UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, DC 20549

FORM 8-K
CURRENT REPORT
Pursuant to Section 13 or 15(d) of
The Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): March 23, 2023

Biohaven Ltd.
(Exact name of registrant as specified in its charter)

British Virgin Islands	**001-41477**	**Not applicable**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

c/o Biohaven Pharmaceuticals, Inc.
215 Church Street
New Haven, Connecticut 06510
(Address of principal executive offices, including zip code)
(203) 404-0410
(Registrant's telephone number, including area code)
Not applicable
(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading symbol	Name of each exchange on which registered
Common Shares, no par value	BHVN	New York Stock Exchange

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☒

Item 7.01 Regulation FD Disclosure

On March 23, 2023, Biohaven Ltd. published an updated investor presentation (the "Presentation") to its website. A copy of the Presentation is attached as Exhibit 99.1 to this Current Report on Form 8-K, and is incorporated herein by reference.

The information in this Current Report on Form 8-K, including the information set forth in Exhibit 99.1, is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in such a filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number*	Exhibit Description
99.1	Investor Presentation, dated March 2023
104	The cover page of this Current Report on Form 8-K formatted as Inline XBRL.

* The XBRL instance document does not appear in the interactive data file because its XBRL tags are embedded within the inline XBRL document.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: March 23, 2023

Biohaven Ltd.

By: /s/ Matthew Buten
Matthew Buten
Chief Financial Officer


biohaven
Investor Presentation
March 2023
BHVN
LISTED
NYSE
DAYS MATTER

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 for our BHV-2100, troriluzole, BHV-5500, BHV-1100, BHV-1200, Taldefgrobep Alfa, BHV-8000, BHV-7000 and BHV-7010 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 ARM™, MATE™, MODE™, TRPM3, TDP-43, UC1MT, TYK2/JAK1, and Kv7. 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; 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 or best in class therapies; and the effectiveness and safety of Biohaven's product candidates. 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 is 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.

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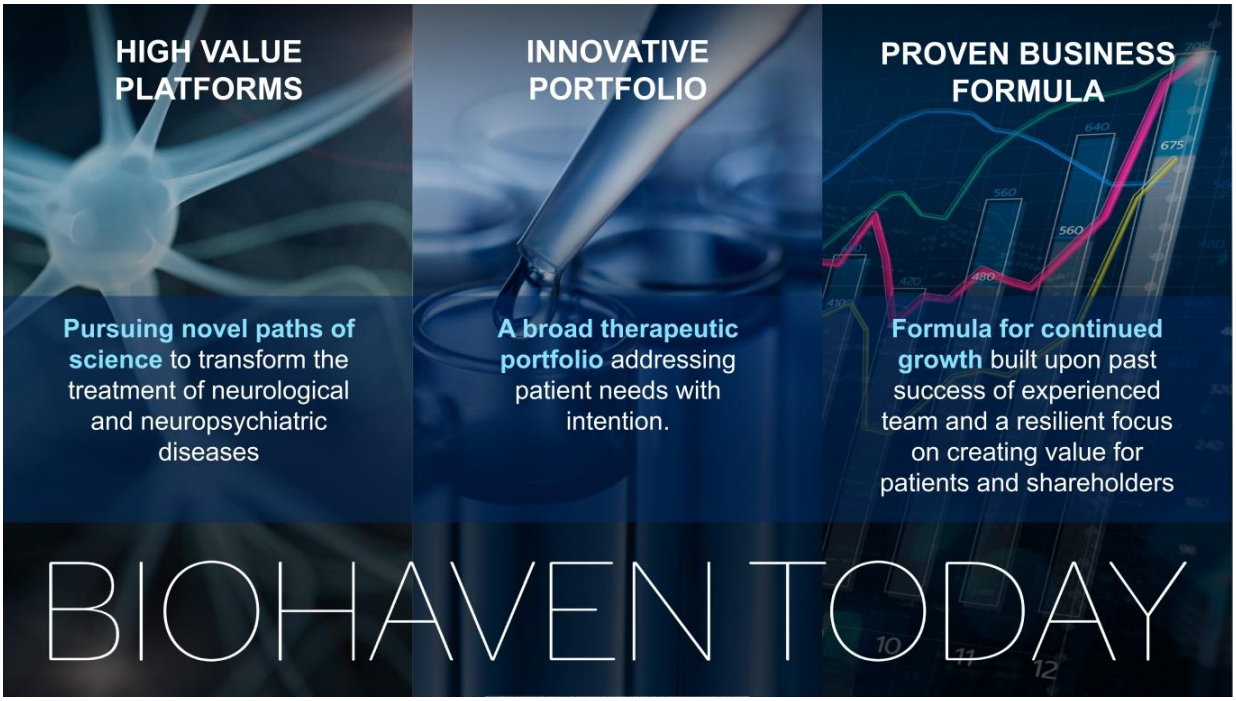
WE SUCCEEDED BY FOLLOWING THE
THE SCIENCE
FOR PATIENTS
#1
MIGRAINE
TREATMENT
IN ITS CLASS
Nurtec® ODT
(rimegepant)
orally disintegrating tablets 75 mg
Pfizer
Pfizer
Acquisition of
CGRP Platform
$13B


Through state-of-the-art drug discovery and passionate scientists
— LARRY
With a powerful R&D engine
— JAVIER
With a commitment to transforming patients' lives
— KATRINA
We never forget patients need us.
— JEREMY
Because we are passionate about the work we do everyday
— FRANCINE
OUR EMPLOYEES KNOW
WE WILL DO IT AGAIN.


HIGH VALUE PLATFORMS
Pursuing novel paths of science to transform the treatment of neurological and neuropsychiatric diseases
INNOVATIVE PORTFOLIO
A broad therapeutic portfolio addressing patient needs with intention.
PROVEN BUSINESS FORMULA
Formula for continued growth built upon past success of experienced team and a resilient focus on creating value for patients and shareholders
BIOHAVEN TODAY


INDICATION
PRECLINICAL
PHASE 1
PHASE 2
PHASE 3
FILED
ION CHANNEL: Kv7 ACTIVATOR
Kv7
BHV-7000 | Epilepsy, Bipolar Disorder
BHV-7010 | Epilepsy, Mood Disorders
ION CHANNEL: TRPM3 INHIBITOR
TRPM3
BHV-2100 | Chronic Pain Disorders
INFLAMMATORY: TYK2/JAK1 INHIBITOR
TYK2/JAK1
BHV-8000 | Immune-Mediated Brain Disorders
GLUTAMATE PLATFORM
Troriluzole
BHV-4157 | Spinocerebellar Ataxia (SCA)
BHV-4157 | Obsessive-compulsive Disorder (OCD)
MYOSTATIN PLATFORM
Taldefgrobep Alfa
BHV-2000 | Spinal Muscular Atrophy (SMA)
BISPECIFIC TARGETED CELL THERAPY
CD-38
BHV-1100 | Multiple Myeloma
DISCOVERY RESEARCH
IgG Degrader
IgA Degrader
Next-Gen ADC Platform


