bridgebio

hope through
rigorous science

Corporate Presentation

October 29th, 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/Beyonttra (acoramidis), the advantages of our business model and overall strategy, the timing of ongoing clinical trials, including the topline results from FORTIFY, the Phase 3 registrational study for BBP-418 for LGMD2I/R9, the topline results from CALIBRATE, the Phase 3 registrational study for encaleret in patients with ADH1, the anticipated timing for topline data for Phase 3 trial of infigratinib in achondroplasia, BridgeBio Oncology Therapeutics' and Gondola Bio's clinical trials and pipelines, the clinical, therapeutic and market potential of our clinical development programs and our 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/EMA, PMDA and MHRA-approved Beyonttra, the potential of acoramidis to be used to delay the onset of, or prevent ATTR, , the potential expansion opportunity for infigratinib and encaleret, our financial position, including our expectations regarding reaching regulatory and commercial milestones, 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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Attruby® (acoramidis) 356 mg tablet

…is FDA-approved for ATTR-CM in the US

BEYONTTRA® (acoramidis) 356mg tablet

…is approved in the EU, Japan, and UK

Two positive Phase 3 trial readouts announced this week

Positive Phase 3 results announced

fortify

Phase 3 trial met all primary and key secondary interim analysis endpoints in **Limb-Girdle Muscular Dystrophy 2I/R9**

Positive Phase 3 results announced

CALIBRATE

Phase 3 trial met all primary and key secondary endpoints in **Autosomal Dominant Hypocalcemia Type 1**

PROPEL3

Phase 3 trial of infigratinib in **Achondroplasia** fully enrolled; topline data expected in Q1 2026

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 program must be:

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

Inspiration – "Strategy: A History" by Lawrence Freedman

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

Inspiration – "Financing drug discovery for orphan diseases" by Andrew Lo

The right approach: decentralized R&D, centralized infrastructure

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

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

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

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

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

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

Inspiration – "Scale" by Geoffrey West

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

Genetics

Macromolecules

Clinical Diagnosis

Therapeutic Modalities

Regulatory

nature medicine — **Genetic association analysis of 77,539 genomes reveals rare disease etiologies**

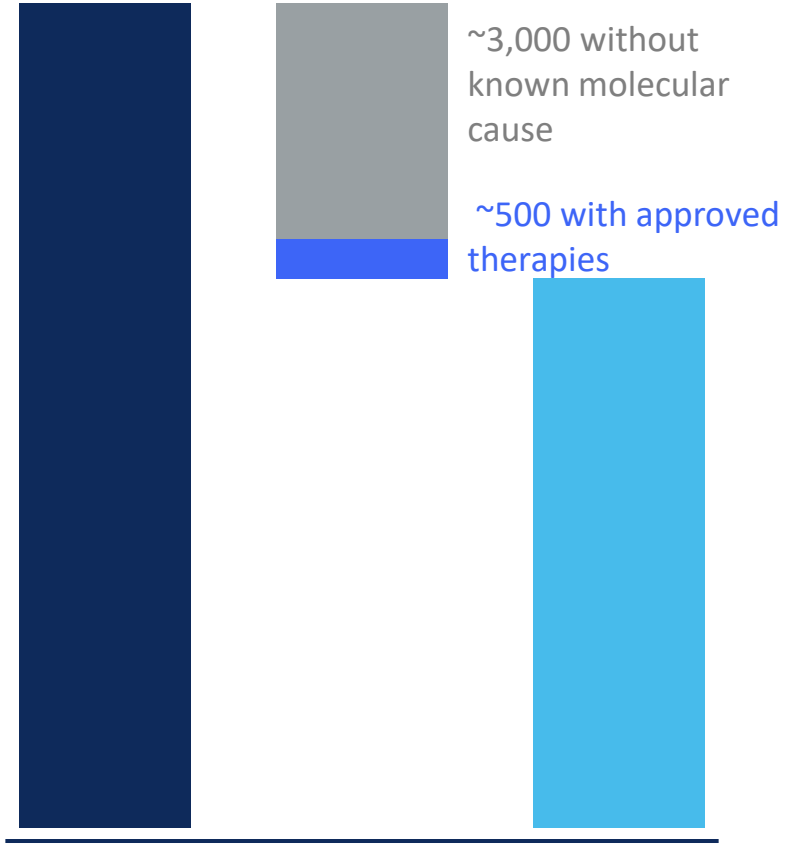
Science JOURNALS AAAS — **Accurate proteome-wide missense variant effect prediction with AlphaMissense**

nature reviews genetics — **The expanding diagnostic toolbox for rare genetic diseases**

nature biotechnology — **New drug approvals reached an all-time high in 2023, with five gene therapies, the first CRISPR–Cas9-edited therapy and a disease-modifying Alzheimer's drug.**

FDA — **FDA issues final guidance on rare disease drug development**

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

Greene, D. et al. Nat Med. 2023; Cheng, J. et al. Science. 2023; Kernohan, K.D., Boycott, K.M. Nat Rev Genet. 2024; Senior, M. Nat Biotechnol. 2024; FDA; NIH; RareX; Science; Nature; NEJM; NHGRI

Our leadership team has world-renowned drug hunters and operators

Neil Kumar, PhD
Founder and
Chief Executive Officer
THIRD ROCK VENTURES · MyoKardia

Thomas Trimarchi, PhD
President and
Chief Financial Officer
REGENERON · Goldman Sachs

Christine Siu
Chief Executive Officer,
Muscular Dystrophy
THIRD ROCK VENTURES · GBT

Ananth Sridhar
Chief Operating Officer,
Cardiorenal
REGENERON · Genentech

Justin To
Chief Executive Officer,
Skeletal Dysplasias
flatiron · McKinsey & Company

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

Jonathan Fox, MD, PhD
Chief Medical Officer,
Cardiorenal
MyoKardia · AstraZeneca

Uma Sinha, PhD
Chief Scientific Officer
GBT · PORTOLA PHARMACEUTICALS

Adora Ndu, PharmD, JD
Chief Regulatory Officer & EVP
Portfolio Strategy and Management
BIOMARIN · FDA

Josh Loehrer
Chief People Officer
Clarify · McKinsey & Company

Damian Wilmot
Chief Legal Officer
VERTEX · GOODWIN

Maricel Apuli
Chief Accounting Officer
pwc · GUARDANT

Charles Homcy, MD
Founder and Chairman of
Pharmaceuticals
MyoKardia · GBT

Frank McCormick, PhD
Founder and Chairman of
Oncology
ONYX PHARMACEUTICALS · UCSF

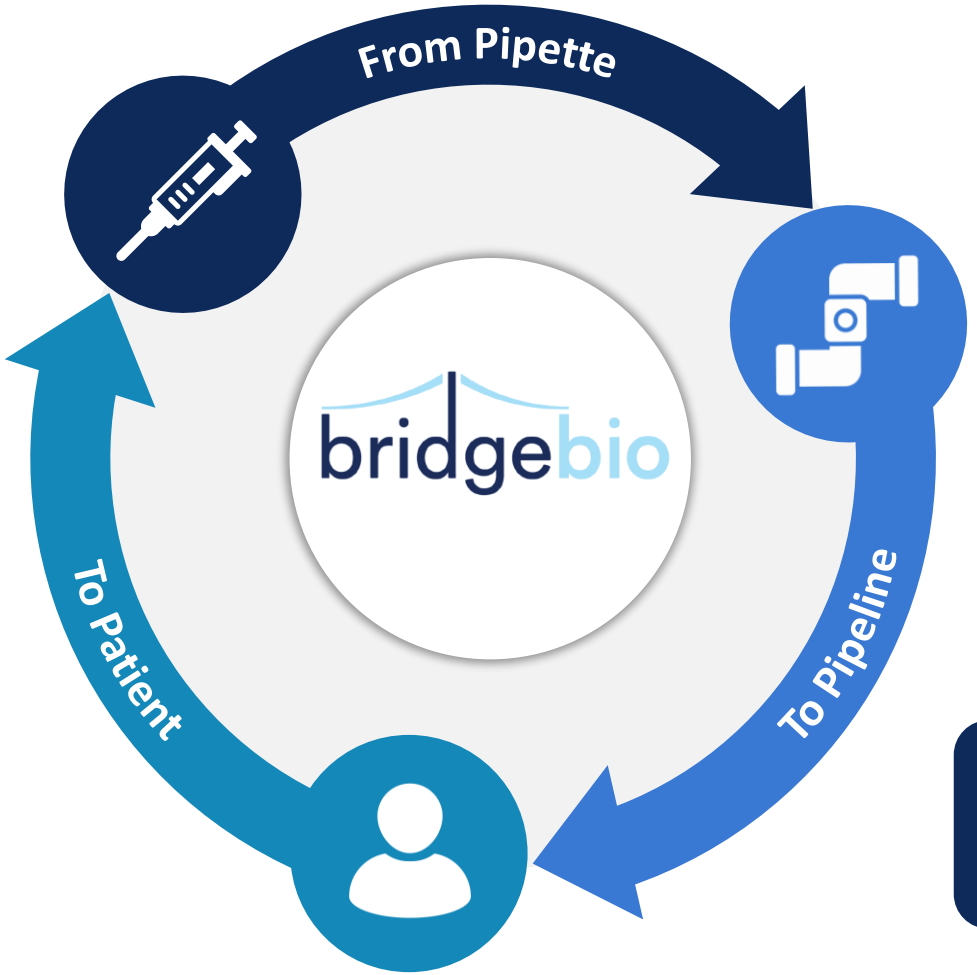
Richard Scheller, PhD
Chairman of R&D
Genentech · 23andMe

Len Post, PhD
Advisor
BIOMARIN · LEAD THERAPEUTICS

Phil Reilly, MD, JD
Advisor
THIRD ROCK VENTURES · bluebirdbio

Robert Zamboni, PhD
Chemistry
MERCK FROSST

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



Attruby™ (acoramidis) 356 mg tablet

Nulibry® (fosdenopterin) for injection

TRUSELTIQ™ (infigratinib) capsules

Obtained Approval for 3 Medicines

> 2,000 patients in trials

> 30 trials
> 30 countries
> 500 sites

3 fully enrolled pivotal trials

+ 2 Positive Phase 3 Results

> 70 papers published

> 35 academic partnerships

19 INDs created

< $10M and **< 3 yrs** to IND

We leverage lean operating teams that advance medicines quickly and efficiently for patients

Data on file.

We target well-described diseases at their source, underpinning our industry-leading probability of technical success

Our approach

1. Characterize the genetic source of the disease

2. Define the function of the genetic drivers

3. Drug the target at its source

Probability of Technical Success (POTS)

Clinical Dev. Stage	Industry Benchmark[1]	BBIO Historical[2]
Phase 1	52%	**86%**
Phase 2	29%	**71%**
Phase 3	58%	**83%**
Cumulative	9%	**51%**

[1] Biotechnology Innovation Organization (BIO), QLS Advisors, and Informa Pharma Intelligence, *Clinical Development Success Rates and Contributing Factors 2011–2020* (2021).
[2] Internal data on `file (2015 – 2025). Includes programs initiated at BridgeBio that continue to be advanced at sister companies BridgeBio Oncology Therapeutics and GondolaBio.

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)					✓	500,000+	$20B+
Nulibry (fosdenopterin)	Molybdenum Cofactor Deficiency (MoCD) Type A					✓	<100	Partnered
Truseltiq (infigratinib)	Cholangiocarcinoma					✓	37,000	Partnered
Infigratinib	Achondroplasia (ACH)					*Fully Enrolled*	55,000+	$2B+
	Hypochondroplasia (HCH)				*Observational Run-in Fully Enrolled*		55,000+	$2B+
BBP-418	Limb-Girdle Muscular Dystrophy Type 2I/R9 (LGMD2I/R9)					*Topline Readout*	7,000+	$1B+
Encaleret	Autosomal Dominant Hypocalcemia Type 1 (ADH1)					*Topline Readout*	25,000+	$1B+
	Chronic Hypoparathyroidism (HP)						200,000+	$1B+
BBP-812	Canavan Disease				*Phase 1/2 Pivotal*		1,000	TBD
BridgeBio Oncology Therapeutics	3 Oncology Programs (various indications)		**~17% ownership***				Various	Various
GondolaBio	17 Rare Disease Programs (various indications)			**~29% 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

- Beyonttra: EU, Japan, UK approvals
- Q1 Earnings Call
- Q2 Earnings Call
- Secured $300M royalty financing via HCRx and Blue Owl

2H 2025

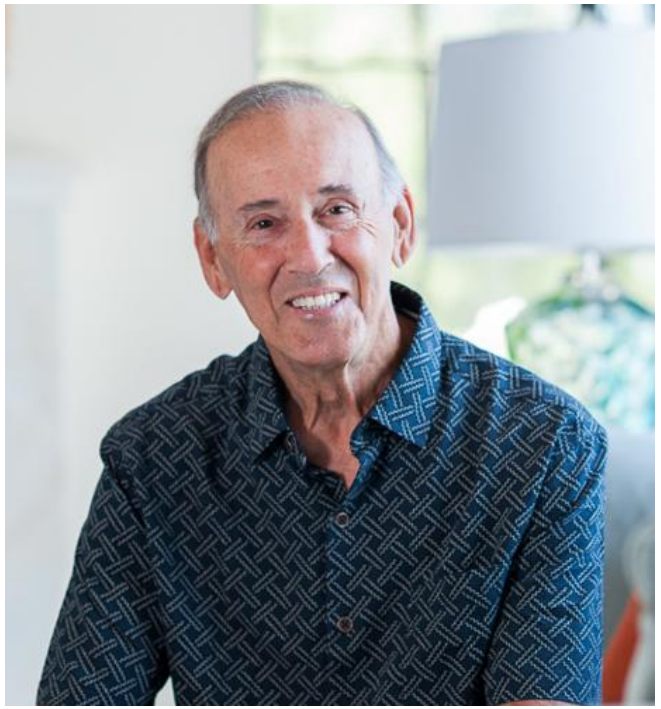
- Q3 Earnings Call
- Encaleret: LPLV, Topline
- BBP-418: LPLV, Topline
- Infigratinib: LPLV

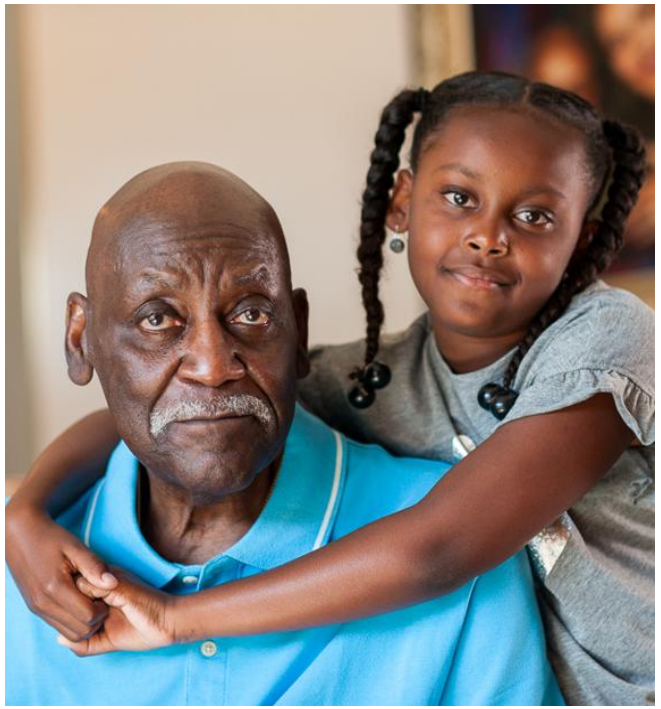
bridgebio vision for 2030:

