BRIDGEBIO

Corporate Presentation

February 24, 2026

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 RECLAIM-HP Phase 3 study of encaleret in chronic hypoparathyroidism, the Phase 2/3 registrational study for encaleret in pediatric patients with ADH1, the Phase 2 study of infigratinib in hydrochondroplasia, the Phase 1/2 study of BBP-812 in Canavan disease, 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, the anticipated timing of submission of NDAs and BLAs to the FDA and corresponding submissions to foreign regulatory authorities and results of our interactions with the FDA, our speed of creating new and meaningful drugs and related impact on patients, the sustainability and velocity of our engine to deliver medicines, our value creation potential for patients, the potential market sizes and opportunities for the indications we are pursuing, the safety, efficacy, ease of access 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 of our depleter program to reverse ATTR, the potential of our pipeline of molecules, 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 products and product candidates, the affordability, maximum pricing 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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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**

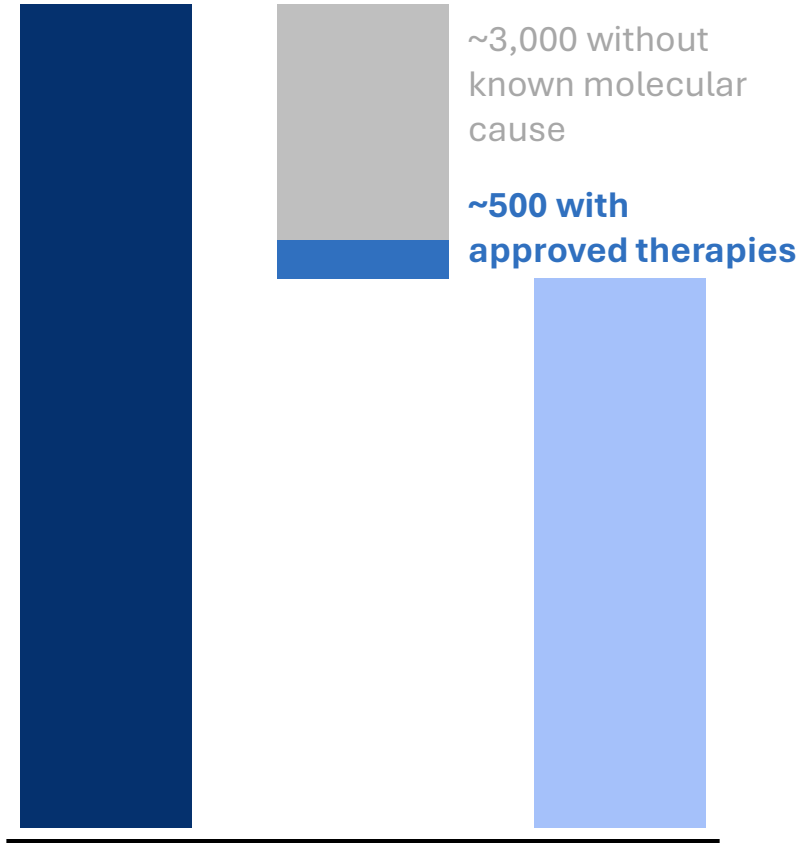
Science JOURNALS AAAS — **Accurate proteome-wide missense variant effect prediction with AlphaMissense**

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

We have built a sustainable, high velocity engine to deliver hope and medicines to the communities that we serve

> 9,500
individuals impacted by our therapies

Obtained Approval for 3 Medicines

Attruby™ (acoramidis) 356 mg tablet

Nulibry® (fosdenopterin) for injection

TRUSELTIQ™ (infigratinib) capsules

3 Positive Phase 3 Results

fortify ✓

CALIBRATE ✓

PROPEL3 ✓

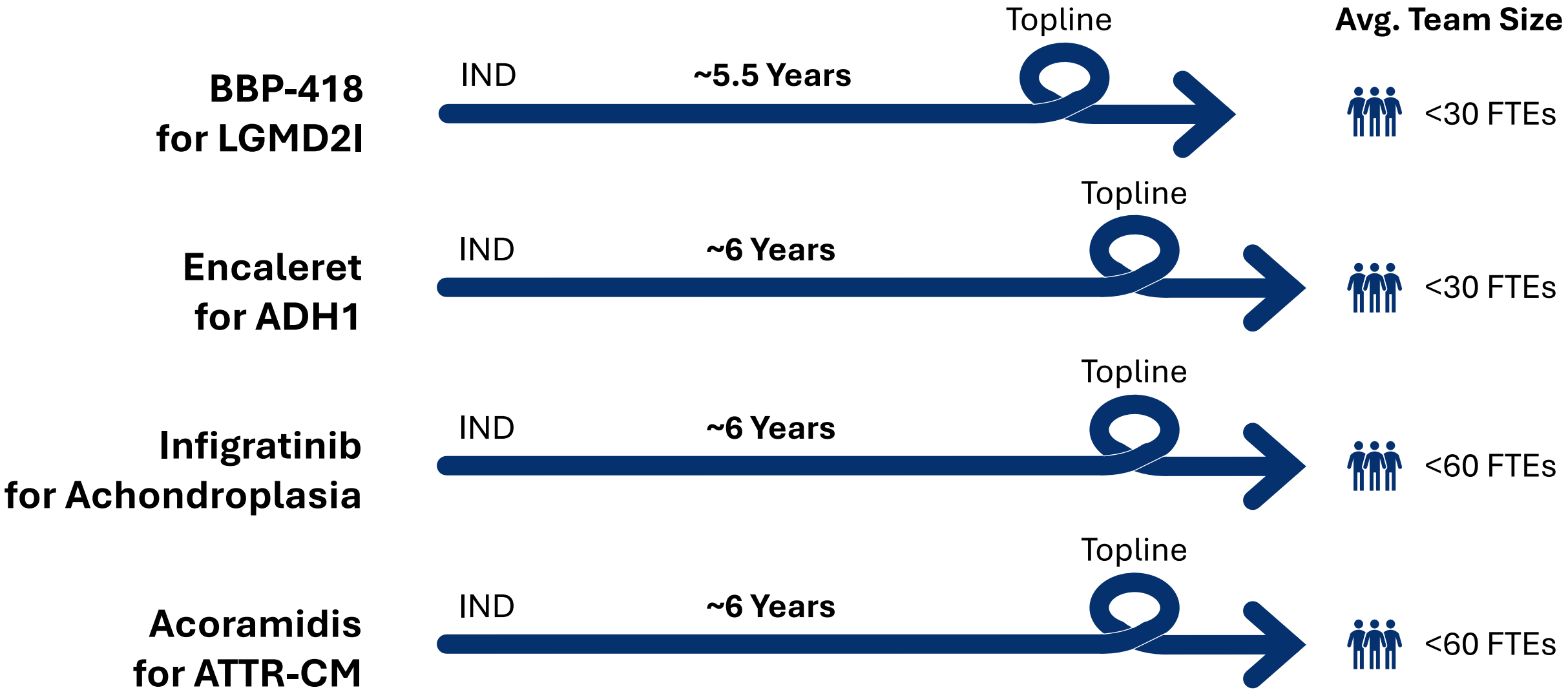
CANaspire

+ 1 Ongoing Pivotal Trials

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

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

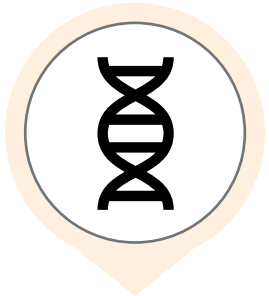
We leverage lean operating teams that advance medicines quickly and efficiently for people living with rare diseases



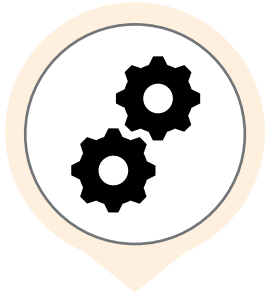
Source: internal estimates. IND: Investigational New Drug application; FTE: Full Time Employee.