TAIYE | Associate Scientist
Key Programs
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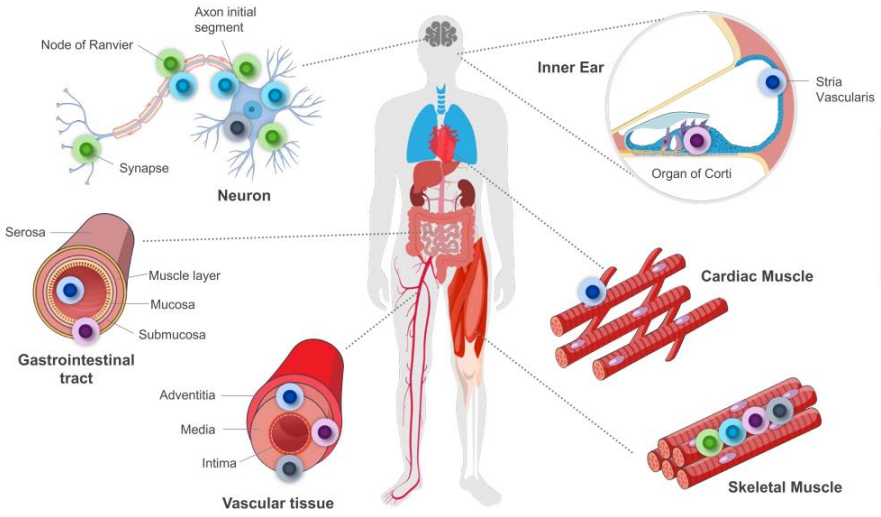
Kv7 Ion Channels:
Key Regulators of Neuronal Function



Source: PHYSIOLOGY 26: 365–376, 2011; doi:10.1152/physiol.00009.2011 (Maria Virginia Soldovieri,1, Francesco Miceli,2,3 and Maurizio Taglialatela1,2)

Kv7

5 FAMILY SUBTYPES

Primary localizations:

- Kv7.1: cardiac
- Kv7.2: CNS **BHV-7000/7010 activator**
- Kv7.3: CNS **BHV-7000/7010 activator**
- Kv7.4: smooth muscle and inner ear
- Kv7.5: vascular tissue, neurons, skeletal muscle

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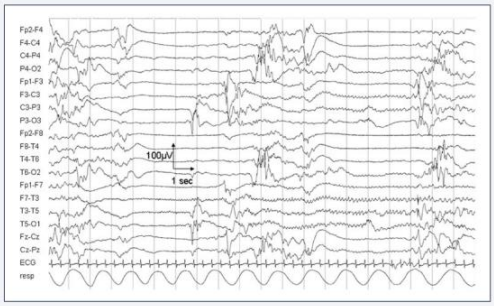
Kv7 Potassium Channels Regulate Neuronal Excitability and Loss of Function Causes Epilepsy

Impaired Kv7 Channel Activity Causes Certain Types of Epilepsy

Loss-of-function mutations in Kv7.2 channel



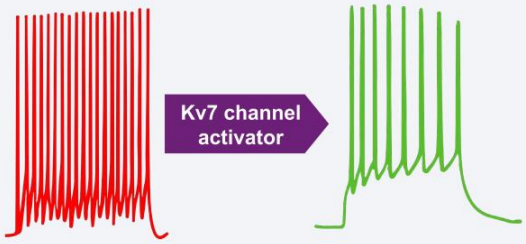
Abnormal EEG with Epileptic Activity

Dalen Meurs-van der Schoor, Front Pediatr (2014).

Kv7 Activation Normalizes Action Potential Firing

Increased frequency of action potential firing

Normal frequency of action potential firing



Hyperexcitability — Epilepsy and Mood Disorders

Adapted from Wulff, Nat Rev Drug Discov (2009) .

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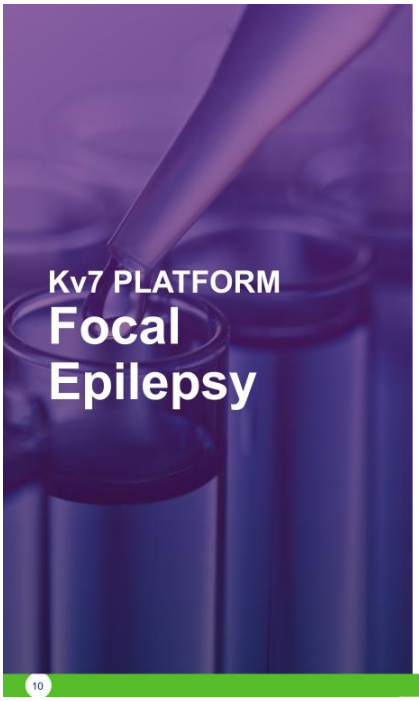

Potential Best-in-Class, Fast-Follower Approach
Clinically validated mechanism of action

Pursuing Differentiation
Preclinical data suggests wide therapeutic index in the clinic by dialing out GABA risk (*somnolence, sedation, dizziness*)

Translatability
Preclinical data in epilepsy models is predictive of clinical efficacy

Broad Potential in Adjacent Indications
Warrants further evaluation in BPD, depression, pain, others

Patent Protection
Both BHV-7000 and BHV-7010 covered until 2039

Status Update
BHV-7000 Phase 1 SAD/MAD study completed (Canada)
BHV-7010 IND anticipated in 2023

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1st and 2nd Gen Kv7 Activators Show Clinical Anti-seizure POC, But Off-target Activities, Opportunity for 3rd Gen Kv7 Differentiation

EZOGABINE

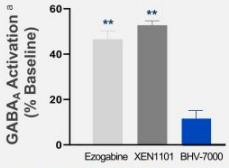
- **Unstable** when exposed to light
- **Label warnings** for skin discoloration
- **Black box warning** for retinal abnormalities/vision loss

FDA Drug Safety Communication: Potiga (ezogabine) [04-26-2013]

BHV-7000 is chemically stable to photo-oxidation

XEN1101

- **XEN1101** and ezogabine **significantly greater** $GABA_A$ receptor allosteric activators than BHV-7000 in vitro[a]
- $GABA_A$ receptor activation: somnolence, dizziness, fatigue, diplopia

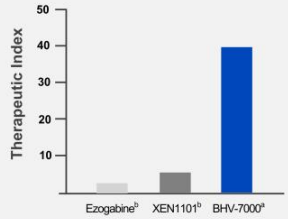


** significantly different from BHV-7000, all tested at 10 µM

BHV-7000 is selective for Kv7 over $GABA_A$ receptors [a]

BHV-7000

- **Potent activator** of Kv7 channels
- **Effective and well-tolerated** in preclinical seizure assays



BHV-7000 has a wide therapeutic index preclinically [a]

a. Biohaven data on file (2022). **b.** Calculated as ratio of TD_{50} (rotarod) to ED_{50} (MES seizure assay) data presented by Xenon at Epilepsy Foundation Pipeline Conference, San Francisco (Feb 2018).
Preclinical results are consolidated from separate reports and graphed together.
Data presented is not the result of any head-to-head clinical trials and does not mean or suggest that BHV-7000 is clinically more safe and effective.

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BHV-7000 Exhibits Highly Differentiated Preclinical Profile

	Ezogabine	XEN1101	BHV-7000
Kv7.2/7.3 Activator	✓ Activator, clinical and preclinical anti-seizure activity	✓ Activator, clinical and preclinical anti-seizure activity	✓ **Activator, clinical and preclinical anti-seizure activity**
$GABA_A$ Activity "dialed-out"	✗ $GABA_A$ activity present	✗ $GABA_A$ activity present	✓ **Negligible $GABA_A$ activity**
Wide Therapeutic Index	✗ <3x reported[a,b]	✗ <5x reported[a]	✓ **>40x[b]**

a. Calculated as ratio of TD_{50} (rotarod) to ED_{50} (MES seizure assay) data presented by Xenon at Epilepsy Foundation Pipeline Conference, San Francisco (Feb 2018). **b.** Biohaven data on file (2022).
Preclinical results are consolidated from separate reports and not a result from head-to-head comparisons Data presented is not the result of any head-to-head clinical trials and does not mean or suggest that BHV-7000 is clinically more safe and effective.

