bridgebio

hope through
rigorous science

J.P. Morgan Presentation

January 12, 2026

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Thank you to our investors for enabling us to deliver hope and medicines to the patients that we serve

> 8,500
patients impacted by our therapies

Attruby™ (acoramidis) 356 mg tablet | Nulibry® (fosdenopterin) for injection | TRUSELTIQ™ (infigratinib) capsules

Obtained Approval for 3 Medicines

2 Positive Phase 3 Results

fortify ✓ | CALIBRATE ✓ | PROPEL3 | CANaspire

+ 2 Ongoing Pivotal Trials

15 active trials in ecosystem	**> 70** papers published	**> 35** academic partnerships	**19** INDs created	**< $40M** spend to proof-of-concept data

Fortify results reflect interim analysis data. BridgeBio does not commercialize Nulibry but receives royalties. Truseltiq was withdrawn from the market voluntarily after receiving FDA approval.

Today we will review commercial/late-stage programs and early-stage progress

- Attruby revenue
- Late-stage data and regulatory progress
- Early-stage research and our potentially best-in-class EPP program

NEW INFORMATION

Continued Attruby commercial momentum

$146M[1]

Q4 2025 Net Product Revenue

[1]Represents preliminary, unaudited results for the fourth quarter ended December 31, 2025, based on management's current expectations and subject to completion of year end audit procedures. See Forward Looking Statements and Disclaimer on slide 2 regarding risks and uncertainties that could cause actual results to differ.

NEW INFORMATION

Continued Attruby commercial momentum and patient impact

6,629

Unique U.S. patient prescriptions for Attruby

1,632

Unique U.S. prescribing HCPs

>25%

Estimated share of NBRx for Attruby

As of December 31st, 2025

We continue to study the impact of Attruby across clinical dimensions

First and ONLY…

…approved product with a label specifying **near-complete stabilization** of TTR

1 month

Reduction in cumulative cardiovascular outcomes **within the first month** of treatment in patients with ATTR-CM

42%

Reduction in **composite of all-cause mortality** and **recurrent cardiovascular-related hospitalization** events at Month 30

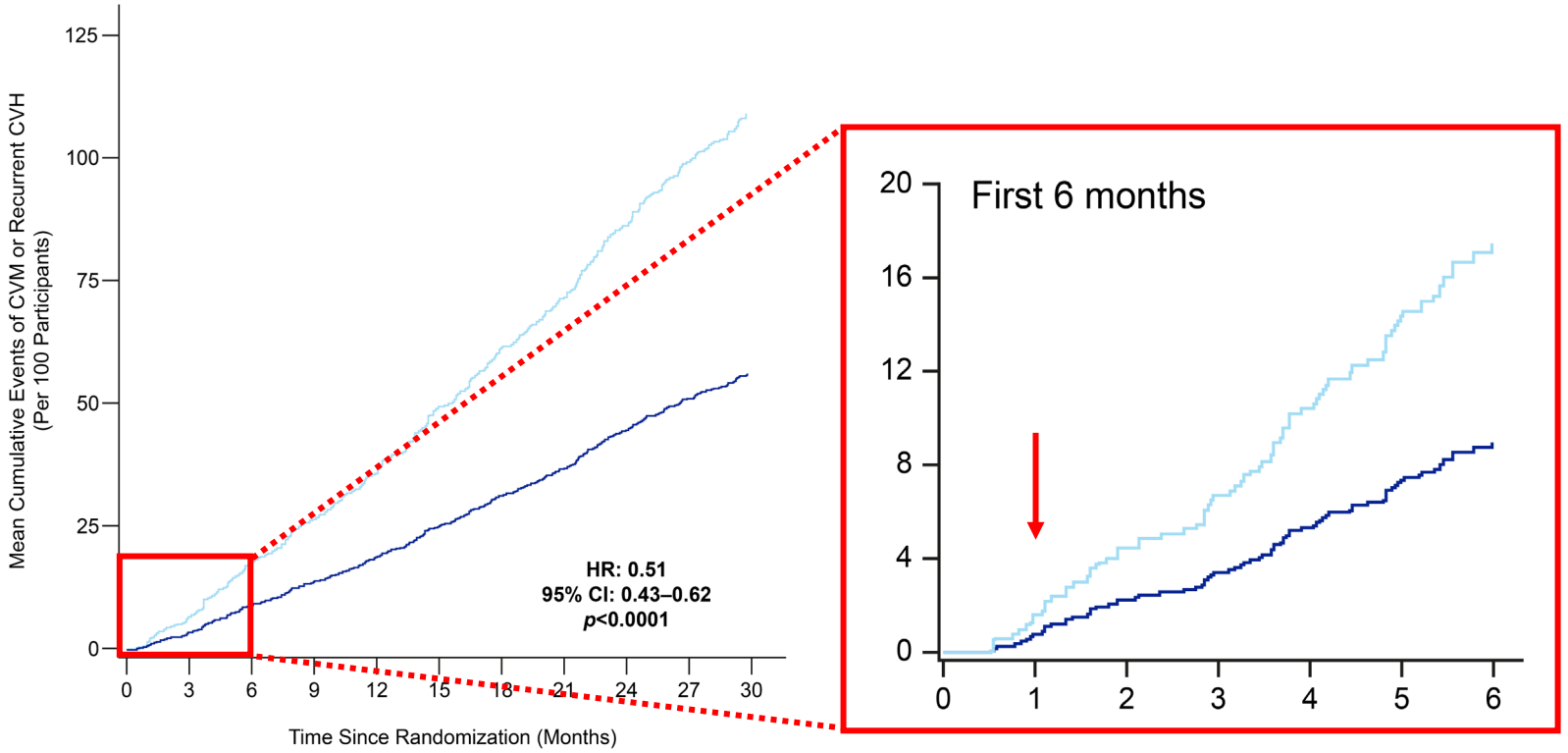
50%

Reduction in the **cumulative frequency** of **cardiovascular-related hospitalization** events at Month 30

Attruby significantly reduced the risk of CVM or recurrent CVH through Month 30 vs. placebo by 49% with separation of curves starting by Month 1

Estimated Mean Cumulative Events of CVM or Recurrent CVH Through Month 30

(mITT Population, Acoramidis, n = 409; Placebo, n = 202)



	Acoramidis (n = 409)	Placebo (n = 202)
Participants with CVM or recurrent CVH, n (%)	136 (33.3)	98 (48.5)
Hazard ratio (95% CI)[a]	**0.51** (0.43, 0.62)	
***p* value**	< 0.0001	

Source: Masri et al. (2025) Early, Long-Term Reduction in CV-Outcomes With Acoramidis, JACC; HFSA 2025 Presentation (Masri) Acoramidis Reduces Cumulative Cardiovascular Outcomes Within the First Month of Treatment in Transthyretin Amyloid Cardiomyopathy: Results From ATTRibute-CM

Data are for the mITT population in the ATTRibute-CM study, defined as all the participants who had undergone randomization, received at least one dose of acoramidis or placebo, and had at least one efficacy evaluation after baseline; participants with eGFR < 30 mL/min/1.73 m2 were excluded

aModified Andersen-Gill model with a robust variance estimator, with treatment, age, NYHA class, genotype, eGFR, and log-transformed baseline NT-proBNP as covariates

CI, confidence interval; CVH, cardiovascular-related hospitalization; CVM, cardiovascular mortality; eGFR, estimated glomerular filtration rate; mITT, modified intention-to-treat; NT-proBNP; N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association

Attruby's advantages stem from the fact that it is the most potent stabilizer

Acoramidis is the only stabilizer with "near complete" stabilization in the label

- ✓ **Sees more target** **(superior free fraction)**
- ✓ **Binds more target** **(superior kd2)**
- ✓ **Glues the target together stronger** **(enthalpic binding mode)**

The multiplicity of advantages to superior stabilization continue to be better understood and elucidated in novel research

RESEARCH ARTICLE | BIOCHEMISTRY

Mass spectrometry footprinting reveals how kinetic stabilizers counteract transthyretin dynamics altered by pathogenic mutations

Francisca Pinheiro, Ravi Kant, Saketh Chemuru, +5, and Salvador Ventura

Edited by Jeffery Kelly, Scripps Research, La Jolla, CA; received July 25, 2025; accepted November 19, 2025

December 31, 2025 | 123 (1) e2519908122 | https://doi.org/10.1073/pnas.2519908122

"Our thermodynamic analysis further supports the notion that binding enthalpy (ΔH), not affinity (Kd or ΔG), better predicts the conformational stabilization imparted by kinetic stabilizers…. These results underscore the need to prioritize enthalpy-driven interactions during stabilizer design."

"Given the variability in stoichiometry in the experiments between tafamidis and AG10 and TTR, **the data always tell the same story, that AG10 is better than tafamidis** as would be expected from the determined binding constants."

– Prof. Jeffery Kelly (inventor of tafamidis) in email correspondence with Dr. Isabella Graef, February 12, 2013. Bold added.

Nelson et al., Amyloid 2020; 28(1), 24–29. Miller, M. et al. J Med Chem. 2018;61:7862-7876. Pinheiro, F. et al. Proc Natl Acad Sci U.S.A. 2026; 123(1) e2519908122. AG10 is the former internal development code for Attruby/Beyonttra (acoramidis).

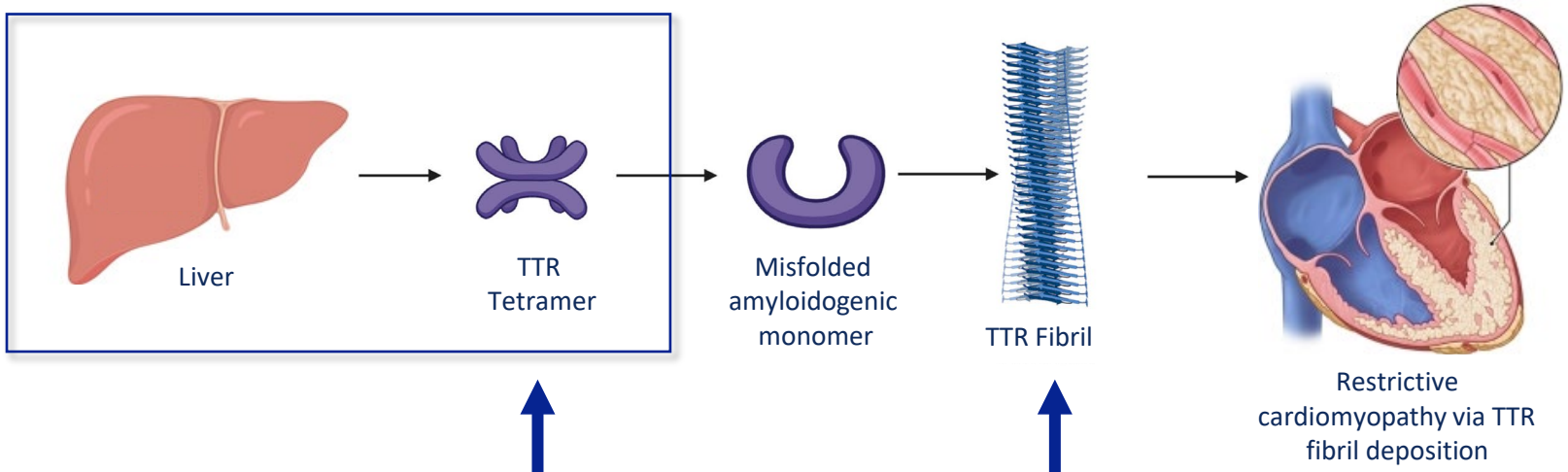
Along with work on Attruby, BridgeBio has initiated a depleter program to explore the potential of ATTR-CM disease reversal

NEW PROGRAM

ATTR-CM Disease Pathophysiology

Existing TTR therapies target upstream tetramer stabilization and synthesis…

…but unmet need remains for patients with existing amyloid deposits for downstream clearance



Attruby

Near-complete stabilizer that prevents dissociation of TTR tetramers into monomers

Depleter

Monoclonal antibody that clears existing amyloid deposits via macrophage-mediated phagocytosis