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%	**86%**
Cumulative	9%	**52%**

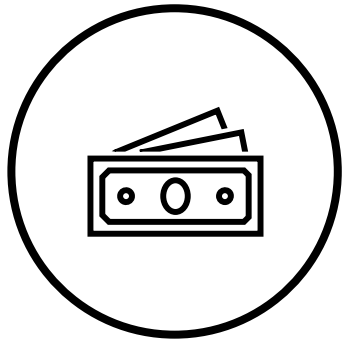
Source: [1] Biotechnology Innovation Organization (BIO), QLS Advisors, and Informa Pharma Intelligence, *Clinical Development Success Rates and Contributing Factors 2011–2020* (2021).
[2] Internal data on file (2015 – 2025). Includes programs initiated at BridgeBio that continue to be advanced at sister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

A pipeline of products that sing across the BrigeBio ecosystem

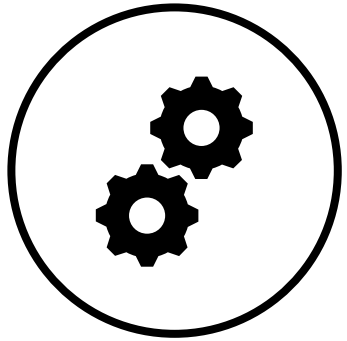
Program	Indication	Pre-Clinical	Phase 1	Phase 2	Phase 3	Approved	Patients (US + EU)	Market Opportunity
Attruby (acoramidis)	Transthyretin Amyloidosis (ATTR-CM)					✓	500,000+	$20B+
Nulibry (fosdenopterin)	Molybdenum Cofactor Deficiency (MoCD) Type A					✓	<100	Partnered
Truseltiq (infigratinib)	Cholangiocarcinoma					✓	37,000	Partnered
Infigratinib	Achondroplasia (ACH)					*Topline Readout*	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
Evanesco (depleter)	Transthyretin Amyloidosis (ATTR-CM)						500,000+	$20B+
ADPKD (small molecule)	Autosomal Dominant Polycystic Kidney Disease						300,000	TBD
BridgeBio Oncology Therapeutics	3 Oncology Programs (various indications)						Various	Various
GondolaBio	17 Programs (various indications)						Various	Various

As of December 31, 2025, BridgeBio has an 18.2% ownership stake in BridgeBio Oncology Therapeutics and a 27.5% ownership stake in GondolaBio. BridgeBio Oncology Therapeutics and GondolaBio are independent companies from BridgeBio. BridgeBio's interest in GondolaBio is subject to reduction as additional tranches of capital contributions are funded. Truseltiq was withdrawn from the market voluntarily after receiving FDA approval.

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



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



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

1H 2026

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

2H 2026

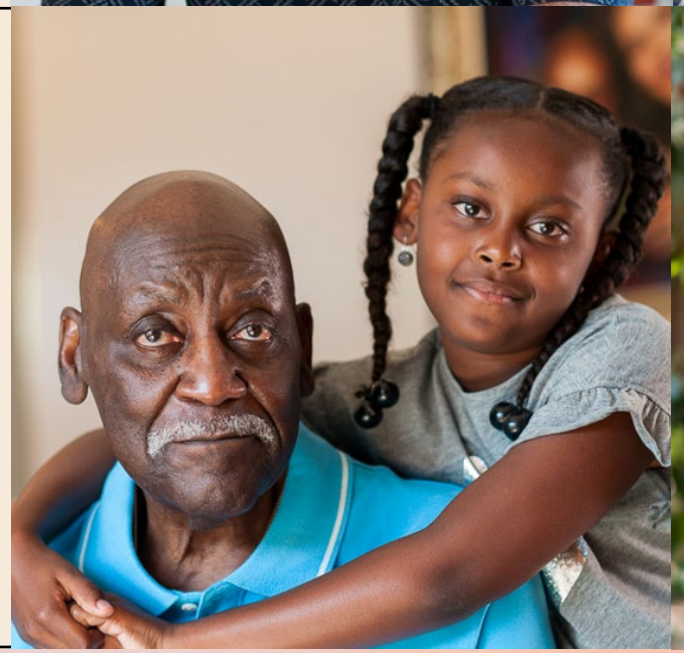
- **Infigratinib: NDA submission**
- **Infigratinib: HCH P2 data readout**
- **Encaleret: Initiate P3 CHP trial**

1H 2027

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

ACH=achondroplasia. HCH=hypochondroplasia. CHP=chronic hypoparathyroidism. Dates reflect anticipated timing.

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

Continued Attruby commercial momentum

$146M

Q4 2025 Net Product Revenue

NEW INFORMATION

Continued commercial momentum and patient impact

7,804

Unique U.S. patient prescriptions for Attruby

1,856

Unique U.S. prescribing HCPs

>25%

Estimated share of NBRx for Attruby

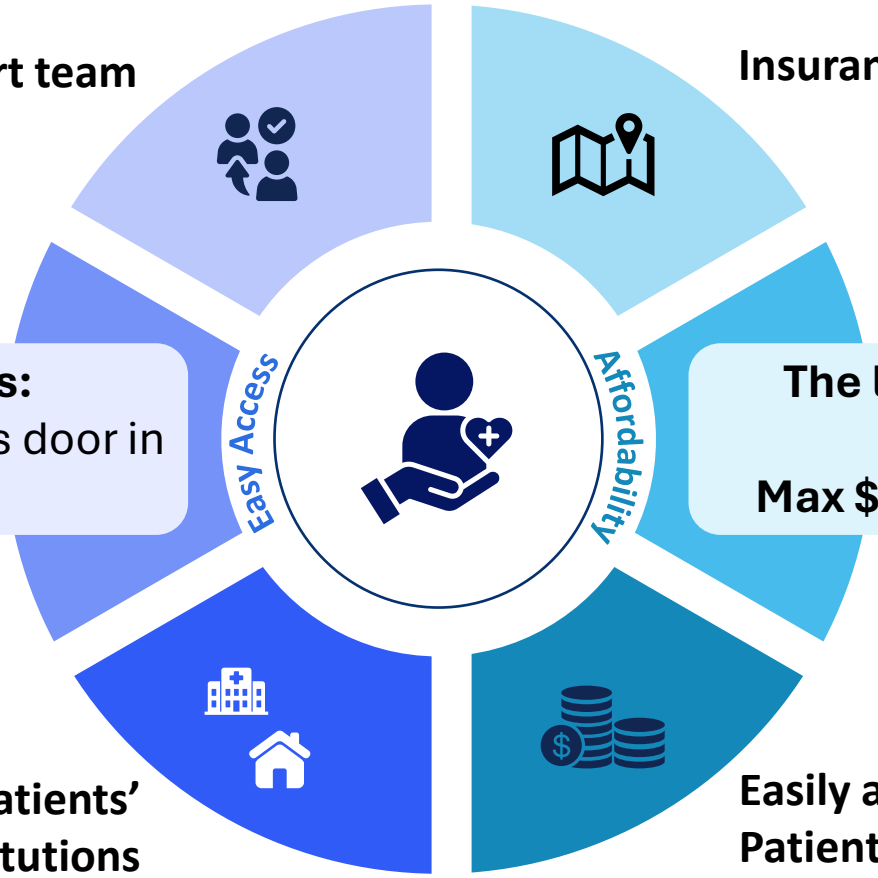
As of Feb 20th, 2026

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 $175 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