De-risked PYS **>$8B**

Lives impacted **>100k lives**

Attruby®
Approved for ATTR-CM





Key Attruby highlights

Attruby® (acoramidis) 356 mg tablet

- First and only approved product with a label specifying **near-complete stabilization** of TTR
- **Effect seen as early as 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

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

Dedicated patient support team

Insurance navigation and support

Ensuring easy and fast access:
Attruby can be delivered to a patient's door in <48 hours

The least expensive ATTR treatment:
$0 OOP for many patients
Max $167 per month for Medicare Part D

Drug available direct to patients' homes or through institutions

Easily accessible Copay and Patient Assistance Program

Nationally, more than 90% of Medicare Part D patients have access to Attruby with approved prior authorization (PA)

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

Free Trial	28-day supply for patients new to Attruby *(newly diagnosed or switching from another therapy)*
Distribution Network	Independent and rare/orphan disease–focused Specialty Pharmacies (SPs) where Institutional SPs can keep prescription
Field Access Teams	Dedicated Attruby Field Reimbursement Managers and Patient Access Liaisons
Patient Assistance Programs	Easily accessible Copay and Patient Assistance Program

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, continued commercial momentum

5,259

Unique U.S. patient prescriptions for Attruby

1,355

Unique U.S. prescribing HCPs

$108.1M

Q3 Attruby net product sales

As of October 25, 2025

Q3 2025

Beyonttra® (acoramidis) has seen rapid EU uptake with leading guideline endorsements

BEYONTTRA®
(acoramidis) 356mg tablet

Beyonttra (acoramidis) Demonstrates Robust Commercial Uptake in Germany, Exceeding Projections

47% NBRx share	Achieved by Germany 6 months into launch (as of October 15, 2025)
Market Share	Prescriber feedback from several leading German centers indicates >50% acoramidis initiation among newly eligible ATTR-CM patients; a few centers describe near-exclusive use
National Tender	Denmark awarded the national tender to Beyonttra, establishing it as the only first-line treatment for ATTR-CM and suggesting that patients switch from tafamidis
Strong Uptake	Germany's strong uptake signals powerful Demand ahead for soon-to-launch EU markets

Global Guidelines Endorse Beyonttra (acoramidis) for Clinical Confidence, Credibility, and Impact

Acoramidis endorsed with highest tier recommendation and highlighted as near-complete stabilizer while tafamidis described as partial stabilizer

References: 1. JCS/JHFS 2025 Guideline; J Card Fail. 2025 Mar 27; S1071-9164(25)00100-9;2. iCARDIO Alliance HF 2025, Chopra V. et al., Heart Lung Circ 34(7):e55–e82. 3. Marques N, Aguiar Rosa S, Cordeiro F, et al. Portuguese recommendations for the management of transthyretin amyloid cardiomyopathy (Part 1 of 2): Screening, diagnosis and treatment. Rev Port Cardiol. 2025;44(1):7-48. doi:10.1016/j.repc.2024.12.002. 4. IQVIA™ LRx (Germany). NBRx share for acoramidis; period ending 15 Oct 2025. Definitions: JCS=Japanese Circulation Society; JHFS=Japanese Heart Failure Society; MHLW=Ministry of Health, Labour & Welfare; ROW=Rest of World; NBRx=New to brand prescriptions.

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 auto-immune 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

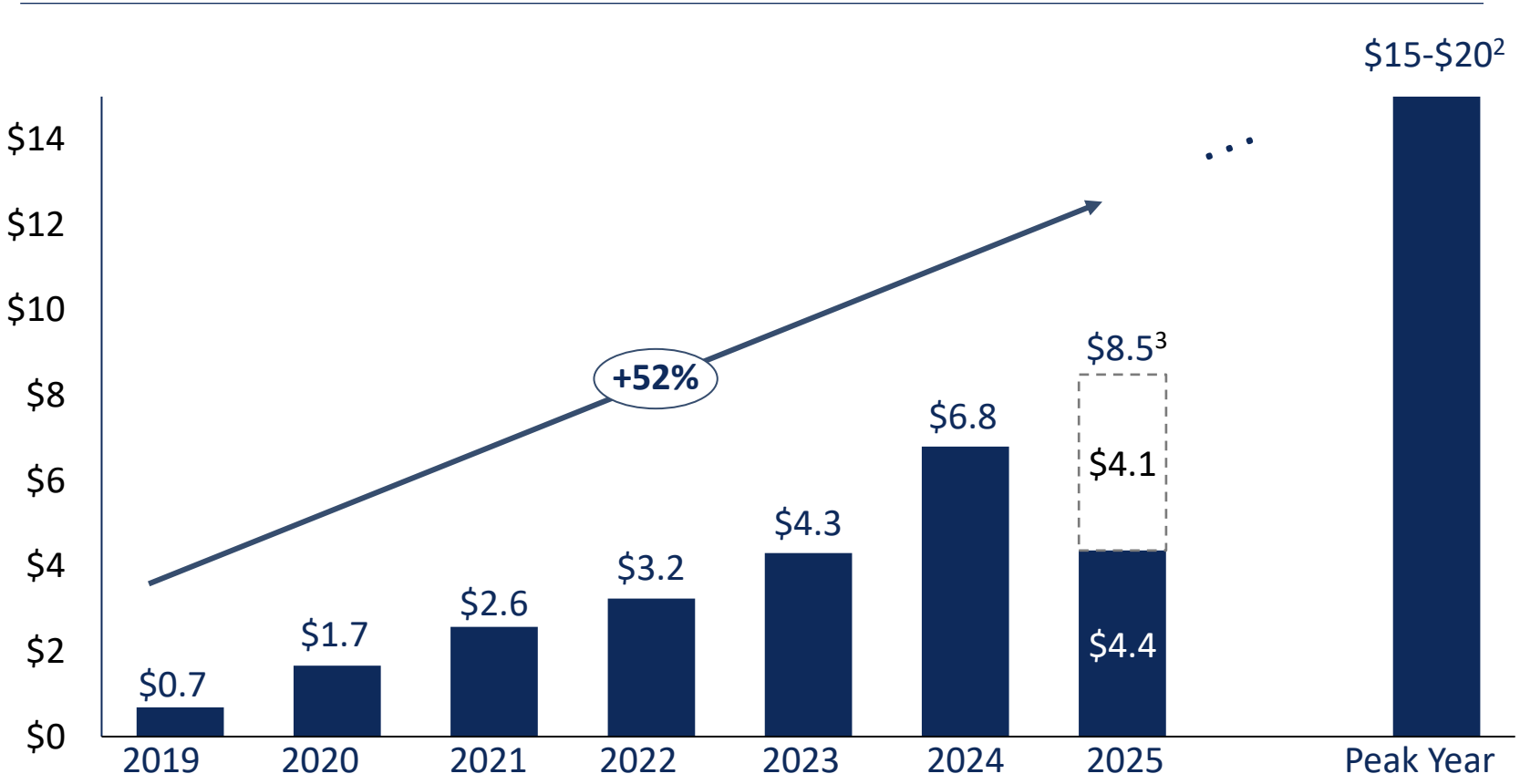
Liz Arnold
Head of Commercial Strategy

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 sales[1]



Market growth drivers include:

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

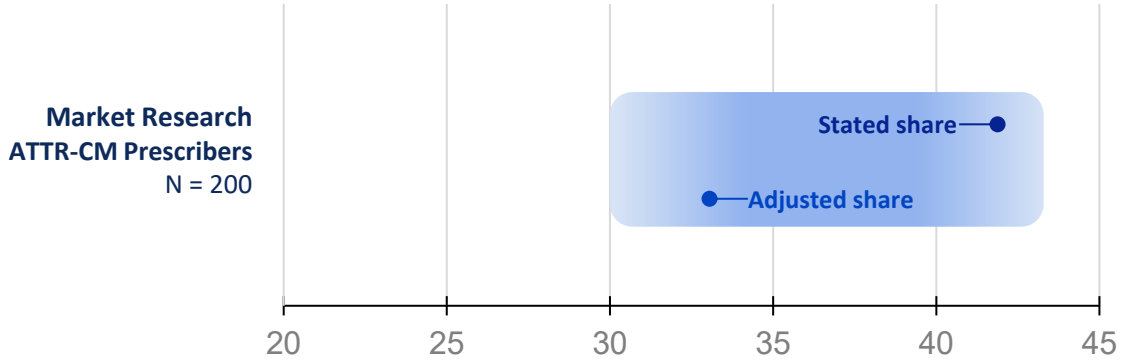
[1]ATTR market includes all approved drugs for ATTR-PN and ATTR-CM.
[2]Consensus estimates of $15B-$20B ATTR market.
[3]Extrapolated from Q1, Q2, and disclosed Q3 numbers (annualized)

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

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

Attruby Market Share as % of Future Competitive Market

(August 2024 Research)



- 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

Note: Stated share adjusted for historical performance of demand study market research on new product launches.
BridgeBio Market Research (N=200), Advisory board, IQVIA LAAD data set

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

2024

ATTRibute-CM

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

2025+

ATTRibute-CM

ATTR-CM
WT and hereditary
Attruby lifecycle management and further analyses of pivotal study data

actearly

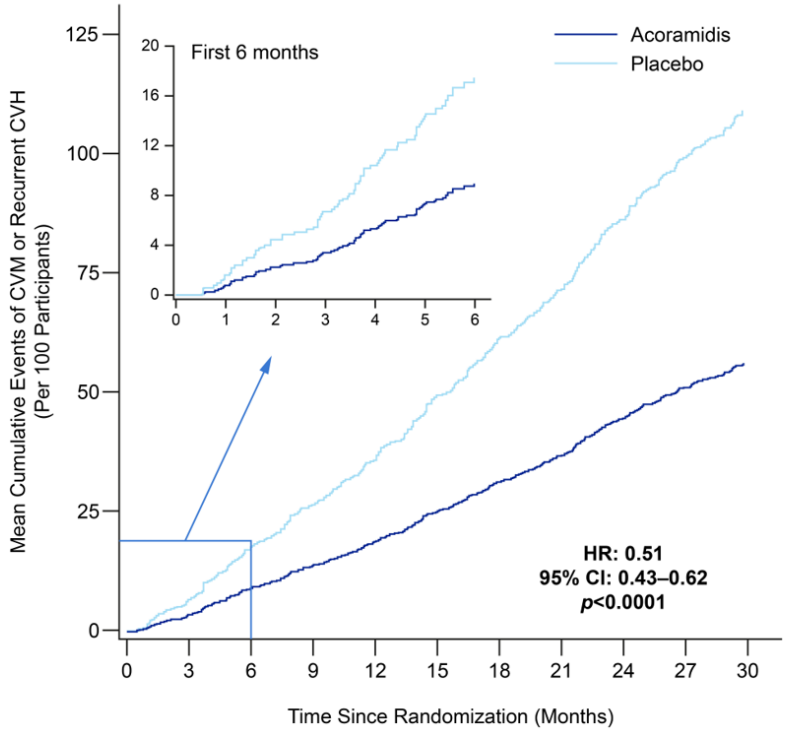
ATTR-CM
Hereditary
Prevention study

Additional real-world evidence generation and publication plan to further differentiate Attruby

Multi-prong data generation plan in place to solidify Attruby's differentiated clinical profile

Acoramidis significantly reduced cumulative cardiovascular outcomes within the first month of treatment

Figure: 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

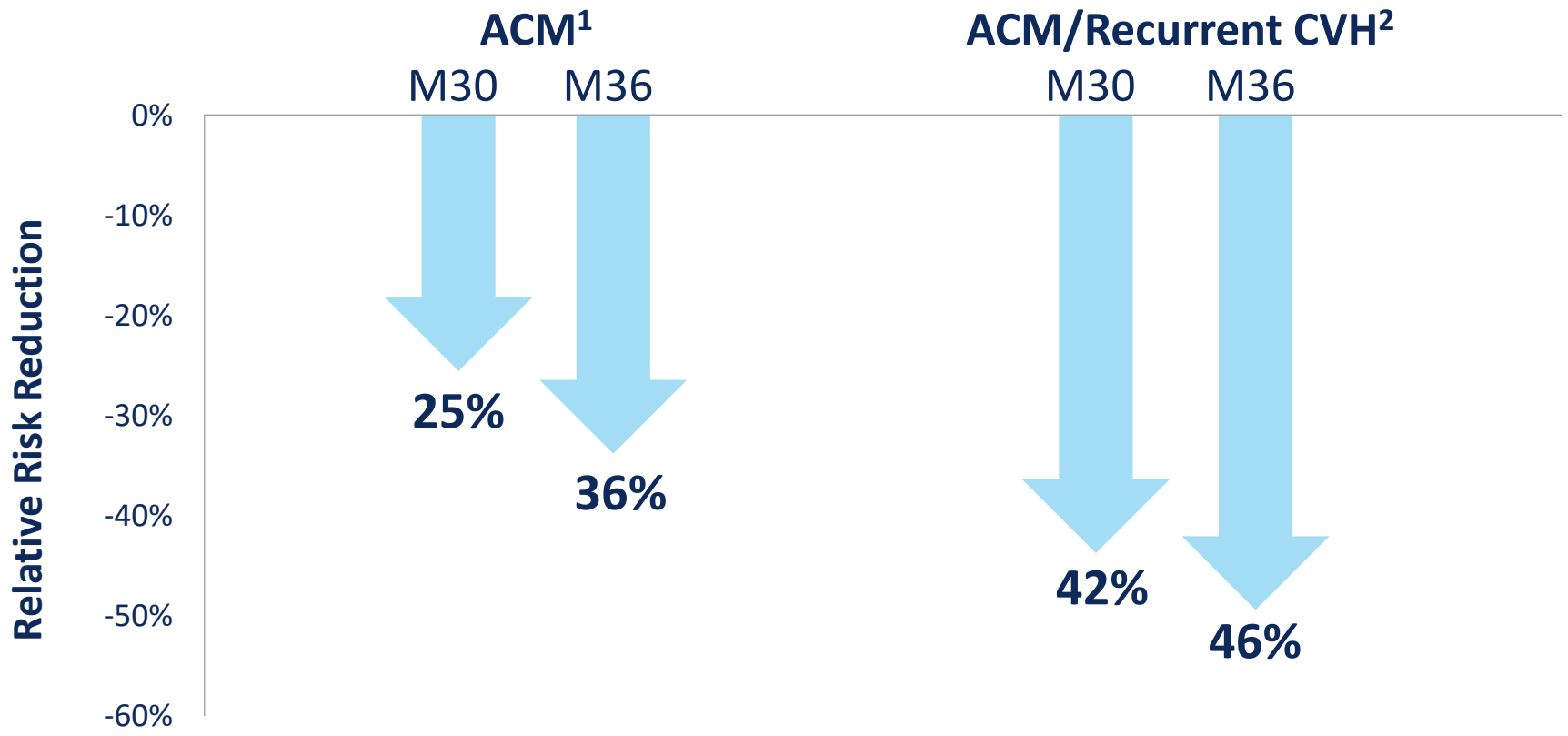
Data are for the mITT population in the ATTRibute-CM study, defined as all the participants who had undergone randomization, received at least one dose of acoramidis or placebo, and had at least one efficacy evaluation after baseline; participants with eGFR < 30 mL/min/1.73 m^2 were excluded.