Led by Renowned Antibody Expert

Dr. Richard Scheller

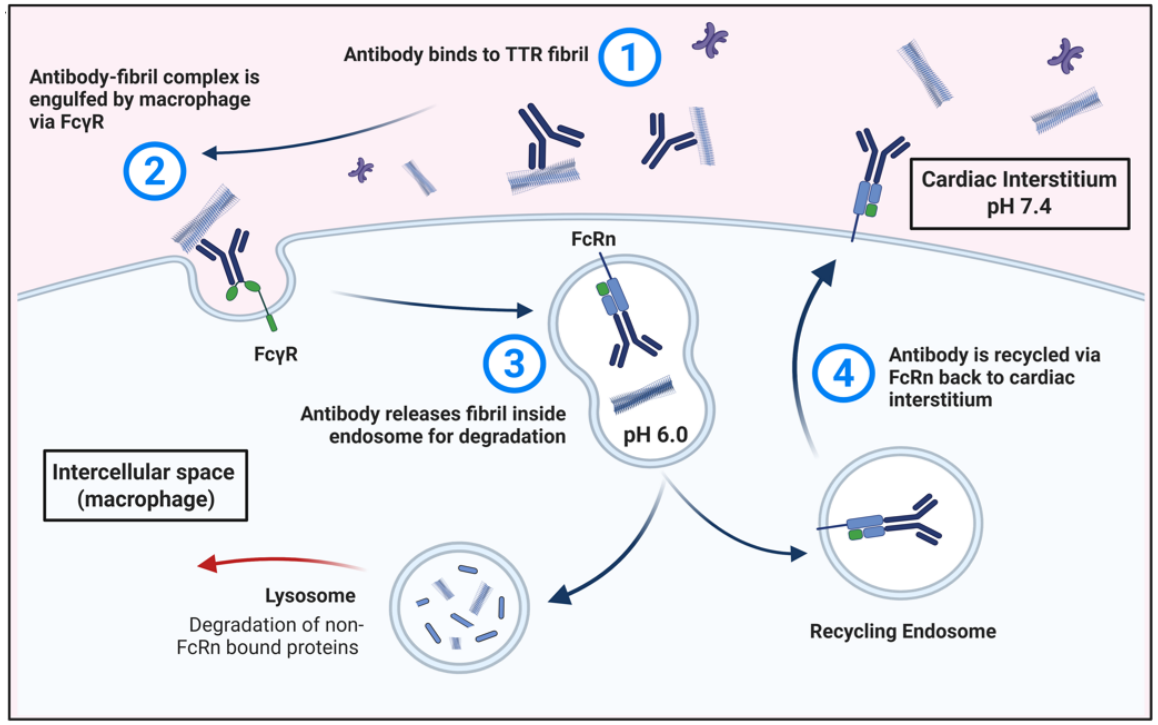
- Former Professor, Stanford
- Former CSO, Genentech
- Former EVP and Executive Committee, Roche Genentech
- Chairman of R&D, BridgeBio

Source: Wanniarachchige et al., 2025

BridgeBio's depleter is engineered across 4 novel properties for potential differentiation on clinical efficacy and dosing convenience

NEW PROGRAM

Depleter Mechanism of Action



BridgeBio's Differentiated Target Properties

1. **Improved fibril:tetramer binding ratio**
   - >10× preferential binding to misfolded TTR fibrils vs. native TTR tetramers

   ➔ **Binds more target**

2. **Faster macrophage recruitment**
   - First depleter to activate Fcγ receptors to boost macrophage activity

   ➔ **Clears more target**

3. **pH sensitivity**
   - Intentionally designed for pH-dependent antigen release inside macrophages

   ➔ **Extends antibody half-life**

4. **Half-life extension**
   - First depleter engineered for enhanced FcRn binding

   ➔ **Extends antibody half-life**

Program expected to advance into the clinic in 2027–2028

Limitations of first-generation depleters highlight the opportunity for a next-generation depleter to better serve patient needs

NEW PROGRAM

		Competitors		BridgeBio	
	Property	Company A	Company B	BridgeBio's Depleter	Why it Matters
1	Improved fibril:tetramer binding ratio	✗	Limited	✓ >10× preferential binding to misfolded TTR fibrils vs. native TTR tetramers	• Maximizes on-target engagement with misfolded TTR fibrils • Minimizes unintended clearance of physiologic TTR tetramers
2	Faster macrophage recruitment	✗	✗	✓ First depleter to activate Fcγ receptors to boost macrophage activity	• Accelerates amyloid clearance • Potentially enables earlier time to separation on clinical endpoints
3	pH sensitivity	Limited	✗	✓ Intentionally designed for pH-dependent antigen release inside macrophages	• Enables antibody recycling after phagocytosis
4	Half-life extension	✗	✗	✓ First depleter engineered for enhanced FcRn binding	• Extends circulating half-life • Enables more convenient dosing vs. monthly IV infusions

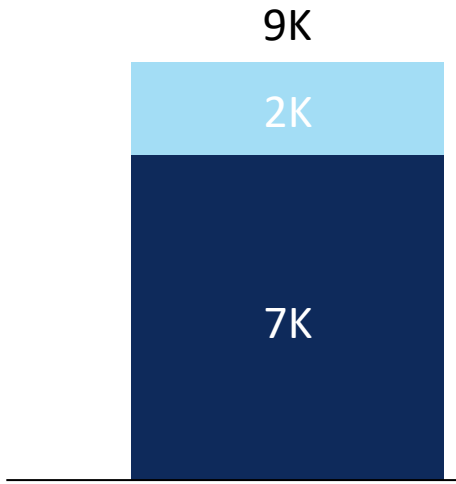
Note: Green = favorable; orange = unfavorable; grey = in-between.

BBP-418

Status: Positive Phase 3 Interim Result in LGMD2I/R9

Addressable people by indication

(current population with addressable mutations)



Fukuyama congenital muscular dystrophy *(Japan)*

LGMD2I/R9[1,2] *(US + EU)*

(Phase 3 fully enrolled and positive interim analysis topline announced as of Oct. 2025)

Analogous markets to LGMD2I/R9

Product	Vyjuvek® beremagene geperpavec-svdt	EXONDYS 51 (eteplirsen) Injection	SKYCLARYS® (omaveloxolone)
Indication	Dystrophic Epidermolysis Bullosa	DMD w/ amenable exon 51 mutations	Friedreich's Ataxia
Prevalence (US)	~4K[3]	~1.3-2K[4]	~4-5K[5]
Projected peak year sales (US)[6] / Market cap	$640M / $7.6B	$541M / $2.5B	$565M / $7.3B[7]

[1] Liu, et al. Genetics in Medicine. 2019; [2] Includes all patients with potentially treatable mutations in FKRP, FKTN, and ISPD; [3] Eichstadt et al Clin Cosmet Investig Dermatol; [4] Cure Duchenne and Sarepta; [5] MDA and Friedreich's Ataxia Research Alliance; [6] Evaluate; [7] M&A value of Reata

Unprecedented, clinically meaningful improvement across all pre-specified endpoints and well-tolerated safety profile at interim analysis

MET PRIMARY AND ALL KEY SECONDARY ENDPOINTS VS. PLACEBO

1.8x
increase in glycosylated αDG at 3 months
(p<0.0001)

82%
decrease in CK at 12 months
(p<0.0001)

Faster ambulation
compared to deterioration on placebo
(p<0.0001)

Improved pulmonary function
compared to deterioration on placebo
(p=0.0071)

Well tolerated
safety profile
Low discontinuation rate; higher in PBO arm

An appreciable share of patients achieved normal levels on some measures in just 1 year

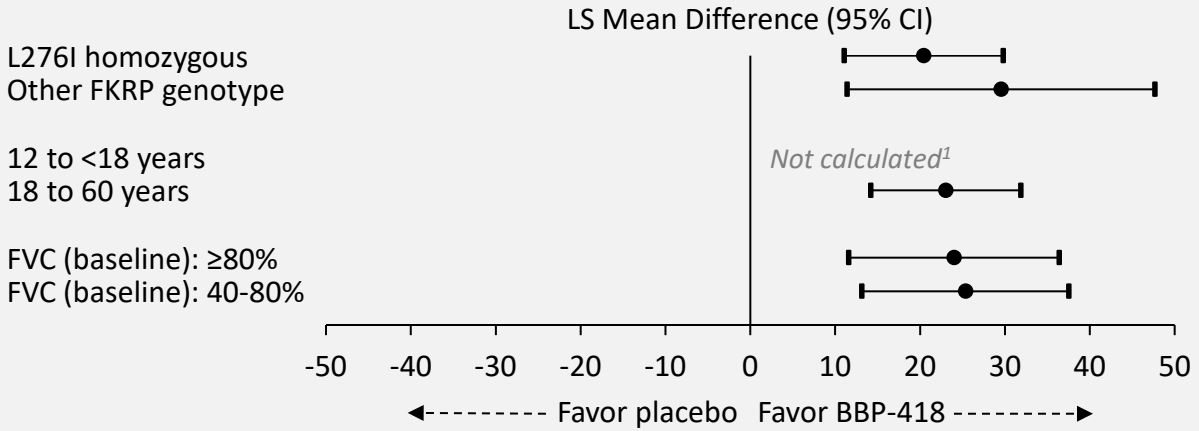
αDG = alpha dystroglycan; PBO = placebo; CK = creatine kinase
Source: Data on file

Planned subgroup analyses show consistent benefit of BBP-418 vs. placebo in all subgroups across α-controlled efficacy endpoints at 12 months

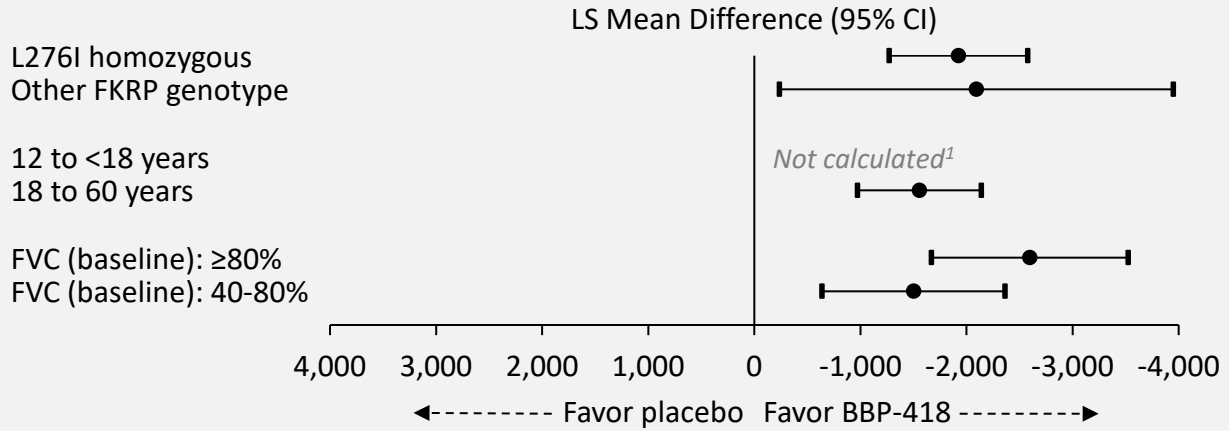
NEW INFORMATION

Biomarker

Glycosylated αDG (% of control)

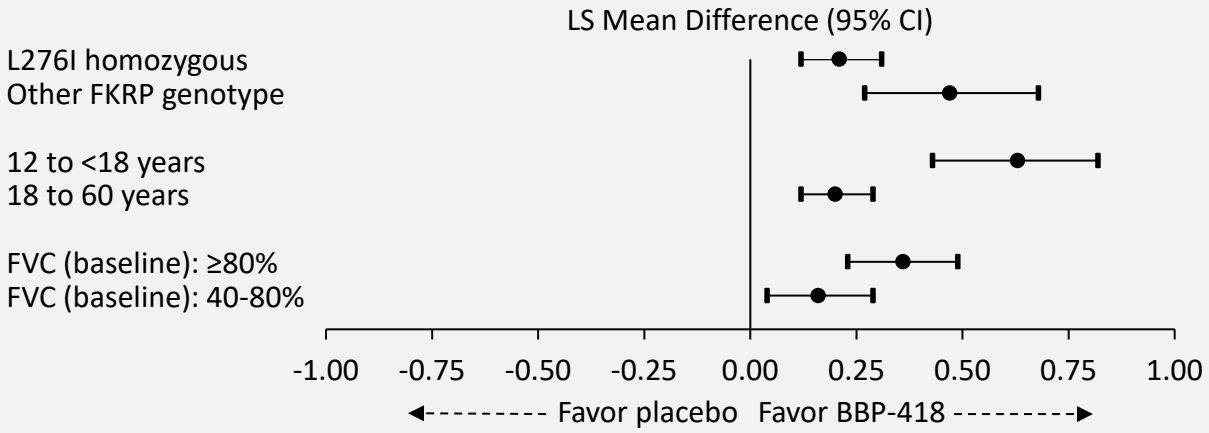


Serum creatine kinase (U/L)