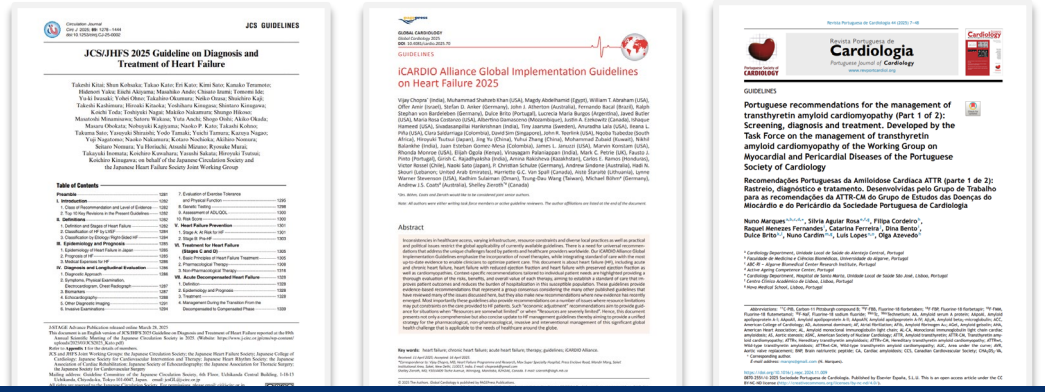
Beyonttra® (acoramidis) has seen rapid EU uptake with leading guideline endorsements

BEYONTTRA®
(acoramidis) 356mg tablet

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

50% **NBRx share**	Achieved by Germany 8 months into launch (as of November 31, 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.
98% Volume Share	Denmark awarded the national tender to Bayer, establishing Beyonttra 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 the 15 EU countries with reimbursement and for future launches.

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

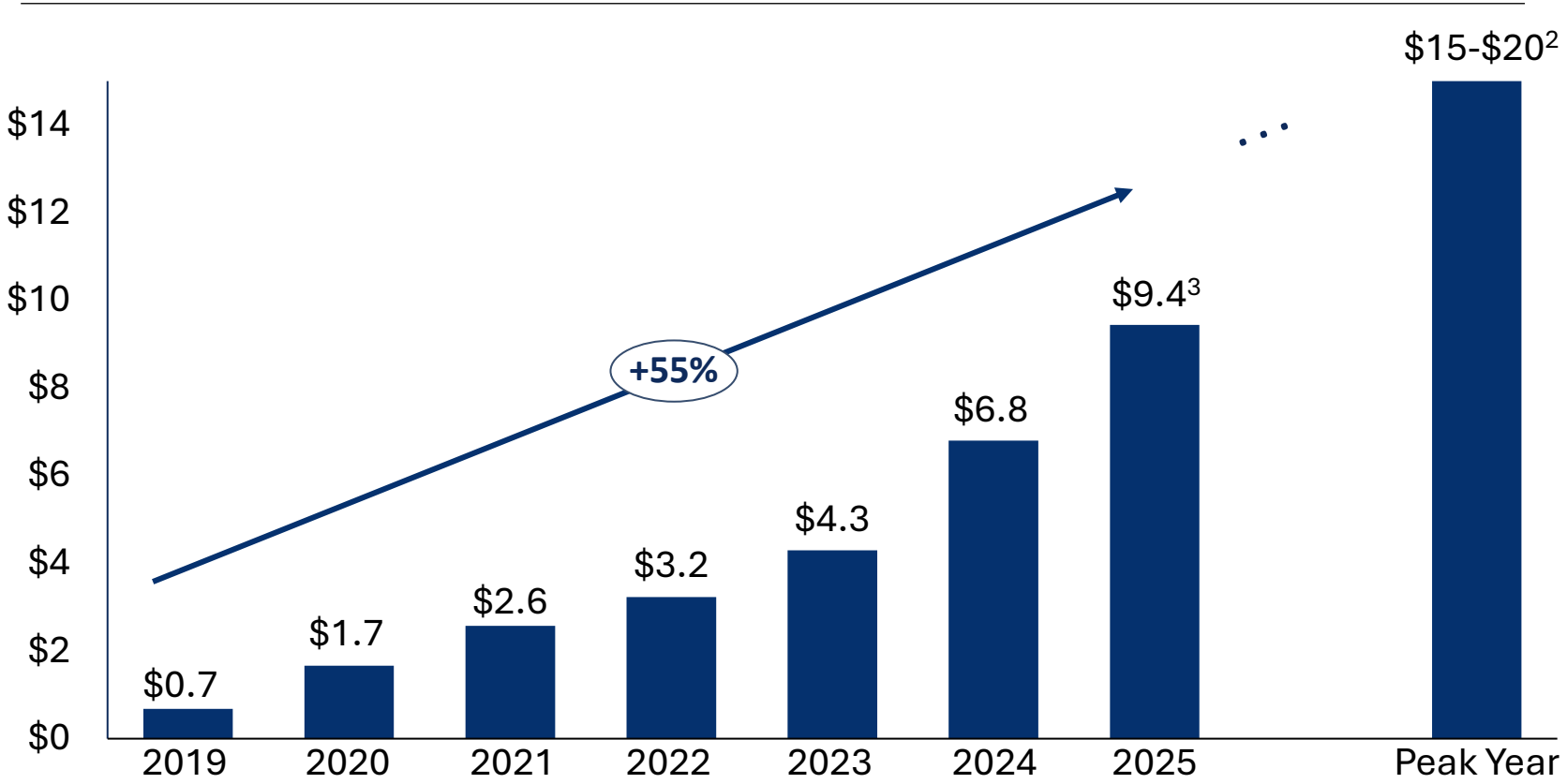


Acoramidis endorsed with highest tier recommendation and highlighted as near-complete stabilizer while tafamidis described as partial stabilizer

Beyonttra is partnered with Bayer in the EU. 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 August 31 2025, reporting 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.

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

Global annual ATTR market sales[1]



[1]ATTR market includes all approved drugs for ATTR-PN and ATTR-CM.
[2]Consensus estimates of $15B-$20B ATTR market.
[3]Based on 2025 numbers

Market growth drivers include:

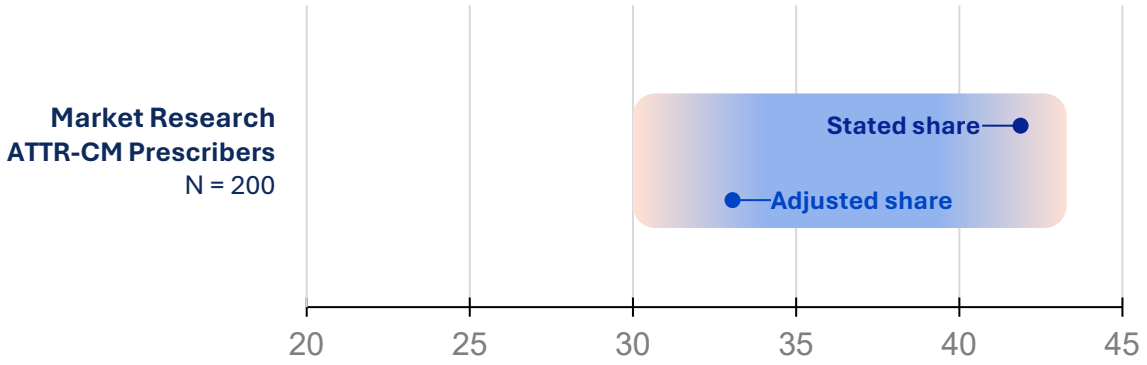
- With **more sponsors**, there is expanding disease awareness
- Increased global adoption of **non-invasive diagnostic tools**