CVM was defined as death adjudicated as cardiovascular or undetermined cause and included receipt of a heart transplantation or cardiac mechanical assist device implantation. CVH was defined as non-elective admission to an acute care setting for cardiovascular-related morbidity (>24 hours) and urgent outpatient visits (<24 hours) for decompensated heart failure requiring intravenous diuretics.

[a]Modified 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

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



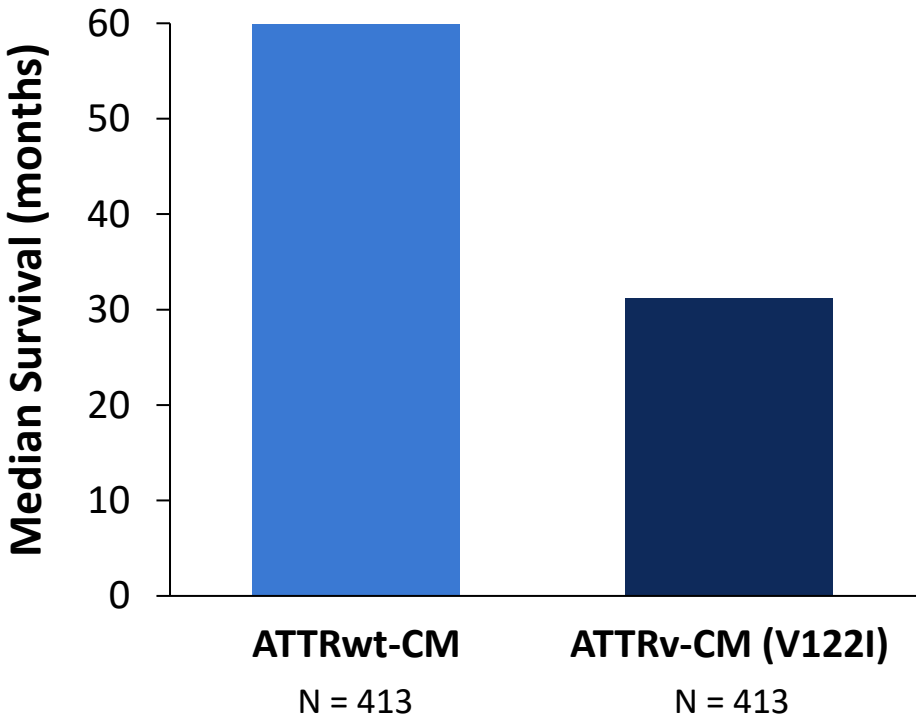
Attruby resulted in a **statistically significant** ACM and ACM/Recurrent CVH relative risk reduction at **both Month 36 and Month 42**

Source: Judge et al., Circ 2024. [1]Relative Risk Reduction based on percent of participants with ACM event at M30 and M36. [2]Relative Risk Reduction is calculated by (1-Relative Risk Ratio from the negative binomial regression analysis) x 100%. Note: Study limitations include lack of a true control treatment arm, and the exclusion of participants who elected not to enroll in the OLE due to desire to receive SOC.

Attruby delivers outstanding results in patients with poor prognosis

Natural History

V122I ATTRv-CM has an aggressive phenotype and poor prognosis[1]



ATTRibute-CM mITT Population

Statistically significant benefit on composite ACM or first CVH in ATTRv-CM participants vs. placebo[2]

	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

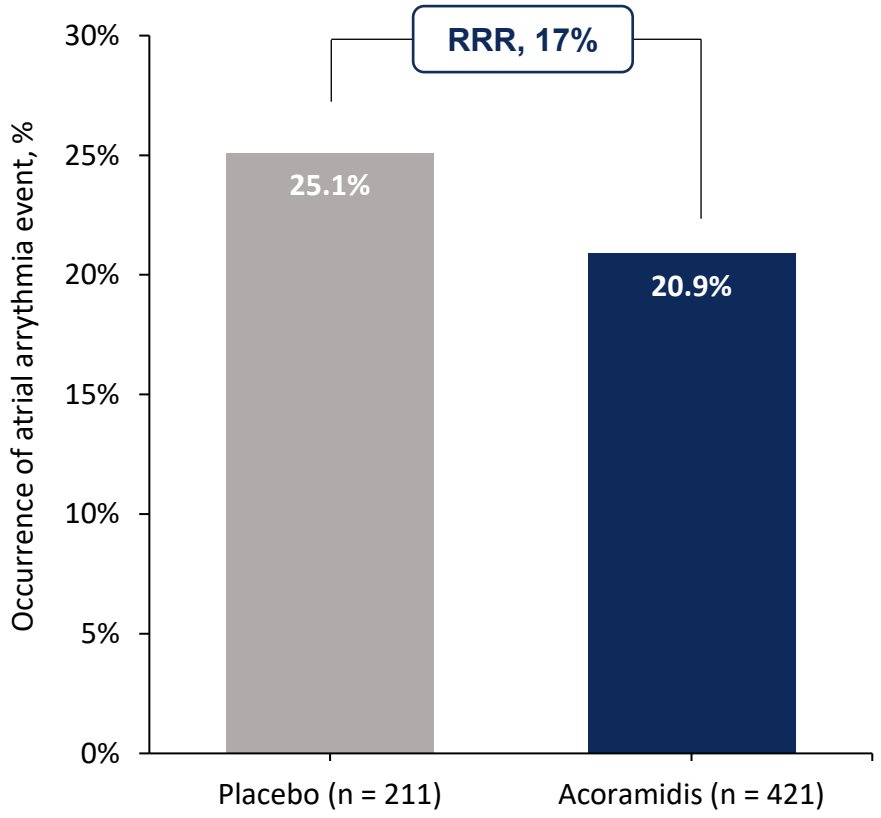
Unprecedented improvements in QOL and Functional Capacity in Variant ATTR-CM[3]

	LS Mean Difference Acor vs. Placebo	p value
6MWD	+86.7 meters	0.0048
KCCQ-OS	+20.3 points	0.0019

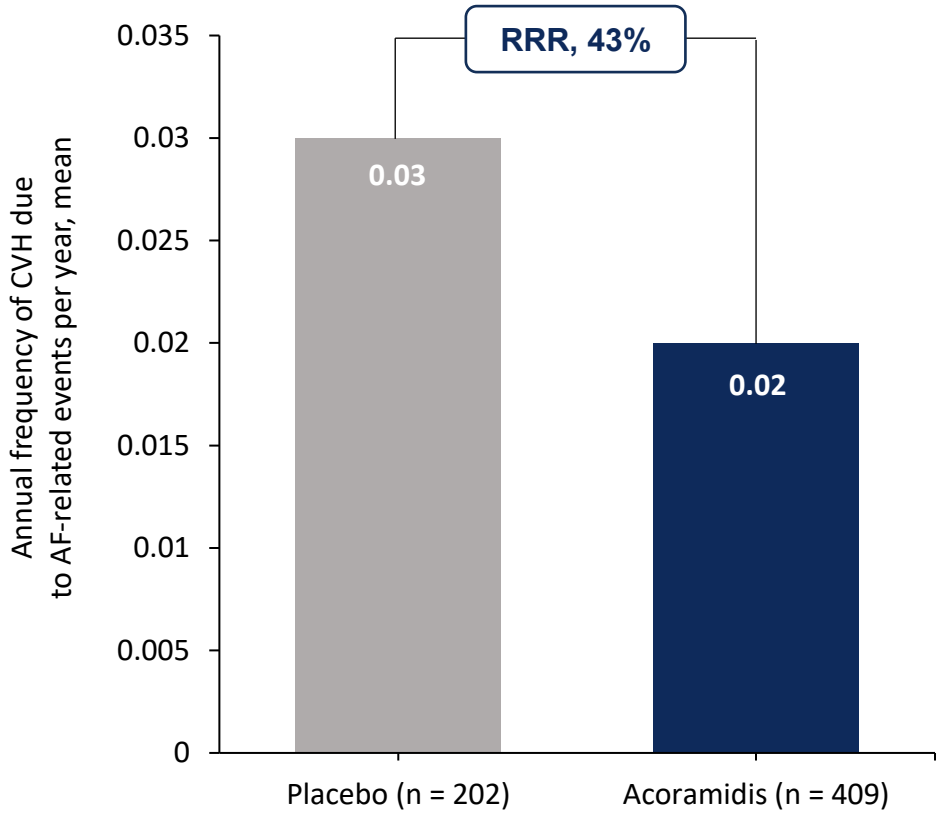
[1]Razvi, Y. et al., Eur Jour Heart Fail., 2024;26(2):383-393. [2]BridgeBio Data on File. [3]Fontana et al., Effect of acoramidis on functional capacity and quality of life in patients with variant ATTR-CM: Results from ATTRibute-CM Presented at ESC-HF 2025. Overall population and ATTRv-CM data vs. placebo. V122I, also known as V142I under an alternative naming convention, are both the same variant.

Acoramidis reduced the risk of AF adverse events and AF-related cardiovascular hospitalizations

Incidence AF Adverse Event



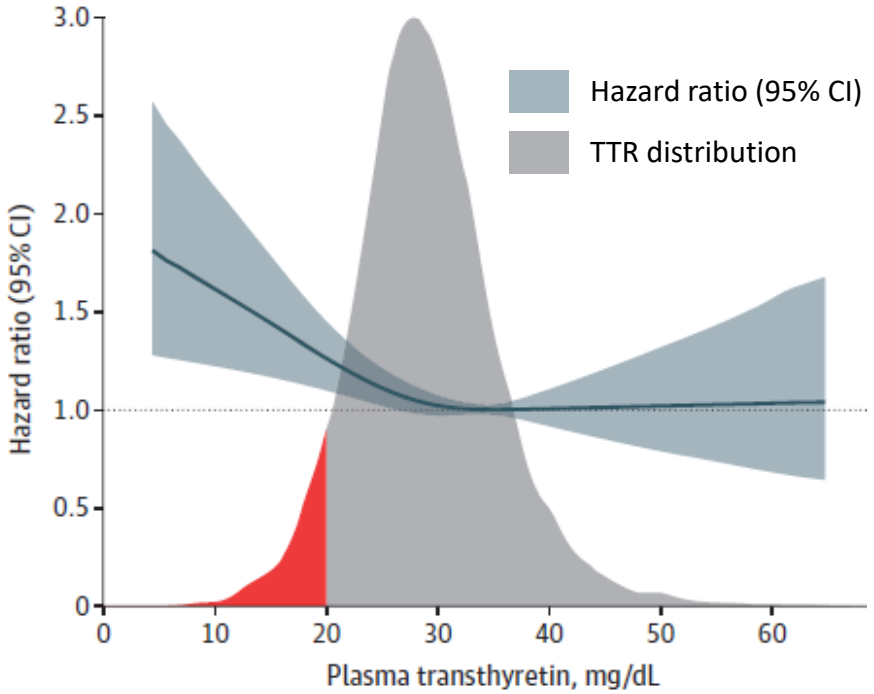
Annual Frequency of CVH Due to AF



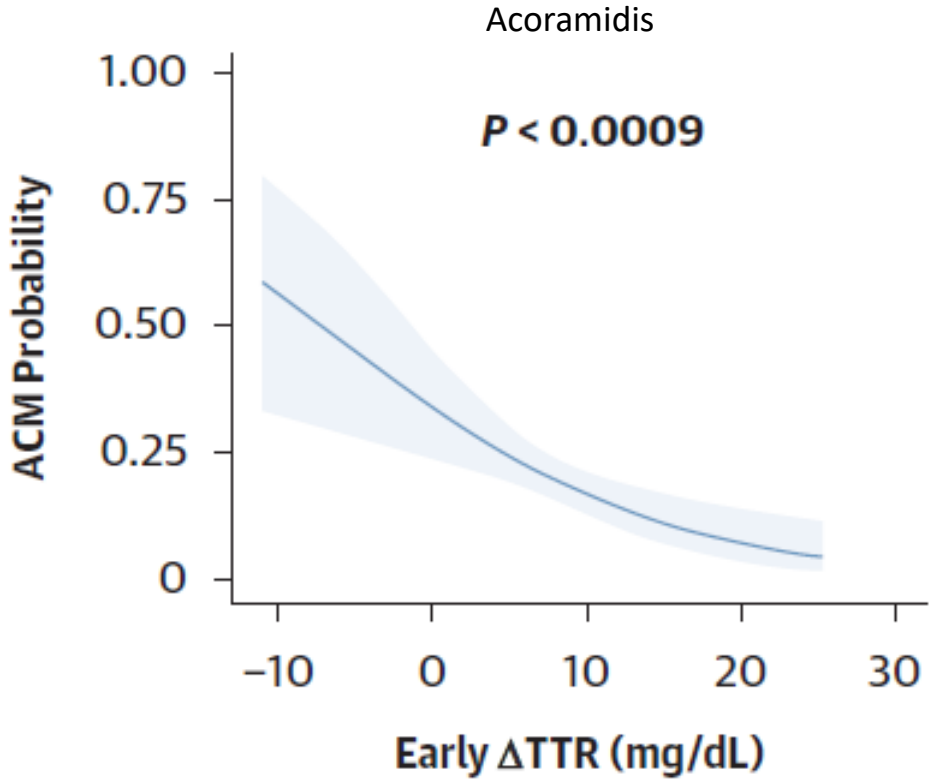
Source: Alexander, KM et al. Presented at: The Annual Congress of the Heart Failure Association of the European Society of Cardiology 2025;
Note: AF, atrial fibrillation; AFL, atrial flutter; AF-type adverse event covers the MedDRA preferred terms Atrial fibrillation, Atrial flutter, and Cardiac flutter.