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BHV-7000 Phase I SAD/MAD Study Update: Dosing Completed

Preliminary Safety

- Single doses up to 100 mg and multiple doses up to 40 mg daily x15 days generally well-tolerated
- Most AEs mild and resolved spontaneously
- No serious or severe AEs
- No dose limiting toxicities

Preliminary Pharmacokinetics

- Target concentrations for efficacy exceeded based on preclinical MES model
- High fat meal had no effect on exposures

Pooled Adverse Events*	N = 61
Headache	7 (11.5)
Back pain	3 (4.9)
Constipation	2 (3.3)
Abdominal discomfort	2 (3.3)
Urinary frequency	2 (3.3)

Pooled adverse events from SAD and MAD cohorts occurring in more than 1 subject (preliminary blinded data including BHV-7000 and placebo groups)

* MedDRA® Preferred Term

BHV-7000 Shows Favorable CNS-Associated AE Profile Compared to Xen1101 Across Pooled MAD Cohorts

Pooled Adverse Events[1]	BHV7000-101 MAD pooled (active, n=17)	Xen1101 MAD pooled[2] (active, n=18)
Somnolence	0%	39%
Headache	18%	39%
Balance disorder	0%	17%
Dizziness	0%	17%
Memory impairment	0%	28%
Sensory disturbance	0%	11%
Speech disorder	0%	33%

[1]MedDRA® Preferred Term within the System Organ Class of "Nervous System Disorders"
[2]Data from 2019 73rd Annual American Epilepsy Society Meeting poster, Abstract # 3.31, published 11.25.19
Data presented is not the result of any head-to-head clinical trials and does not mean or suggest that BHV-7000 is clinically more safe and effective.

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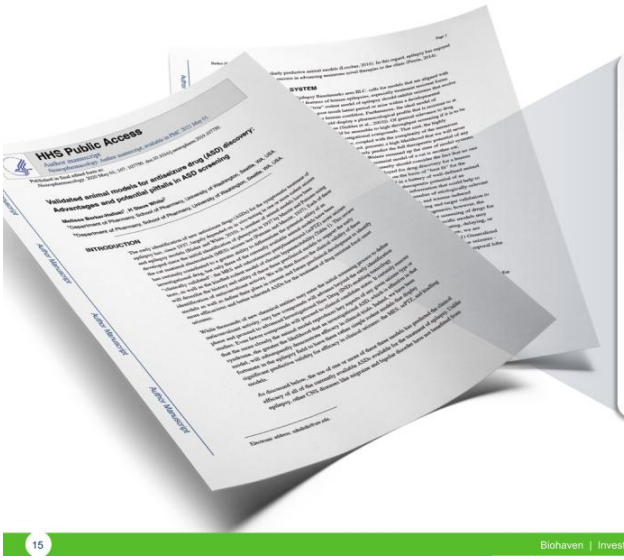
Maximal Electroshock Seizure Model: Strongly Positive Predictive Translation to the Clinic



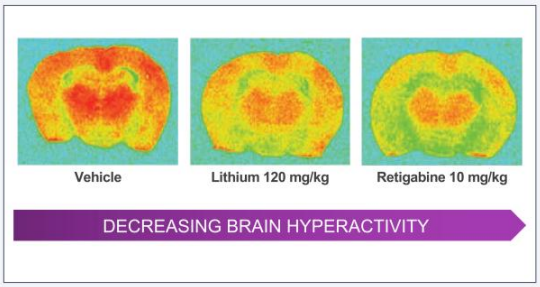
Predictive Efficacy: Efficacy results from the maximal electroshock (MES) model are **predictive of efficacy in the epilepsy patient population**.

Other CNS diseases have not benefitted from such similarly predictive animal models (Loscher, 2016).

Preclinical and Clinical Data Suggest a Role for Kv7 in Bipolar Disorder and Depression

Mania

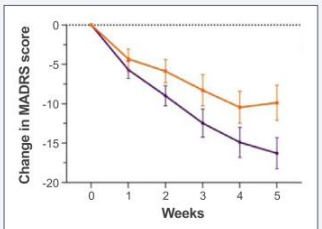
In mania models, Kv7 activation reduces hypermetabolism and hyperactive behavior[1]



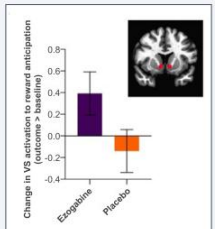
Retigabine normalizes a mania model hypermetabolic signature to **a higher degree than lithium**

Depression

Robust MDD efficacy likely to translate to bipolar disorder[2]



~7pt decrease vs placebo with ezogabine in depression symptoms



fMRI study: ezogabine effects mediated by the limbic system

3/3 positive studies of fluoxetine in bipolar depression indicate effects in MDD translate to bipolar disorder[3]

1. Kristensen et al. J Neurochem (2012). 2. Costi et al. Am J Psychiatry (2021); 178(5):437-446. 3. Golberg et al. J Clin Psychiatry (2021) 82(1):20ed13649

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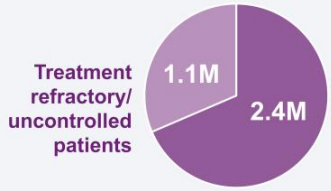
Blockbuster Peak Sales Potential for Kv7 Platform

Epilepsy

3.5M EPILEPSY PATIENTS
adult and pediatric, US[1–3]



200–300 KCNQ2-DEE
incidence in live births

Mood Disorders

7–11M
BIPOLAR DISORDER (US)[4]

34.5M
MAJOR DEPRESSIVE DISORDER (US)[5]

- ✓ **Easy to Prescribe**
  Convenient dosing, no titration, no food effect
- ✓ **Safe/Tolerable**
  Side effects negatively impacted other recent epilepsy launches
- ✓ **Biohaven Proven Commercial Expertise in Neuroscience**

1. https://www.cdc.gov/epilepsy/data/index.html (1.2% of the population has epilepsy, 60% is focal onset). 2. Kammerman S. West J Med. 2001 Aug;175(2):99-103. 3. Hauser WA. Epilepsia. 1991 Jul-Aug;32(4):429-45. 4. https://www.nimh.nih.gov/health/statistics/bipolar-disorder (2.8% of US adults have had BPD within last year and 4.4% of population over their lifetime). 5. Hasin DS. JAMA Psychiatry. 2018 Apr 1;75(4):336-346.

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TRPM3 (BHV-2100): A Novel Peripheral Target for Neuropathic Pain

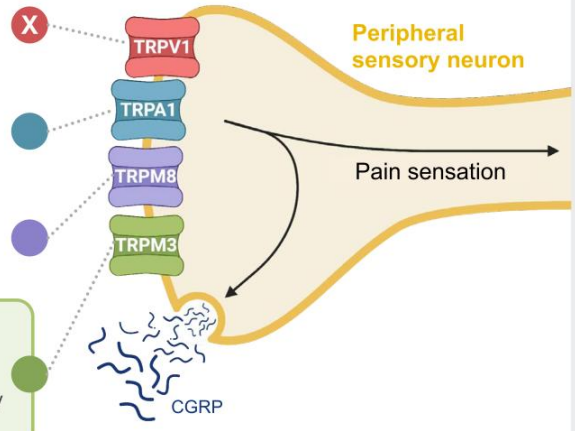
Differentiated from existing programs targeting TRPV1 and TRPA1

Appeared effective, but **class terminated for hyperthermia** despite significant investment

Difficult target to drug with PK/solubility issues; remains in active drug development (Lilly)

Conflicting data for pro- and anti-nociceptive role; remains in active drug development

Promising as a novel target: expressed at similar levels as TRPV1 and TRPA1 in peripheral neurons but unlikely to have thermal liabilities



- Current SoC comes with liabilities — addiction (opioids), sedation
- Pre-clinical data shows reversal of pain in various models, without sedative effects
- IND planned for 2H 2023

See Koivisto et al, Nature Reviews Drug Discovery 2022 for background on TRP channel drug development.