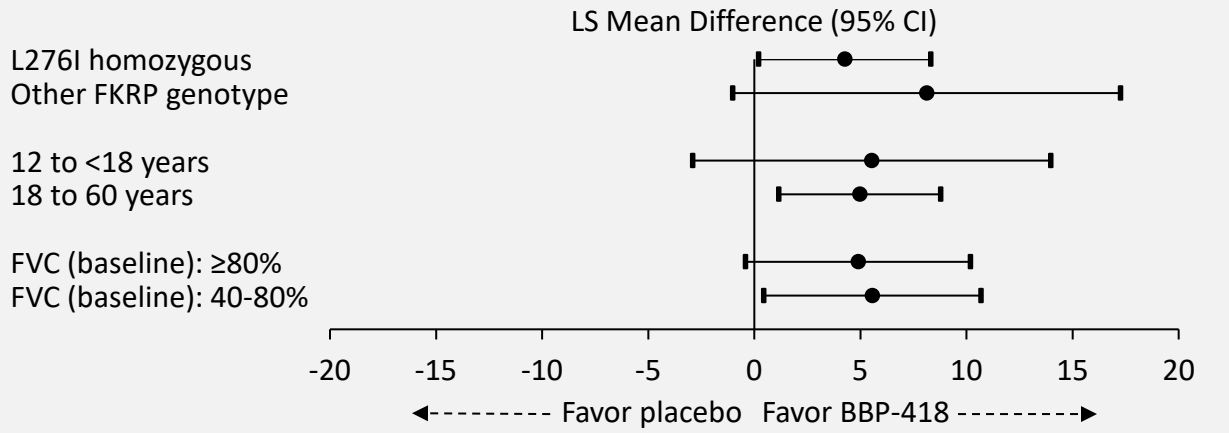


Functional

100-meter timed test (m/s)



Forced vital capacity (% predicted)



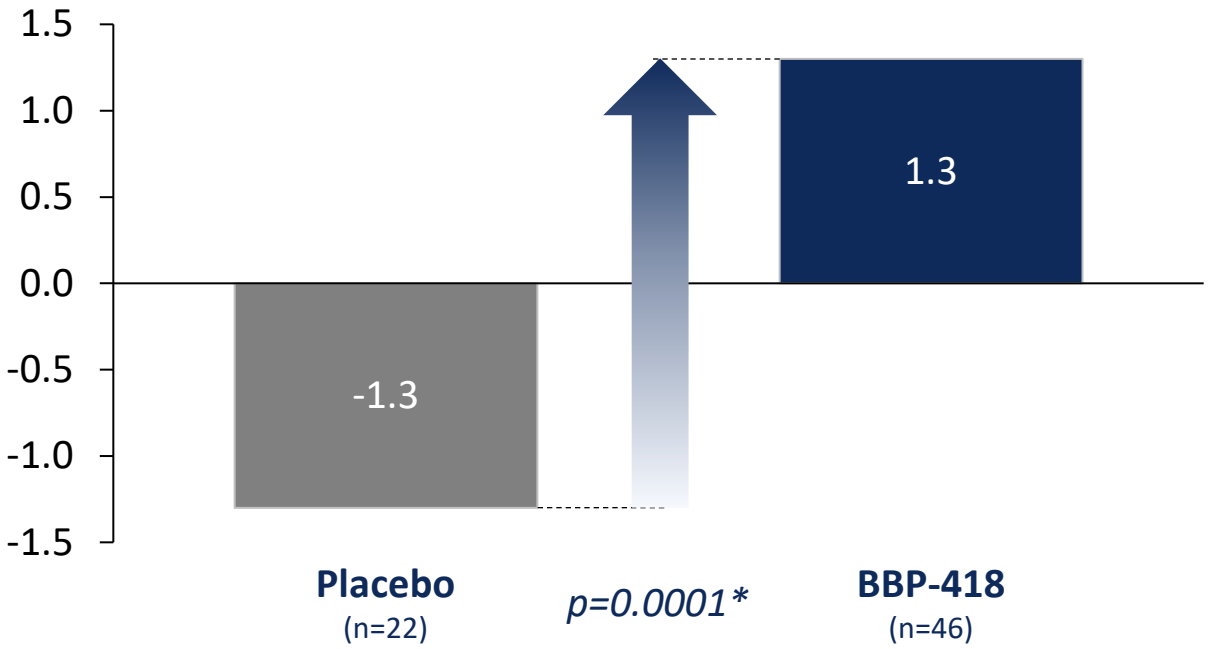
Note: [1] Pre-specified inferential statistics not performed for subgroups representing <15% of all participants in relevant analysis set; αDG=alpha dystroglycan; PBO=placebo; FKRP= Fukutin Related Protein; FVC=forced vital capacity. Source: Data on file

BBP-418 treated patients experienced highly clinically meaningful 2.6 point benefit on NSAD relative to placebo even at early 12-month timepoint

NEW INFORMATION

Improved gross motor function

Change from baseline in NSAD (points)



Even a 1-point difference in NSAD can mean…

Requiring someone's help to get up	Able to get up from a fall
Requiring assistance or mobility aids	Able to use stairs or steps
Asking for help to get up from toilet	Able to toilet independently

NSAD *(primary endpoint at 36 months)* benefit is highly clinically meaningful even at 12 months

** Nominally statistically significant based on exploratory analysis; analysis not part of alpha-controlled hierarchy at interim analysis*

NSAD = North Star Assessment for Limb-Girdle Type Muscular Dystrophies; Least-Squares Mean Change from Baseline at 12-month timepoint
Source: Data on file.

NEW INFORMATION

We completed a successful meeting with the FDA and they recommended orienting our NDA toward traditional and full approval

- All data from the Phase 3 FORTIFY study were presented to the agency, including key sensitivities
- FDA acknowledged the data "…demonstrate consistent treatment effects on multiple efficacy endpoints"
- FDA recommends orienting NDA toward traditional approval

We anticipate filing an NDA with the FDA in 1H 2026

Encaleret

Status: Positive Phase 3 Result in ADH1

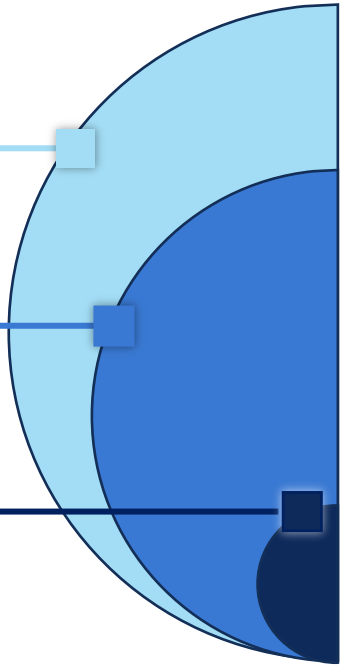
Addressable people by indication in US

(current population with ADH1)

12K Individuals with ADH1
~12K ADH1 prevalent population in the US based on 4 population databases[1-2]

9K Symptomatic
73% of patients with ADH1 are symptomatic[3]

3K – 5K Currently Addressable
40-60% of symptomatic ADH1 patients are estimated to be diagnosed today; anticipate increase



An analogous market to ADH1 is XLH

(X-linked Hypophosphatemia)

	XLH	ADH1
Prevalence (US)	12K[4]	12K
Disease burden	Hypophosphatemia	Acute - hypocalcemia Chronic - hypercalciuria
Standard of care	Vitamin D, daily phosphate[5]	Vitamin D, daily calcium
Registrational endpoint	Serum phosphate	Serum and urine calcium
Projected peak year sales	$2B+[6]	$1B+

[1]Dershem, J. et al. Amer. J. Hum. Genetics, 2020. [2] Chang, J.B. et al. Amer. J. Hum. Genetics, 2025. [3]Roszko, K.L. et al. JBMR, 2022.
[4]Dahir, K. et al. J. Endocr. Soc., 2020. [5]Munns, C.F. et al., JBMR Plus., 2023. [6]Evaluate Pharma.

CALIBRATE achieved & exceeded all criteria set forth as an upside target, with a 76% responder rate following 24 weeks of encaleret treatment

Upside Target Clinical Profile	Outcome Observed
Statistically significant primary analysis result compared to conventional therapy	**Primary endpoint met (p <0.0001) demonstrating superiority to conventional therapy**
At Week 24, ≥50% of study participants achieve target serum and urine Ca on encaleret	**76% (34 out of 45) achieved target serum and urine Ca on encaleret vs. 4% on conventional therapy**
Majority of participants randomized to encaleret able to remain independent from conventional therapy[1]	**Among encaleret responders at Week 24, none required conventional therapy during Period 3[1]**
At Week 24, mean iPTH within normal range on encaleret	**>90% of participants administered encaleret achieved iPTH above the lower limit of the reference range**
Comparable safety and tolerability profile to conventional therapy	**Encaleret was well-tolerated; no discontinuations related to study drug**

[1]Requirement for conventional therapy defined as oral calcium >600 mg/day and/or active vitamin D during Period 3.
Ca = Calcium; iPTH = Intact Parathyroid Hormone. Encaleret is an investigational drug. Its safety and efficacy have not been fully evaluated by any regulatory authority.

>90% of CALIBRATE participants administered encaleret demonstrated a pharmacologic response

Primary Analysis – Within Group	Week 4 SoC (N=45)	Week 24 Encaleret (N=45)	p-value[3]
Number of Participants Meeting The Primary Endpoint (Responder status)[1,2]	2	34	
Proportion, %	4%	76%	
Difference in Proportion of Responders (95% CI)	71% (58%, 84%)		<0.0001

Key Secondary Analysis – Within Group	Week 4 SoC (N=45)	Week 24 Encaleret (N=45)	p-value[3]
Number of Participants With iPTH ≥ LL Reference Range	3	41	
Proportion, %	7%	91%	
Difference in Proportion of Responders (95% CI)	84% (74%, 95%)		<0.0001

[1]The primary endpoint assessed responder status of participants who achieved both corrected serum calcium and 24-hour urine calcium in the target range at the completion of the maintenance periods.
[2]Participants randomized to receive encaleret who required doses of elemental calcium >600 mg/day for >7 days during Period 3 were evaluated as non-responders. [3]Analyzed by McNemar's test.
CI = Confidence Interval; iPTH = Intact Parathyroid Hormone; LL = Lower Limit

BridgeBio is pioneering efforts to enable the successful launch of encaleret, potentially the first calcilytic molecule to be approved for any condition

NEW INFORMATION

Expanded availability of testing through sponsored genetic testing program

Detect Hypopara

BridgeBio sponsored genetic testing available for providers & patients at no cost

Enabled creation of new ICD-10 code dedicated to ADH1 & ADH2

E20.810

Billable ICD-10 code associated with ADH1 & ADH2

Enabled update to treatment guidelines specifying need for genetic testing[1]