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

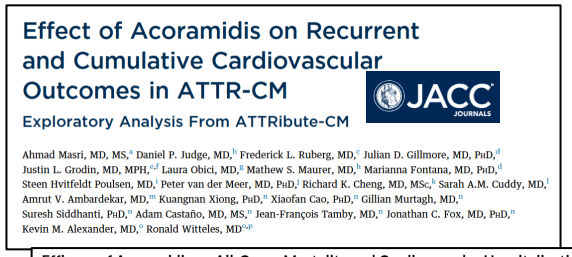
> *Very impressive treatment effect and best data to date on what happens in a contemporary ATTR-CM population."*
>
> HCP - Central

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

We continue to research the differentiated clinical role of Attruby

11 published manuscripts across 8 journals and 47 abstracts across 9 conferences in 2025

Clinical Outcomes


Effect of Acoramidis on Recurrent and Cumulative Cardiovascular Outcomes in ATTR-CM

Exploratory Analysis From ATTRibute-CM

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Ahmad Masri, MD, MS,[a] Daniel P. Judge, MD,[b] Frederick L. Ruberg, MD,[c] Julian D. Gillmore, MD, PhD,[d] Justin L. Grodin, MD, MPH,[e,f] Laura Obici, MD,[g] Mathew S. Maurer, MD,[h] Marianna Fontana, MD, PhD,[d] Steen Hvitfeldt Poulsen, MD,[i] Peter van der Meer, MD, PhD,[j] Richard K. Cheng, MD, MSc,[k] Sarah A.M. Cuddy, MD,[l] Amrut V. Ambardekar, MD,[m] Kuangnan Xiong, PhD,[n] Xiaofan Cao, PhD,[n] Gillian Murtagh, MD,[n] Suresh Siddhanti, PhD,[n] Adam Castaño, MD, MS,[n] Jean-François Tamby, MD,[n] Jonathan C. Fox, MD, PhD,[n] Kevin M. Alexander, MD,[o] Ronald Witteles, MD[o,p]

Efficacy of Acoramidis on All-Cause Mortality and Cardiovascular Hospitalization in Transthyretin Amyloid Cardiomyopathy

JACC JOURNALS

Editorial Comment: Transthyretin Amyloid Cardiomyopathy

Authors: Daniel P. Judge, Kevin M. Alexander, Francesco Cappelli, Marianna Fontana, Pablo Garcia-Pavia, Simon D.J. Gibbs, Martha Grogan, Mazen Hanna, Ahmad Masri, Mathew S. Maurer, Laura Obici, Prem Soman, Xiaofan Cao, Ted Lystig, Jean-François Tamby, Suresh Siddhanti, Adam Castaño, Leonid Katz, Jonathan C. Fox, Kenneth W. Mahaffey, and Julian D. Gillmore

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Publication: JACC • Volume 85, Number 10

Continuous Acoramidis Treatment Significantly Reduced Risk of All-Cause Mortality and Cardiovascular-Related Hospitalization Through Month 42 in Participants with Wild-Type and Variant Transthyretin Amyloidosis Cardiomyopathy

Martha Grogan,[1] Amrut Ambardekar,[2] Justin L Grodin,[3] **Lily K Stern,**[4] Prem Soman,[5] Marianna Fontana,[6] Pablo Garcia-Pavia,[7] Kuangnan Xiong,[8] Suresh Siddhanti,[8] Jean-François Tamby,[8] Jonathan C Fox,[8] Nowell Fine,[9] Mathew Maurer[10]

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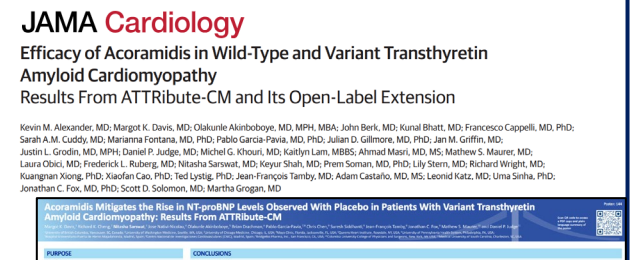
Presenter: Lily K Stern

Variant


JAMA Cardiology

Efficacy of Acoramidis in Wild-Type and Variant Transthyretin Amyloid Cardiomyopathy

Results From ATTRibute-CM and Its Open-Label Extension

Kevin M. Alexander, MD; Margot K. Davis, MD; Olakunle Akinboboye, MD, MPH, MBA; John Berk, MD; Kunal Bhatt, MD; Francesco Cappelli, MD, PhD; Sarah A.M. Cuddy, MD; Marianna Fontana, MD, PhD; Pablo Garcia-Pavia, MD, PhD; Julian D. Gillmore, MD, PhD; Jan M. Griffin, MD; Justin L. Grodin, MD, MPH; Daniel P. Judge, MD; Michel G. Khouri, MD; Kaitlyn Lam, MBBS; Ahmad Masri, MD, MS; Mathew S. Maurer, MD; Laura Obici, MD; Frederick L. Ruberg, MD; Nitasha Sarswat, MD; Keyur Shah, MD; Prem Soman, MD, PhD; Lily Stern, MD; Richard Wright, MD; Kuangnan Xiong, PhD; Xiaofan Cao, PhD; Ted Lystig, PhD; Jean-François Tamby, MD; Adam Castaño, MD, MS; Leonid Katz, MD; Uma Sinha, PhD; Jonathan C. Fox, MD, PhD; Scott D. Solomon, MD; Martha Grogan, MD

Acoramidis Mitigates the Rise in NT-proBNP Levels Observed With Placebo in Patients With Variant Transthyretin Amyloid Cardiomyopathy: Results From ATTRibute-CM

Acoramidis Reduces All-Cause Mortality and First Cardiovascular Hospitalization in Participants With Variant Transthyretin Amyloid Cardiomyopathy: Results From the ATTRibute-CM Study

Margot K. Davis,[1] Jan M. Griffin,[2] Nitasha Sarswat,[3] Justin L. Grodin,[4] Kevin M. Alexander,[5] Daniel P. Judge,[2] Julian D. Gillmore,[6] Francesco Cappelli,[7] Richard Wright,[8] **Prem Soman,**[9] John Berk,[10] Xiaofan Cao,[11] Jean-François Tamby,[11] Adam Castaño,[11] Jonathan C. Fox,[11] Keyur Shah,[12] Martha Grogan[13]

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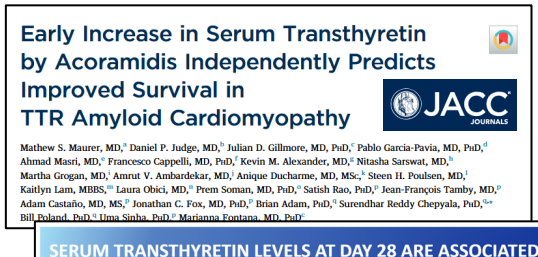
Biomarkers


Early Increase in Serum Transthyretin by Acoramidis Independently Predicts Improved Survival in TTR Amyloid Cardiomyopathy

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SERUM TRANSTHYRETIN LEVELS AT DAY 28 ARE ASSOCIATED WITH CARDIOVASCULAR OUTCOMES: INSIGHTS FROM THE ATTRibute-CM STUDY

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ACORAMIDIS IMPROVES SERUM TTR LEVELS IN PATIENTS WITH WILD-TYPE OR VARIANT TRANSTHYRETIN AMYLOID CARDIOMYOPATHY – RESULTS FROM ATTRibute-CM