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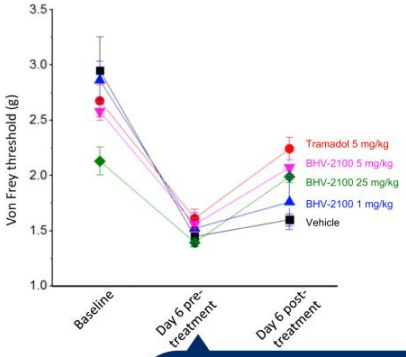
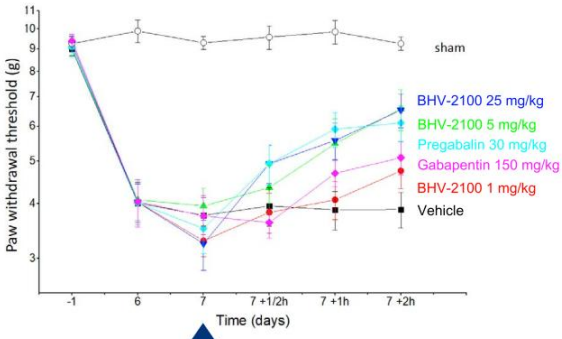
TRPM3 (BHV-2100) Reduces Pain in Preclinical Models of Chemotherapy and Diabetic Neuropathy

Encouraging evidence of pain reduction without the sedation observed with high dose tramadol/gabapentin



Drug administered 6 days after oxaliplatin in mice



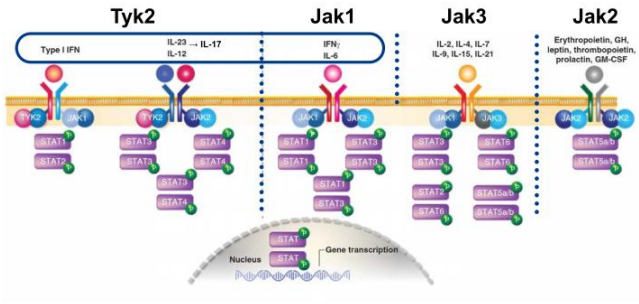
Drug administered 7 days after diabetic induction with STZ in rats

Source: Biohaven data on file

BHV-8000: TYK2/JAK1 in NeuroInflammatory Disease

BHV-8000[1] has potential for first-in-class and best-in-class dual TYK2/JAK1 inhibition

- ✓ Most advanced, selective TYK2/JAK1 inhibitor with good brain penetration in animal models, to the best of our knowledge
- ✓ Dual inhibition of TYK2 and JAK1 can effectively block Th17 cell generation, Type I IFN signaling and inflammation
- ✓ JAK inhibitors are more efficacious anti-inflammatory agents than anti-TNFs



Modified from Gonciarz et al 2021 Immunotherapy (2021) 13(13), 1135–1150

1. Formerly TLL-041

Cellular Drivers In Neuroinflammation: Predominant TYK2/JAK1 Effects

Microglia

- **IFN-γ**

- IL-β
- TNF downstream of IFN-γ
- IL-8
- GM-CSF, MCP-1

Microglia are the resident macrophages of the CNS, playing an important role in neuroinflammation, repair and maintenance

Astrocytes

- **IFN-γ**
- **IL-12**

- TNF
- IL-8

Abnormal astrocytic activity may exacerbate inflammatory reactions and contribute to tissue damage

Lymphocytes, other leukocytes

- **IL-23**
- **IL-17 downstream of IL-23**

- IL-2, IL-4

Strong evidence for Th17 lymphocyte involvement in the pathogenesis of multiple sclerosis and Parkinson's disease

TYK2/JAK1 cytokines

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Properties of TYK2/JAK1 Selective Inhibitors Ideal for Treatment of Neuroinflammation

Brain penetrance

- Biologics are not brain penetrant
- Requires specially designed small molecule to reach site of action
- Existing agents exclude all lymphocytes from the CNS
  - TYK2/JAK1 inhibitor expected to selectively deplete CD4+ Th17 cells at the site of action

Dose-limiting tolerability issues of JAK2 and JAK3 inhibition minimized

- Serious cardiovascular events
- Severe immunosuppression
- Anemia, cancer, death

Small molecules allowing convenient at-home, daily oral administration

Potential adverse events of central immunomodulation rapidly addressed following withdrawal vs. long exposure of biologics

BHV-8000: Phase-1 Ready, Brain-Penetrant TYK2/JAK1 Inhibitor for Neurological Disorders

BHV-8000

✓ **The most advanced brain-penetrant, selective, dual TYK2/JAK1 inhibitor**
- Good brain penetration in pre-clinical species
- Excellent selectivity over JAK2 and JAK3, avoiding associated toxicities
- Wide estimated therapeutic windows

✓ **Mechanistic proof-of-concept has been demonstrated in preclinical models:**
- Inhibited LPS-induced proinflammatory cytokine production in mouse microglia
- Efficacy in Th17-dependent EAE mouse models
- Completely blocks dsRNA induced pSTAT1 signaling, considered important in Parkinson's disease, AD and ALS

✓ **Demonstrated good safety pharmacology and toxicology**
- Completed IND enabling toxicology studies

✓ **Pharmacokinetics potentially suitable for once daily dosing**

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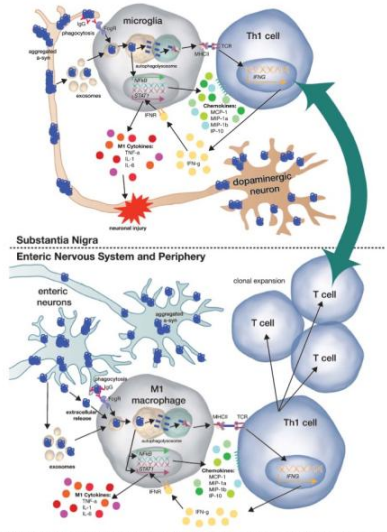
TYK2/JAK1 Pathways in Parkinson's Disease

- α-synuclein aggregates in dopaminergic neurons of substantia nigra
- Microglia present α-syn fragments with MHC-II leading to immune response, and activated T cells entering the CNS
  - T cells release IFN-γ, activating microglia which release further proinflammatory cytokines
- Activated T cells and microglia injure and kill DA neurons
- Strong evidence implicates IL-17 and Th17 cells in Parkinson's
- TYK2/JAK1 kinase inhibitors reduce Th17 cells (through IL-23 inhibition) and reduced microglial activation through inhibition of IFN-γ signaling

BHV-8000 is ideally suited to reducing both important axes of neuroinflammation in Parkinson's



Reish and Standaert, a-syn induction of inflammation in PD; Fu et al. Journal of Neuroinflammation (2022) 19:98

TYK2/JAK1 Inflammatory Pathways in Multiple Sclerosis

- Multiple sclerosis is an inflammatory disease in which humoral immune and cell-mediated immune responses target CNS antigens
- IL-17A-defective mice are highly resistant to induction of EAE
- PKM2 activators mediate potent inhibitory effects in EAE model due to Th17 cell effects
- In a meta-analyses of literature, TNF-α, IL-15, IL-12, IL-23/IL-17, and IFNγ were elevated in or predictors of MS patients vs. controls
- Secukinemab (IL-17A) demonstrates an effect in RRMS
- Brain penetrant TYK2/JAK1 kinase inhibitors reduce Th17 cells (IL-17 and IL-23) and target IL-12 signal transduction

