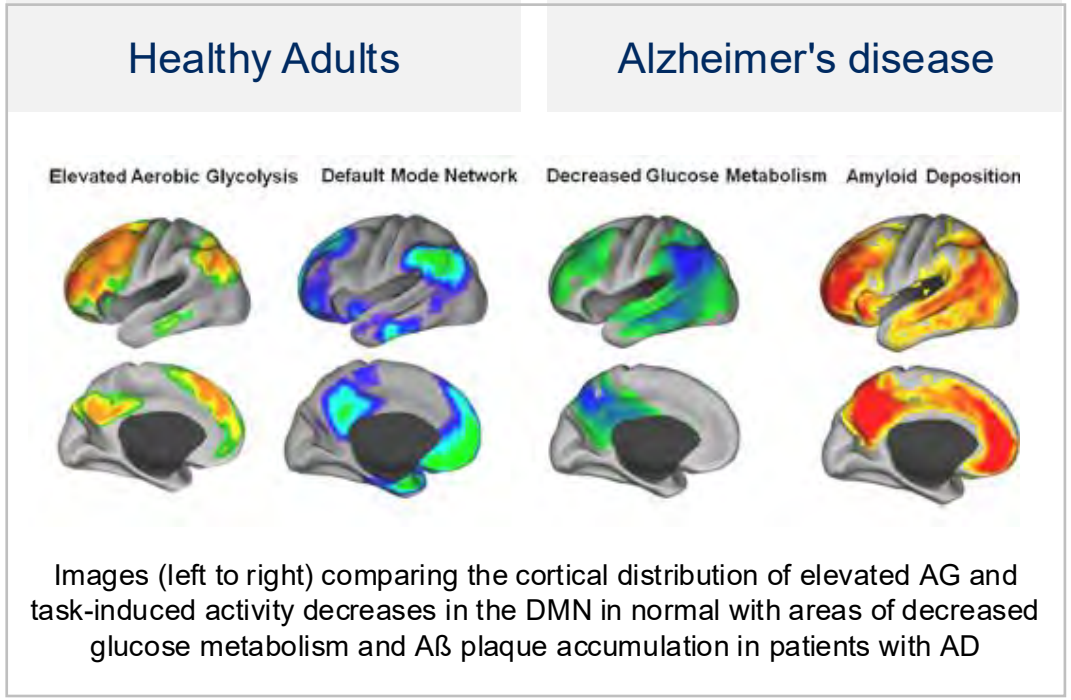
20% of **oxygen consumption**

Aerobic Glycolysis is Reduced in the Aging Brain

DISCOVERY

- **Aerobic glycolysis**, the metabolic process of glucose primarily promoted by astrocytes in the brain, is reduced in the aging brain[1]
- Reduction of aerobic glycolysis is pronounced in frontal and temporal regions[2]