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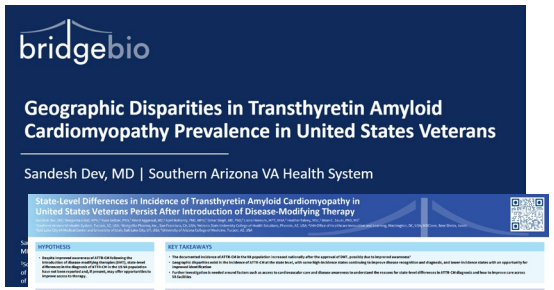
HEOR/Other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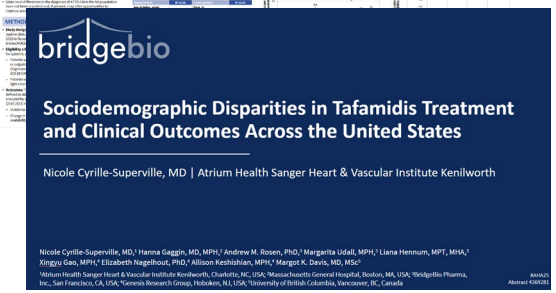
Geographic Disparities in Transthyretin Amyloid Cardiomyopathy Prevalence in United States Veterans

Sandesh Dev, MD | Southern Arizona VA Health System

State-Level Differences in Incidence of Transthyretin Amyloid Cardiomyopathy in United States Veterans Persist After Introduction of Disease-Modifying Therapy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Sociodemographic Disparities in Tafamidis Treatment and Clinical Outcomes Across the United States

Nicole Cyrille-Superville, MD | Atrium Health Sanger Heart & Vascular Institute Kenilworth

Nicole Cyrille-Superville, MD,[1] Hanna Gaggin, MD, MPH,[2] Andrew M. Rosen, PhD,[2] Margarita Udall, MPH,[2] Liana Hennum, MPT, MHA,[2] Xingyu Gao, MPH,[2] Elizabeth Nagelhout, PhD,[2] Allison Keshishian, MPH,[2] Margot K. Davis, MD, MSc[3]

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Abstract 4169285

2026 Themes

- Variant
- Arrhythmias
- Early Tx Effect
- Biomarkers
- Prevention / ACT-EARLY
- And more…

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

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

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

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

RESEARCH ARTICLE | BIOCHEMISTRY

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

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

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

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

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

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

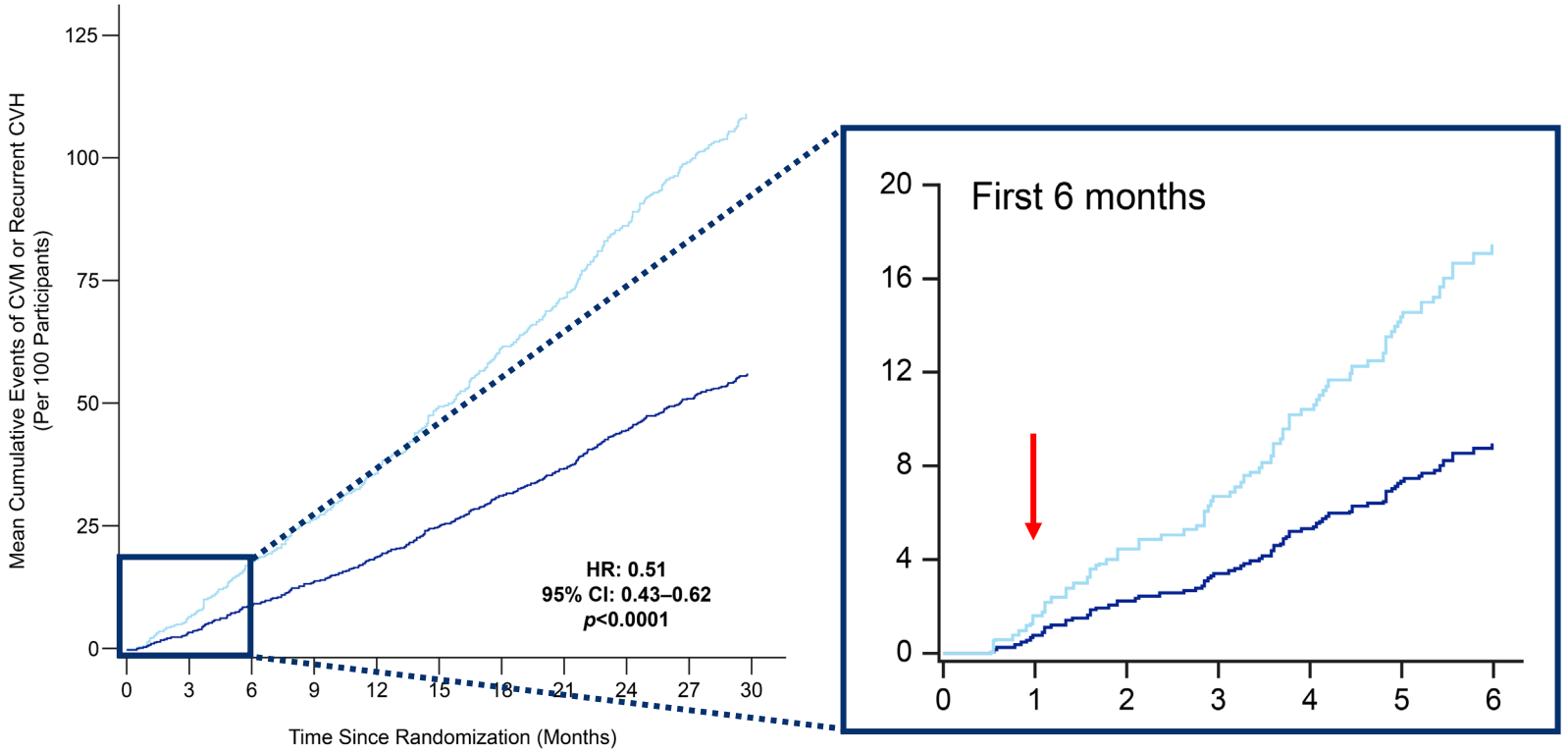
– Prof. Jeffery Kelly (inventor of tafamidis) in email correspondence with Dr. Isabella Graef, February 12, 2013. Bold added.

