

hope through rigorous science

Corporate Presentation

January 2025









Forward Looking Statements and Disclaimer

The presentation contains forward-looking statements. Statements made or presented may include statements that are not historical facts and are considered forward-looking within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. Words such as "believe," "anticipate," "plan," "expect," "intend," "will," "may," "goal," "potential," "should," "could," "aim," "estimate," "predict," "continue" and similar expressions or the negative of these terms or other comparable terminology are intended to identify forward-looking statements, though not all forward-looking statements necessarily contain these identifying words. We intend these forward-looking statements to be covered by the safe harbor provisions for forward-looking statements contained in Section 27A of the Securities Act and Section 21E of the Exchange Act. These forward-looking statements, including express and implied statements relating to the commercial success of Attruby, the timing of ongoing clinical trials, including BridgeBio Oncology Therapeutics' and Gondola Bio's clinical trials, the clinical, therapeutic and market potential of our clinical development programs and our pipeline, BridgeBio Oncology Therapeutics' pipeline and Gondola Bio's pipeline, our speed of creating new and meaningful drugs and related impact on patients, the efficiency of our engine to rapidly and efficiently deliver medicines, our value creation potential for patients, the potential market sizes and opportunities, the safety, efficacy and mechanisms of our newly FDA-approved Attruby (acoramidis) and other later-stage products including infigratinib, BBP-418 and encaleret, the timing of approval of Attruby for ATTR-CM in the European Union and Japan, our financial position, including our expectations regarding reaching regulatory milestones and the receipt of milestone payments, the potency and safety of our product candidates, the potential benefits of our product candidates, the potential for greater patient access to medications, the affordability and availability of insurance coverage of our medications. and the timing and expectations regarding results of our various clinical trials, reflect our current views about our plans, intentions, expectations and strategies, which are based on the information currently available to us and on assumptions we have made. Such statements reflect the current views of the Company with respect to future events and are subject to known and unknown risks, including business, regulatory, economic and competitive risks, uncertainties, contingencies and assumptions about the Company, including, without limitation, risks inherent in developing therapeutic products, and those risks and uncertainties described under the heading "Risk Factors" in the Company's most recent Annual Report on Form 10-K filed with the U.S. Securities and Exchange Commission ("SEC") and in subsequent filings made by the Company with the SEC, which are available on the SEC's website at www.sec.gov. In light of these risks and uncertainties, many of which are beyond the Company's control, the events or circumstances referred to in the forward-looking statements, express or implied, may not occur. The actual results may vary from the anticipated results and the variations may be material. You are cautioned not to place undue reliance on these forward-looking statements, which speak to the Company's current beliefs and expectations only as of the date of the presentation. Except as required by law, the Company disclaims any intention or responsibility for updating or revising any forward-looking statements made or presented at the presentation in the event of new information, future developments or otherwise. No representation is made as to the safety or effectiveness of the product candidates for the therapeutic use for which such product candidates are being studied.

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Indicated for the treatment of adult patients with ATTR-CM to reduce cardiovascular death and cardiovascular-related hospitalization



Three Phase 3 Trials are now fully enrolled



Trial enrolled **112 patients** to evaluate BBP-418 in **Limb-Girdle Muscular Dystrophy 2I/R9**



Trial enrolled **70 patients** to evaluate encaleret in **Autosomal Dominant Hypocalcemia Type 1**



Trial enrolled **114 participants** to evaluate infigratinib in **Achondroplasia**

BridgeBio's objective function

Patient impact...

Objective: max
$$\int_{0}^{t} \sum_{Drugs \ i=1}^{N} \frac{\Delta QALY \ (i)}{patient} * patients \ (i)$$

BridgeBio maximizes the speed of creating
as many new and meaningful drugs
that have a profound impact on as
many patients as possible

...through sustainable value creation

Each project must be:

- Based on beautiful science with a high probability of technical success (POTS)
- NPV positive (driven by ROIC, g, WACC)

BridgeBio is a new type of biopharmaceutical company

From: To:

Slow and bureaucratic decision making



Rapid and decentralized decision making

Expensive platforms with long lead times before proof-of-concept data



Assets selected to target genetic diseases at their source

High fixed costs



Variablized and flexible costs

Limited sources of capital



Strategic toolkit of financing options at the levels of the portfolio and affiliate companies

Incentives at the portfolio level



Incentives at the level of each asset to preserve focus at the level of biology

The right approach: decentralized R&D, centralized infrastructure



Build "minimum viable companies" to de-risk programs as quickly and efficiently as possible



Build **central infrastructure** for functions with economies of scale, such as commercial



Leverage hyper-experienced R&D practitioners who are focused on the science of each individual program





Leverage seasoned company builders and centralized capital allocators to take the best possible shots on goal

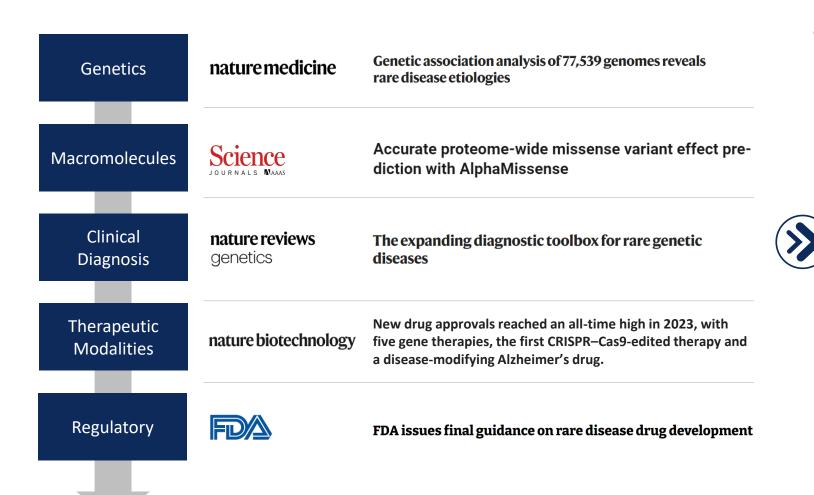


Provide investors with increased choice in where to participate in our ecosystem

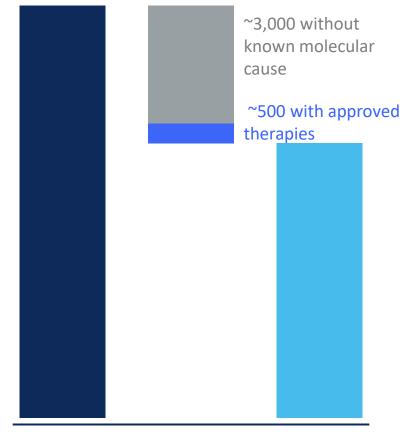


Provide investors with a de-risked portfolio of assets; enable access to low-cost debt

The right space: capitalizing on a scientific revolution to treat a massive unmet need for genetic diseases



There are **10,000+ counted rare diseases** affecting 450 million+ people globally



This leaves hundreds of millions of people across 6,500 diseases with known molecular cause who are **anxiously waiting** for therapies