Additional data: Elevated TTR levels are associated with improved survival

Higher TTR concentration associated with greater life expectancy (N≈102K individuals)[1]



Each 5 mg/dL rise in serum TTR is associated with ~32% lower odds of death by 30 months



[1]Figure adapted from Christoffersen, M. et al., JAMA Cardiology, 2024. Figure 3D: Cardiovascular mortality, adjusted multifactorially.
[2]Maurer et al. Early Increase in Serum Transthyretin by Acoramidis Independently Predicts Improved Survival in TTR Amyloid Cardiomyopathy (JACC 2025)

Recruitment continues for ACT-EARLY, the first ATTR primary prevention clinical trial, opening a new potential therapeutic paradigm for patients

actearly

Current therapeutic paradigm: slow the progression of disease in patients with diagnosed ATTR

130+ **ATTR Variants**

~600 **Participants**

~100 **Global Sites**

~24 **Countries**

ACT-EARLY is exploring whether acoramidis has the potential to be used to delay the onset of, or prevent, ATTR

Encaleret

encaleret for Autosomal Dominant Hypocalcemia Type 1 (ADH1)

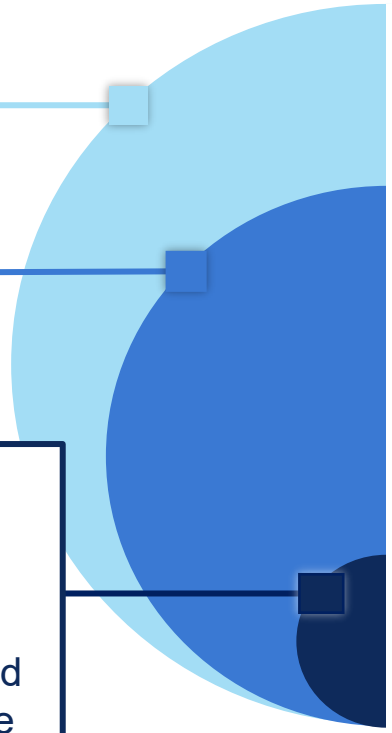
A sincere THANK YOU to the patients, families, advocates, investigators, clinical research staff, referring providers, and collaborating research partners

There are no therapies currently indicated to treat ADH1, a serious and rare genetic condition

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]

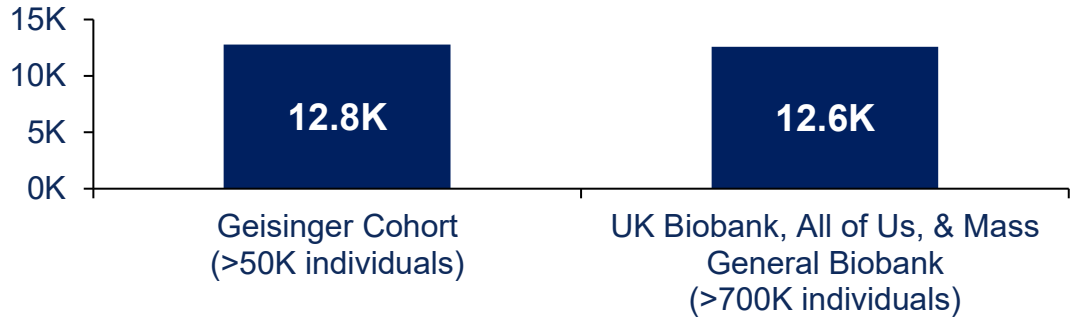
3K – 5K Currently Addressable
40-60% of symptomatic ADH1 patients are estimated to be diagnosed today; anticipate increase



An analogous ADH1 market is XLH

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

ADH1 variant frequency estimates in literature[1-2]



[1]Dershem, et al. Amer Jour of Hum Genetics, 2020. [2] Chang, et al. Am J Hum Genet., 2025. [3]Roszko, et al. JBMR. 2022. [5]Dahir, et al. Jour Endo Soc., 2020. [6]Evaluate Pharma.
XLH = x-linked hypophosphatemia.

Encaleret is an investigational, potential first-in-class therapy that targets the underlying disease mechanism of ADH1

Design Principles

Only investigational treatment directly targeting ADH1 at its source
Potential to restore physiologic mineral homeostasis that is disrupted by CaSR oversensitivity

Address common symptomatology
Designed to normalize PTH, serum Ca, and urine Ca levels, potentially correcting the root cause of neuromuscular and renal consequences

Convenient oral dosing
First potential targeted treatment for ADH1 in a convenient form for patients and providers

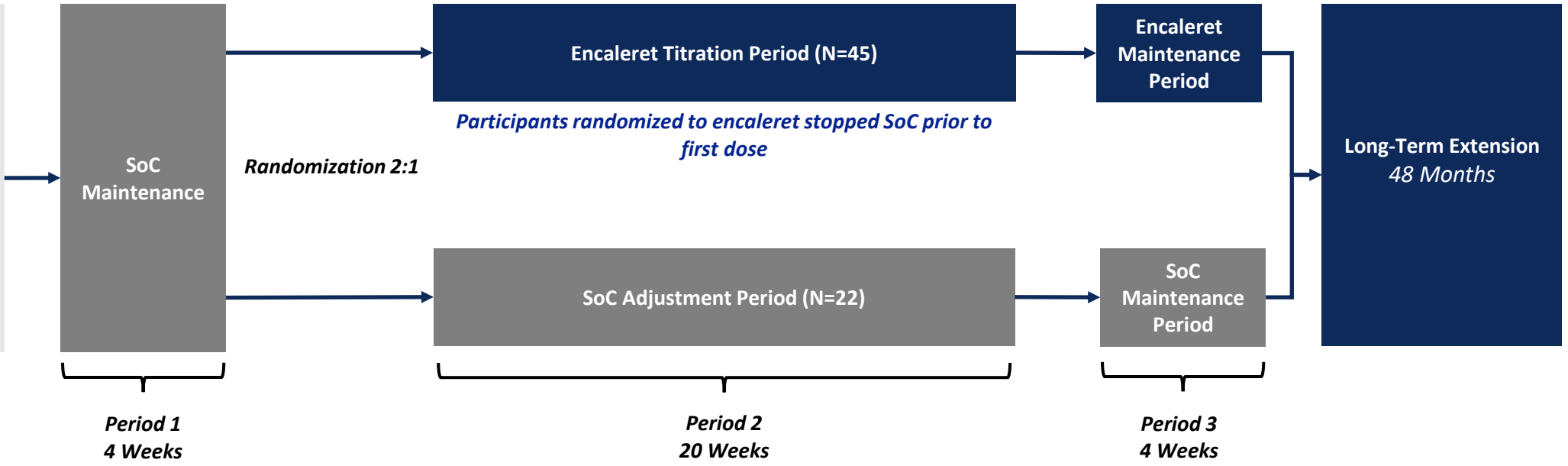
CaSR = calcium-sensing receptor; PTH = parathyroid hormone; Ca = calcium.

Phase 3 study (CALIBRATE) of encaleret in ADH1

CALIBRATE

Screening and SOC Optimization Period

- Age ≥ 16 years of age
- Documented pathogenic variant, or variant of uncertain significance, of the *CASR* gene associated with biochemical findings related to hypoparathyroidism



Primary Composite Endpoint:

- Proportion of participants achieving:
  - Corrected Ca[1] within the target range of 8.3-10.7 mg/dL

    AND
  - 24-hour urine Ca within the reference range (<300 mg/day for men & <250 mg/day for women)

Key Secondary Endpoint:

- Proportion of participants achieving iPTH above the lower limit of the reference range

Select Secondary Endpoints:

- 1,25-$(OH)_2$ Vitamin D, magnesium, and phosphate
- Bone turnover markers
- Renal ultrasound and renal function

[1]Albumin-corrected calcium.
SoC = standard of care; a combination of oral activated Vitamin D and/or calcium supplements. *CASR* = calcium-sensing receptor gene. iPTH = intact parathyroid hormone.

76% of participants randomized to encaleret met the primary endpoint, achieving serum calcium and 24-hour urine calcium in the target ranges

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

Key Secondary Analysis – Between Group at Week 24 (Encaleret vs. SoC) responder status confirmed a statistically significant result (p<0.0001)[5]

[1] Albumin-corrected serum calcium. [2]The primary endpoint assessed responder status of participants who achieved both corrected calcium and 24-hour urine calcium in the target range at the completion of the maintenance periods. [3]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. [4]Analyzed by McNemar's test. [5]Analyzed by Barnard's unconditional exact test.
CI = Confidence Interval.

Encaleret was well-tolerated with no TEAEs resulting in encaleret or study discontinuation

	Period 1	Periods 2 and 3	
	SoC N=67	SoC N=22	Encaleret N=45
Participants experiencing any Serious TEAE	2 (3%)	3 (14%)	4 (9%)
Serious Related TEAE	1 (2%)	0 (0%)	1 (2%)
Participants experiencing any TEAE	30 (45%)	14 (64%)	40 (89%)
Mild	23 (34%)	6 (27%)	21 (47%)
Moderate	4 (6%)	6 (27%)	16 (36%)
Severe	3 (5%)	2 (9%)	3 (7%)
Related TEAE	4 (6%)	0 (0%)	16 (36%)
TEAE of Hypocalcemia	4 (6%)	3 (14%)	3 (7%)
TEAE of Hypercalcemia	3 (5%)	0 (0%)	10 (22%)
TEAE Leading to Study Discontinuation	0 (0%)	0 (0%)	0 (0%)

For each category, participants are included only once, even if they experienced multiple events in that category. Relatedness assessed on the basis of the investigational product being administered in the respective study period reported.
TEAE = Treatment-Emergent Adverse Event

Encaleret was found to restore physiologic mineral homeostasis through its action on the CaSR

- 76% of participants randomized to encaleret achieved serum and urine calcium in the target range compared to the same individuals on conventional therapy (difference 71%, p<0.0001)[1,2]
  - Among encaleret responders at Week 24, none required conventional therapy during Period 3[3]
- Clinically meaningful restoration of intact PTH in participants administered encaleret compared to the same individuals on conventional therapy (difference 84%, p<0.0001)[1]
- Clinically meaningful increase in corrected serum calcium (p<0.0001) and decrease in 24-hour urine calcium excretion (p<0.0001) at Week 24
- Similar changes for the above parameters were also demonstrated between treatment arms
- Encaleret was well tolerated, with no discontinuations related to study drug

[1]Analyzed by McNemar's test. [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]Requirement for conventional therapy defined as oral calcium >600 mg/day and/or active vitamin D during Period 3.
Encaleret is an investigational drug. Its safety and efficacy have not been fully evaluated by any regulatory authority.

NDA submission planned in the first half of 2026 with two additional registrational studies planned for initiation next year

Advance encaleret towards registration in ADH1

Submit New Drug Application to FDA
1H 2026

Submit Marketing Authorization Application to EMA
2H 2026

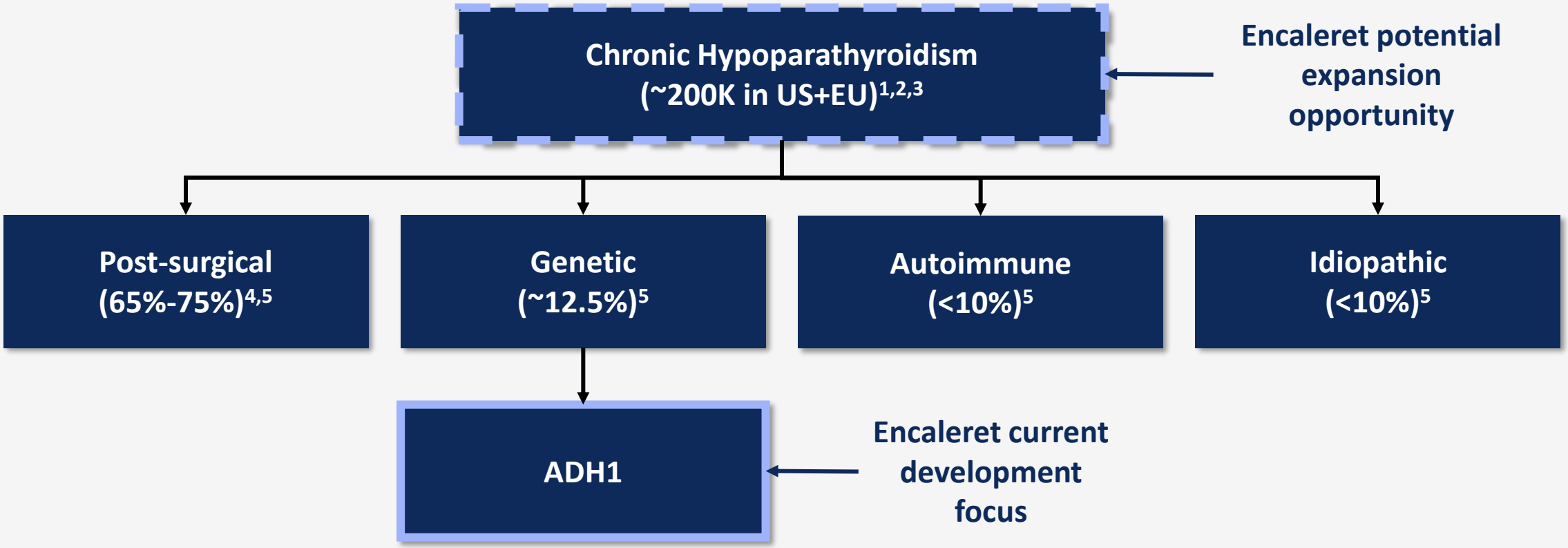
Generate clinical evidence for encaleret in additional patient populations

Initiate Phase 2/3 study in Pediatric ADH1
1Q 2026

Initiate Phase 3 study in chronic hypoparathyroidism
2026

CALIBRATE results planned for presentation at relevant medical conference in 1H 2026

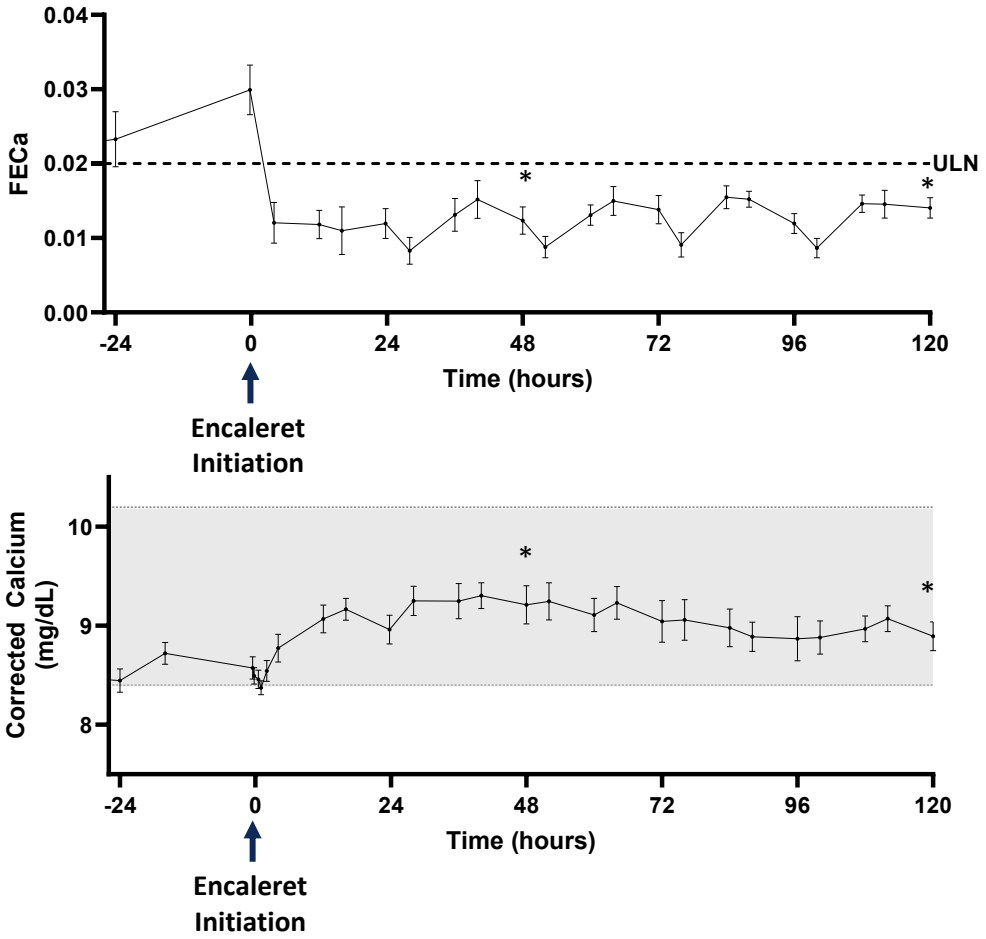
Encaleret also has potential as an oral medication for other etiologies of chronic hypoparathyroidism (HP)