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

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

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

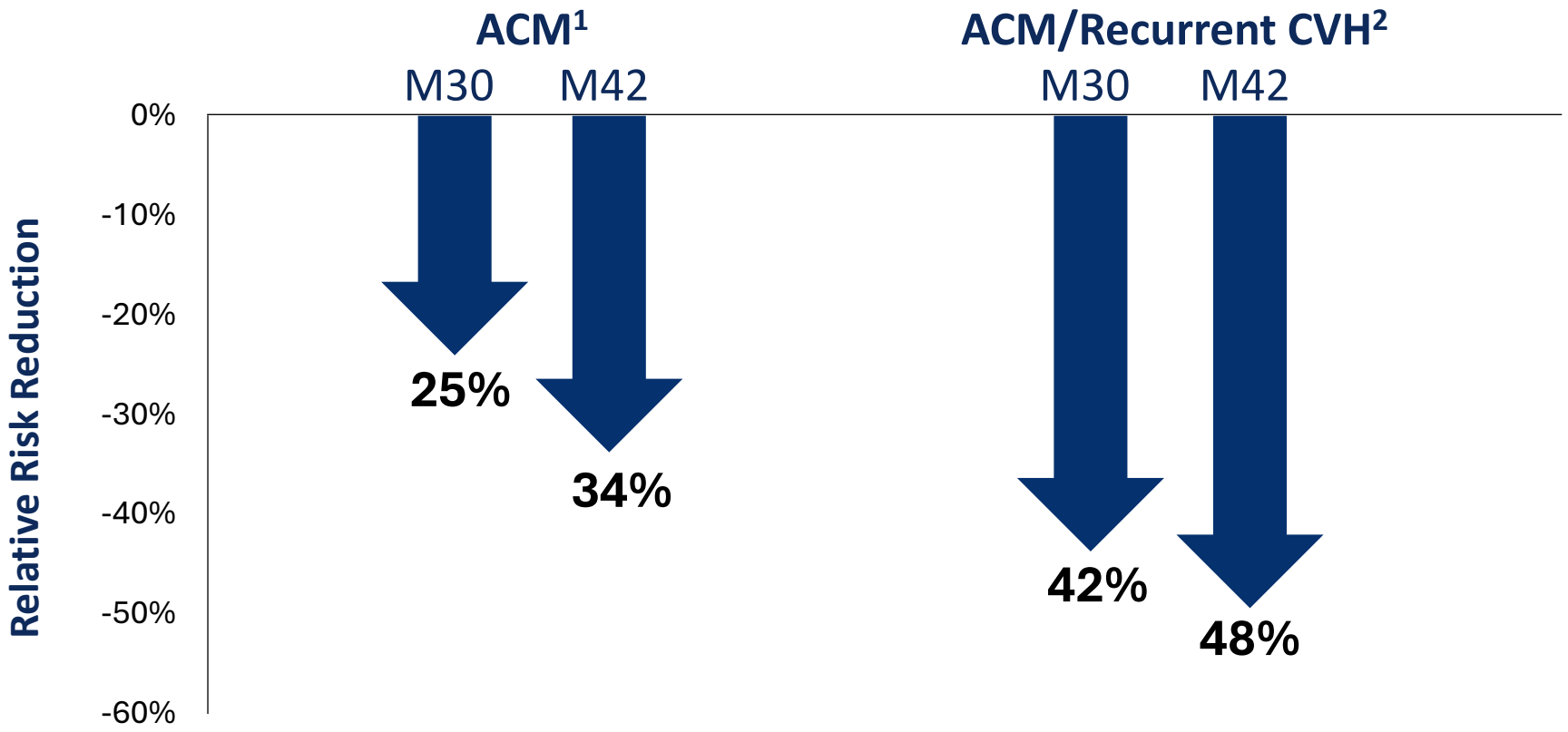
(mITT Population, Acoramidis, n = 409; Placebo, n = 202)



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

Source: Masri et al. (2025) Early, Long-Term Reduction in CV-Outcomes With Acoramidis, JACC; HFSA 2025 Presentation (Masri) Acoramidis Reduces Cumulative Cardiovascular Outcomes Within the First Month of Treatment in Transthyretin Amyloid Cardiomyopathy: Results From ATTRibute-CM; Data are for the mITT population in the ATTRibute-CM study, defined as all the participants who had undergone randomization, received at least one dose of acoramidis or placebo, and had at least one efficacy evaluation after baseline; participants with eGFR < 30 mL/min/1.73 m2 were excluded 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



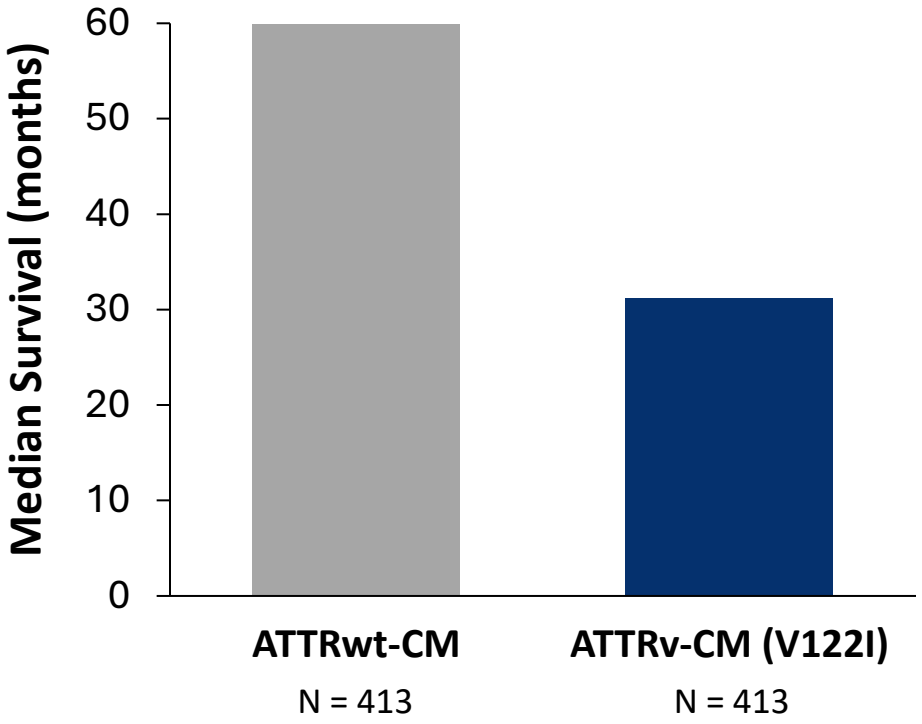
Attruby resulted in a **statistically significant** ACM and ACM/Recurrent CVH relative risk reduction at **both Month 30 and Month 42**

Source: Judge et al., Circ 2024. [1]Relative Risk Reduction based on percent of participants with ACM event at M30 and M42. [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
V122I	35 (5.7%)	0.31 (0.12-0.81)	0.016

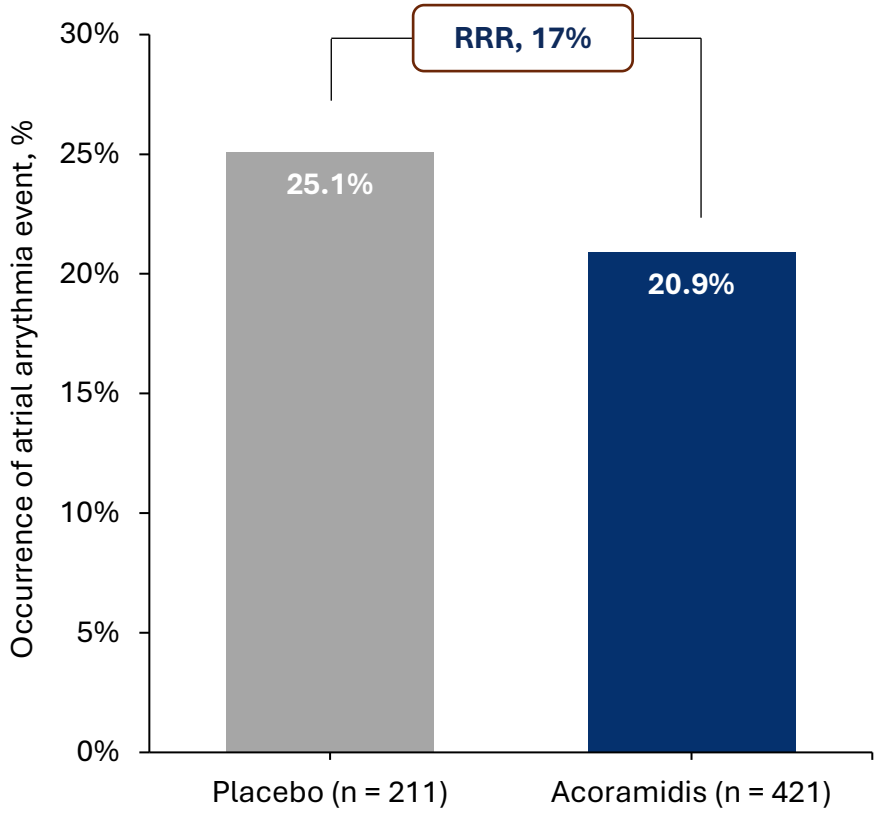
Unprecedented improvements in QOL and Functional Capacity in Variant ATTR-CM[3]