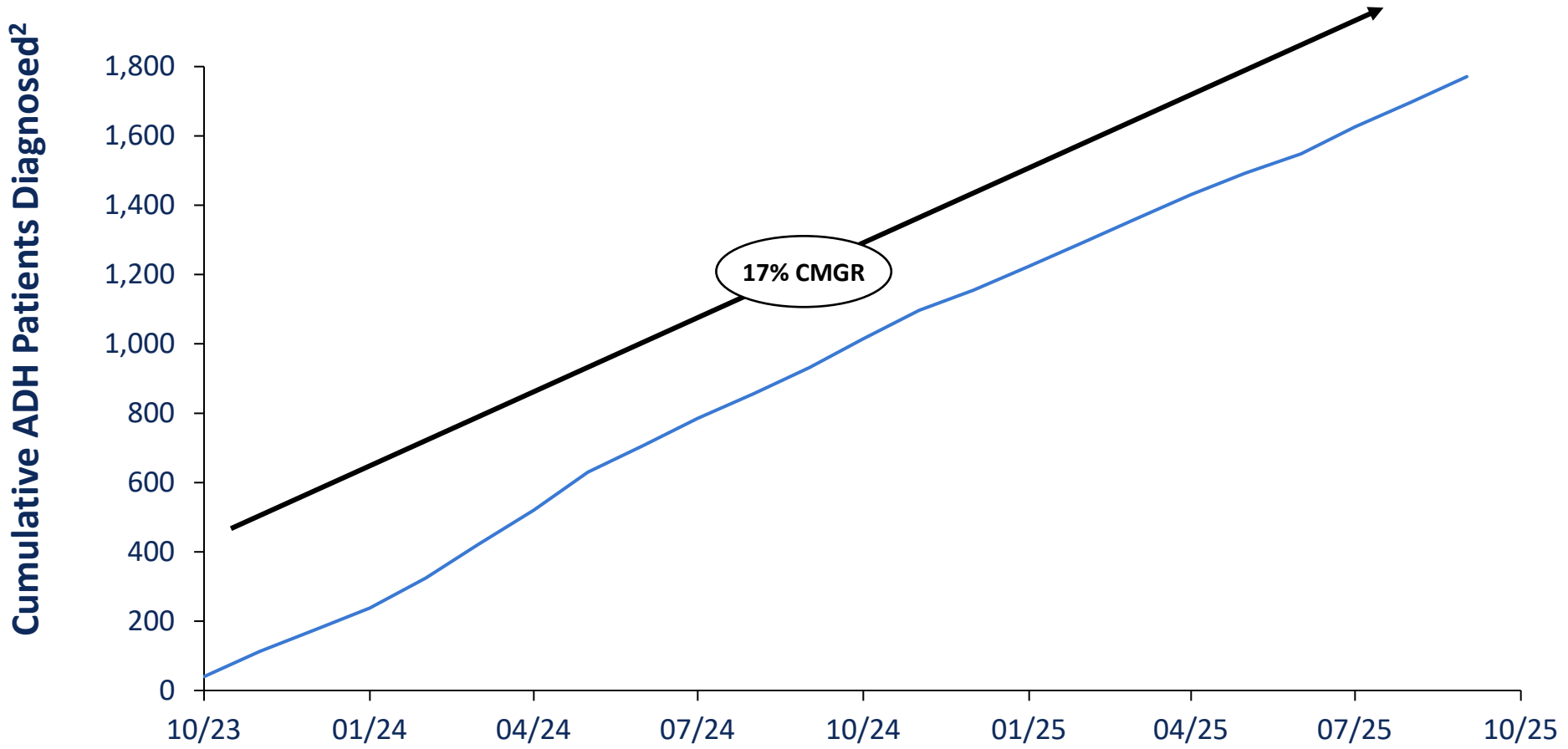
"We recommend genetic testing and/or family screening in a patient with nonsurgical HypoPT without other obvious aetiology."

Encaleret has a first-to-market opportunity to potentially establish a new standard of care in ADH1 while benefiting from ecosystem tailwinds supporting patient identification

[1]Included in updated best practice recommendations for diagnosis and management of hypoparathyroidism (Khan, A. A. et al., Metabolism, 2025.) and European Society of Endocrinology (ESE) treatment guidelines (Bollerslev, J. et al., Eur. J. Endocrinol., 2025.)

Over a 24-month period, >1,700 unique patients were diagnosed with ADH in the US

NEW INFORMATION



~50% of HCPs diagnosing ADH patients manage ≥5 non-surgical hypoparathyroidism patients[3]

[1]Compounded Monthly Growth Rate (CMGR) since the code's introduction in Oct. 2023. [2]Komodo Claims Data, October 2023-September 2025. ICD10 Code E20.810 (Autosomal Dominant Hypocalcemia). [3]Based on analysis of Symphony and Definitive Healthcare Claims Data for ICD-10 Code E20.810.

Encaleret has the potential to be an orally administered option for patients with chronic hypoparathyroidism (CHP)

NEW INFORMATION

Encaleret has the potential to normalize blood and urine calcium in CHP patients

- CHP patients present similarly as ADH1 patients (i.e., hypocalcemia and hypercalciuria)
- Current guidelines specify normalization of blood and urine calcium as therapeutic goals[1,2]
- In a Phase 2 study (N=10) presented at the ASBMR 2025 meeting, encaleret demonstrated a PTH-independent effect to normalize blood and 24-hour urine calcium in 80% of study participants within 5 days[3]

Announcing the RECLAIM-HP Phase 3 Study of Encaleret in CHP *Initiating in 2026*

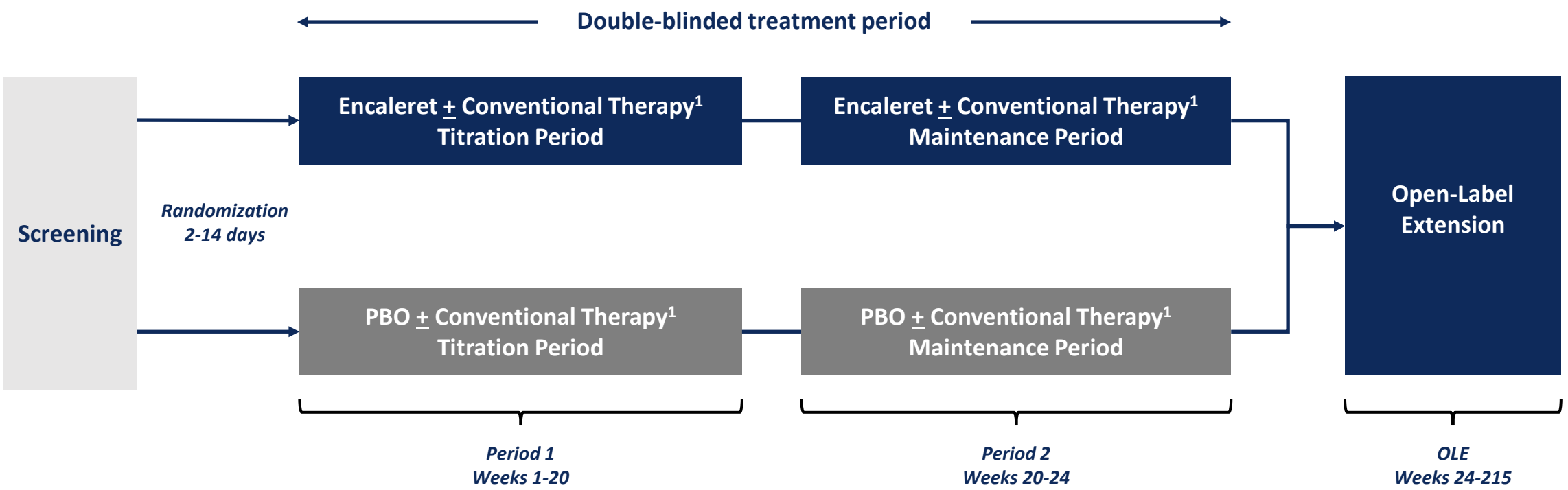
- Completed successful End of Phase 2 interaction with the FDA
- Phase 3 registrational trial to evaluate encaleret in CHP
- Primary endpoint will assess achievement of target blood and urine calcium

[1]Bollerslev, J. et al., Eur Jour of Endocinol., 2025. [2]Khan, A.A. et al., Metabolism, 2025. [3]Hartley, I.R. et al., presented at ASBMR 2025 Annual Meeting.
ASBMR = American Society for Bone and Mineral Research

RECLAIM-HP Phase 3 study of encaleret in chronic hypoparathyroidism to initiate in Summer 2026

NEW INFORMATION

RECLAIM-HP: Global, multi-center, randomized, double-blind, placebo-controlled study



Primary Endpoint Proportion of participants achieving albumin-corrected blood and urine calcium within target range

[1]Conventional therapy includes a combination of oral activated vitamin D and/or calcium supplements
PBO = Placebo

Infigratinib

Status: LPLV in Phase 3 in achondroplasia

55,000 individuals with achondroplasia in US/EU

Represents diagnosed and addressable ACH population with open growth plates

$5B+ potential global market

[1]CDC birth estimates; EU Eurostats birth estimates; Foreman, et al. Am J Med Genet. 2020.; Bober, et al. Gene Reviews. 2020.; Wenger, et al. Gene Reviews. 2020.; Al-Namman, et al. J Oral Biol Craniofac Res. 2019. [2]Achondroplasia market includes all approved drugs.

We have achieved LPLV on PROPEL 3 and expect topline in Q1, and we have made significant operational progress on expansion opportunities

NEW INFORMATION

PROPEL 3

Pivotal Phase 3 Study in children and adolescents (3-<18 years) with achondroplasia and open epiphyses

Last participant last visit completed; Topline expected Q1 2026

PROPEL I&T

Phase 2 Study In Infants & Toddlers from birth to less than age 3 with achondroplasia

First participant enrolled

ACCEL 2/3

Phase 2 study followed by a Phase 3 study in children and adolescents with hypochondroplasia

Full enrollment completed for Phase 2 portion; data expected 2H 2026

Infigratinib: Defining characteristics of a potentially best-in-class program in the ACH landscape

Designed to target achondroplasia at its genetic source: FGFR3 overactivation
Addresses not just overactivation of the MAPK pathway (chondrocyte hypertrophy), but also STAT1 (chondrocyte proliferation) and all other downstream pathways

Achieved profound efficacy in animal models, beyond just long bone growth
In mouse models of achondroplasia, treatment with infigratinib showed an increase in proximal and distal long bone length (femur +21%, humerus +12%, tibia +33%, ulna 22%, and radius +24%) and foramen magnum area (+17%)[5]

Demonstrated the largest degree of efficacy (across multiple dimensions[1]) across any clinical trial for ACH[2]
1. Mean change from baseline in AHV: +2.51 cm/yr at M12
2. Mean absolute AHV: >6 cm/yr at M12
3. Mean change in height Z-score compared to ACH growth charts: +0.36 SD at M12
4. Mean improvement in upper-to-lower body segment ratio (proportionality): Decrease of 0.12 (P=0.001)

Received the only Breakthrough Designation from the FDA for ACH
Met the regulatory requirement of showing preliminary evidence of substantial improvement over SoC

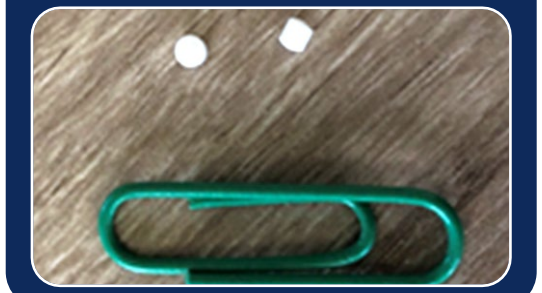
Designed to be taken as a daily oral, avoiding side effects associated with CNPs and repeated injections
Avoids symptomatic hypotension[1], injection site reactions[1], and the psychosocial burden of receiving/administering repeated injections[3,4]

[1]Savarirayan et al, NEJM, 2024; [2]For monotherapy trials in ACH; [3]Antal et al, Journal of Pediatric Psychology, 2011; [4]Jacobse et al, European Journal of Pediatrics, 2019; [5]Komla-Ebri et al, J Clin Invest. 2016.

Infigratinib sprinkle capsules are being developed for oral administration[1,2]

Capsules
(17 mm long*)

Granules
(2 mm long)



- Infigratinib is being studied in children over 3 years of age with achondroplasia (0.25 kg/mg/day) as a sprinkle capsule
- Capsules can be swallowed whole or content (granules) sprinkled on soft food
- The dosage strength of each capsule depends on how many granules are inside
- Each child's dose is based on their weight

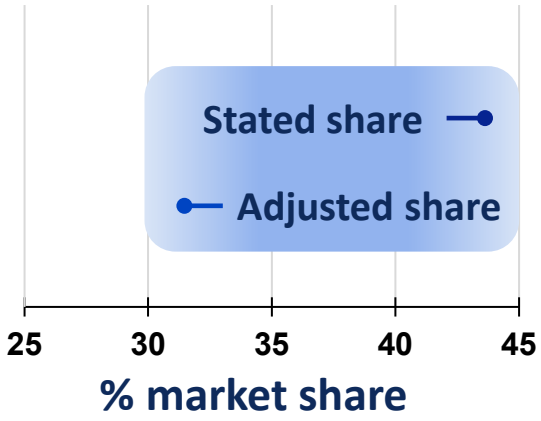
[1]Savarirayan R, et al. N Engl J Med. 2024;392(9):865–874. [2]BridgeBio data on file.
*Infigratinib is an investigational agent that is not approved for use by any regulatory authority. Size 2 capsules are shown in photo.