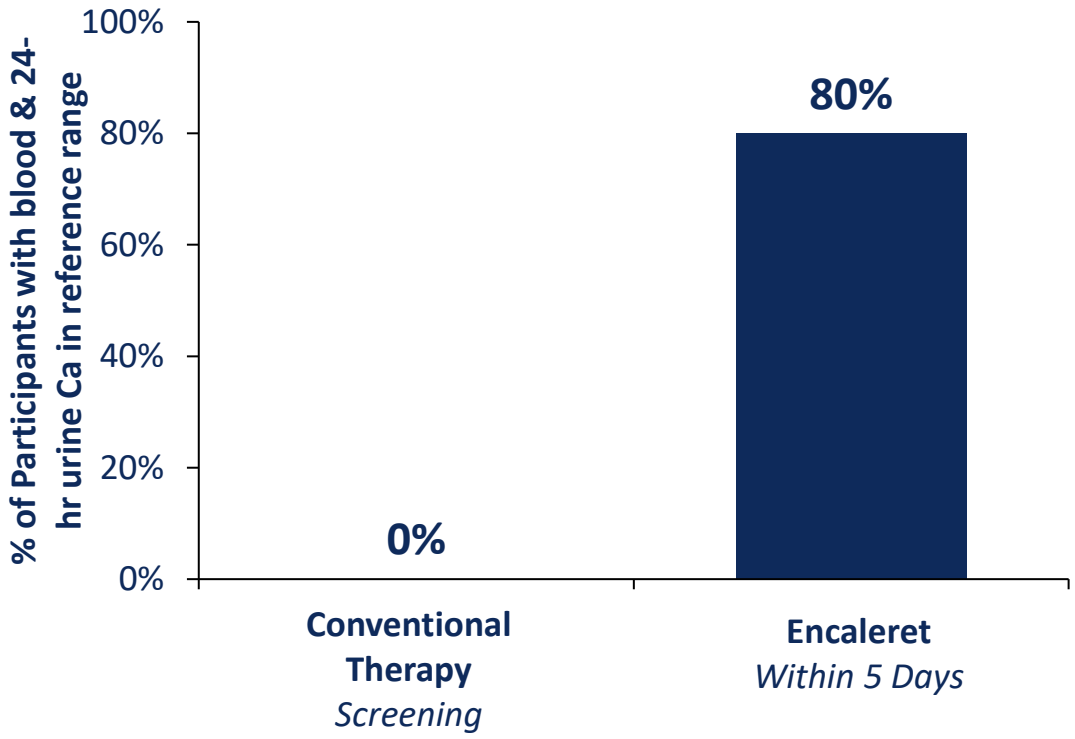


[1]Powers et al., J. Bone Miner. Res., 2013. [2]Clarke et al., J. Clin. Endocrinol. Metab., 2016. [3]Underbjerg et al., J. Bone Miner. Res., 2013. [4]Mannstadt et al., Nat. Rev. Dis. Primers., 2017. [5]Kiam et al., J. Clin. Endocr., 2022.

Preliminary data supports potential expansion to chronic HP, showing rapid, PTH-independent, effect on blood and urine calcium

Proof-of-principle phase 2 study of encaleret in post-surgical hypoparathyroidism (PSH), N=10





*p<0.05, 48hr or 120hr compared to baseline at 0hr .
FECa= fractional excretion of calcium, a measure of renal calcium handling; ULN = upper limit of normal. Gray shading reflects normal range.
Source: Hartley I.R. et al., presented at ASBMR 2025 Annual Meeting.

Proof-of-concept study findings are suggestive of a potential path for improved calcium control in a convenient pill form

Potential Differentiating Features For Encaleret

- Orally administered tablet
- Benefit in 24-hour urinary calcium excretion
- Avoidance of long-term PTH-mediated impact on bone health

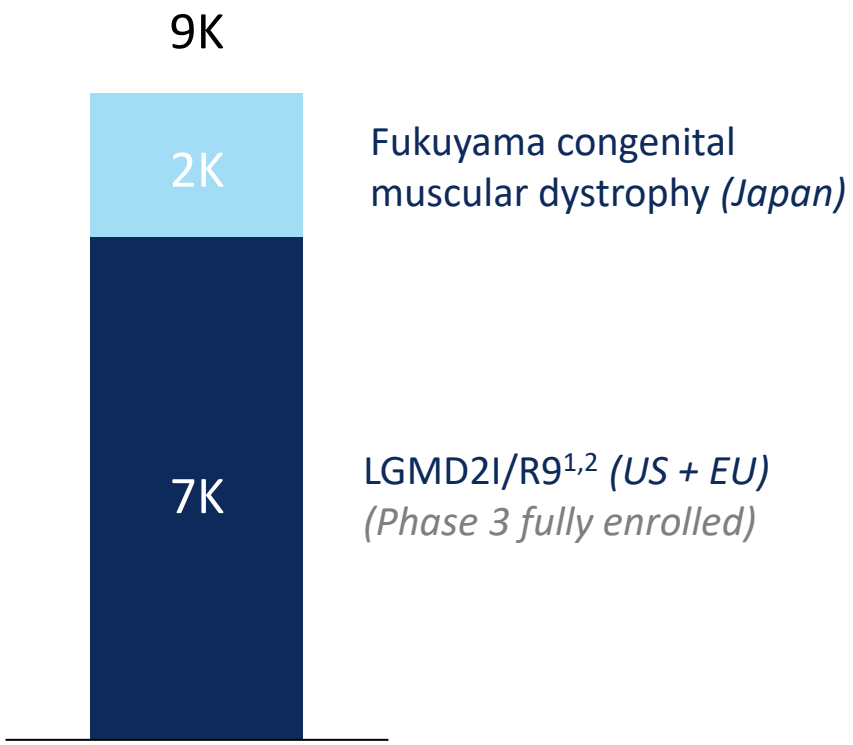
BBP-418

BBP-418 for Limb-Girdle Muscular Dystrophy Type 2I/R9 (LGMD2I/R9)

A sincere THANK YOU to patients, families, advocates, investigators, clinical research staff, and collaborating research partners

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 therapies** 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+

[1]Liu, et al. Genetics in Medicine. 2019. [2]Includes all patients with potentially treatable mutations in FKRP, FKTN, and ISPD.

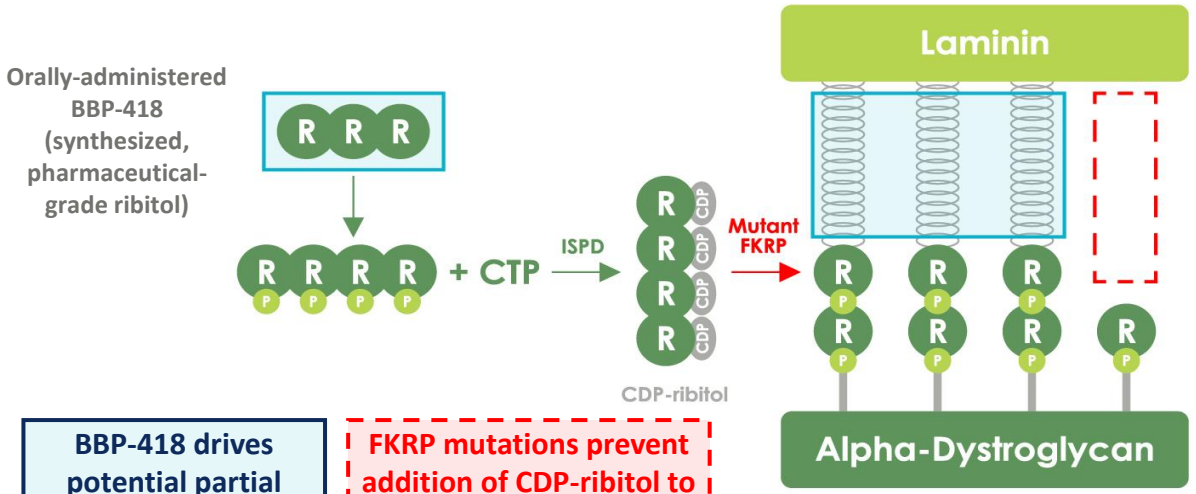
BBP-418 is an oral, disease-modifying therapy that targets LGMD2I/R9 at its source by restoring glycosylation of alpha-dystroglycan

Mechanism

FKRP glycosylates alpha-dystroglycan (αDG), 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 αDG in muscle cells, increasing cell susceptibility to damage

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



BBP-418 drives potential partial restoration of glycosylation of αDG

FKRP mutations prevent 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

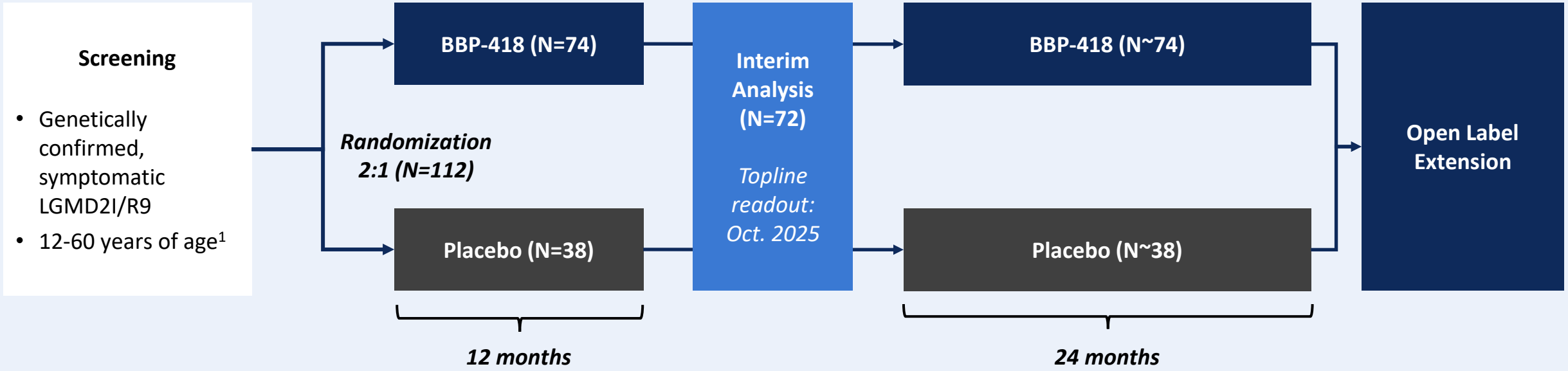
Avoid safety concerns with FKRP modulation

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 mitigate safety concerns

FORTIFY is an ongoing randomized, placebo-controlled Phase 3 study; we reported topline results from a planned interim analysis in Oct. 2025



Pre-specified Interim Endpoints[2]:

- Glycosylated αDG (*primary*)
- Serum creatine kinase (CK)
- Ambulatory measure: 100MTT
- Pulmonary function: FVC

Final Analysis Endpoints[3]:

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

Placebo & BBP-418 arms were stratified by age group (adult vs. pediatric), ambulatory status, and genotype (L276I homozygous vs. Other FKRP genotype)

Note: [1] Ph. 3 trial population limited to 18-60 years in EU region; [2] Alpha-protected efficacy endpoints as defined in statistical analysis plan (SAP); [3] Exploratory endpoints include cardiac endpoints such as HS Troponin I
Source: Table 14.1.1.1

Interim analysis topline data reflect highly statistically significant results on all pre-specified endpoints, including biomarker and clinical measures at 12 mo.

BBP-418 in LGMD2I/R9: FORTIFY Phase 3 interim analysis topline results

Primary endpoint (3 months)	p-value
Change from baseline in glycosylated αDG, % of control	p<0.0001

Key secondary endpoints (12 months)	p-value
Change from baseline in glycosylated αDG, % of control	p<0.0001
Change from baseline in serum CK, U/L	p<0.0001
Change from baseline in 100MTT, m/s	p<0.0001
Change from baseline in FVC, % predicted	p=0.0071

αDG = alpha-dystroglycan; CK = Creatine Kinase; 100MTT = 100-meter Timed Test; FVC = Forced Vital Capacity

Unprecedented and consistent improvement across primary and all key secondary efficacy endpoints combined with well-tolerated safety profile

fortify

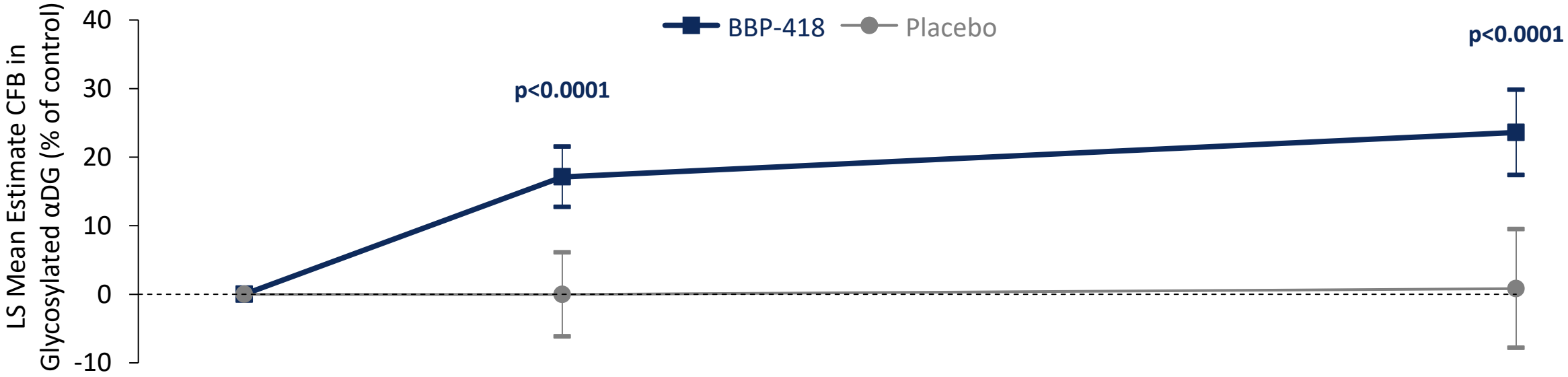
Ph. 3 FORTIFY interim analysis

Type	Endpoint	Upside case target	Outcome observed
Primary *(3 months)*	Glycosylated αDG	• Statistically significant increase vs. placebo • 1.5x CFB in BBP-418 treated vs. approx. no change in placebo	✓ **Highly statistically significant increase** (**1.8x CFB; absolute increase of 17%** of control) at 3 months ✓ Increase **sustained at 12 months**
	Creatine kinase (CK)	• Average decline of ≥50% CFB in BBP-418 treated	✓ **Highly statistically significant** average **decline of 82%** in BBP-418 treated
Key secondary *(12 months)*	• Ambulatory measures (100MTT) • Pulmonary (FVC)	• Trends in one or more measures favoring BBP-418 treated vs. placebo	✓ **Statistically significant** and **clinically meaningful improvement** in BBP-418 treated ✓ 100MTT: **Increase in velocity** of 0.14 m/s from baseline and 0.27 m/s vs. placebo ✓ FVC: **Increase in ventilatory capacity** of ~3% predicted volume from baseline and a difference of ~5% predicted volume vs. placebo
Safety		• Well-tolerated *(consistent with Ph. 2 results)*	✓ Well-tolerated; consistent with Ph. 2 results

CFB = Change From Baseline; αDG = alpha-dystroglycan; 100MTT = 100-Meter Timed Test; FVC = Forced Vital Capacity

Interim analysis showed statistically significant increase (+17% of control) from baseline in glycosylated αDG for BBP-418 treated, which was sustained at 12 mo.