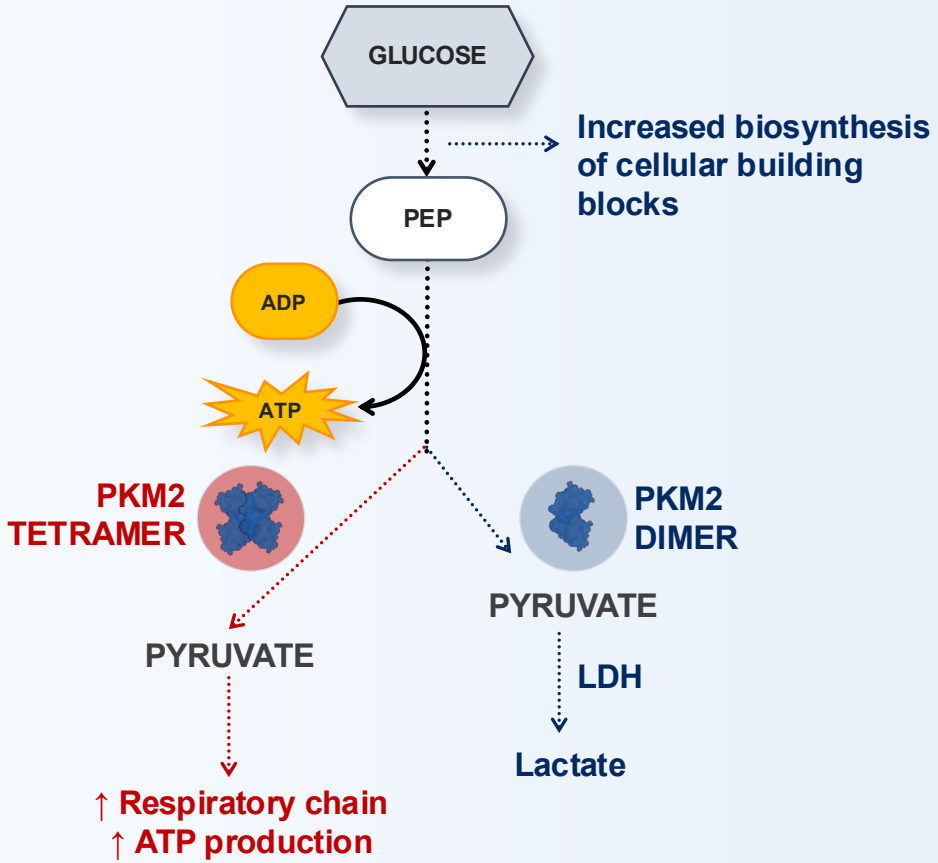


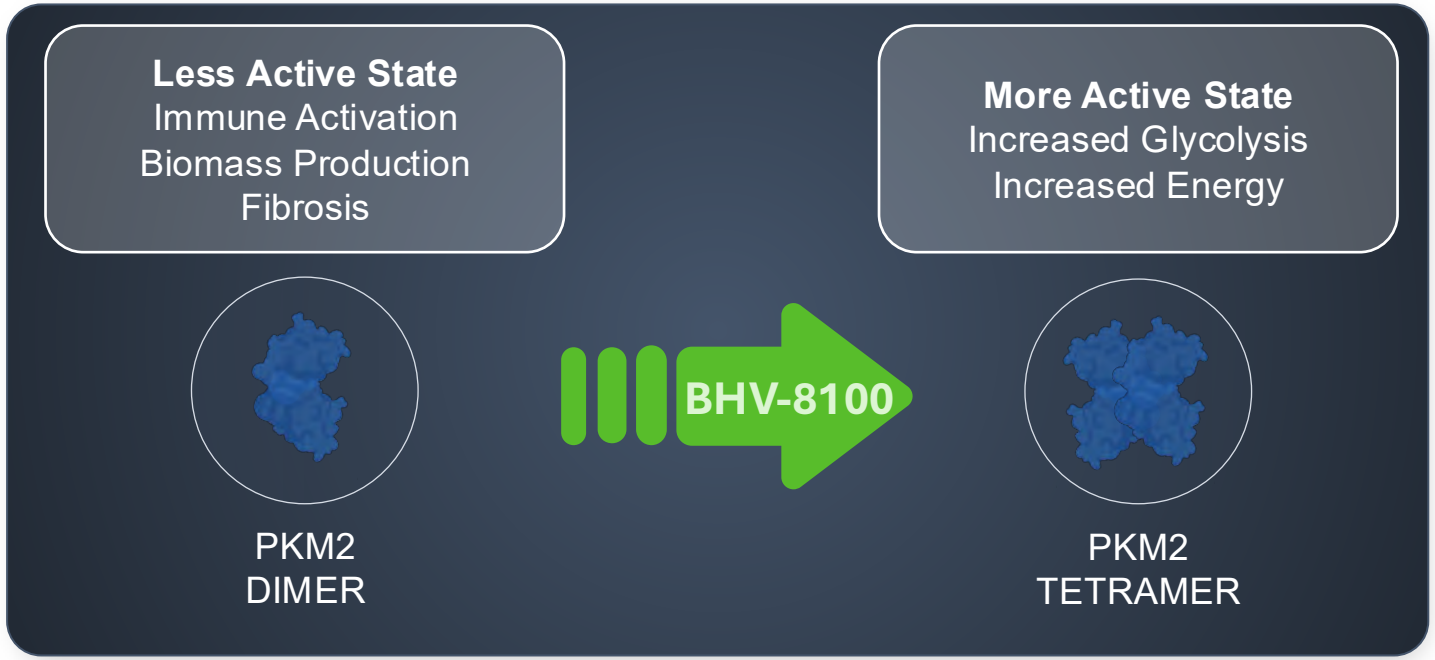
Images (left to right) comparing the cortical distribution of elevated AG and task-induced activity decreases in the DMN in normal with areas of decreased glucose metabolism and Aß plaque accumulation in patients with AD