BHV-8000 is ideally suited to reducing neuroinflammation in MS

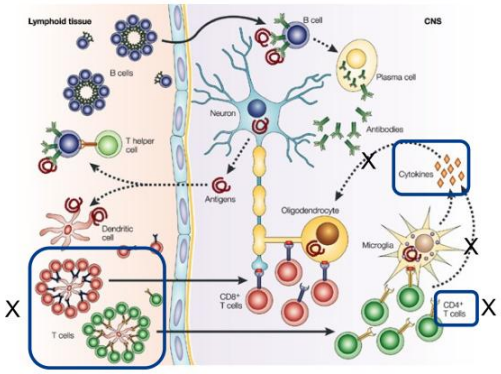


Figure modified from https://u.osu.edu/multiplesclerosis/pathophysiology-and-clinical-presentation-correct-diagnosis/

McGinley et al, Immunity 52:342-356, Palle et al, Med Sci, 5:23, 2017;
Bai et al, Frontiers in Neuroscience, 10.3389, Oct 4, 2019, Havrdova, Multiple Sclerosis Journal, 18_509, 2012

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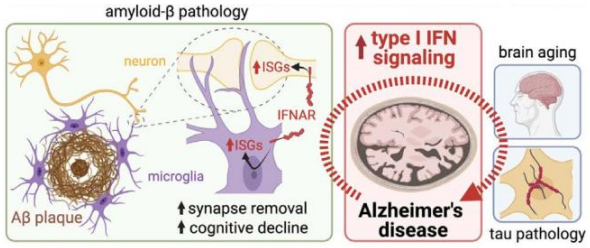
TYK2/JAK1 Inflammatory Pathways in Other Neuroinflammatory Disorders

- Alzheimer disease
  - Neuroinflammation is a key event in AD pathogenesis
  - Cytokines and chemokines affect TAU phosphorylation and amyloid deposition
  - Aβ induces the glial cell expression of several pro-inflammatory cytokines, such as interleukin-1β (IL-1β), IL-6, tumor necrosis factor-α (TNF-α) and interferon-γ (IFN-γ)
- Amyotrophic lateral sclerosis
  - TDP-43 inclusions may trigger Type I interferon in Amyotrophic Lateral Sclerosis and FTD
- Autoimmune encephalomyelitis
  - Multiple cytokines overlapping with TYK2/JAK1 cytokines suggests likely therapeutic role



Jonas J. Neher, Immunity 55, May 10, 2022

BHV-8000 reduces several key cytokines driving Alzheimer's pathology

Domingues et al, Curr Alzheimer Res. 2017 Aug; 14(8): 870–882.

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BHV-8000 Summary

Selective inhibition of TYK2/JAK1 has profile for best-in-class immunomodulation in neuroinflammatory disorders

CNS immunomodulation in neuroinflammatory disorders will interrupt the cycle of inflammation, neuronal injury and demyelination, and will complement other approaches directly addressing neurodegeneration such as amyloid, a-synuclein, tau and mitochondrial targeting therapies

Strong evidence supports potential efficacy in Parkinson's disease, multiple sclerosis, Alzheimer disease and possibly further neuroinflammatory diseases

Selectivity for TYK2/JAK1 mitigates non-selective JAK class liabilities, largely related to JAK2 and JAK3 inhibition, and offer potential to improve benefit-risk for the highly selective BHV-8000 dual kinase inhibitor

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TRORILUZOLE

SCA and OCD

SPINOCEREBELLAR ATAXIA (SCA)

- Reduction in fall risk + treatment effect in SCA3 genotype
- Safe and well-tolerated profile
- **Regulatory engagement planned for H1 2023**

OBSESSIVE COMPULSIVE DISORDER (OCD)

- Compelling mechanistic rationale for cortico-striatal glutamate abnormalities in OCD patients
- Strong PoC in Phase 2
- Well-characterized in 1,000+ patients
- Two Phase 3 studies ongoing; **enrollment completion anticipated YE 2023**

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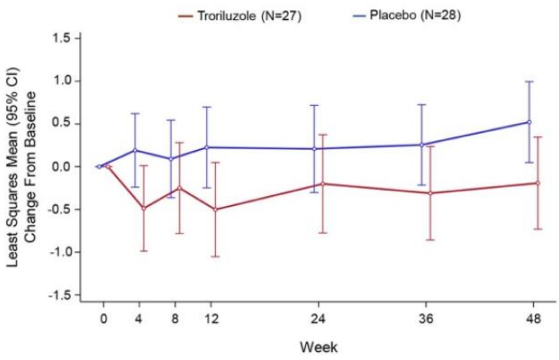
Troriluzole Phase 3 Results: Treatment Effect in SCA3 Genotype

- Post-hoc analysis suggests **troriluzole has disease-stabilizing effect in SCA3 genotype** (most common genotype, pre-identified randomization stratum)
  - In SCA3 patients (N=89 / 41%), numerical treatment benefit observed on change from baseline on f-SARA at week 48
  - LS mean change difference -0.55, nominal p-value = 0.053, 95% CI: -1.12, 0.01
  - In subgroup of SCA3 patients able to walk w/o assistance at baseline (N=55), greater numerical treatment benefit observed on change from baseline on f-SARA at week 48 (Figure)
  - LS mean change difference -0.71, nominal p-value = 0.031, 95% CI: -1.36, -0.07)
  - In SCA3 patients, reduction in fall risk also observed compared to placebo
- Primary endpoint was not met in overall population (n=213)



Reduction in fall risk + treatment effect in SCA3 patients suggest troriluzole has clinically meaningful benefit

f-SARA: Functional Scale for the Assessment and Rating of Ataxia

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BHV-4157 Troriluzole Treated OCD Patients: Strong Signal Observed in Phase 2 POC Supports Advancement to Phase 3

STUDY BHV-4157-202

Patients with moderate-to-severe OCD (Y-BOCS score ≥ 19) and inadequate response to standard of care

SAMPLE SIZE

226 subjects

RANDOMIZATION

1:1

DOSE

Troriluzole 200 mg QD vs Placebo QD (in patients on standard of care)

PRIMARY OUTCOME

Y-BOCS, precedented outcome measure accepted by FDA

Y-BOCS, Yale-Brown Obsessive Compulsive Scale (FDA accepted outcome measure)

Table 1: Troriluzole Effect on OCD in Phase 2/3 Trial[1]

Y-BOCS Total Change from Baseline	Week 4 (N=115[a], 111[b])	Week 8 (N=108[a], 96[b])	Week 12 (N=102[a], 99[b])
a. Placebo[a]	-2.9	-3.6	-4.9
b. Troriluzole[b]	**-3.4**	**-5.1***	**-5.9**
p-value	0.451	0.041	0.220

1. BHV-4157-202 Final Unblinded Analysis YBOCS Total Change from Baseline by Week LSMeans from MMRM Model MITT Data Set

Table 2: Troriluzole Effect on Patients with Severe OCD[1]

Y-BOCS Total Change from Baseline	Week 4 (N=47[c], 49[d])	Week 8 (N=45[c], 42[d])	Week 12 (N=43[c], 44[d])
a. Placebo[c]	-3.5	-3.1	-4.6
b. Troriluzole[d]	**-4.1**	**-6.0***	**-7.0**
p-value	0.584	0.035	0.084

1. Patients at baseline with median Y-BOCS total scores > 26 (severe OCD symptoms).
* $p < 0.05$ versus placebo

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Pivotal Phase 3 Study Initiated July 2022

- Large pre-clinical and clinical safety package licensed from BMS
- Limited additional work needed to support BLA submission
- 34 sites total, ~20 open