Our leadership team has world-renowned drug hunters and operators



Neil Kumar, PhD Founder and Chief Executive Officer







Thomas Trimarchi, PhD President & **Chief Operating Officer**

Goldman Sachs

REGENERON



Brian Stephenson, PhD, CFA Chief Financial Officer





Uma Sinha, PhD Chief Scientific Officer







Robert Zamboni, PhD Chemistry





Jonathan Fox, MD, PhD Chief Medical Officer, Cardiorenal





Ananth Sridhar Chief Operating Officer, Cardiorenal

REGENERON Genentech



Christine Siu Chief Executive Officer, Muscular Dystrophy







Justin To Chief Executive Officer, Skeletal Dysplasias



McKinsey & Company



Eric David, MD, JD Chief Executive Officer, Gene Therapy organovo McKinsey & Company



Charles Homcy, MD Founder and Chairman of **Pharmaceuticals**







Frank McCormick, PhD Founder and Chairman of Oncology







Richard Scheller, PhD Chairman of R&D







Len Post, PhD Advisor





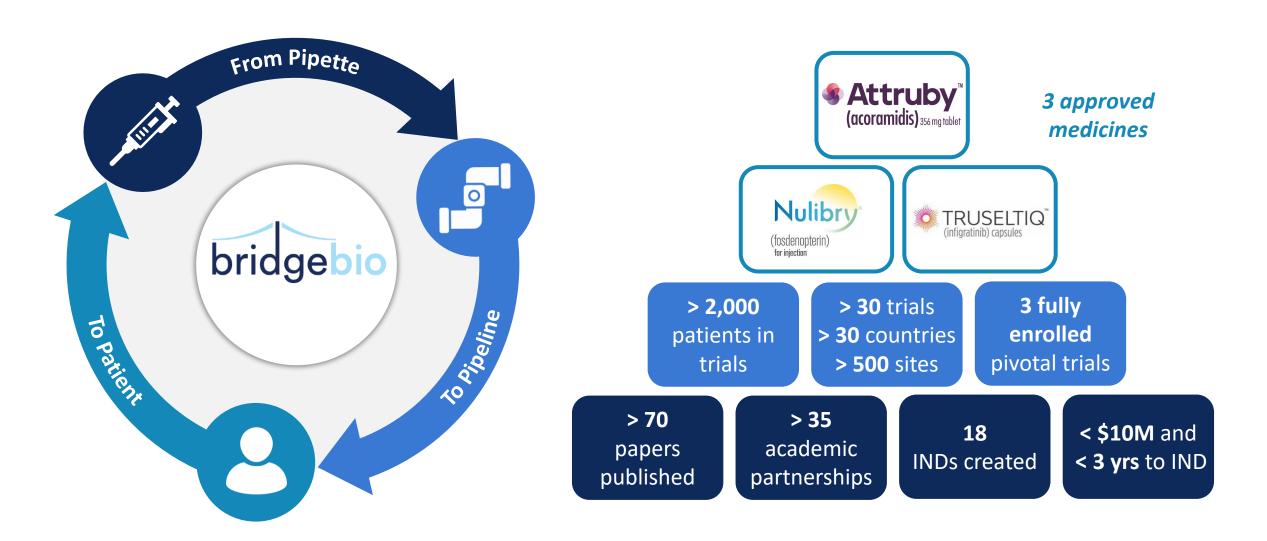


Phil Reilly, MD, JD Advisor





We have built a sustainable, high velocity engine to deliver medicines



A pipeline of products that sing across the BridgeBio ecosystem

| Program | Indication | Pre- clinical | Phase 1 | Phase 2 | Phase 3 | Approved | Patients (US + EU) | Market Opportunity |
|------------------------------------|--|------------------|----------------|----------------------|---------|-------------------|-----------------------|-----------------------|
| Attruby (acoramidis) | Transthyretin Amyloidosis (ATTR-CM) | | | | | \checkmark | 500,000+ | \$20B+ |
| Nulibry (fosdenopterin) | Molybdenum Cofactor Deficiency (MoCD) Type A | | | | | \checkmark | <100 | Partnered |
| Truseltiq (infigratinib) | Cholangiocarcinoma | | | | | \checkmark | 37,000 | Partnered |
| Infigratinib — | Achondroplasia (ACH) | | | | | Fully Enrolled | 55,000+ | \$2B+ |
| iiiigiatiiib | Hypochondroplasia (HCH) | | | | | | 55,000+ | \$2B+ |
| BBP-418 | Limb-Girdle Muscular Dystrophy Type 2I/R9 (LGMD2I/R9) | | | | | Fully Enrolled | 7,000+ | \$1B+ |
| Encaleret — | Autosomal Dominant Hypocalcemia Type 1 (ADH1) | | | | | Fully Enrolled | 25,000+ | \$1B+ |
| Encaleret | Post-Surgical Hypoparathyroidism (PSH) | | | | | 200,000+ | \$1B+ | |
| BBP-812 | Canavan Disease | | | Phase 1/2 Pivotal 1, | | 1,000 | TBD | |
| BridgeBio Oncology Therapeutics | Oncology, various | | 38% | 38% ownership* | | Various | Various | |
| GondolaBio | Rare disease, various | | 45% ownership* | | Various | Various | | |

^{*} BridgeBio Oncology Therapeutics and GondolaBio are separate, independent companies from BridgeBio. BridgeBio's initial interest in GondolaBio is subject to reduction as additional tranches of capital contributions are funded.

We are well-financed and expect to hit numerous milestones in 2025

1H 2025

- Acoramidis: EU approval
- Acoramidis: Japan approval
- Q1 Earnings Call

2H 2025

- Q2 and Q3 Earnings Calls
- Encaleret: LPLV, Topline
- BBP-418: LPLV, Topline
- Infigratinib: LPLV



bridgebio vision for 2030:



De-risked PYS

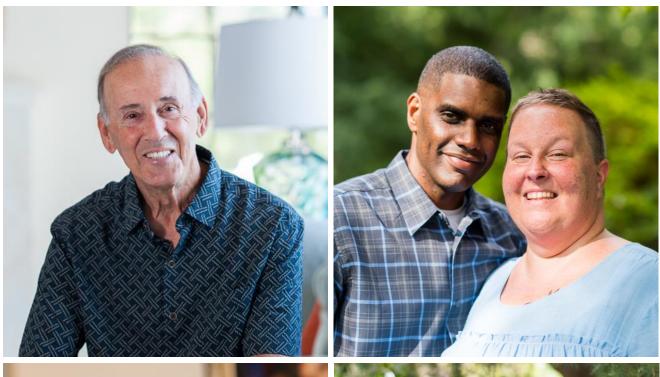
>\$8B



Lives impacted

>100k lives

Attruby Now Approved







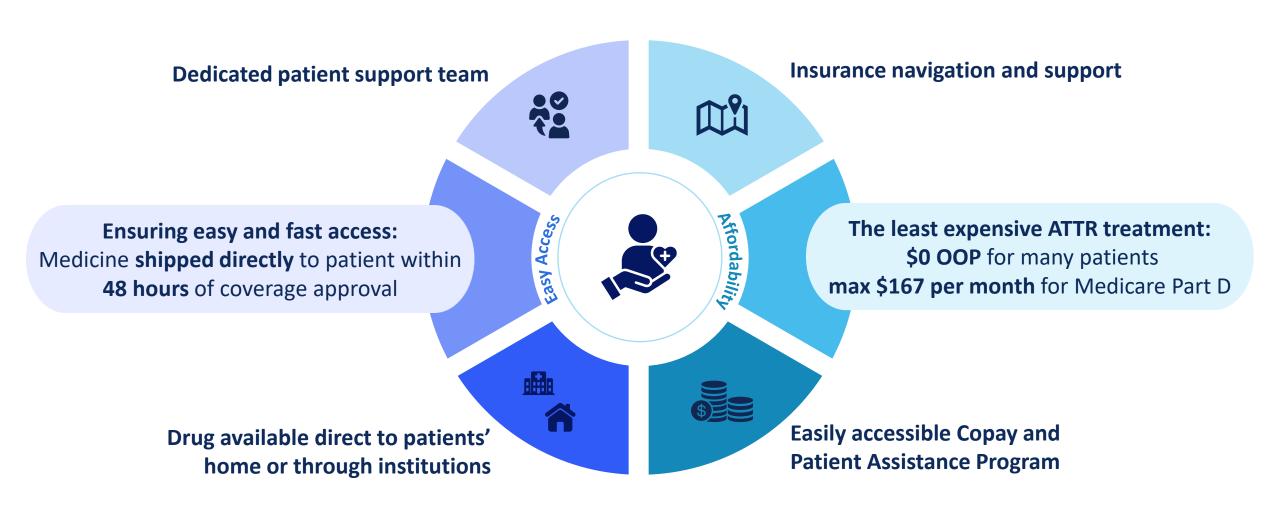
Attruby is now FDA Approved – Key Highlights



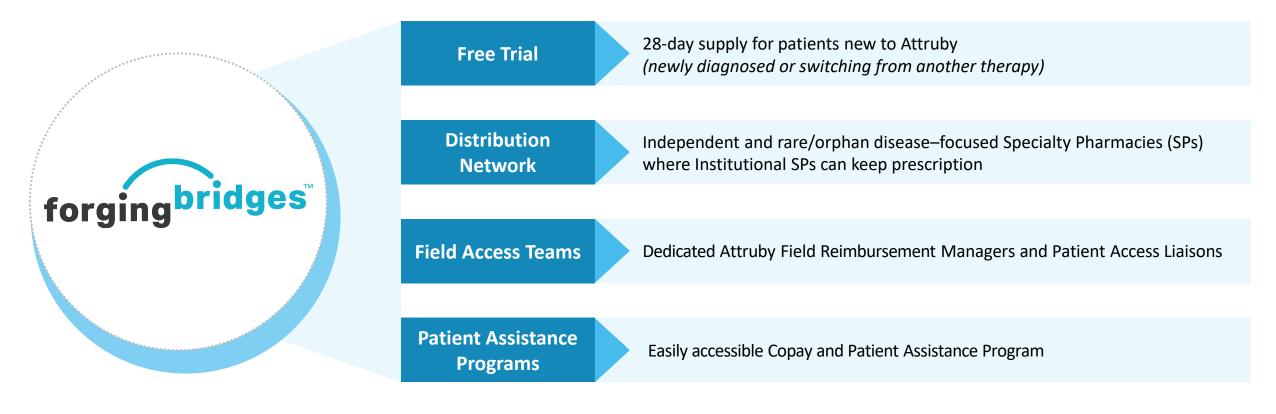


- Effect seen as early as 3 months the most rapid benefit seen in any Phase 3 study of ATTR-CM to date
- **42% reduction** in composite of all-cause mortality and recurrent cardiovascular-related hospitalization events at Month 30
- **50% reduction** in the cumulative frequency of cardiovascular-related hospitalization events at Month 30

Our responsibility to patients spans beyond R&D – we are committed to ensuring the best access and affordability of any ATTR-CM medicine



We make it easy through simplified, differentiated, and generous access programs



Commitment to clinical trial patients

US patients who participated in the acoramidis clinical trials may receive Attruby at no cost for the duration of their medically indicated treatment

Performance to date indicates strong commercial momentum *As of January 13th, 2025*



430

Attruby scripts written to date



248

Unique prescribing HCPs



77%

Medicare lives in equal formulary position to tafamidis

Positive feedback all around on market access

Pricing:

You priced responsibly and have a good label; we will have you at parity with tafamidis. Also, I have never seen a company offer free drug to their trial patients. Shows commitment." - Payer

66

You did the right thing by being less than tafamidis... both your label and price will play a role here." - Payer

Limited Distribution Network:

I want to reemphasize our gratitude for LDN inclusion.
We objectively see key metrics in the patient journey improve as a result of dispensing access...Eager to leverage our resources to impact patient care in a positive way." –Manager of Health System SP

You are a smart company to set up your distribution this way.

I wish all companies would do this!" – Director of Health System SP

ForgingBridges:

"BridgeBio's thoughtful resources and the structure of the network...aligns with the health system's mission of putting patients first. – **Pharmacist**

"Holy Smokes!? I wish all manufacturers would provide this information. Do you have any idea how much time this is going to save our team?

Pharmacist

"Given the combination of strong efficacy and safety data, alongside BridgeBio's very health system friendly approach towards access for this

plan to begin preferring Attruby.

- AD Specialty Clinical Program Development

product (in comparison to the many challenges we have had with Pfizer...), we

99

"It takes less than 5 minutes- the easiest enrollment process of any drug we use. – **RN Heart Failure Coordinator**

Our commercial infrastructure is deployed across the US to ensure full provider coverage

ATTR-CM Prescriber Market



8K+ prescribing HCPs, ~100 COEs hold significant influence with prescribers & communicate overall product value

BridgeBio Coverage



Dedicated commercial & US-field-based team with singular focus on serving patients with ATTR-CM



Salesforce is built to adequately cover every HCP and institution prescribing in ATTR-CM



Robust segmentation to maximize effectiveness

Our Attruby team has experienced industry leaders who have built and launched blockbuster drugs



Matt Outten

Chief Commercial Officer

- Broad commercial leadership expertise with success across multiple competitive markets
- Led \$5B+ portfolio, 12 FDA approvals spanning 6 disease states and 7 indications (IMBRUVICA, Pharmacyclics)
- Commercial lead on \$21B pharma M&A deal



Julie Everett

Chief Business Officer

- Successfully led cross-functional teams through multiple rare disease launches, including VOXZOGO and PALYNZIQ (BioMarin)
- Led commercial strategy/execution across ~\$1B portfolio
- ~Decade of strategy consulting leadership focused on launch excellence and lifecycle maximization (Trinity)



John Whang

Chief Medical Affairs Officer

- Orchestrated multiple successful launches with pioneering therapies in competitive segments – STELARA (Janssen), REPATHA (Amgen), and CAMZYOS (BMS)
- 8+ launches as strategy consultant (McKinsey)
- Demonstrated strategic innovation (Heartline Study J&J / Apple collab) and consistently built outstanding organizations