1. Goyal. Cell Metab. 2017. **2.** Vlassenko. Clin Transl Imaging. 2015.

Activation of Pyruvate Kinase Increases Glucose Consumption and ATP Production While Reducing Deleterious Metabolism

DISCOVERY



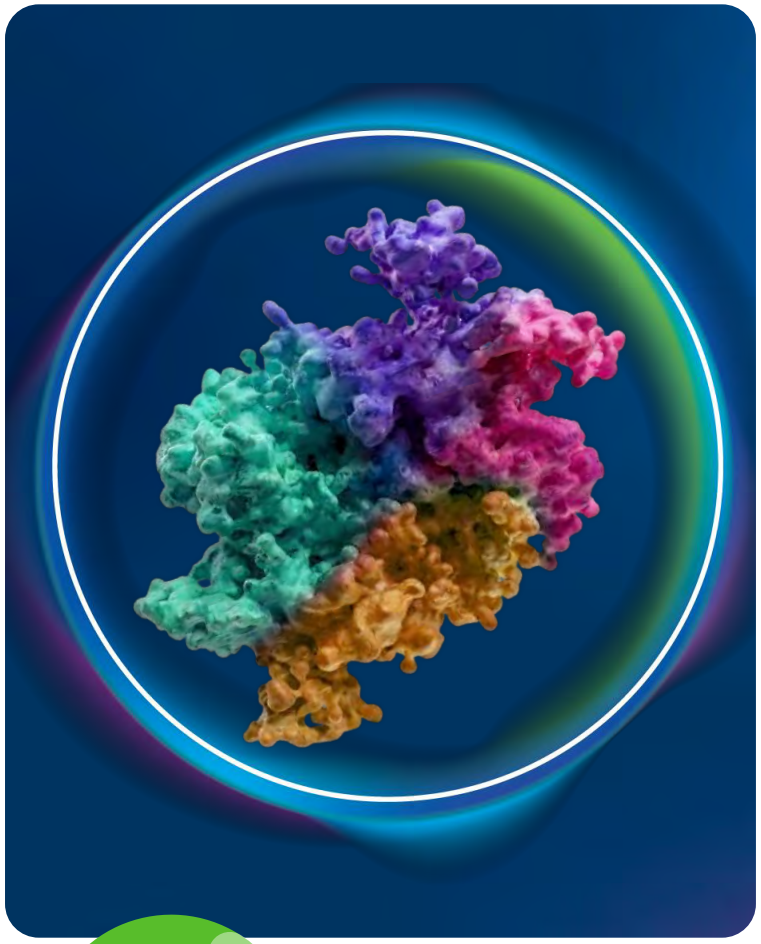


- BHV-8100 stabilizes enzymatically active tetramer state
- Increased energy in CNS
- Decreased neuroinflammation, angiogenesis and fibrosis
- Reduced deleterious metabolic intermediates

Source: Cancer Letters. 2015.

BHV-8100: First Brain-Penetrant PKM2 Activator

DISCOVERY



Oral small molecule medicine with multiple potential indications

- Neurodegeneration
- Retinal degeneration and inflammation
- Neuroinflammation
- Age-related changes: central/peripheral
- Brain ischemia
- Peripheral inflammation