BridgeBio has developed a validated evidence-based perspective to forecasting market share performance

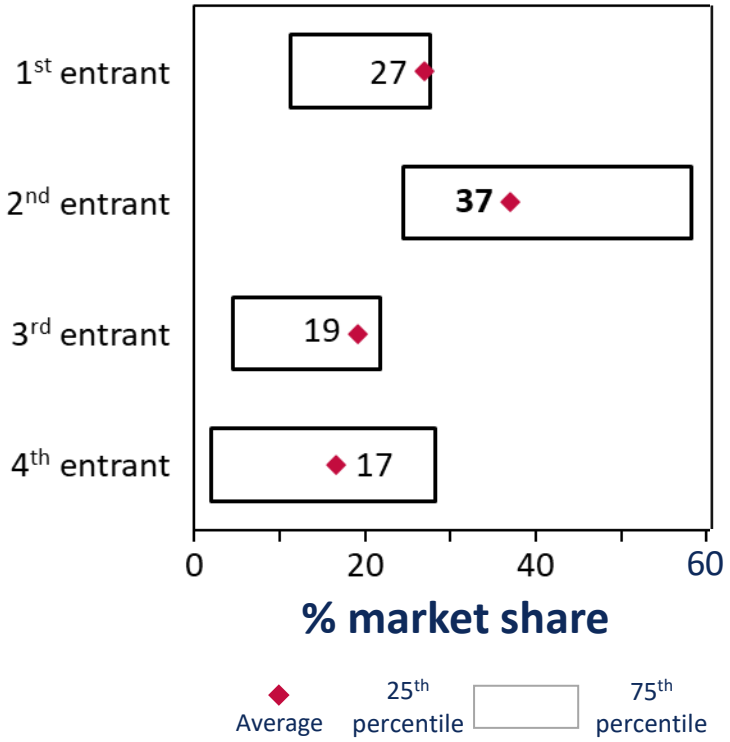
Comprehensive market surveys indicated 30-40%+ Attruby share

Attruby future competitive market share

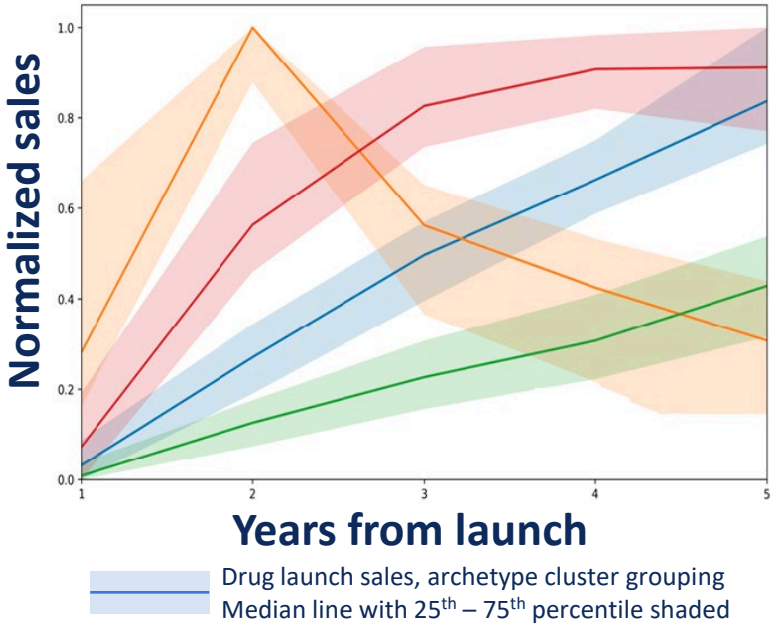
ATTR-CM experienced prescribers
N = 200



Analog research shows 2nd entrants achieve average ~37% share



ML-based empirical analysis enables high fidelity drug launch modeling



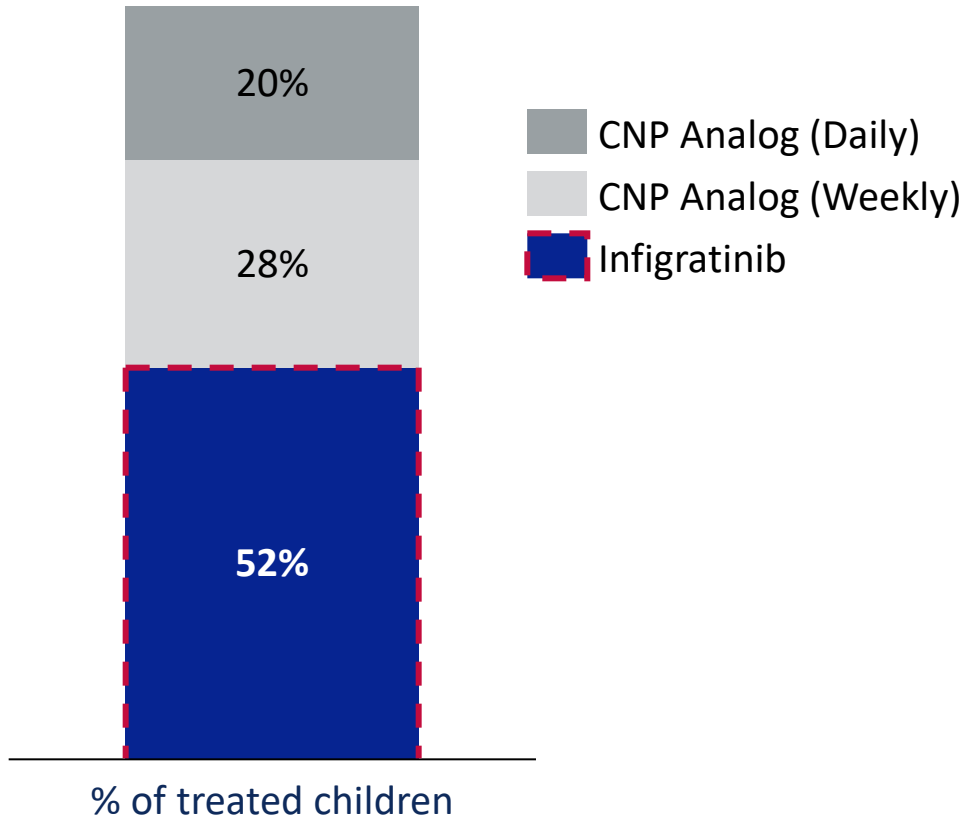
*We have analyzed **>900 drug launches** and built a **proprietary algorithm** capable of categorizing launch profiles into **archetypes** and **predicting future revenue***

Note: Stated share adjusted for historical performance of demand study market research on new product launches.
Source: BridgeBio market research HCP surveys (n = 184 & n=200), Evaluate Pharma, PhAST Rx, Advisory board, IQVIA LAAD data set

Our market research indicates that infigratinib could capture >50% of treated market share, primarily driven by the favorable oral administration and MOA

Attribute	TPP for testing market share
Indication	• Children **(3 – 18 years)** with achondroplasia and open epiphyses
MOA	• **Selective FGFR1-3 tyrosine kinase inhibitor**
Dosing and Administration	• **Once daily capsules** (containing minitablets swallowed whole/chewed/sprinkled on soft foods)
Primary Endpoint	• Statistically significant improvement in change from baseline in annualized height velocity (AHV): **+1.5 cm/year** vs. placebo
Safety & Tolerability	• Well-tolerated AE profile: No injection site reactions or symptomatic hypotension. Less than 10% rate of hyperphosphatemia.

Potential share for ACH children
% of treated children
(N = 95 HCPs; represents ~37% of current market)



[1]BridgeBio market research (Analyses from ACH demand forecast Aug – Oct 2025); % of children; patient-weighted responses from n=95 HCPs; Q: Based on the information you just reviewed, please think about how you might decide to prescribe pharmacological therapy to the next 10 children you see who fall into each of the following clinical scenarios. For each scenario, how many of these children would you expect to prescribe each of the following treatment options?

BBP-812

Status: Pivotal trial ongoing in Canavan disease

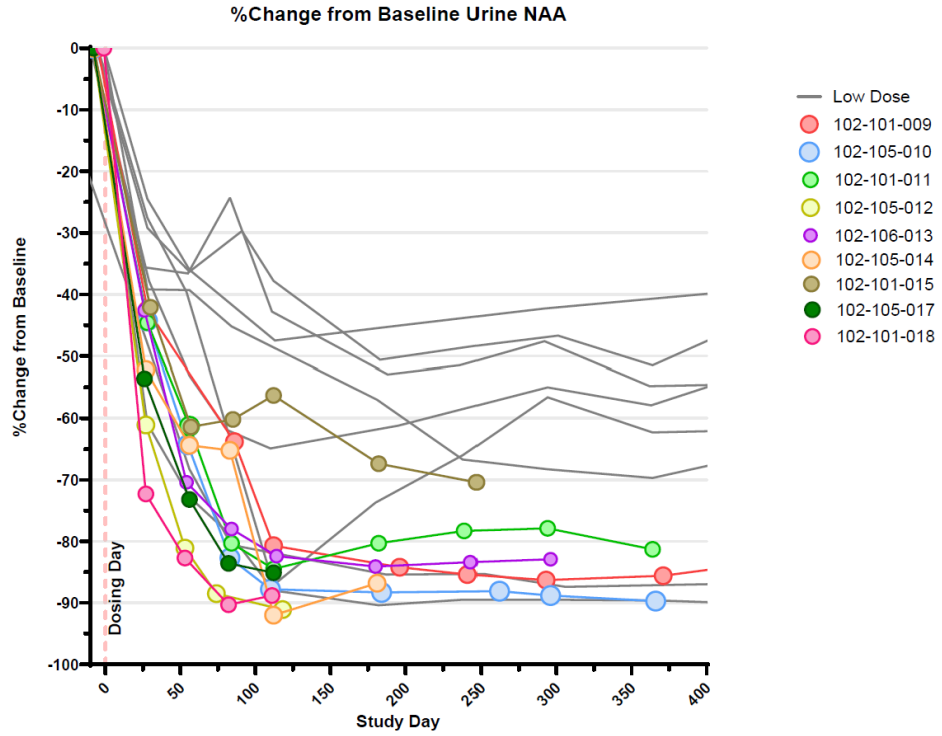
Canavan disease is a fatal, neurodegenerative and ultra-rare pediatric disease with no approved therapies

- Canavan disease (CD) is an ultra-rare neurodegenerative disease with ~1,000 patients across the US and EU
- CD is usually fatal within the first two decades of life, and >25% of patients die by the age of 10 years[1]
- Children with CD exhibit global and severe cognitive, motor, and language impairment, missing or regressing on most developmental milestones
- Children with CD require around the clock care – they cannot hold their heads up, sit, crawl, walk, are generally unable to speak, and suffer from seizures and spasticity
- There are no therapies available for Canavan disease

Current path to a potential BLA filing in 2027 based on reductions in urine NAA (surrogate endpoint) supported by motor function improvements