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

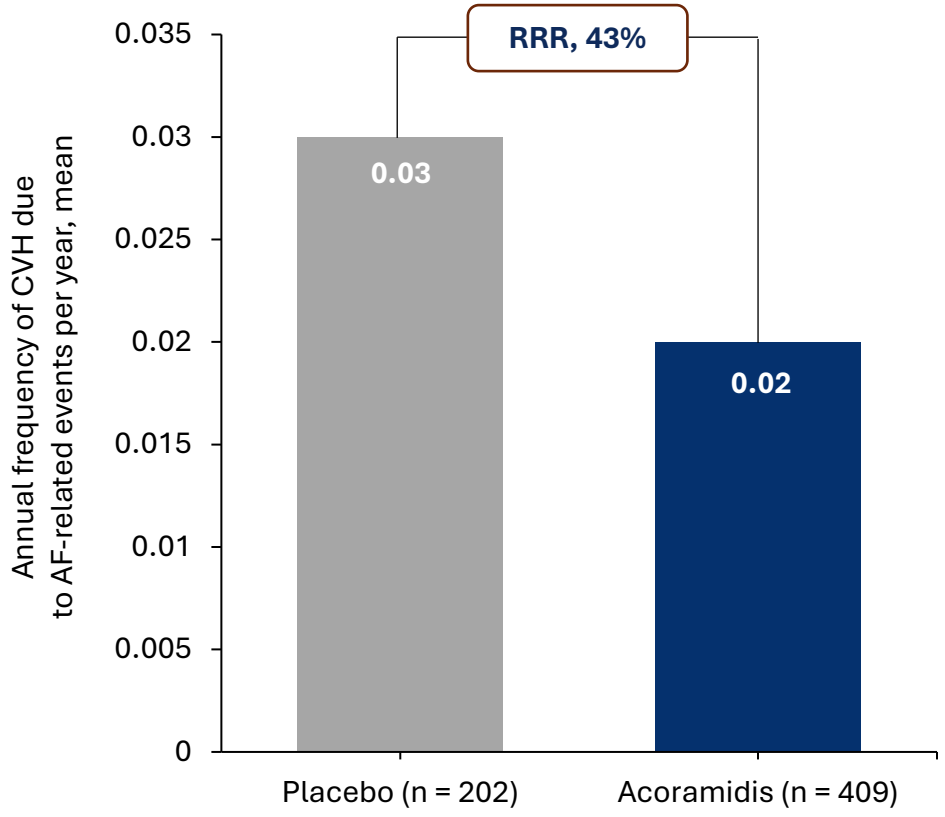
[1]Razvi, Y. et al., Eur Jour Heart Fail., 2024;26(2):383-393. [2]Alexander et al. JAMA Cardiol. [3]Fontana et al., Effect of acoramidis on functional capacity and quality of life in patients with variant ATTR-CM: Results from ATTRibute-CM Presented at ESC-HF 2025. Overall population and ATTRv-CM data 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



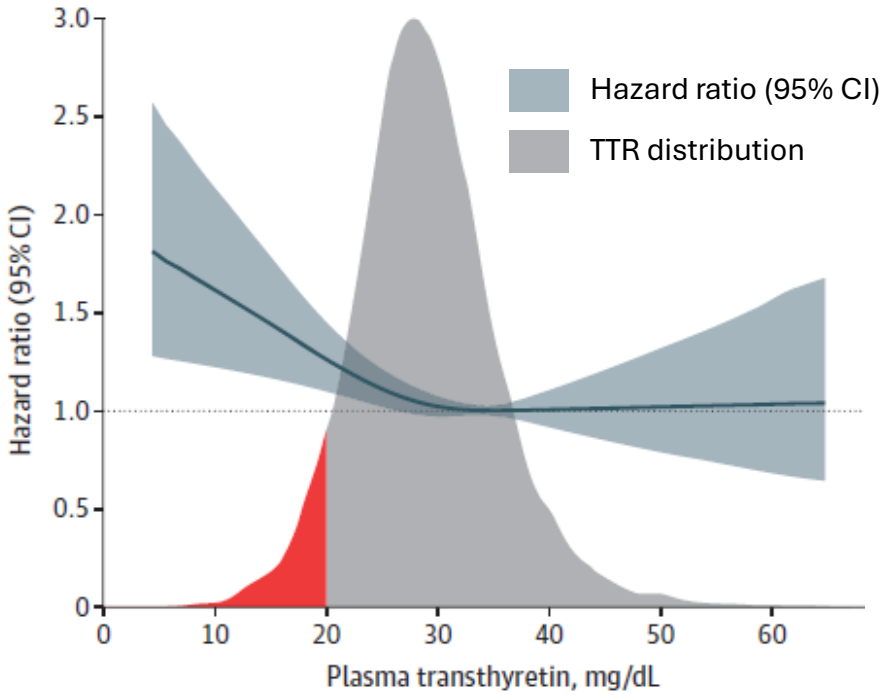
Annual Frequency of CVH Due to AF



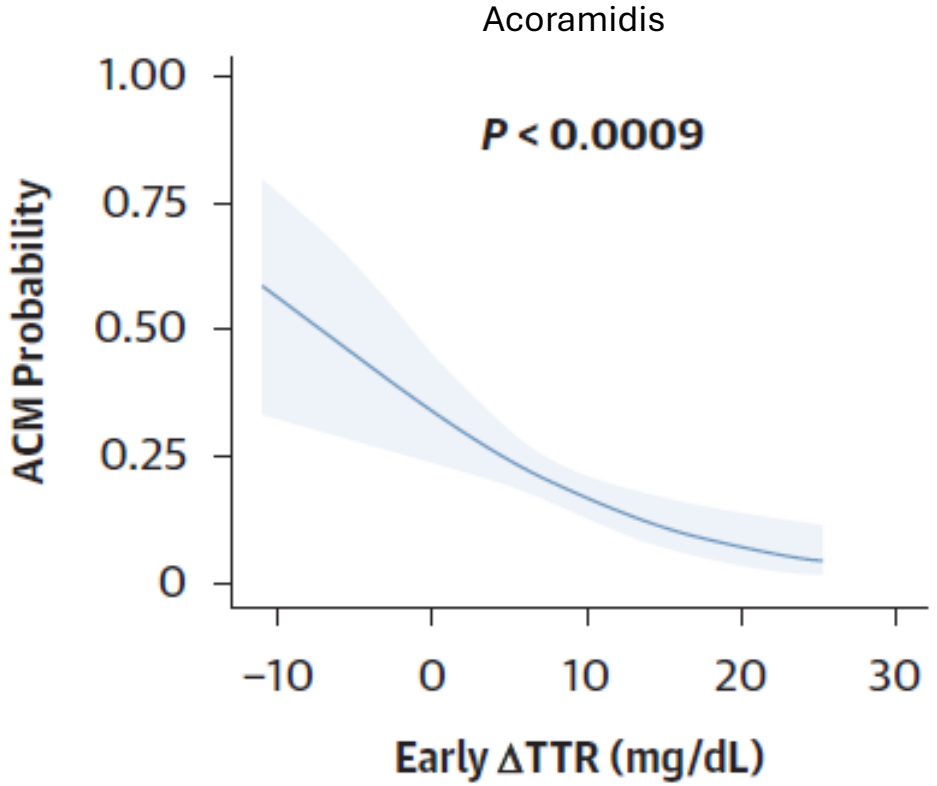
Source: Alexander, KM et al. Presented at: The Annual Congress of the Heart Failure Association of the European Society of Cardiology 2025;
Note: AF, atrial fibrillation; AFL, atrial flutter; AF-type adverse event covers the MedDRA preferred terms Atrial fibrillation, Atrial flutter, and Cardiac flutter.