POC for Mechanism of Action

Supported by clinical data and multiple disease models

Muscle and Bone Improvements

Observed in non-clinical studies

Strong Supporting Efficacy Signal

Muscle mass growth in healthy subjects

Large Safety Database

Established in prior pediatric studies

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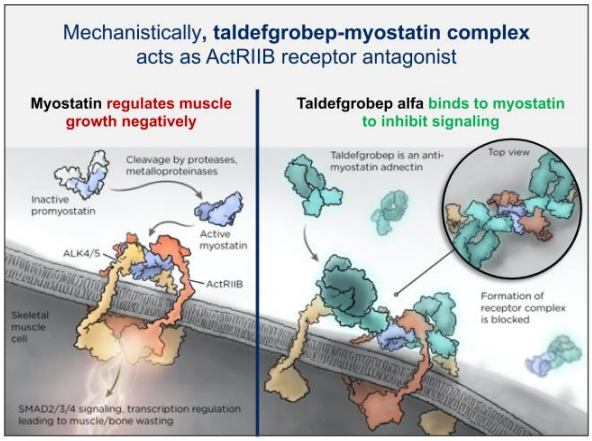
T-Alfa: Targeting a Differentiated Regulatory Pathway of Muscular Growth

The Most Advanced Phase 3 Ready Myostatin Inhibitor



- **Safety** studied with more than 300 subjects dosed in prior clinical studies in pediatric and adult populations
- Non-clinical studies showed **muscle and bone improvements**
- **POC** for mechanism of action supported by clinical data and multiple disease models
- **Phase 3 study initiated in July 2022**

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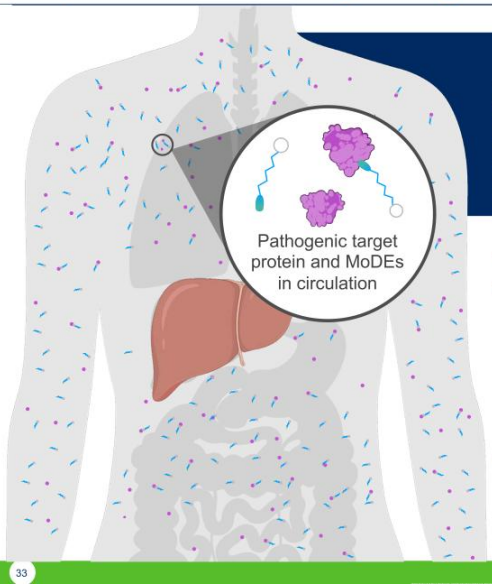
Bispecific Platform: IgG Degradation via MoDE™



Molecular Degraders of Extracellular Proteins (MoDE): small molecules bind extracellular target proteins and cause them to be removed from the body ***through the liver***

- Harnesses the body's own machinery for degrading proteins
- Extracellular protein targets are **eliminated** via the asialoglycoprotein receptor (ASGPR)

- Protein targets are degraded via endolysosomal proteolysis

* formerly BH 2640

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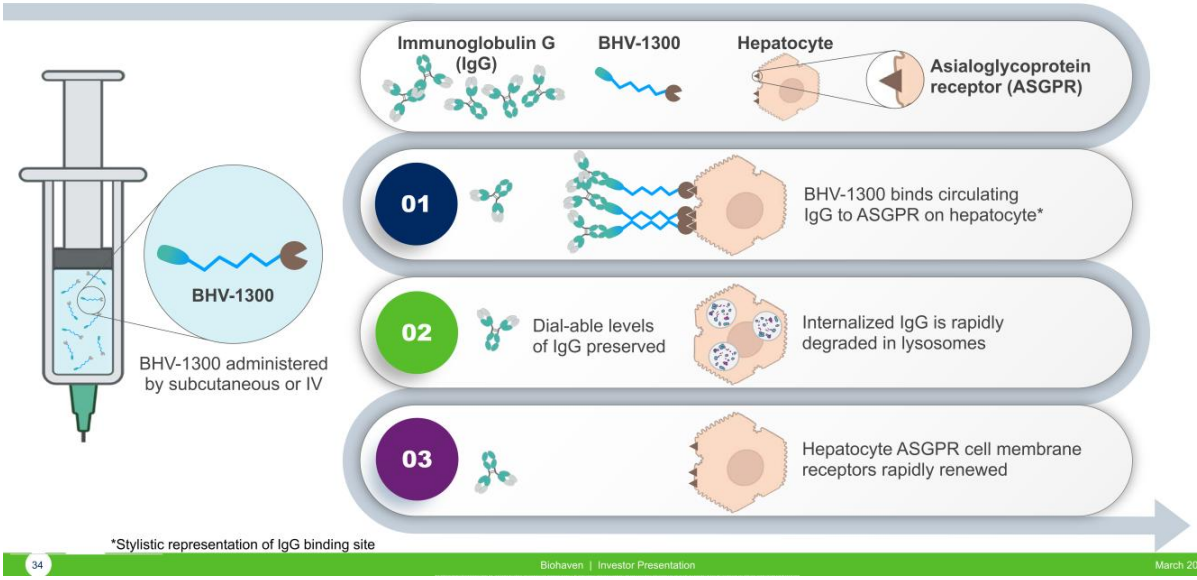
Hepatic ASGPR Receptor Harnessed for Efficient and Safe Removal of Pathogenic Targets – Pan IgG Degradation



*Stylistic representation of IgG binding site

Hepatic ASGPR Receptor Harnessed for Efficient and Safe Removal of Pathogenic Targets

- High capacity ASGPR hepatocellular receptors internalize plasma proteins with specific motifs
- Bispecific ASGPR-binders with target-binder effectively removes [pathogenic] target from the circulation
- IgG may be more rapidly removed from the circulation than FcRN inhibitory antibody or antibody fragments, without causing hypoalbuminemia or dyslipidemia
  - Improved, dialable potency (deeper IgG/IgA reductions possible)
  - Improved pharmacodynamics (faster onset of action)
  - Improved safety profile (fewer side-effects, rapid drug elimination)

BHV-1300: A highly optimized Biohaven ASGPR binder advancing as drug candidate

- ✓ Balances liver removal of unbound to target-bound drug
- ✓ Optimizes safety vs efficacy
- ✓ Improves kinetics of target removal
- ✓ Suitable Target Product Profile for a rapid onset medication with weekly or less frequent SC administration

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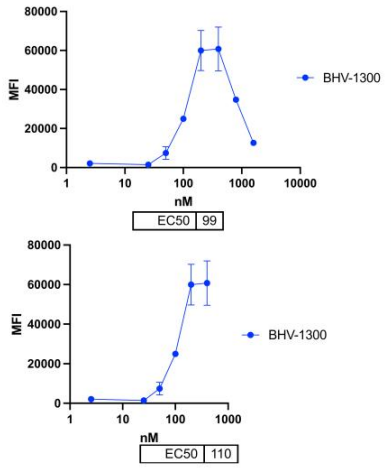
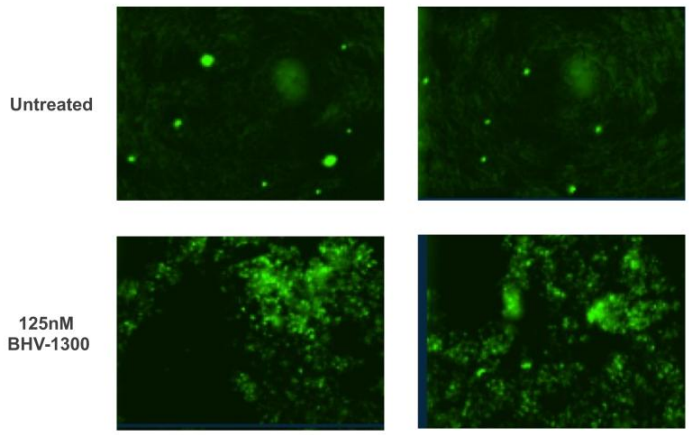
In vitro BHV-1300 Mediates Removal, Uptake and Degradation of IgG