Ana Merz VP, Sales

Launched IMBRUVICA (\$5B+, 12 FDA approvals, 6 disease states, 7 indications in 10 years) and **EPKINLY (3L+ DLBCL)**



Sean Doherty

SVP, Marketing

Broad global sales and marketing launch experience including in rare, infectious, and autoimmune diseases



Scott Collins

SVP, Market Access

Extensive market access experience with consistent coverage across rare disease and oncology, leading large field-based access teams



Hudson Boyer

VP, Commercial Analytics & Ops

Launches in rare disease, hematology, and immunology; strategy consulting and equity analyst background



Liz Arnold

Head of Commercial Strategy

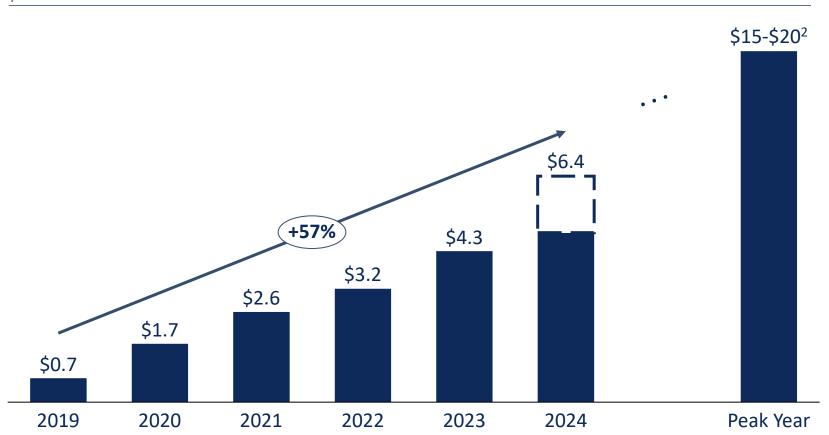
Multiple global launches, expertise in strategy, consulting, and marketing across rare disease, hematology, and OTC

Our BridgeBio team is committed to providing industry-leading access and white glove service for all parties looking to bring Attruby to patients with ATTR-CM.

ATTR is a multi-billion-dollar market primed for continued expansion

Global annual ATTR market sales¹

\$B

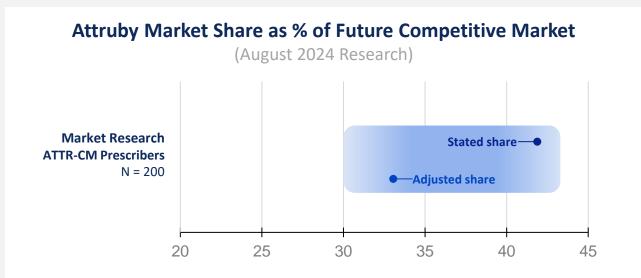


Market growth drivers include:

- With more sponsors, there is expanding disease awareness
- Increased global adoption of noninvasive diagnostic tools

New demand study shows significant Attruby market potential vs. partial stabilizer and partial knockdown

Surveyed HCPs expect 30 - 40%+ peak market share for Attruby



- Survey of 200 HCPs with a history of ATTR-CM prescribing in Rx data
- Included competitive profiles of stabilizer and knockdown products
- Conducted by third-party consulting firm in August 2024, post competitive data release

HCP sentiment towards Attruby is positive

Acoramidis showed dramatic reduction in cardiovascular hospitalizations, and improvement in patient QoL."

HCP - Northeast

There is a mortality benefit and there are also quality of life benefits. It is an oral medication, so that will be well-liked by patients."

HCP - West

Very impressive treatment effect and best data to date on what happens in a contemporary ATTR-CM population."

HCP - Central

BridgeBio is committed to advancing Attruby's scientific narrative to ensure as many appropriate patients as possible can benefit



ATTRibute

ATTR-CM WT and hereditary

Extended Ph. 3 data disclosures at ACC, ESC-HF, ESC, HFSA, and AHA

ATTRibute-CM Ph. 3 OLE Month 42 safety and efficacy data



ATTRibute 8

ATTR-CM WT and hereditary

Attruby lifecycle management and further analyses of pivotal study data

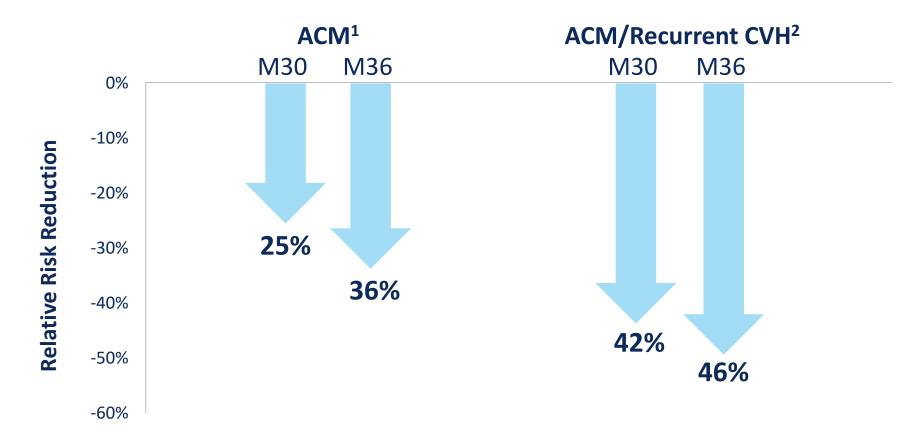


ATTR-CM Hereditary

Prevention study

Additional real-world evidence generation and publication plan to expand scientific share of voice

Recently published data from the OLE further support Attruby's statistically significant benefit on ACM and ACM/Recurrent CVH

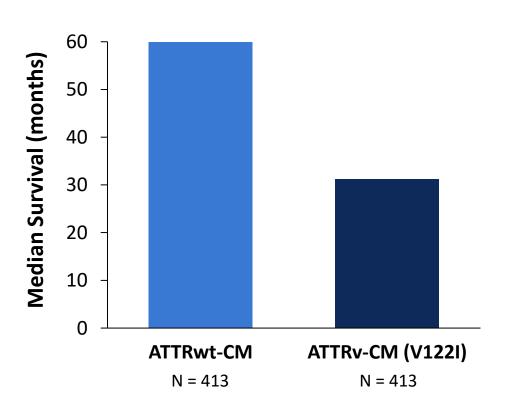


Attruby resulted in a **statistically significant** ACM and ACM/Recurrent CVH relative risk reduction at **both Month 36 and Month 42**

Looking Ahead: Attruby Delivers Outstanding Results in Patients with Poor Prognosis