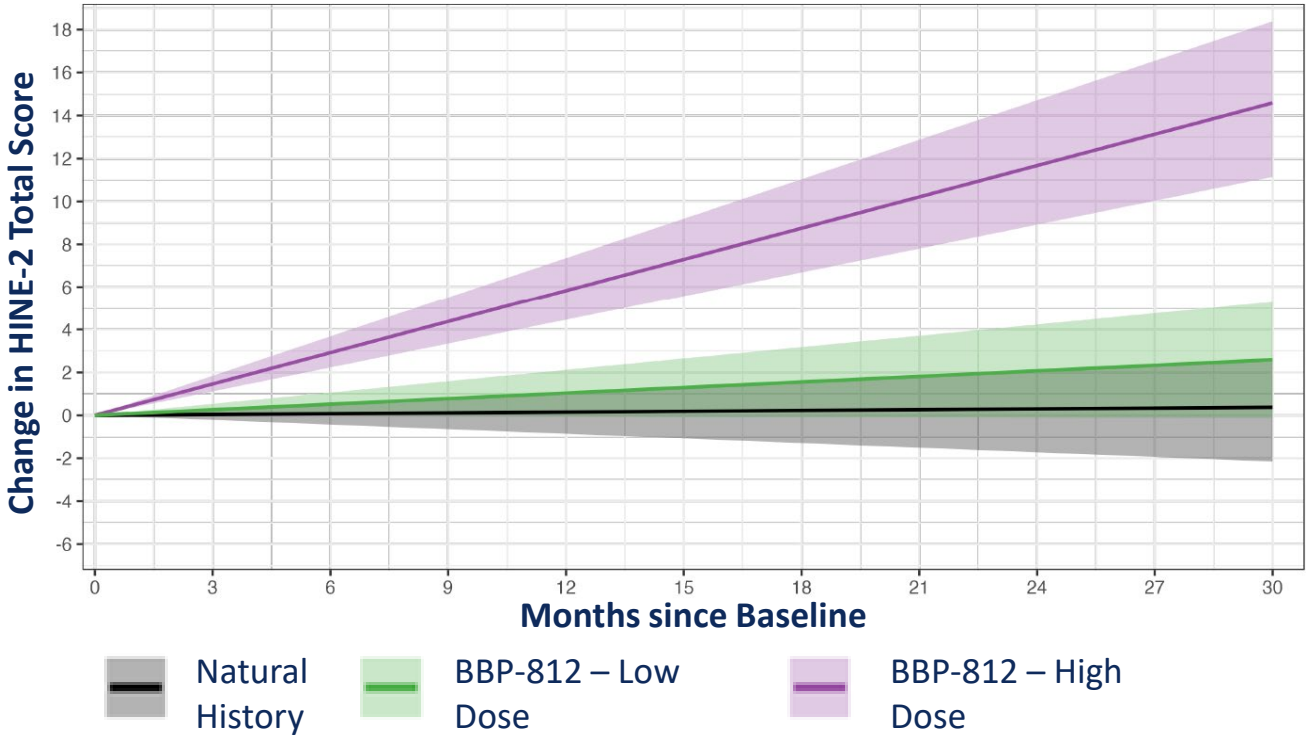
NEW INFORMATION

Urine N-acetylaspartic acid (NAA) levels



- **BBP-812 dose-dependently reduces urine NAA** to levels associated with only mild disease
- **FDA is open to the use of urine NAA as a surrogate endpoint** to support accelerated approval of BBP-812

Hammersmith Infant Neurological Examination (HINE-2) Trajectory



- Trajectory analysis shows **clear, dose-dependent separation in HINE-2 total score** with BBP-812 vs. natural history study
- Children are also showing **improvement on key motor metrics such as sitting, head control, and reaching / grasping**

Source: 10 December 2025 data cut, N = 18

Centralized scale + disease-level focus = ability to have multiple, focused launches

Benefits of centralized commercial model

Build once, scale many
Build commercial capabilities once and leverage across launches

Cost synergies
Leverage fixed costs; Limited incremental additional costs per rare disease launch

Compound launch expertise
Apply cross-launch insights in both competitive and first-in-class markets to systematically de-risk execution

Benefits of execution at the decentralized program level

Deep program expertise
Dedicated affiliate teams stay with programs over time, building deep clinical and community knowledge

Tailored launch strategies
Each program can pursue the right access, positioning, and sequencing approach

Sharper execution
Smaller, specialized markets benefit from tailored approaches

Our future: It’s still day 1 in genetic disease

We are at day 1 of genetic medicine

Today, missing heritability is being explained

"..we show that 12,111 independent SNPs that are significantly associated with height account for nearly all of the common SNP-based heritability."

- Yengo et al, Nature 2022

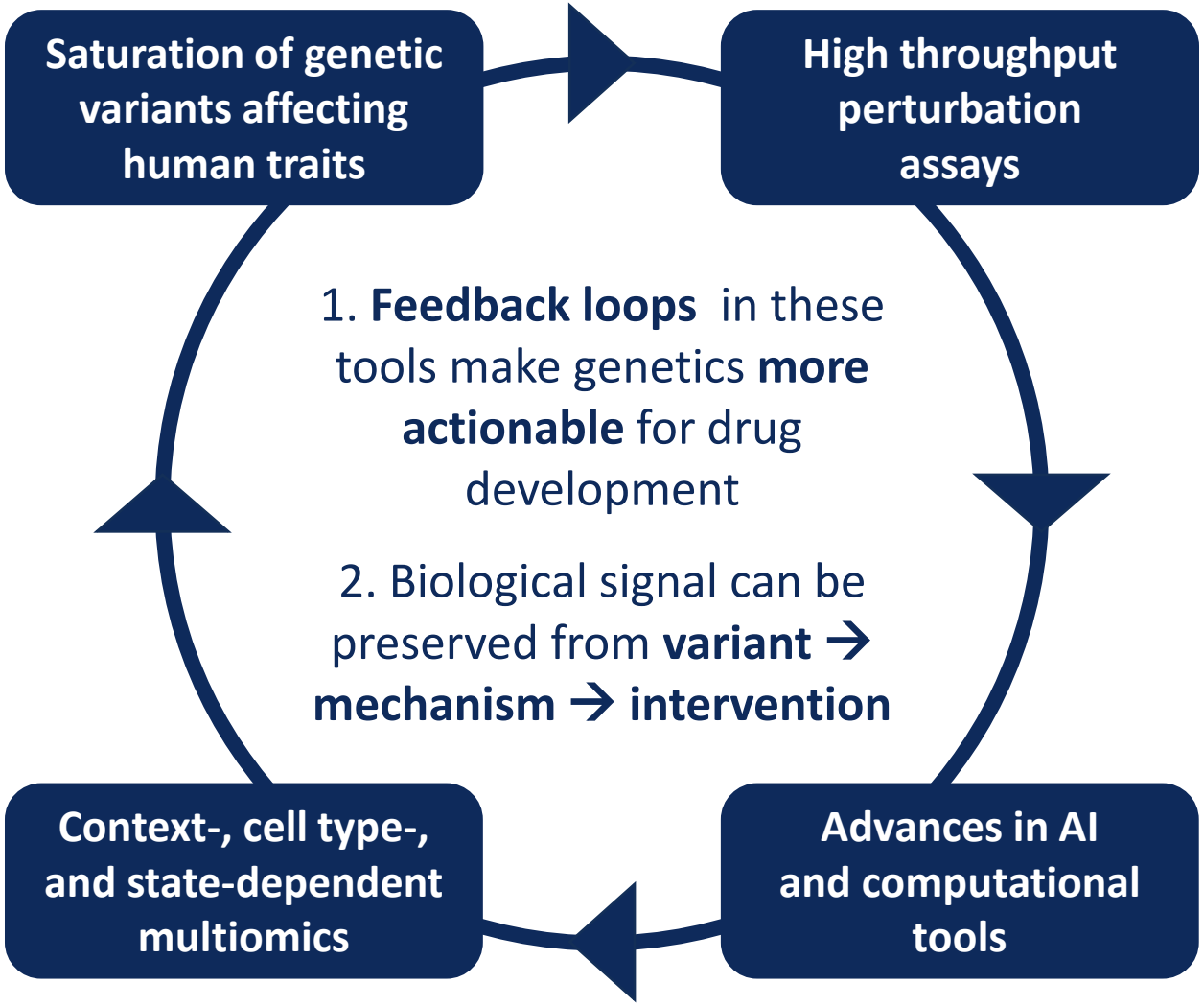
"We identified 15 traits with no significant difference between WGS-based and pedigree-based heritability estimates, suggesting their heritability is fully accounted for by WGS data."

- Wainschtein et al, Nature 2025

Maher Nature 2008; Manolio Nature 2009.

From the missing heritability to the missing mechanism

- Larger Biobanks: UKB, AOU, ADG, etc.
- WGS for non-coding rare variants
- Long-read sequencing for complex SVs



- Perturb-seq to perturb every single gene across the genome
- MAVE to perturb every possible variant within a gene

- Cell atlases: Human Cell Atlas, Tabula Sapiens
- Tissue mapping: HuBMAP
- Large-scale compendia: CellxGENE, TenK10K

- Variant effect prediction: Alphamissense
- Pangenome graphs
- Disease prediction: MILTON

Example #1: The GondolaBio pipeline features a diverse set of programs across therapeutic areas and modalities

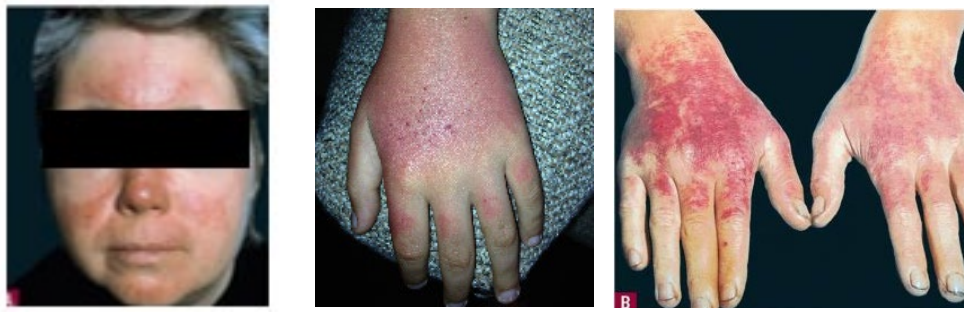
NEW INFORMATION

Indication	Patient Population (US+EU)	Discovery	Lead Op	IND Enabling	Phase 1	Phase 2
Erythropoietic Protoporphyria (EPP)	25k					
Autosomal Dominant Polycystic Kidney Disease (ADPKD)	300k					
Alpha-1 Antitrypsin Deficiency (AATD)	200k					
Charcot-Marie-Tooth 1A (CMT1A)	130k					
Neurofibromatosis Type 1 (NF1)	200k					
Hereditary Pancreatitis	30k					
Fibrous Dysplasia	50k					
Tuberous Sclerosis Complex 1/2 (TSC)	65k					
Genetic Epilepsy Driven by SynGAP1 Mutations	15k					
Dup15q Developmental Epileptic Encephalopathy	20k					
Recurrent Oxalate Kidney Stones	300k					
Best vitelliform macular dystrophy	15k					
Early onset preeclampsia	40k					
+4 discovery programs						

GondolaBio is an independent company from BridgeBio. As of December 31, 2025, BridgeBio has a 27.5% stake in GondolaBio. BridgeBio's interest in GondolaBio is subject to reduction as additional tranches of capital contributions are funded.

Example #2: The Phase 2 Erythropoietic Protoporphyria (EPP) program targets a genetic disease with high unmet need

EPP has severe health consequences…



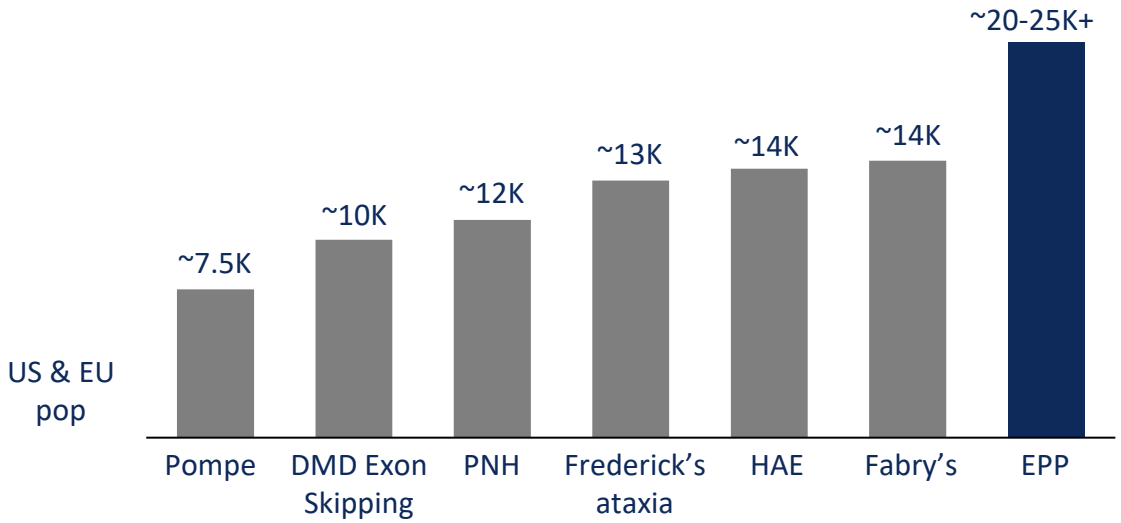
Sunlight causes skin damage and excruciating pain leading to severely impaired lifestyle and quality of life

Liver stress and liver damage are also common (20-30%), with a small portion of patients (5%) experiencing liver failure requiring transplant

Symptoms present at 4 years on average and are lifelong

Standard of care is limited; a tanning agent partially improves symptoms and QoL but does not modify the disease