Change from baseline in glycosylated αDG (+/- 99% CI)



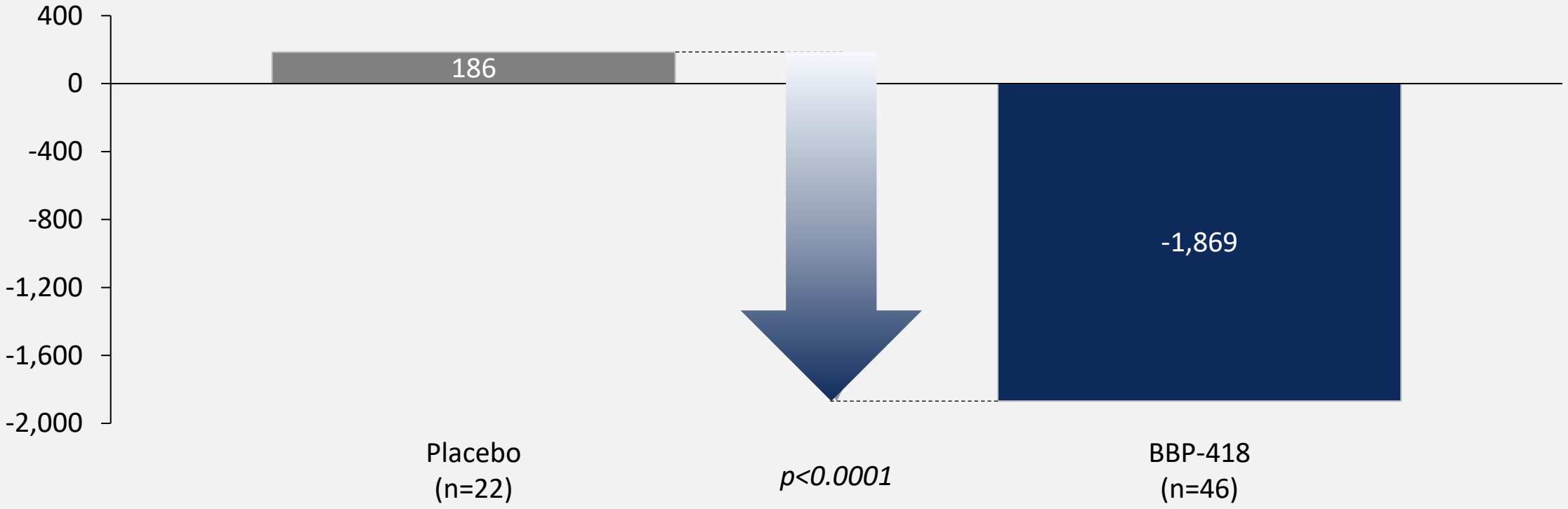
Timepoint	0 mo.	3 mo.	12 mo.
Cohort		All patients	Interim analysis
BBP-418		17.1% (n=71)	23.6% (n=49)
Placebo		-0.04% (n=36)	0.8% (n=22)

LS Mean Estimate CFB: Least-Squares Mean Estimate Change from Baseline; Comparison of BBP-418 change from baseline to Placebo change from baseline is based on a linear mixed model for repeated measures; n = number of participants with observed value
Source: Table 14.2.1.1.1.2.3a.2

In addition, BBP-418 treated patients experienced a large, statistically significant reduction in serum CK of 82% from baseline at 12 months

Reduction in muscle damage

Change from baseline in serum creatine kinase (U/L)

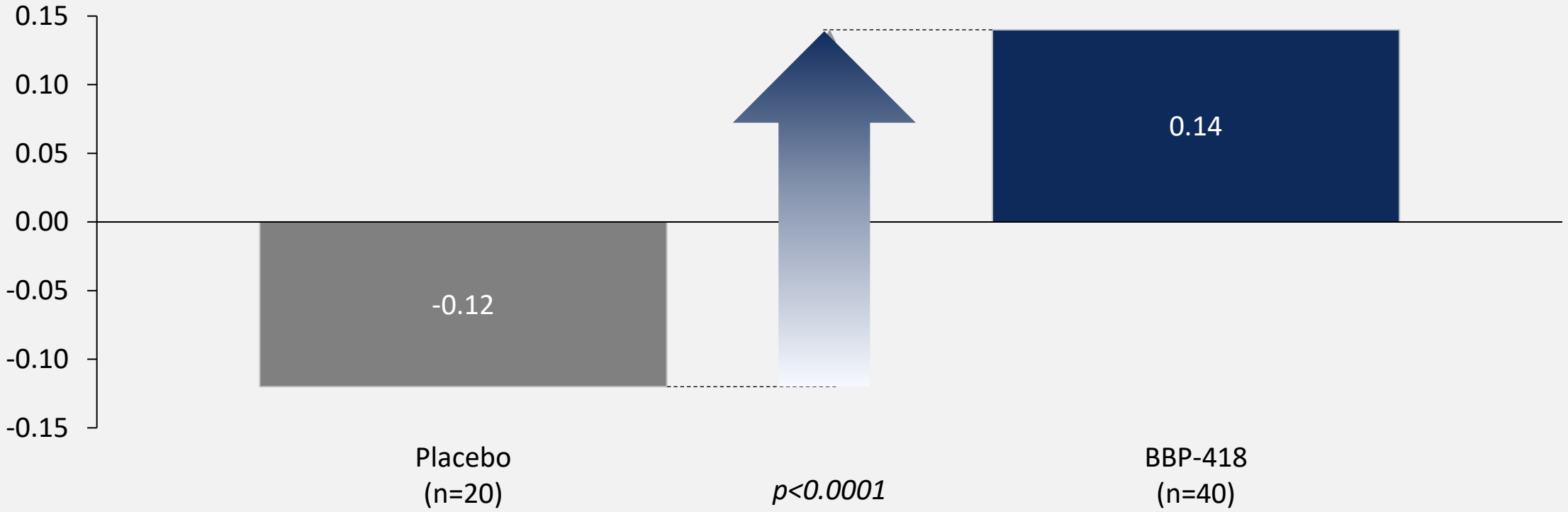


Least-Squares Mean Change from Baseline at 12-month timepoint; Comparison of BBP-418 change from baseline to Placebo change from baseline is based on a linear mixed model for repeated measures; n = number of participants with observed value
Source: Table 14.2.1.2.1.2.1a.2

Results translated to clinical endpoints with a difference of 0.27 m/s between BBP-418 and placebo in 100MTT, translating to a difference of ~14 seconds faster

Improved ambulatory function

Change from baseline in 100-meter timed test (m/s)

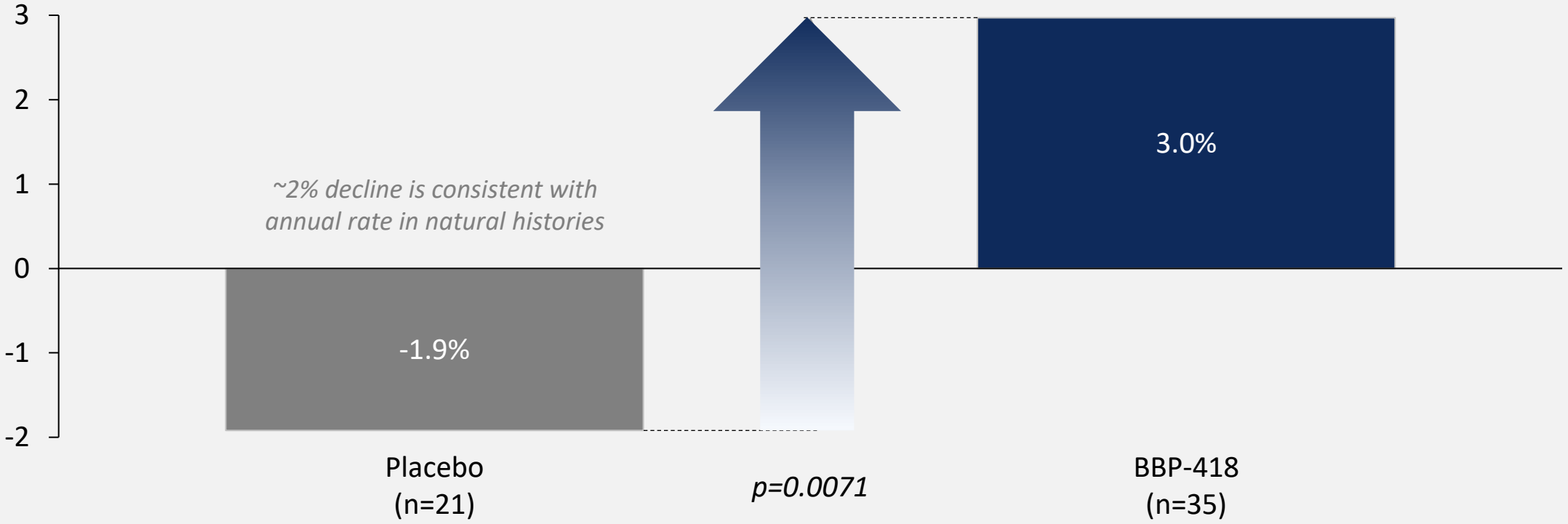


Least-Squares Mean Change from Baseline at 12-month timepoint; Comparison of BBP-418 change from baseline to Placebo change from baseline is based on a linear mixed model for repeated measures; n = number of participants with observed value
Source: Table 14.2.1.3.1.2.2a.2

BBP-418 treated individuals also registered ~3% increase in predicted volume from baseline FVC, resulting in a difference of ~5% predicted volume vs. placebo

Improved pulmonary function

Change from baseline in forced vital capacity, sitting position (% predicted)

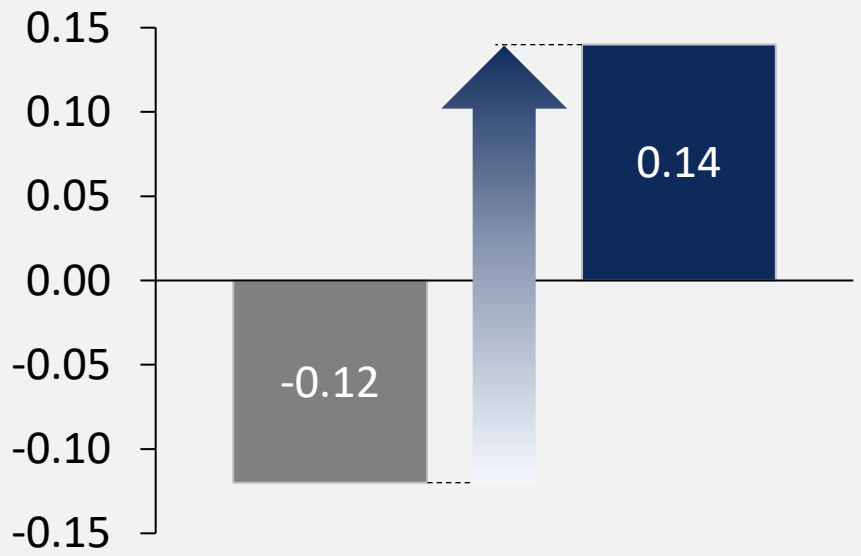


Least-Squares Mean Change from Baseline at 12-month timepoint; Comparison of BBP-418 change from baseline to Placebo change from baseline is based on a linear mixed model for repeated measures; n = number of participants with observed value
Source: Table 14.2.1.4.1.2.2a.2

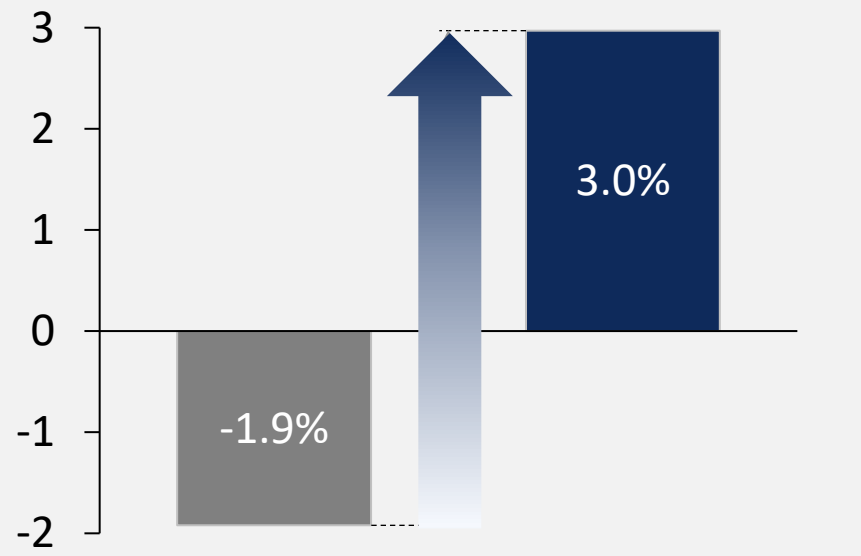
More than stabilization: BBP-418 treated individuals experienced significant, clinically meaningful improvements across clinical endpoints at 12 months

Key: Placebo BBP-418

Improved ambulatory function
CFB in 100MTT (m/s)



Improved pulmonary function
CFB in FVC (% predicted)



CFB = Change From Baseline
Least-Squares Mean Change from Baseline at 12-month timepoint; Comparison of BBP-418 change from baseline to Placebo change from baseline is based on a linear mixed model for repeated measures
Source: Table 14.2.1.3.1.2.2a.2; Table 14.2.1.4.1.2.2a.2

Interim analysis results continue to reflect highly favorable safety profile of BBP-418

- **No new or unexpected safety findings** have been observed; results consistent with Ph. 2
- **Discontinuation rate was low** overall and **higher in the placebo group**
- **No treatment-related serious TEAEs** were observed

Interim analysis continues to support a favorable risk-benefit profile

Next steps

Topline results from Phase 3 FORTIFY study interim analysis
October 2025

Engage FDA prior to NDA submission
Late 2025 / Early 2026

Present Phase 3 FORTIFY study interim analysis results
MDA Clinical & Scientific Conference
March 2026