Natural History

V122I ATTRv-CM has an aggressive phenotype and poor prognosis¹



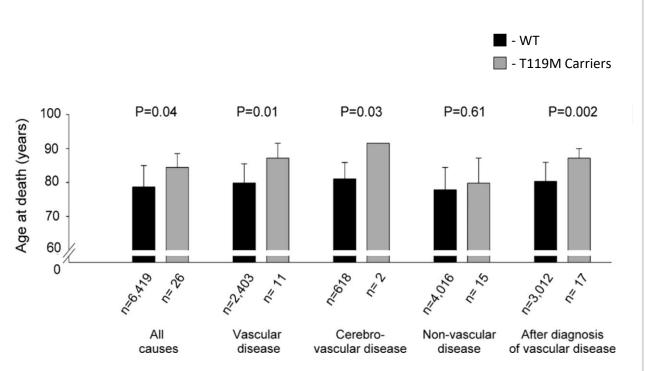
ATTRibute-CM mITT Population

Statistically significant benefit on composite ACM or first CVH in ATTRv-CM participants vs. placebo²

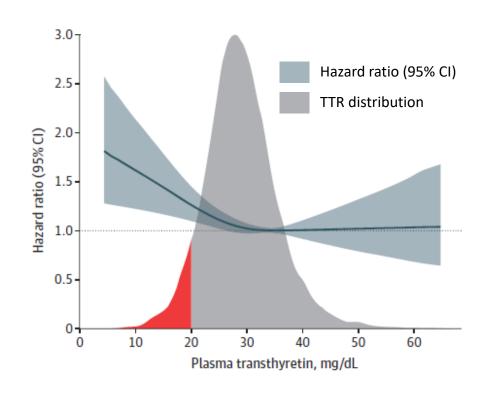
| | N (%) | Hazard Ratio (95% CI) | p value |
|--------------------|------------|--------------------------|---------|
| Overall Population | 611 (100%) | 0.65 (0.50-0.83) | 0.0008 |
| ATTRv-CM | 59 (9.7%) | 0.41 (0.21-0.81) | 0.0109 |
| | | * | |

Additional data: Elevated TTR levels are associated with improved survival

Genetic stabilization of transthyretin associated with improved health outcomes (N≈69K individuals)¹



Higher TTR concentration associated with greater life expectancy (N≈102K individuals)²



Infigratinib



There remains a significant unmet need for many children with skeletal dysplasias; this represents a large (\$4B+) and rapidly growing market

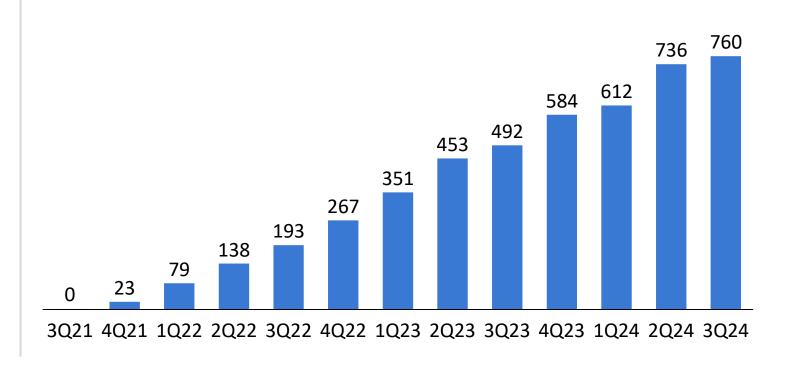
Addressable people by indication in US/EU¹

(current population eligible for treatment)

15-25K 1.5-2.5K Other FGFR-driven conditions Hypochondroplasia 7-10K (Run-in for Phase II Initiated) Achondroplasia 7-10K (Phase III Ongoing)

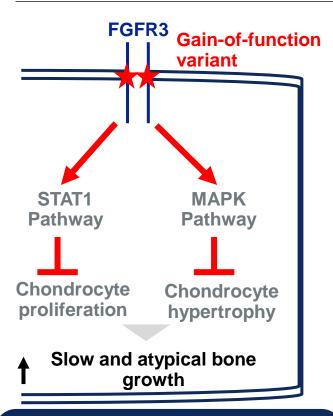
Annualized achondroplasia product sales²

(\$M WW, annualized by quarter)

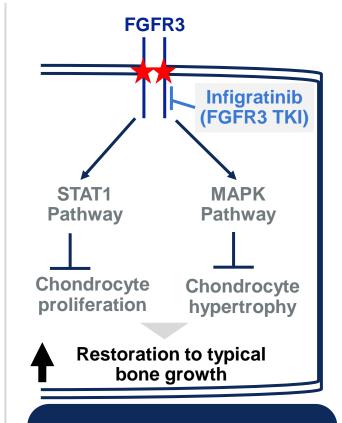


Infigratinib is a best-in-class FGFR3 inhibitor that targets achondroplasia and hypochondroplasia at their source

Mechanism



FGFR3 acts as a "molecular brake" on chondrocyte proliferation and hypertrophy; in ACH or HCH, this brake is **stuck** due to gain-of-function mutations resulting in shortened bones



Infigratinib "releases" the brake, potentially resuming normal chondrocyte function, allowing for restoration of bone growth

Design Principles



Maximize efficacy by targeting disease at the source

For all the manifestations of ACH and HCH, not just height, which matter for families and physicians



Demonstrate safety with low dosing

Avoiding hypotension & injection site reactions with no hyperphosphatemia, ocular effects or VEGFR3 off-target effects

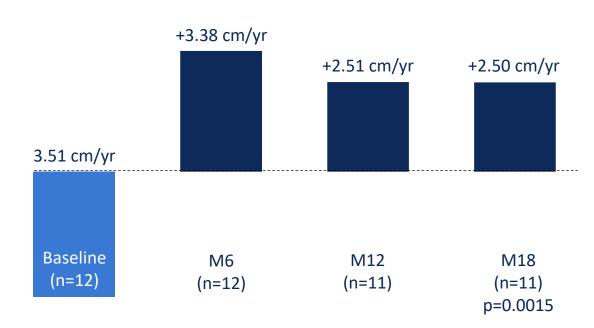


Avoid injections and provide an oral option

For children and families, to reduce burden and pain of treatment

At 18 months, infigratinib has shown persistent improvement in AHV and body segmentation, along with a favorable safety profile

Mean change from baseline in annualized height velocity (AHV)

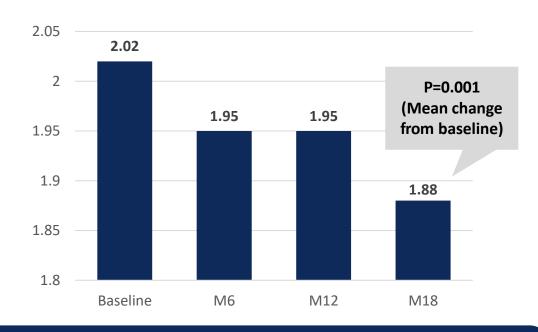


At each timepoint, infigratinib change from baseline AHV is higher than that reported by any other treatment option

At the highest dose, there were no SAEs, most TEAEs were of grade 1 severity, and none were assessed as related to study drug