Uptake of IgG in HepG2



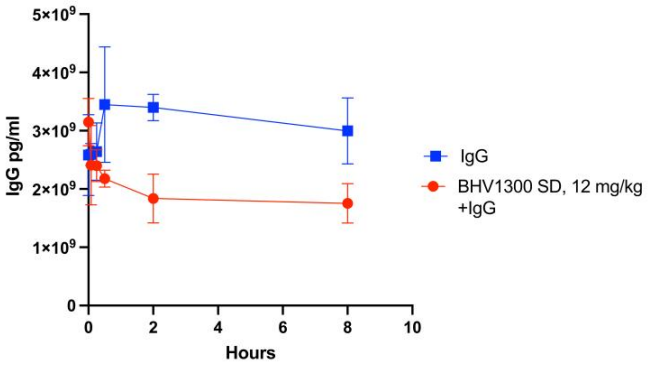
HepG2 cells internalize IgG-BHV-1300 complexes



BHV-1300 Selected for Remarkable Efficiency in Removal of Exogenously Administered Human IgG in Mouse Screen

Approximately 40% IgG removal achieved in 2 hours with a molar ratio of drug-to-target = 1.0

BHV-1300 human IgG depletion in mice, single dose 12 mg/kg



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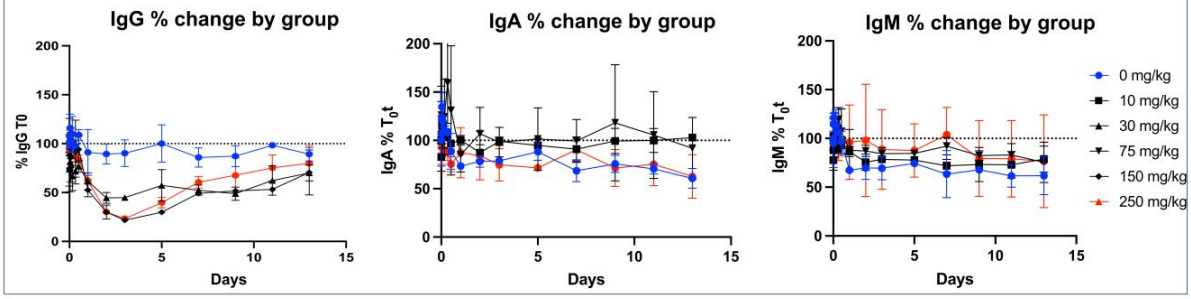
IgG is Specifically Depleted: IgA and IgM Levels Remain Unchanged

BHV-1300 administered IV to cynomolgus macaques



- Preliminary BHVN data and literature consistent with an absence of effects on serum albumin, LDL, HDL or triglycerides, and specificity for targeted IgG species
- Remarkable drug efficiency in mouse given exogenous IgG recapitulated in monkeys with endogenous IgG. Molar ratio of approximately 1.0 allows 60% IgG lowering following a single dose

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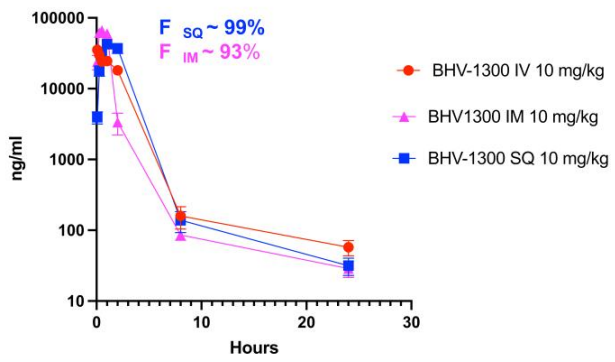
BHV-1300 Administered by SC, IM or IV Routes has Comparable Bioavailability

BHV-1300 levels in plasma after a single dose of 10 mg/kg in the presence of human IgG



Potentially allows at-home, self administration with subcutaneous injection

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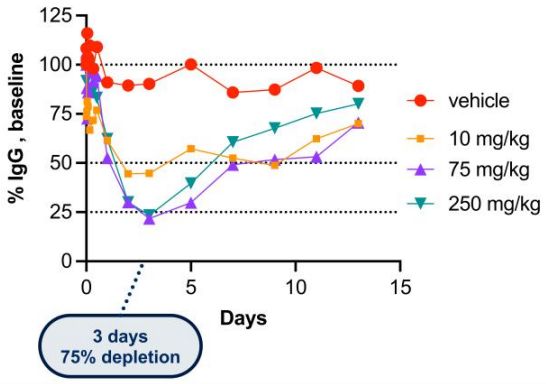
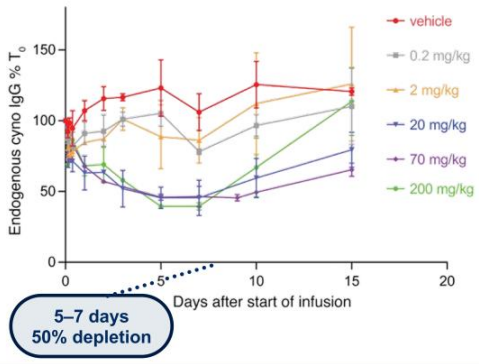
Pan-IgG Degrader (BHV-1300) and SoC (Efgartigimod) Primate Data

BHV-1300: Cynomolgus Pharmacodynamics



Efgartigimod: Cynomolgus Pharmacodynamics



IND ready in H2 2023

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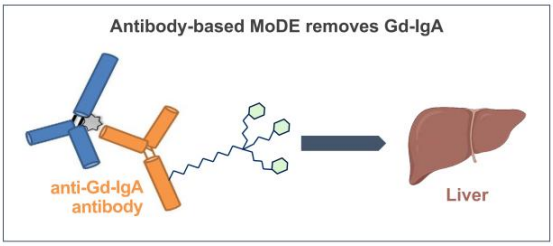
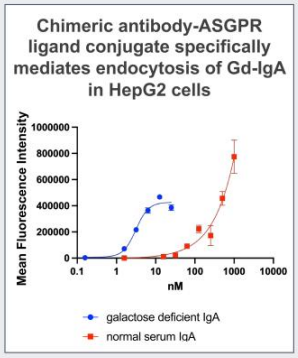
The Journal of Clinical Investigation 2018:128(10):4372-4386. https://doi.org/10.1172/JC197911.

Bispecific Platform: IgA Degrader for IgA Nephropathy

Therapeutic Hypothesis: Galactosyl deficient-IgA (Gd-IgA) containing-immune complexes drive glomerular injury in IgA nephropathy.[1] Specific removal of this pathogenic Gd-IgA with preservation of "normal" IgA (and other Ig's) permits disease remission without incurring an infection risk.