File New Drug Application (NDA) with FDA
1H 2026

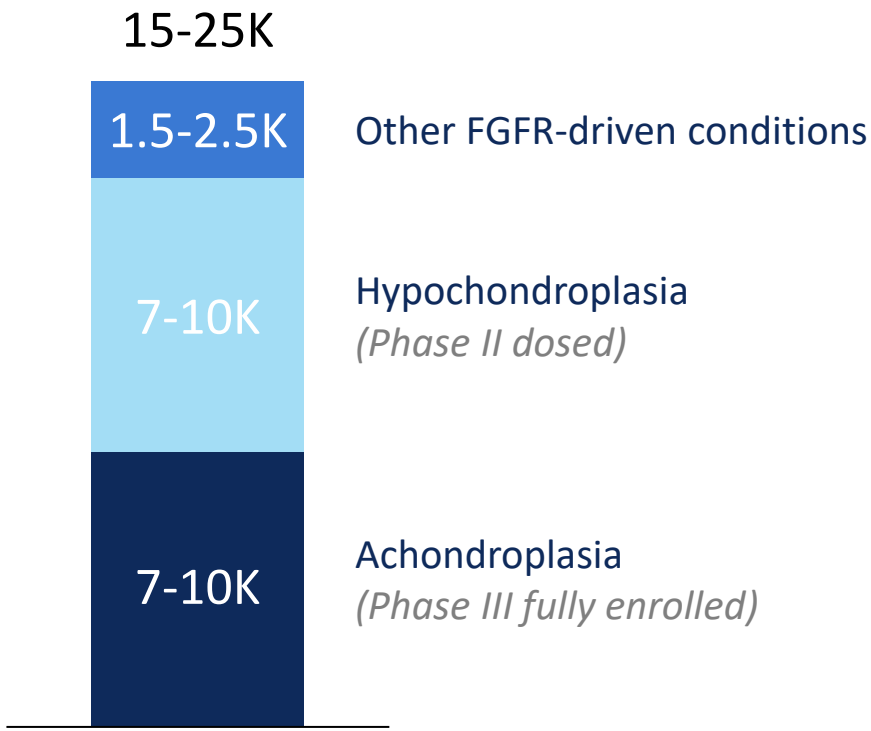
U.S. approval and commercial launch in LGMD2I/R9
Potential to be granted Priority Review Voucher (PRV)
Late 2026 / Early 2027

Infigratinib

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

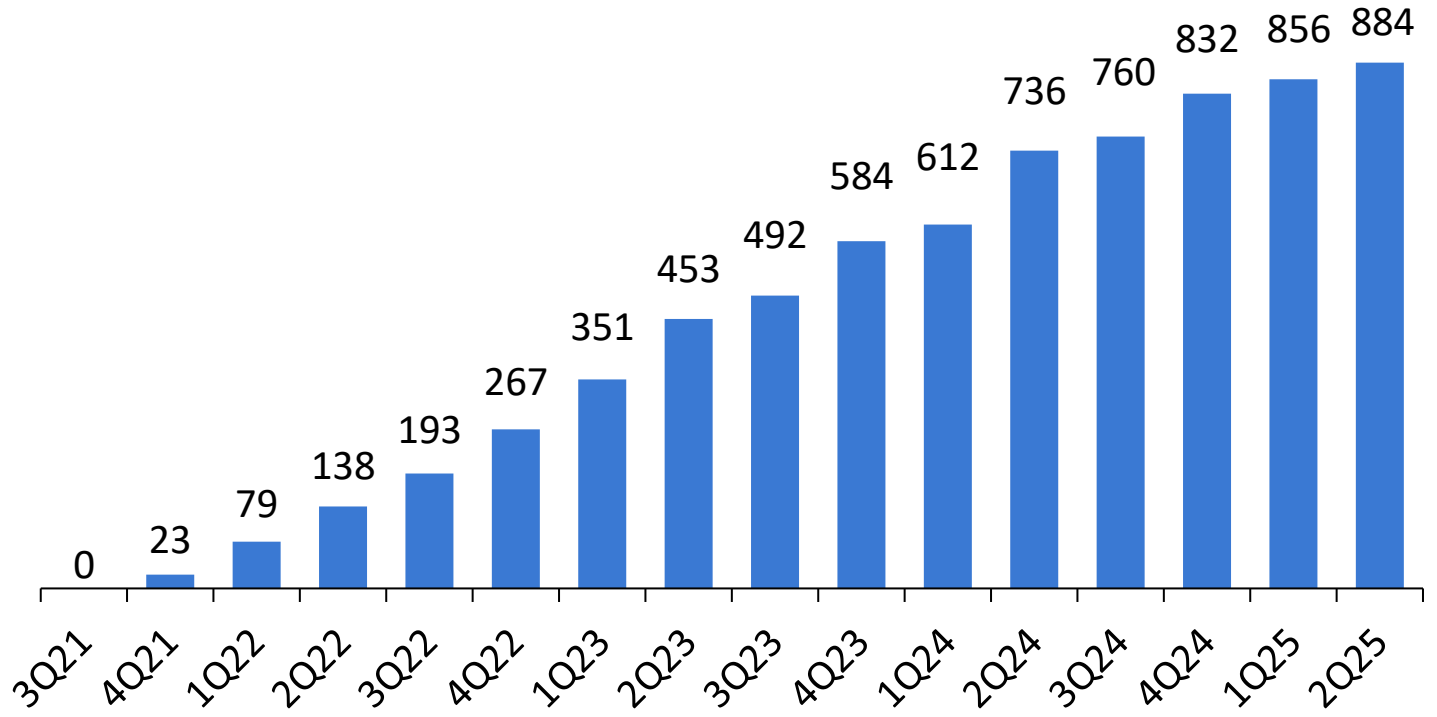
Addressable people by indication in US/EU[1]

(current population eligible for treatment)



Annualized achondroplasia product sales[2]

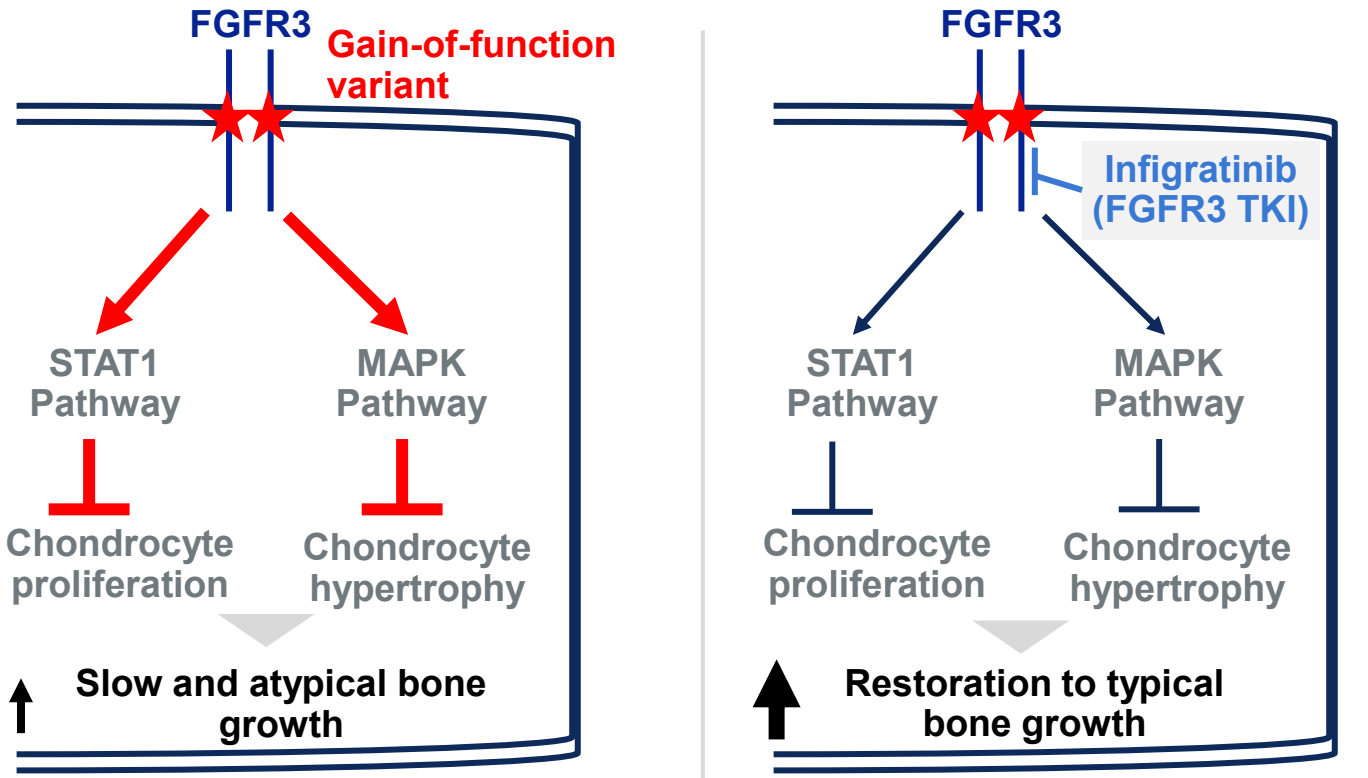
($M WW, annualized by quarter)



[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.

Infigratinib is a potentially 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 condition 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

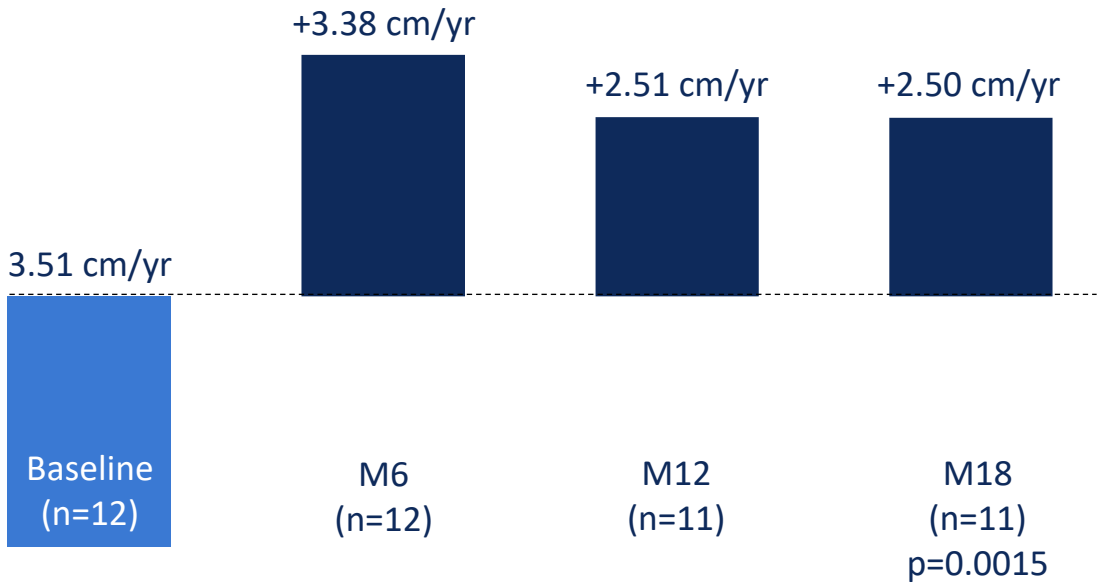
Avoiding hypotension & injection site reactions with no hyperphosphatemia, ocular effects or VEGFR3 off-target effects

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

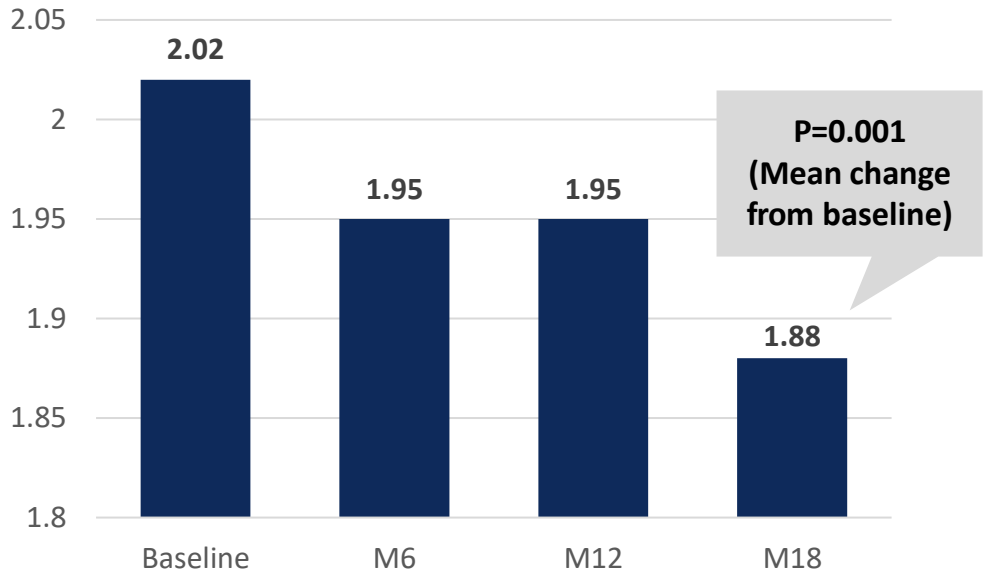
Mean change from baseline in annualized height velocity (AHV)



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

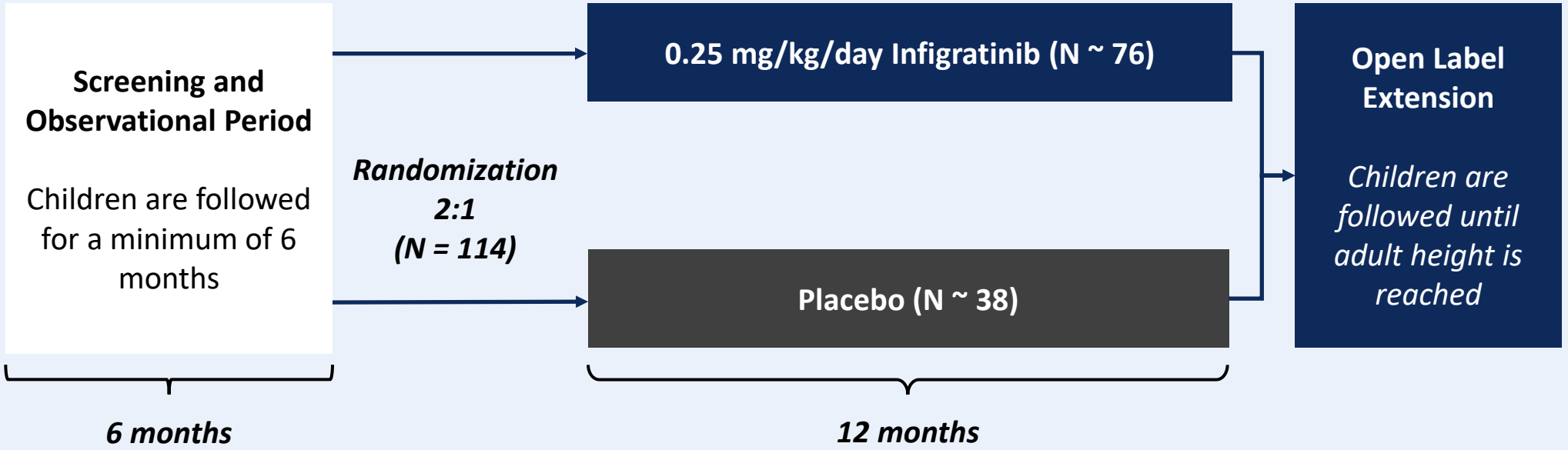
Upper body to lower body segment ratio (Lower value means improved proportionality)