Upper body to lower body segment ratio

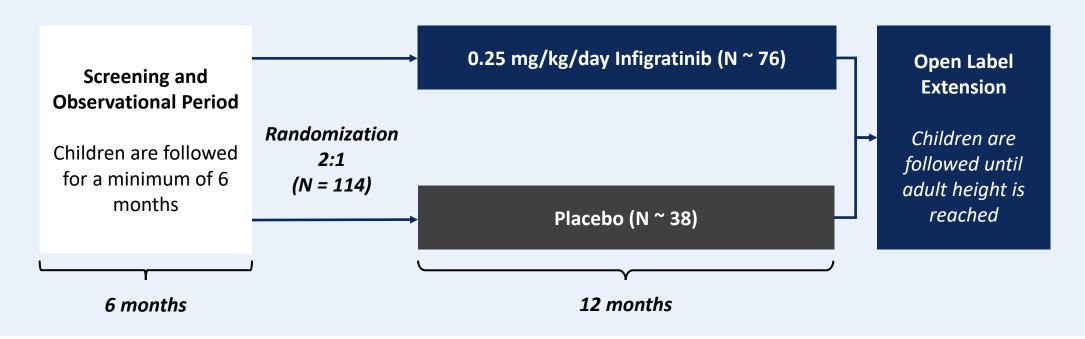
(Lower value means improved proportionality)



Infigratinib shows statistically significant proportionality improvements after 18 months

This has potential for a meaningful effect on body proportionality, and if maintained, can be associated with functionality

We have fully enrolled a Phase 3 study (PROPEL 3) of Infigratinib in **Achondroplasia with LPLV in 2H 2025**



Primary Endpoint:

 Change from baseline in annualized height velocity at Wk 52

Key Secondary Endpoints:

- Change from baseline in height z-score
- Change from baseline in upper to lower body segment ratio

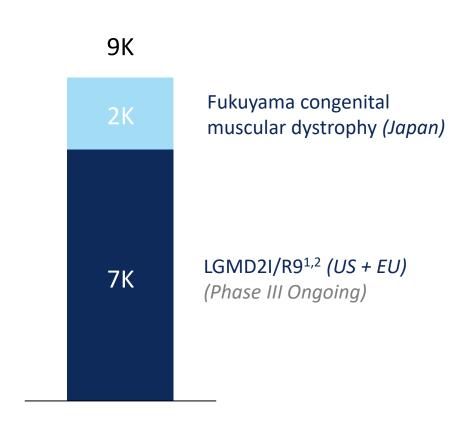
Phase 3 Trial Fully Enrolled; LPLV Expected 2H 2025

BBP-418



LGMD2I/R9 is a progressive neuromuscular disease with high unmet need, representing a >\$1B market opportunity in the US and EU

Addressable patients by indication



Unmet need

- LGMD2I/R9 is an inherited neuromuscular disorder characterized by lower-limb weakness and loss of ambulation as well as respiratory decline and cardiac dysfunction
- No approved disease modifying agents for LGMD2I/R9
- Current standard of care is aimed at symptom management and includes physical therapy, steroids, and pain management
- Standard of care does not prevent continuous and progressive decline in LGMD2I/R9 patients

Market opportunity \$1B+

BBP-418 is a first-in-class, disease-modifying therapy with potential to be the first approved therapy for LGMD2I/R9

Mechanism



FKRP glycosylates alpha-dystroglycan (α DG), stabilizing muscle cells by binding extracellular ligands to act as a "shock absorber" for muscle fibers



Partial loss of function in FKRP results in dysfunctional, hypo-glycosylated α DG in muscle cells, increasing cell susceptibility to damage

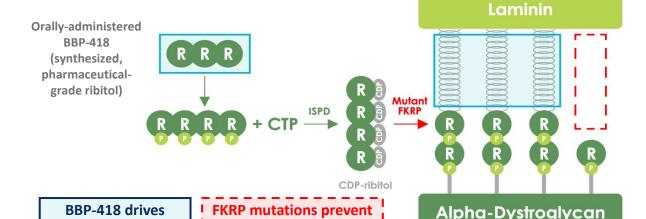


potential partial

restoration of

glycosylation of αDG

Supply supraphysiological levels of synthesized, pharmaceutical grade ribitol upstream aiming to drive residual activity of mutant FKRP enzyme and increase αDG glycosylation levels



addition of CDP-ribitol to

αDG, limiting function as

a "shock absorber"

Design Principles



Provide first disease-modifying therapy

For patients with LGMD2I/R9 and potentially applicable for other α -dystroglycanopathies



Avoid safety concerns with FKRP modulation

Avoid off-target effects using a synthesized version of an endogenous compound with an encouraging safety profile

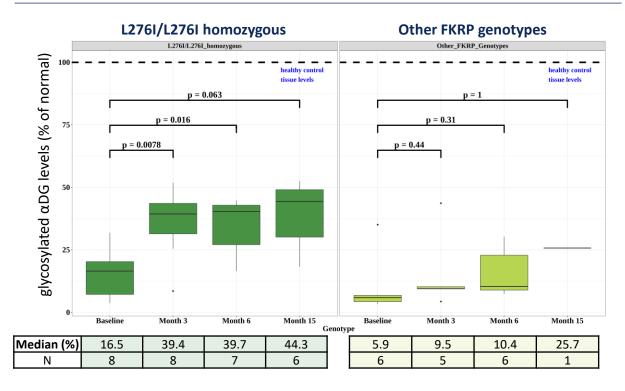


Convenient oral medicine

To reduce burden for patients and avoid safety concerns

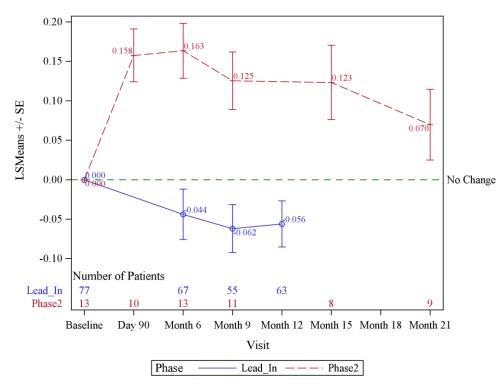
Significant increases in glycosylated αDG in muscle and stabilization of ambulatory measures were observed in a Phase 2 study of BBP-418

Increase in glycosylated αDG in muscle observed post dosing with BBP-418 (median ± 95% CI)



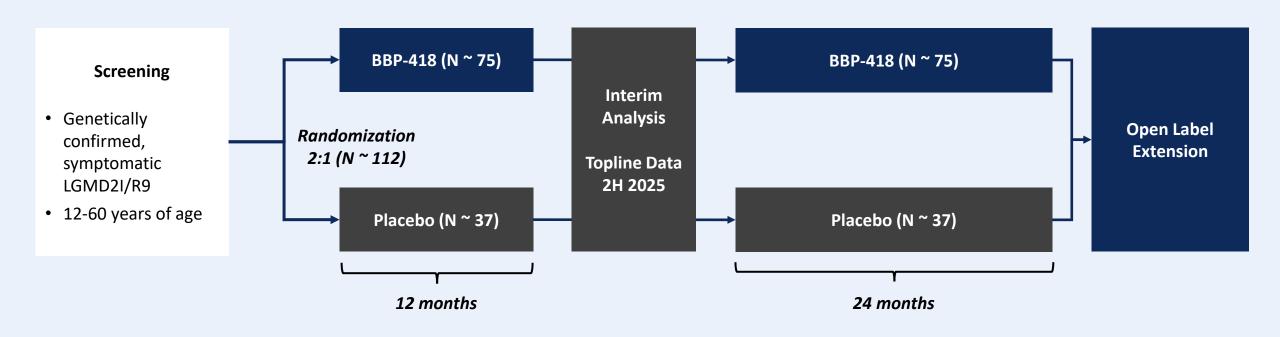
Patient samples were interpolated to standard curve to determine % of normal glycosylation of αDG + 3 mo = Part 1, 90-day, +6 mo = Part 2, Month 3, + 15 mo = Part 3, Month 9 Median and 10–90% percentile are shown, Wilcoxon test was used to determine significance MLB-01-003 Listing 16.4.1 and 16.1.4.2