KEY POINT

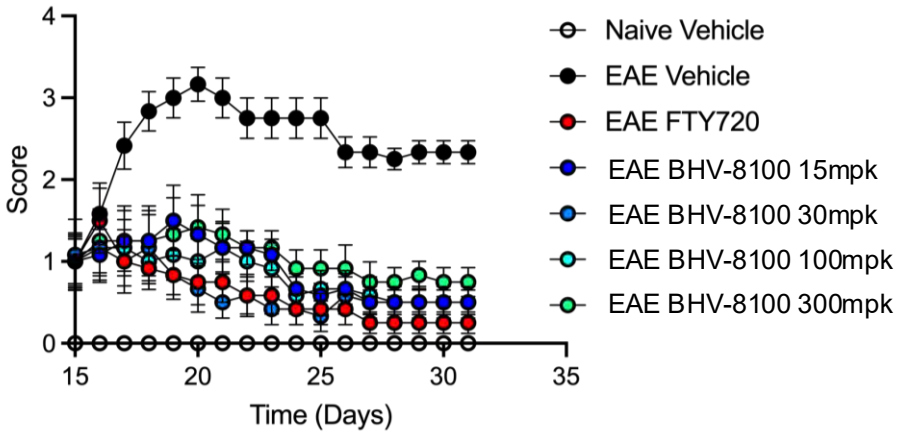
First-in-human dosing at pharmacologically relevant doses initiated 2Q 2026

BHV-8100: Experimental Mitigation of Neuroinflammation and Restoration of Energetic Deficiency

DISCOVERY

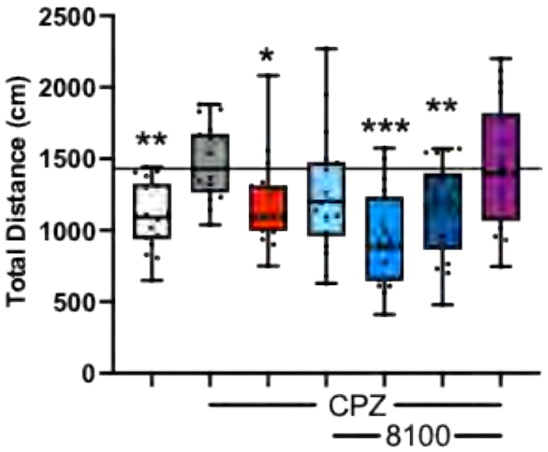
BHV-8100 Shows Robust Reduction of Disease Burden in EAE

Clinical Score (EAE Therapeutic Model)

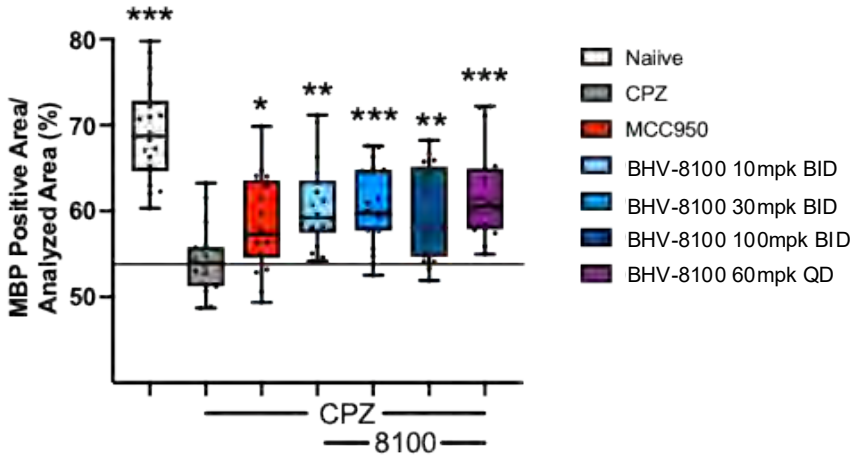


BHV-8100 Demonstrates Striking Efficacy in Cuprizone Model

Distance Traveled



Myelin-Basic Protein in Corpus Callosum



* p < 0.05, ** p < 0.01, *** p < 0.001 relative to CPZ group, MCC950 is an NLRP3 inhibitor.

KEY POINT

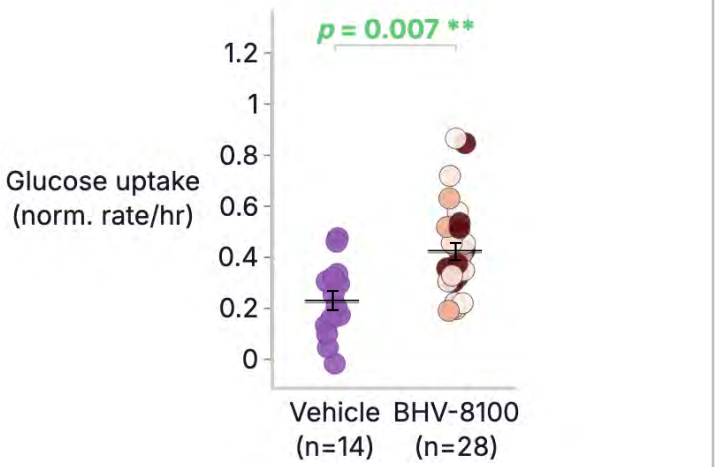
BHV-8100 demonstrates anti-inflammatory efficacy and a strong ability to promote oligodendroglial function in models of EAE