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 and expect topline results in early 2026



Primary Endpoint:

- Change from baseline in annualized height velocity at Wk 52

Key Secondary Endpoints:

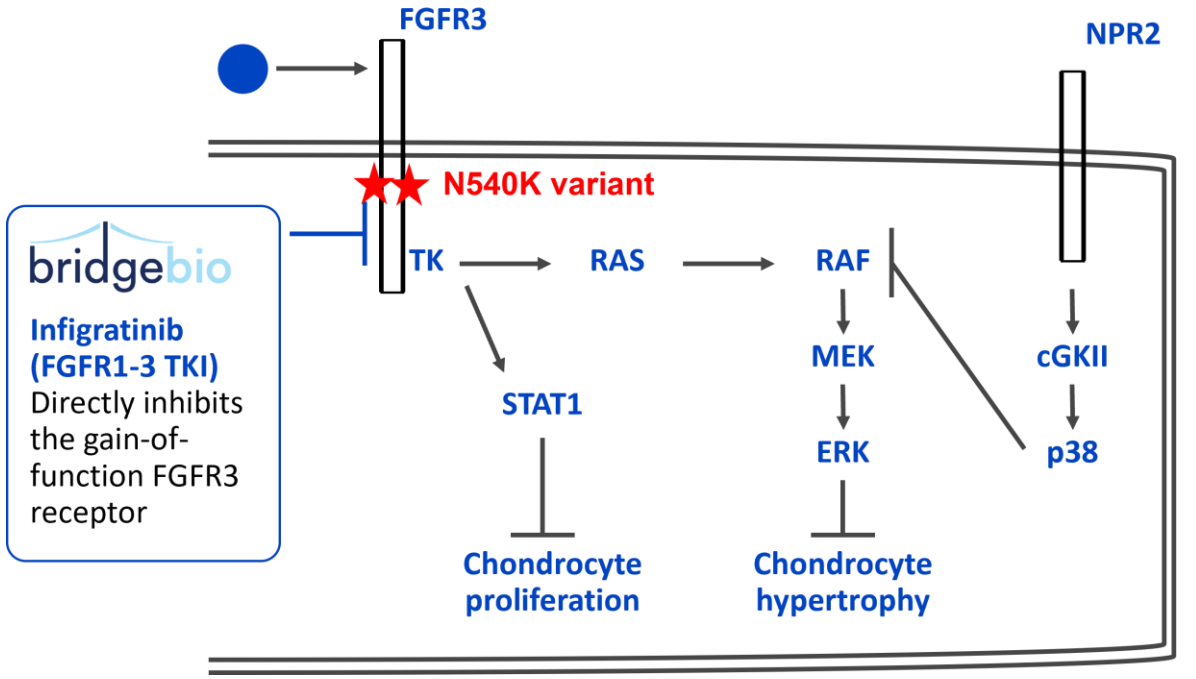
- Change from baseline in height z-score
- Change from baseline in upper to lower body segment ratio

Phase 3 Trial fully enrolled; LPLV expected 2H 2025, with topline results in early 2026

Hypochondroplasia is a FGFR3-related skeletal dysplasia

- **Autosomal dominant condition**
- **Similar incidence to achondroplasia**
- **Greater genetic heterogeneity in *FGFR3* pathogenic variants (e.g. N540K in addition to others)**
- **Clinical features:**
  - Moderate disproportionate short stature
  - Head circumference larger than average
  - Tibial bowing
- **Medical complications are milder and less frequent than in achondroplasia**
  - Motor milestones less delayed
  - Reports of epilepsy, temporal lobe abnormalities & other cognitive functions[2,3]

Infigratinib directly targets FGFR3 signaling



Infigratinib directly targets the underlying cause of hypochondroplasia, FGFR3 overactivity

[1] Rousseau et al. J Med Genet. 1996;33(9):749-752; [2]Linnankivi et al (2012). Am J Med Genet. 158A:3119–3125; [3]Philpott et al (2013). Pediatr Radiol (2013). 43:1190–1195

Hypochondroplasia is a large, mechanistically de-risked expansion opportunity; First participant dosed in Phase 2 study

Rationale for infigratinib

- Directly targets the underlying cause of hypochondroplasia, FGFR3 overactivity[1,2]
- Single-digit nM potency against multiple FGFR3 variants associated with hypochondroplasia, including in vivo improvement of bone growth in an HCH mouse model[1,2]
- Ability for infigratinib to cross the blood-brain barrier[3] to potentially address any neurological or cognitive manifestations[4,5]

Recent progress and upcoming milestones

- Observational run-in study for Phase 2 is fully enrolled significantly ahead of schedule
- Enrollment completion for Phase 2 portion expected in 2H 2025; Phase 2 data expected in 2H 2026

[1]Demuynck, B., & Shah, B. P. (2025). Infigratinib low-dose therapy is an effective strategy to treat hypochondroplasia. Journal of Bone and Mineral Research. PMID: 40581757; [2]Dambkowski C, et al. Journal of the Endocrine Society. 2022;6(Suppl 1):A459–A460.; [3]Cornille et al., Journal of Experimental Medicine, 2022; [4]Linnankivi et al (2012). Am J Med Genet. 158A:3119–3125, [5]Philpott et al (2013). Pediatr Radiol (2013). 43:1190–1195

BBP-812

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

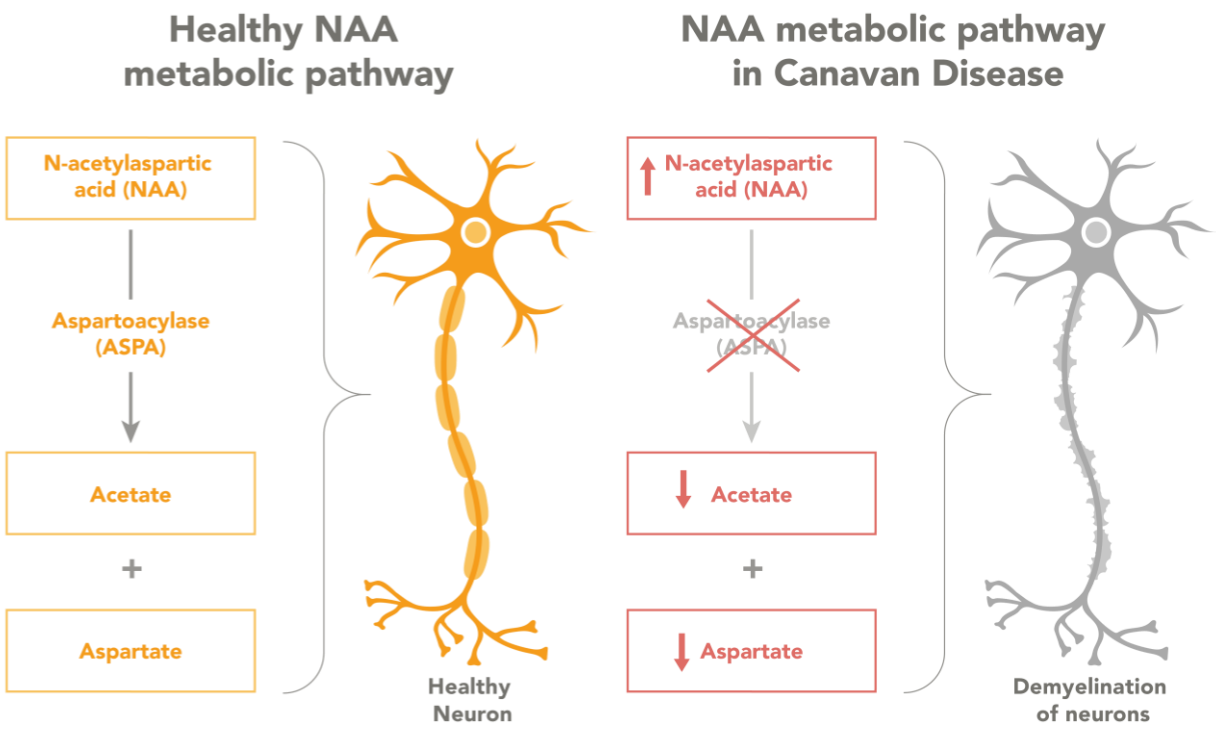
Unmet need

- 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 years[1]
- 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

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

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

Mechanism



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 based on known safety profile

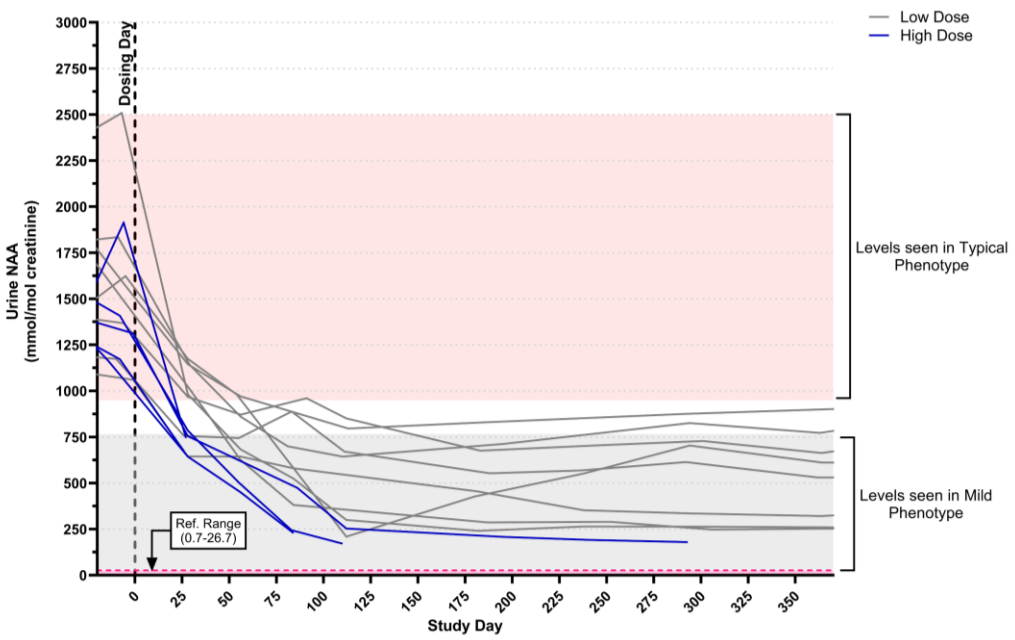
Leverage safety profile from approved IV AAV9 gene therapy

Avoid invasive neurosurgery

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

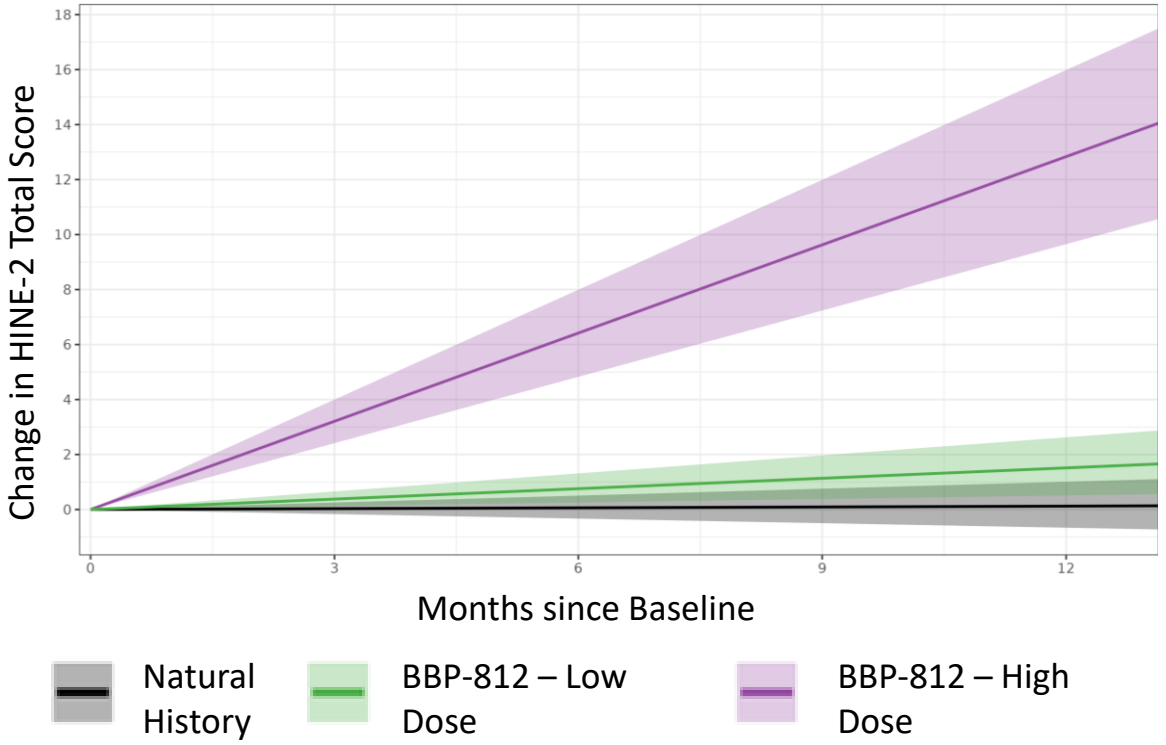
Significant, sustained reductions in NAA levels and improvement in motor function after 12-months of BBP-812 dosing in Phase 1/2 study

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

Hammersmith Infant Neurological Examination (HINE-2) Trajectory



- Trajectory analysis shows clear, dose-dependent separation in HINE-2 total score between individuals dosed with BBP-812 in the treatment study (CVN-102, in purple and green) vs. individuals in the natural history study (shown in gray). Children are progressing in key motor metrics such as sitting, head control, and reaching / grasping.

Preliminary data on file shown; n=14.

BridgeBio
Ecosystem Highlights

BBOT
BridgeBio Oncology Therapeutics

gondolabio

BBOT has progressed a pipeline of molecules into the clinic that are poised to close key activity gaps in the RAS-focused therapeutic space

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	**Phase 1 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 • Decreased risk for hyperglycemia / hyperinsulinemia	**Phase 1 Enrolling**
BBO-11818 **PanKRAS ON / OFF**	• Direct inhibitor of KRAS^{G12X} ON / OFF • Potent panKRAS inhibitor • Directly binds mutant KRAS	**Phase 1 Enrolling**

The GondolaBio pipeline features a diverse set of programs across therapeutic areas and modalities

Indication	Discovery	Lead Optimization	IND Enabling	Phase 1	Phase 2	Est. Patient Pop. (US + EU)
Erythropoietic Protoporphyria (EPP)						25k
Alpha-1 Antitrypsin Deficiency (AATD)						200k
Hereditary Pancreatitis						30k
Neurofibromatosis Type 1 (NF1)						200k
Fibrous Dysplasia						50k
Autosomal Dominant Polycystic Kidney Disease (ADPKD)						300k
Recurrent Oxalate Kidney Stones						300k
Tuberous Sclerosis Complex 1/2 (TSC)						65k
Genetic Epilepsy Driven by SynGAP1 Mutations						15k
Dup15q Developmental Epileptic Encephalopathy (DEE)						20k
Angelman Syndrome						50k
+7 undisclosed programs						

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.