…and affects a large patient population



20-25k+ EPP patients in the US and EU

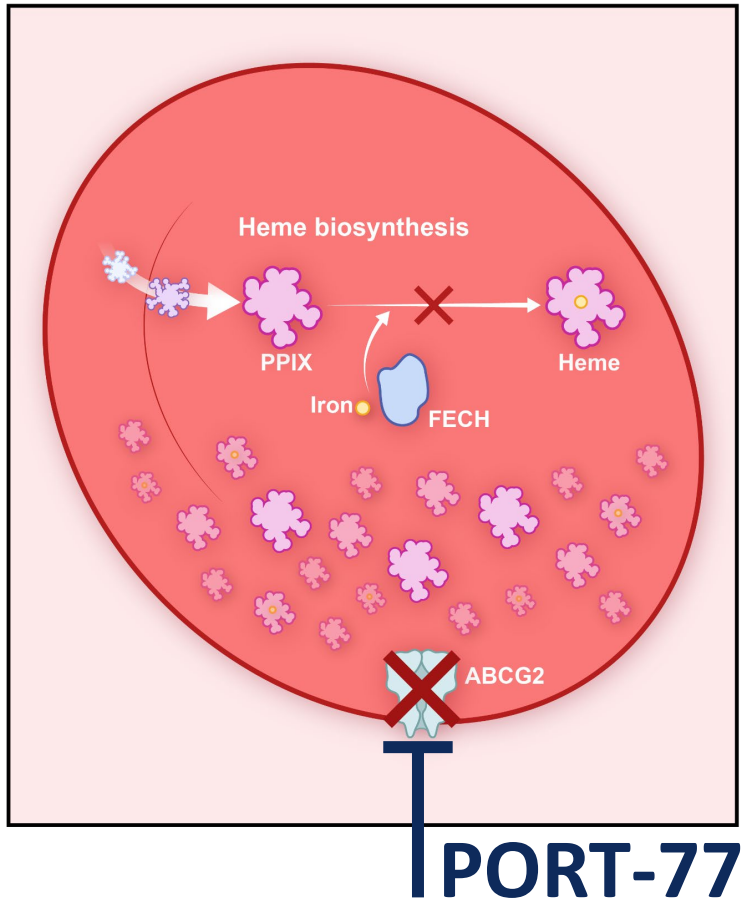
- 10-14k in US estimated from multiple independent claims datasets
- Internal genomic prevalence estimates 45-70k genetic incidence in US + EU, with incomplete penetrance and underdiagnosis

Source: Balwani et al. 2017; Balwani et al. 2019; Prevalence estimates on GnomAD, TopMed, Orphanet, DelveInsights, Canaccord (2025), Deutsche Bank (2025), Dinu et al. (2021), Amicus Corporate Presentation (2025), Komodo Claims

The pathomechanism of EPP is driven by the heme biosynthesis intermediate protoporphyrin nine (PPIX) in plasma and bile

Inhibition of ABCG2 prevents the transport of PPIX out of red blood cells to the plasma

The Portal small molecule PORT-77 is designed as a potentially best-in-class disease modifying therapy to meet three main criteria



Sunlight and liver efficacy

Dual mechanism that lowers plasma and bile PPIX may address both the phototoxic and hepatobiliary impacts of EPP

Avoid CNS side effects

Novel mechanism that does not modulate glycine or other neurotransmitters to prevent headaches, dizziness, or daytime sleepiness

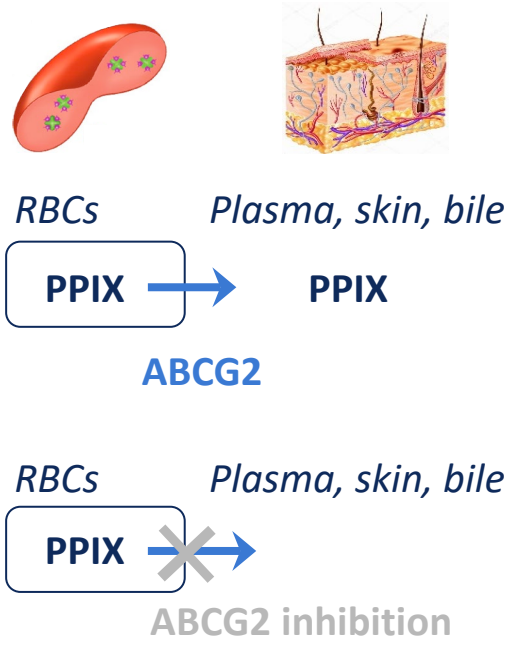
Shorten onset of protection

Rapid ABCG2 inhibition mechanism to maximize speed of protection after dosing

PORT-77 targets EPP at its source by preventing transport of PPIX out of red blood cells into the plasma, skin, and bile

Mechanism

ABCG2 inhibition keeps PPIX out of the plasma, skin, and bile



PORT-77 is a small molecule ABCG2 inhibitor

- Orally bioavailable
- 15nM IC_{50} for ABCG2 inhibition
- Highly selective for ABCG2 over other transporters
- Human half-life ($t_{1/2}$): 10-21 hrs
- Favorable ADME properties
- Composition of matter IP through at least 2044

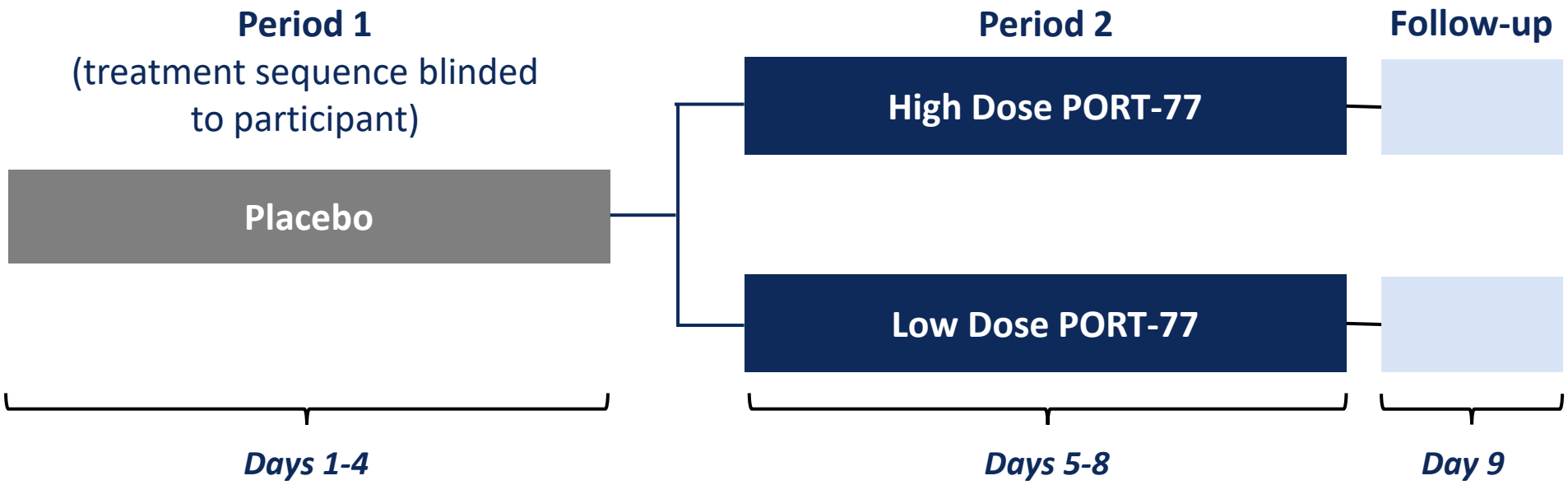
Which addresses all aspects of EPP safely and rapidly

- Addresses both skin and liver symptoms
- Highly safe and well-tolerated in preclinical + phase 1 dosing
- No CNS side effects
- Onset of action in minutes vs weeks

PORT-77 has a potential best-in-disease drug profile

Source: Internal Data

PORT-77 is being investigated in the GATEWAY Phase 2a trial



Design

- Single-blind, randomized, placebo-controlled cross-over
- In-clinic dosing, single-site

Endpoints

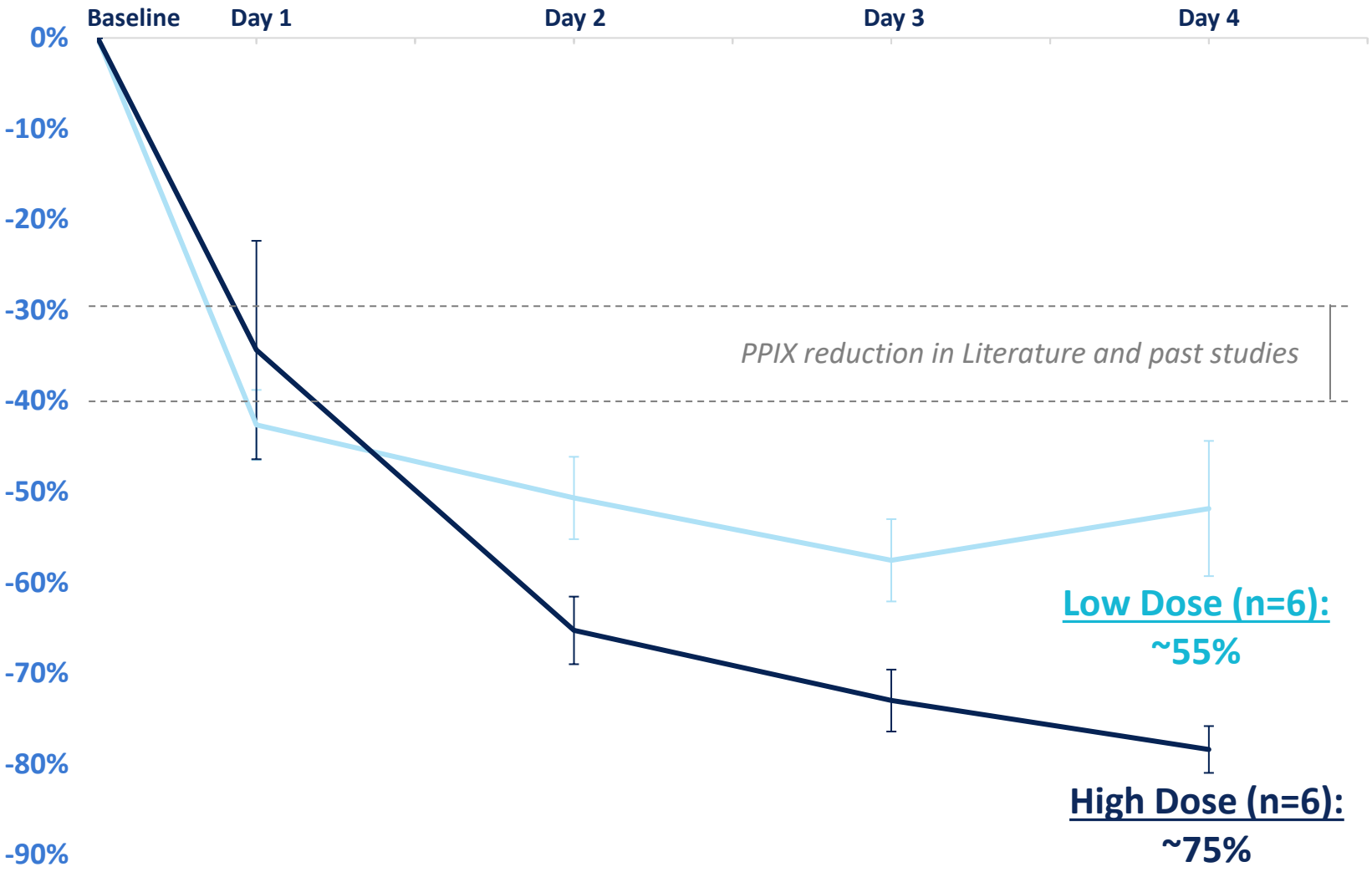
- Change in plasma PPIX as compared to baseline and placebo
- Safety and tolerability
- PK in EPP patients
- Exploratory clinical endpts

Study population

- EPP patients
- Age 18 years or older

PORT-77 has shown the largest PPIX reduction seen to date, reaching steady state within days