Change from baseline in 10-meter walk test (m/s)



Blue line denotes natural history; red line denotes on-treatment data from Ph. 2 study. Stabilization of 100-meter timed test and NSAD was also observed at 21 mo. MLB-01-001 Listing 16.2.1 and MLB-01-003 Listing 16.2.1

We have fully enrolled a Phase 3 study (FORTIFY) of BBP-418 in LGMD2I/R9 and expect topline interim analysis data readout in 2H 2025



Interim Endpoints:

- Glycosylated αDG (*primary*)
- Serum creatine kinase (CK)
- Trends in clinical measures

Final Analysis Endpoints:

- NSAD (primary)
- Ambulatory measures
 - 10MWT
 - 100MTT
- Pulmonary function: FVC
- Upper limb function: PUL 2.0
- QoL measures

Phase 3 Trial Fully Enrolled; LPLV & Topline Results Expected 2H 2025

Encaleret



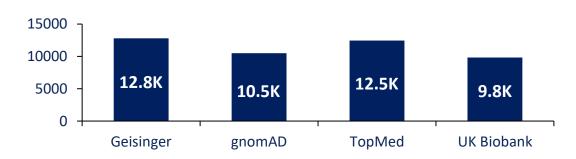
ADH1 is a serious and rare genetic condition for which there are no indicated therapies and diagnosis rates are low

12K ADH1 Carriers ~12K carriers of ADH1 causing variants in the US based on 4 population databases¹⁻² 9K Treatable 73% of patients with ADH1 are symptomatic³ **3K – 5K Currently Addressable** 40-60% of symptomatic ADH1 patients are diagnosed today, anticipate increase

An analogous ADH1 market is XLH

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

Population prevalence estimates in literature¹⁻²



Encaleret is a first-in-class, disease-modifying therapy that targets the underlying disease mechanism of ADH1

Mechanism

Normal CaSR senses and regulates serum Ca levels to maintain homeostasis

Encaleret is a **CaSR antagonist**

designed to decrease the sensitivity of CaSRs to extracellular calcium



ADH1 CaSR is overly sensitive to extracellular calcium



<u>restored</u> in the presence of encaleret

Overactive CaSR causes dysregulation of calcium homeostasis, encaleret has the potential to normalize PTH, serum Ca, and urine Ca levels



Restore PTH

to the normal range



<u>Restore</u>

serum calcium to the normal range



<u>Decrease</u>

urine calcium to the normal range

Design Principles



First and only investigational treatment directly targeting ADH1 at its source

Potential to restore physiologic mineral homeostasis that is disrupted by CaSR oversensitivity



Address common symptomology

Designed to normalize PTH, serum Ca, and urine Ca levels, potentially resolving key symptoms

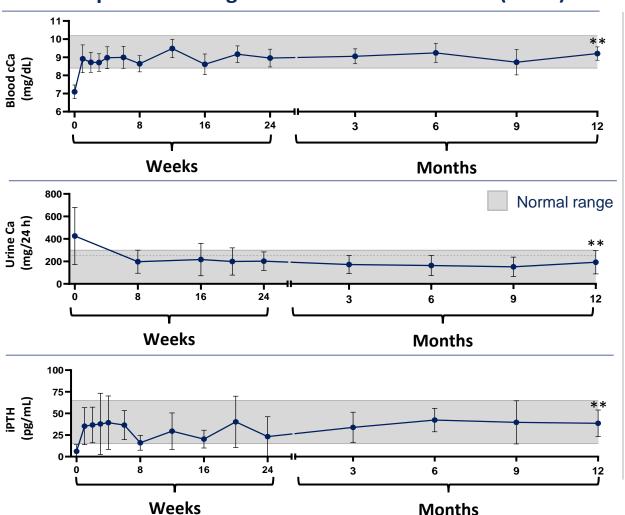


Convenient oral dosing

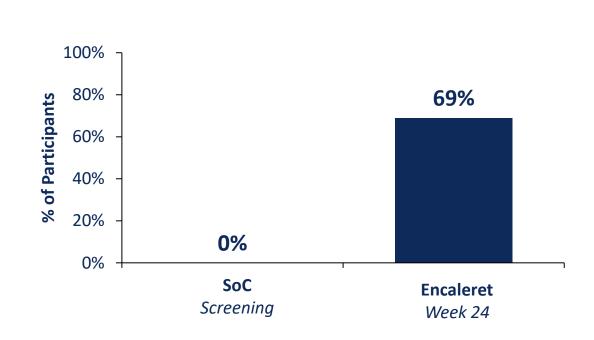
First targeted treatment for ADH1 in a convenient form for patients and families

Phase 2B results demonstrated rapid and sustained normalization of serum Ca, urine Ca, and serum PTH, without need for dose escalation

Mean responses through 18 months of treatment (N=13)

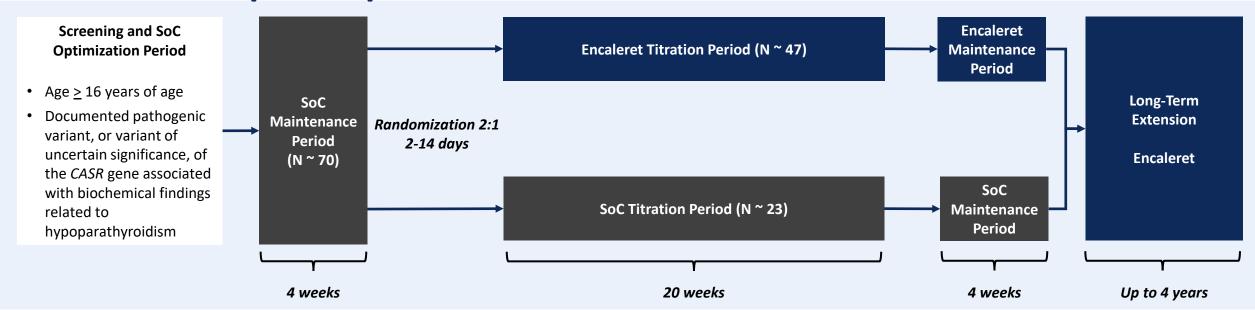


Participants with blood Ca and urine Ca in the target range



Encaleret normalized mean blood Ca, PTH, and urine Ca in participants with ADH1 over an 18-month period representing a meaningful improvement over SoC