BHV-8100 Demonstrates Sustained Efficacy in Human Brains With Documented Alzheimer's Disease and All-cause Dementia

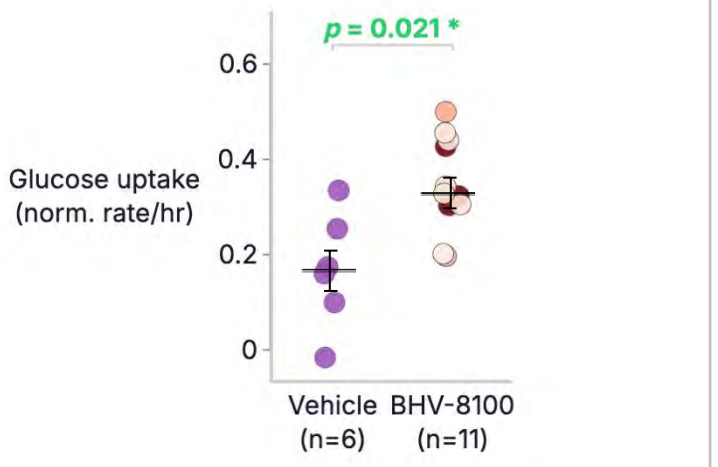
DISCOVERY

Reperfused human brains (Brainex™) allow precise study of brain penetrance, pharmacology, pharmacokinetics, pharmacodynamics and biomarkers in brains with documented diseases

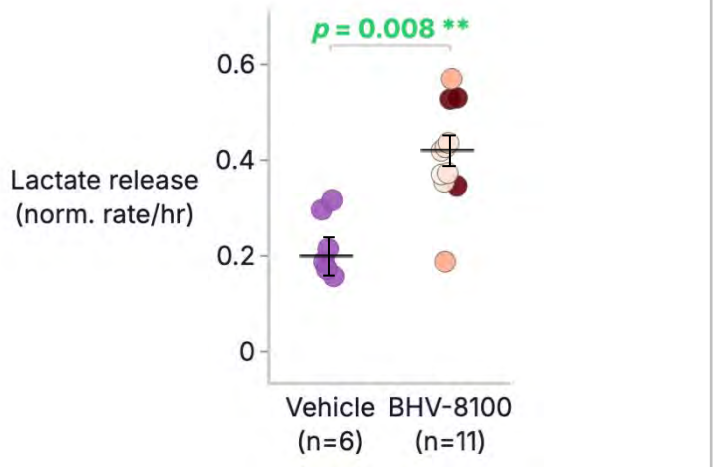
Disease of Cognitive Impairment[1]: BHV-8100 Enhances Glucose Uptake



Alzheimer's Disease: BHV-8100 Enhances Glucose Uptake



Alzheimer's Disease: BHV-8100 Enhances Lactate Production



1. Diverse diagnoses including AD and other disease.

KEY POINT

Precise determinations of brain penetrance and dose-response pharmacology confirm efficacy and guide human dosage

Early FIH Data of BHV-8100 in SAD Shows Safety and Tolerability in Healthy Participants

DISCOVERY

- No SAEs or severe AEs
- Most AEs mild and spontaneously resolving
- No clinically significant ECG changes
- No clinically significant trends in safety labs including LFTs
- PK allows QD dosing & achieves projected efficacious exposures

Preliminary data from ongoing Phase 1 Study BHV8100-101 (as of 29-May-2026)

Financial Update

DAYS MATTER™

1. Includes cash, cash equivalents and marketable securities as of March 30, 2026; **2.** As of April 30, 2026. **3.** 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.

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	

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