NEW INFORMATION



Dose dependent reductions in Plasma PPIX (relevant disease compartment) observed

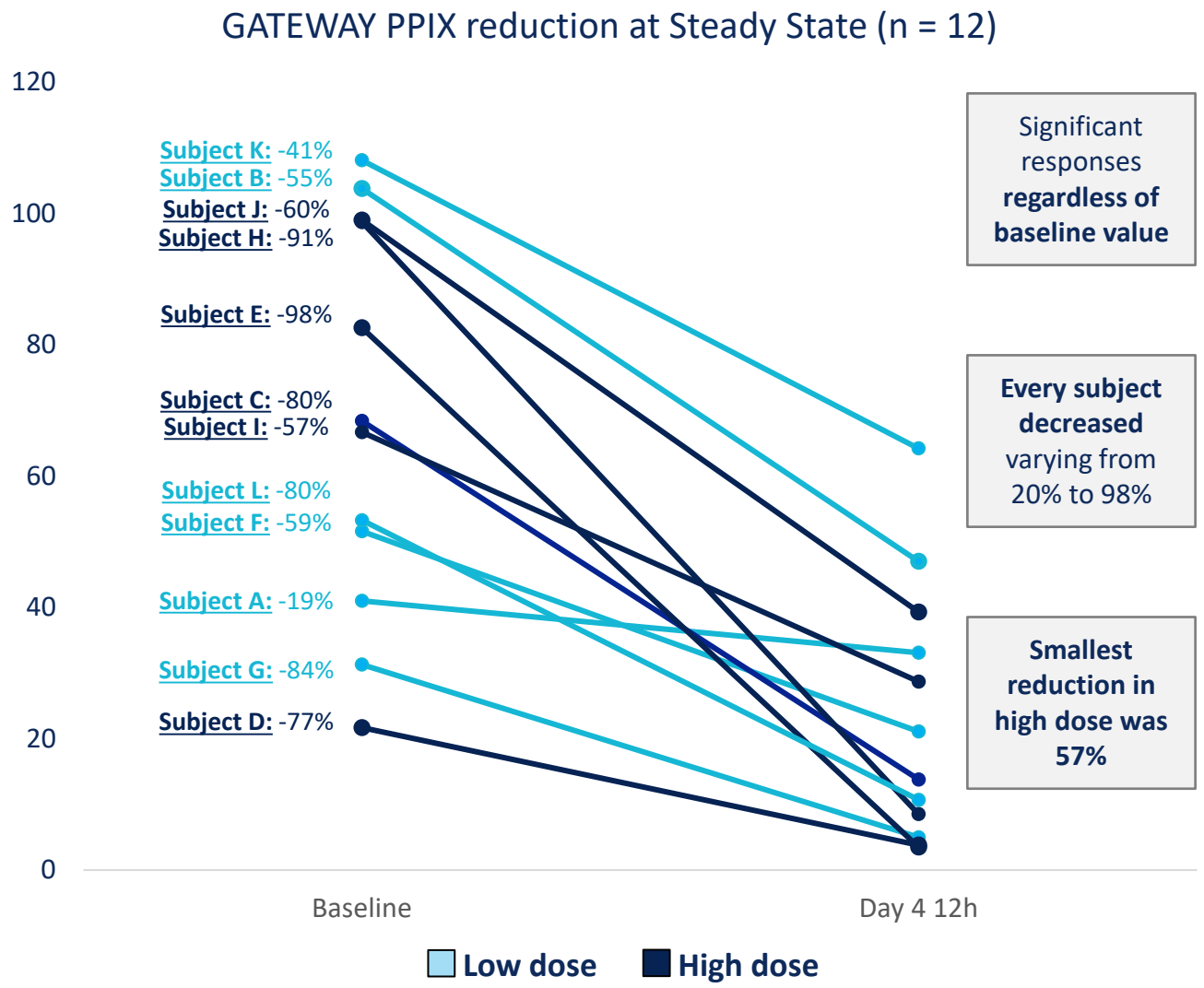
Largest PPIX reduction seen to date with ~75% at high dose

Rapid PPIX reduction to steady state within days

Note: Baseline defined as the pre-dose value on Day 1 of treatment period (D5 of study). Treatment measurements are averages within a day; Source: Internal data

Significant PPIX reduction seen in the Phase 2a, regardless of baseline PPIX value

NEW INFORMATION



Significant responses **regardless of baseline value**

Every subject decreased varying from 20% to 98%

Smallest reduction in high dose was 57%

% PPIX reduction between Baseline and Steady State:		
	Baseline PPIX value (ug/dL)	Steady State (D4 12H)
Subject A	41	-19%
Subject B	104	-55%
Subject F	52	-59%
Subject G	31	-84%
Subject K	108	-41%
Subject L	53	-80%
Subject C	68	-80%
Subject D	22	-77%
Subject E	83	-98%
Subject H	99	-91%
Subject I	67	-57%
Subject J	99	-60%

Note: Baseline defined as the pre-dose value on Day 1 of treatment period (D5 of study). D4 12H was the last timepoint of the treatment period (D8 of study); Source: Internal data.

PORT-77 has a clean safety profile across dose levels with no SAEs or safety and tolerability signals identified to date in the Phase 1 and Phase 2a trials

NEW INFORMATION

	Predose (n=12)	Placebo (n=12)	PORT-77 Treatment (n=12)
Subjects with any TEAE	3 (25%)	7 (58%)	5 (42%)
TEAEs leading to discontinuation	0	0	0
SAEs	0	0	0
Common TEAEs			
Headache	2 (17%)	3 (33%)	0
Nausea	0	4 (33%)	3 (25%)
Loose stools	0	1 (8%)	2 (17%)

No SAEs, no discontinuations, and AEs balanced across placebo and treatment period (likely viral)

Source: Internal data

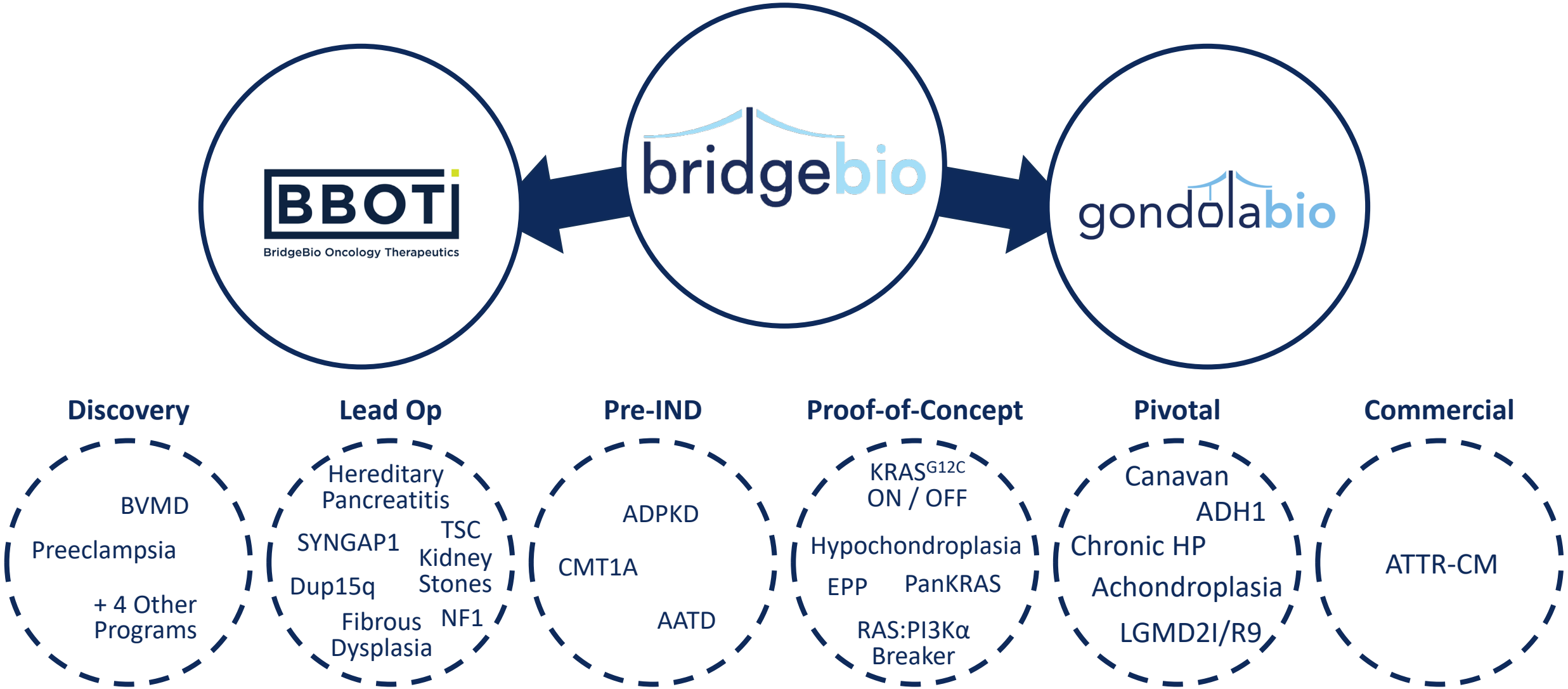
The EPP program has achieved proof-of-concept for a potential best-in-disease profile

Consistent and potential best-in-class PPIX reduction;
Dual-mechanism independently treating sunlight and liver symptoms

Clean safety profile with no notable AEs in treatment group and lack of CNS effects to date

PPIX reduction seen within hours, days to steady-state effect

Continued progress across the BridgeBio ecosystem



As of December 31, 2025, BridgeBio has an 18.2% ownership stake in BridgeBio Oncology Therapeutics and a 27.5% ownership stake in GondolaBio. BridgeBio Oncology Therapeutics and GondolaBio are independent companies from BridgeBio. BridgeBio's interest in GondolaBio is subject to reduction as additional tranches of capital contributions are funded.

Key takeaways today – significant momentum across the portfolio

ATTR-CM	• Attruby: $146M[1] in Q4 net product revenue; 6,629 unique patient prescriptions; and >25% NBRx share • New antibody depleter program announced for ATTR-CM
Infigratinib	• LPLV for Phase 3 achondroplasia trial achieved • LPI for Phase 2 hypochondroplasia trial achieved
BBP-418	• Broad benefit of BBP-418 in all subgroups across α-controlled efficacy endpoints at 12 months • Highly clinically meaningful and stat sig. 2.6 point benefit on NSAD relative to placebo at 12 months • Recommendation from FDA to orient NDA toward traditional approval
Encaleret	• Rapid uptake in diagnosis of ADH1 with >1,700 unique patients identified in claims since October 2023 • FDA alignment on and path forward with Phase 3 RECLAIM-HP trial in Chronic HP; expected to initiate in summer 2026
BBP-812	• Additional data demonstrating dose-dependent reductions in urine NAA and motor function improvements • Current path to potential BLA filing in 2027
GondolaBio	• Positive Phase 2a data for PORT-77 in EPP

[1]Represents preliminary, unaudited results for the fourth quarter ended December 31, 2025, based on management's current expectations and subject to completion of year end audit procedures. See Forward Looking Statements and Disclaimer on slide 2 regarding risks and uncertainties that could cause actual results to differ. Note: Unique patient prescriptions and NBRx share as of 12/31/2025. GondolaBio is an independent company from BridgeBio. As of December 31, 2025, BridgeBio has a 27.5% stake in GondolaBio. BridgeBio's interest in GondolaBio is subject to reduction as additional tranches of capital contributions are funded. NSAD=North Star Assessment for Girdle Type Muscular Dystrophies. EPP = Erythropoietic Protoporphyria

We are well-financed to hit a drumbeat of potential milestones in 2026 and beyond

NEW INFORMATION

BridgeBio ended 2025 with $587.5M[1] in cash, cash equivalents, and marketable securities

Cash burn declined in Q4 2025 relative to Q3 2025, driven by rising revenues and improving operating leverage

1H 2026

- **Infigratinib: ACH Topline**
- **Encaleret: Initiate P2/3 pediatric ADH1**
- **Encaleret: NDA filing**
- **BBP-418: NDA filing**

2H 2026

- **Infigratinib: HCH P2 data readout**
- **Encaleret: Initiate P3 CHP trial**

1H 2027

- **Encaleret: FDA approval and product launch**
- **BBP-418: FDA approval and product launch**

[1]Represents preliminary, unaudited results as of December 31, 2025, subject to completion of year-end audit procedures. See Forward Looking Statements and Disclaimer on slide 2 regarding risks and uncertainties that could cause actual results to differ. ACH=achondroplasia. HCH=hypochondroplasia. CHP=chronic hypoparathyroidism. Dates reflect anticipated timing.