We have fully enrolled the Phase 3 study (CALIBRATE) of encaleret in ADH1 and expect topline results in 2H 2025



Primary Endpoint:

- Proportion of participants achieving:
 - Blood Ca within the target range AND
 - 24-hour urine Ca within the reference range

Select Secondary Endpoints:

- Blood iPTH, 1,25-(OH)₂ Vitamin D, magnesium, and phosphate
- Urine magnesium and phosphate
- Bone turnover markers
- Renal ultrasound and renal function
- ER/urgent care visits and/or hospitalizations
- Quality of life (SF-36)

Phase 3 Trial Fully Enrolled; LPLV & Topline Results Expected 2H 2025 BBP-812



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

Unmet need

- Canavan is an ultra-rare neurodegenerative disease with ~1,000 patients across the US and EU
- Canavan is usually fatal within the first two decades of life, and >25% of patients die by the age of 10 years¹
- Children with Canavan exhibit global and severe cognitive, motor, and language impairment, missing or regressing on most developmental milestones
- They 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



¹Bley A, et al. Orphanet J Rare Dis. 2021 PMID: 34011350

BBP-812 is a first-in-class, disease-modifying therapy that targets Canavan disease directly at its source

Mechanism

Healthy NAA NAA metabolic pathway metabolic pathway in Canavan Disease N-acetylaspartic **▲ N-acetylaspartic** acid (NAA) acid (NAA) **Aspartoacylase** (ASPA) Acetate Acetate **Aspartate Aspartate** Healthy **Demyelination** of neurons Neuron

BBP-812 is an AAV9 gene therapy which directly replaces the mutated ASPA gene that causes Canavan disease

Design Principles



Provide first disease-modifying therapy

Target the condition directly at the source, utilize single registrational study & biomarker for accelerated approval



Provide therapy with known safety profile

Leverage safety profile from approved AAV9 gene therapy (Zolgensma)

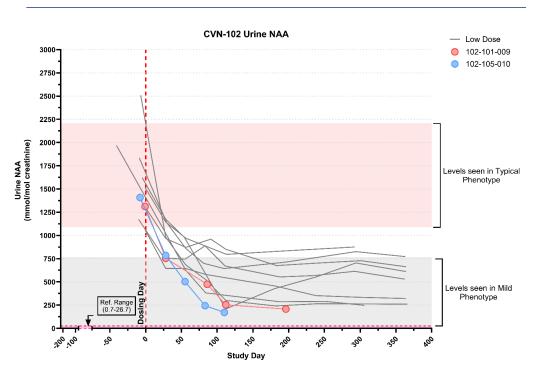


Avoid invasive neurosurgery

Provide a less invasive IV treatment option to minimize burden for patients and their caregivers

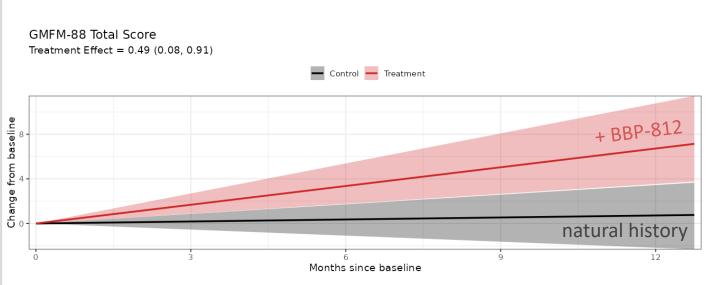
Significant, sustained reductions in NAA levels and improvement in motor function after 12-months of BBP-812 dosing in Phase 1/2 study

Urine N-acetylaspartic acid (NAA) levels



- BBP-812 reduces urine NAA from levels associated with typical Canavan disease to levels associated with mild disease
- Preliminary high-dose data suggest higher BBP-812 doses further reduce urine NAA levels

Gross Motor Function Measure (GMFM-88) Trajectory Analysis



• Trajectory analysis shows clear separation in GMFM-88 Total Score between individuals dosed with BBP-812 in treatment study (CVN-102, in red) vs. individuals in the natural history study (shown in gray).

BridgeBio Ecosystem Highlights





BBOT has progressed two potentially first-in-class molecules into the clinic with a third expected in 1H 2025

| Program | Mechanism of Action | Status |
|---|--|---------------------|
| BBO-8520 KRAS ^{G12C} ON / OFF | First direct inhibitor of KRAS^{G12C} ON Inhibits both KRAS^{G12C} GTP (active) and GDP (inactive) states Differentiates from KRAS^{G12C} GDP (inactive)-only inhibitors | Enrolling |
| BBO-10203 RAS:PI3Kα Breaker | Blocks specific interaction between RAS and PI3Ka RAS driver agnostic (KRAS, HRAS and NRAS) Selectively blocks PI3K / AKT effector signaling in the tumor No hyperglycemia / hyperinsulinemia | Enrolling |
| BBO-11818 PanKRAS ON / OFF | Direct inhibitor of KRAS^{G12X} ON Potent panKRAS inhibitor Directly binds mutant KRAS | IND exp. Q1 2025 |

The GondolaBio pipeline features a diverse set of programs across TAs

| Indication | Discovery | Lead Optimization | IND Enabling | Phase 1 | Est. Patient Pop. (US + EU) |
|--|-----------|----------------------|-----------------|---------|--------------------------------|
| Erythropoietic Protoporphyria (EPP) | | | | | 20k |
| Galactosemia | | | | | 10k |
| Alpha-1 Antitrypsin Deficiency (AATD) | | | | | 200k |
| Neurofibromatosis Type 1 (NF1) | | | | | 200k |
| Hereditary Pancreatitis | | | | | 30k |
| Fibrous Dysplasia | | | | | 50k |
| Congenital Glycosylation Disorder Type Ia (PMM2-CDG) | | | | | 5k |
| Autosomal Dominant Polycystic Kidney Disease (ADPKD) | | | | | 300k |
| Tuberous Sclerosis Complex 1/2 (TSC) | | | | | 65k |
| Genetic Epilepsy Driven by SynGAP1 Mutations | | | | | 10k |

About Attruby and BridgeBio

About Attruby™ (acoramidis)

Attruby is the only near-complete (≥90%) stabilizer of Transthyretin (TTR) approved in the U.S. for the treatment of adult patients with ATTR-CM to reduce cardiovascular death and cardiovascular-related hospitalization. Attruby was generally well-tolerated. The most common side effects were mild and included diarrhea and abdominal pain that were resolved without drug discontinuation. BridgeBio offers an extensive suite of programs to help patients access our medicines. Visit Attruby.com for more information, including full Prescribing Information.

About BridgeBio Pharma, Inc.

BridgeBio Pharma, Inc. (BridgeBio) is a new type of biopharmaceutical company founded to discover, create, test, and deliver transformative medicines to treat patients who suffer from genetic diseases. BridgeBio's pipeline of development programs ranges from early science to advanced clinical trials. BridgeBio was founded in 2015 and its team of experienced drug discoverers, developers and innovators are committed to applying advances in genetic medicine to help patients as quickly as possible. For more information visit bridgebio.com.