- Highly selective, rat mAb conjugate binds Gd-IgA with high affinity
- Antibody humanization ongoing, subsequent conjugation with established degrader technology will allow rapid and efficient hepatic extraction, leaving "normal" IgA





Gd-IgA removal drives efficacy without infection risk[2]

1. Canetta et al, 2014, others. 2. Untested therapeutic hypothesis.

BHVN Bispecific Degrader Platform Summary

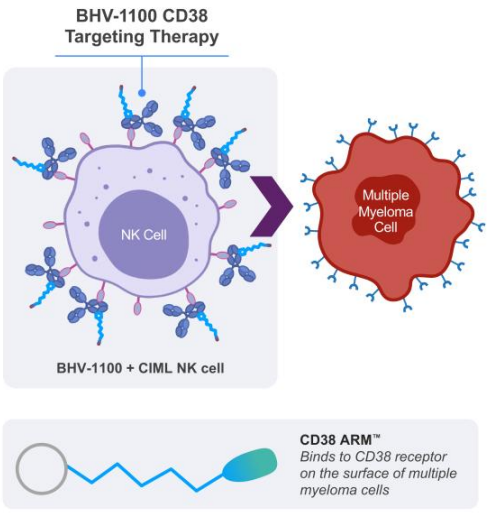
- Pan-IgG degrader (BHV-1300) in nonclinical development with anticipated 2023 IND
  - Mechanism with potential for “pipeline in a product”
- Highly optimized chemistry, drug delivery potential, and differentiated mechanism of action compared to standard of care
- Following pan-IgG degraders, versatile and novel BHVN bispecifics for precision removal of degalactosylated IgA (pathogenic species of IgA in IgA nephropathy) and removal of other pathogenic proteins including specific autoantibodies currently in late discovery

Bispecific Platform:
CD38 Targeted Cell Therapy for Multiple Myeloma

ARM

CD38 Antibody Recruiting Molecule (ARM™)

- Modular bispecific molecules with two moieties, each designed for non-covalent binding to a specific target
- Redirect endogenous antibodies to target cancerous or virally infected cells for immune destruction



Key Potential Advantage Over Biologics

- ✓ Lower manufacturing cost
- ✓ More versatile — smaller and tunable
- ✓ Faster and less expensive to develop
- ✓ Better safety and efficacy
  - Non-immunogenic; better dosing
  - Enhanced PK properties
  - Reduced NK cell fratricide compared to daratumumab

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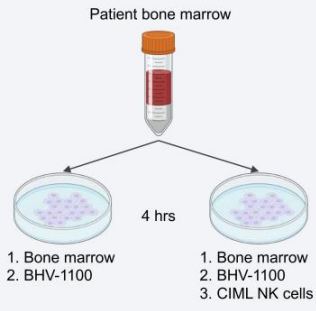
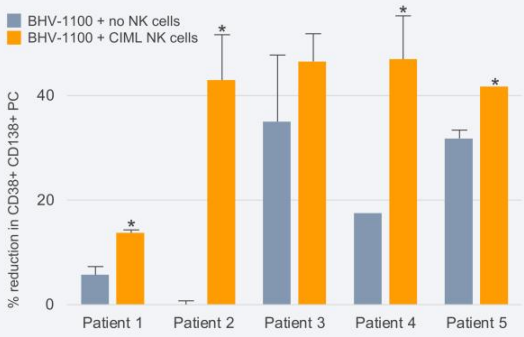
Ex Vivo Patient Study: 4 Out of 5 Patients Showed Reduction in Plasma Cells (Multiple Myeloma)

Using patient bone marrow and a combination therapy



Readout % CD38+ CD138+ plasma cells

% reduction in plasma cells in patient bone marrow +/- autologous CIML NK cells in presence of BHV-1100



ONGOING CLINICAL STUDY

MRD + post-transplant multiple myeloma patients ongoing at Dana Farber Institute

- First patient survival to one year
- Two additional patients randomized

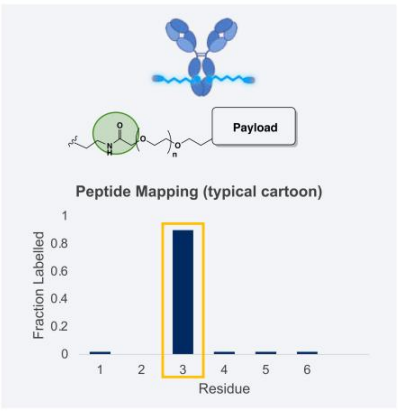
Journal of Clinical Oncology 2020 38:15_suppl, 8523-8523.

Bispecific Platform: Advancing Next-Generation, Site-Specific Antibody Drug Conjugates (ADCs)

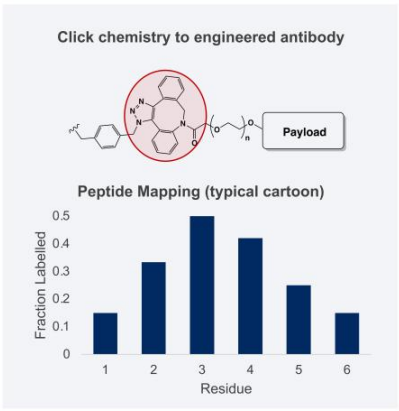
DISCOVERY RESEARCH

Potential for best-in-class

Biohaven third-generation technology



Second-generation ADC-linker technologies



*DAR = Drug antibody ratio

A single residue per heavy chain is available for conjugation

- **Controlled DAR* ratio is critical to therapeutic index**
- **MATE tech precisely defines DAR***

Improved linker stability should yield wider therapeutic index

- **Improved safety:** less systemic, untargeted payload
- **Improved efficacy:** targeted payload delivered to tumor

Uses native antibody: potentially improved CMC vs. current tech

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Capitalization Considerations

CGRP royalties: Pfizer will make royalty payments in low- to mid-teens% in respect of annual U.S. net sales of rimegepant and zavegepant >$5.25B, subject to annual cap ($400M/year)[3]

1. Excludes 9.1 million options ($7.00 strike price). 2. As of December 31, 2022. 3. Cap reached if aggregate annual U.S. 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.*

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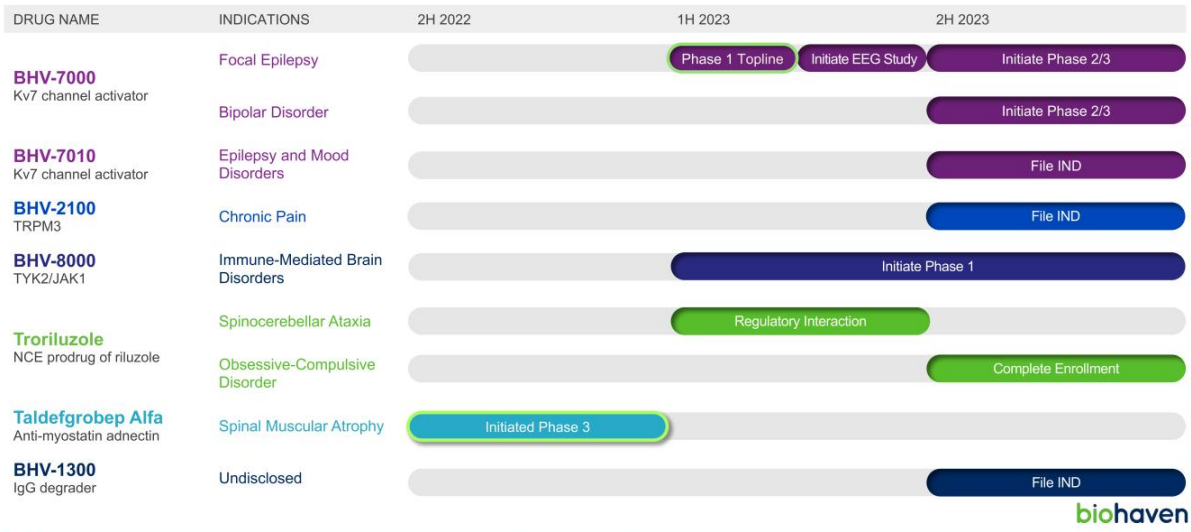
Near-Term Milestones (Anticipated)

Milestone achieved



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HIGH VALUE PLATFORMS
Pursuing novel paths of science
INNOVATIVE PORTFOLIO
A broad therapeutic portfolio
PROVEN BUSINESS FORMULA
Formula for continued growth
BIOHAVEN TODAY