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

130+ ATTR Variants

~600 Participants

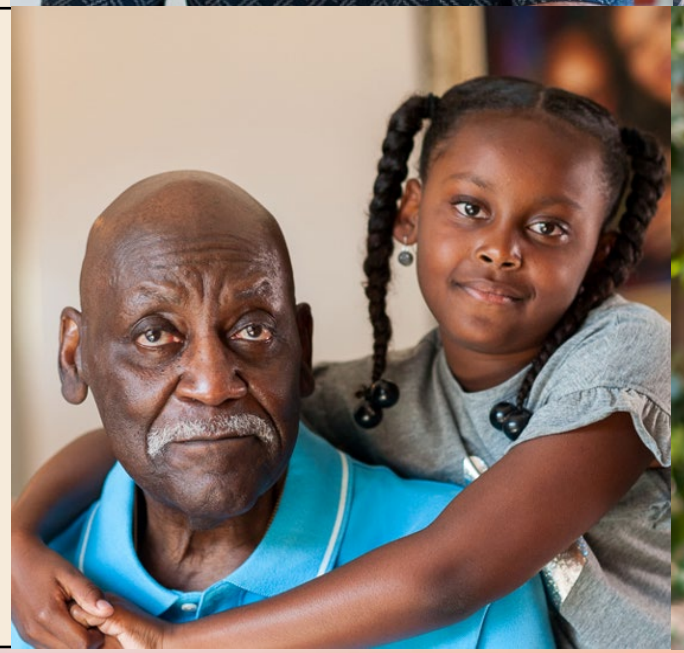
~100 Global Sites

~24 Countries

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

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

Depleter for ATTR-CM

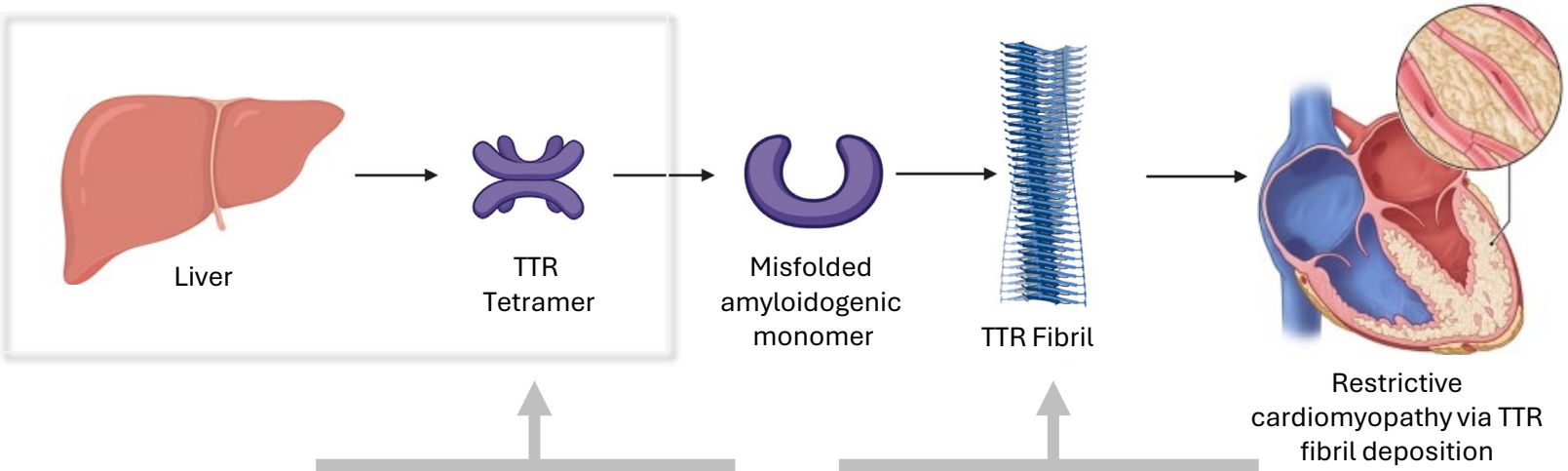


BridgeBio has initiated a depleter program to explore the potential of ATTR-CM disease reversal

ATTR-CM Disease Pathophysiology

Existing TTR therapies target upstream tetramer stabilization and synthesis…

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



Attruby/Beyonttra

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

Depleter

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

Led by Renowned Antibody Expert

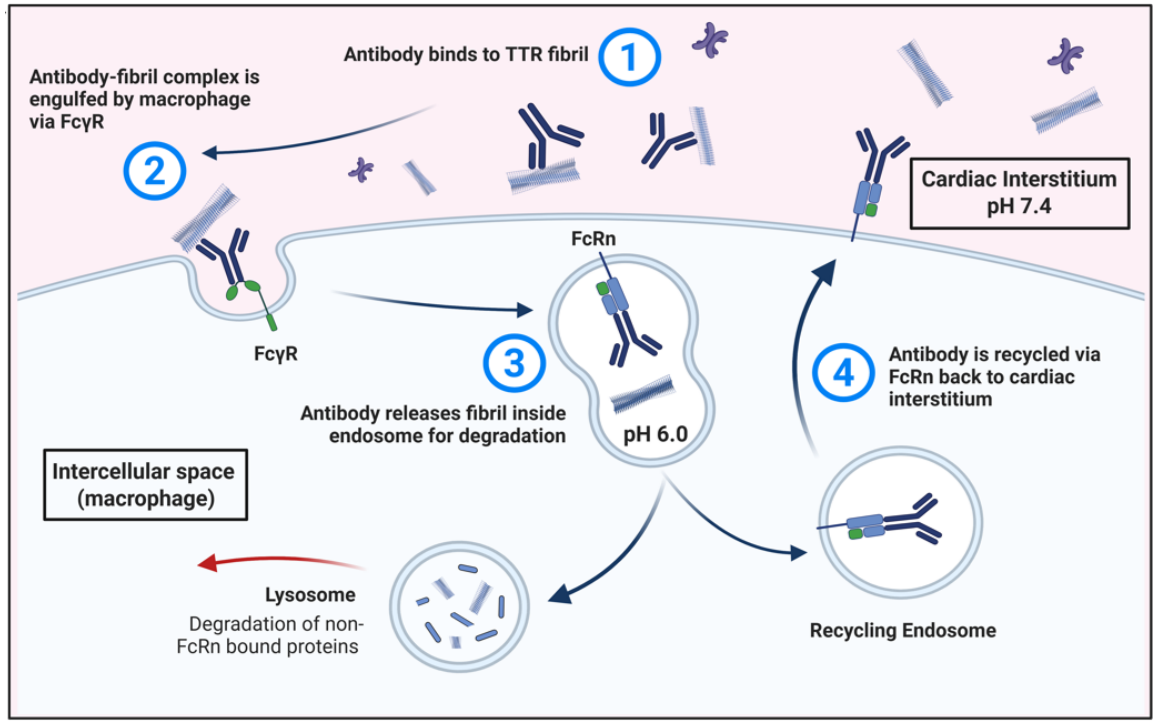
Dr. Richard Scheller

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

Source: Wanniarachchige et al., 2025

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

Depleter Mechanism of Action



BridgeBio's Differentiated Target Properties

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

   ➔ **Binds more target**

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

   ➔ **Clears more target**

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

   ➔ **Extends antibody half-life**

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

   ➔ **Extends antibody half-life**

Program expected to advance into the clinic in 2027–2028

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

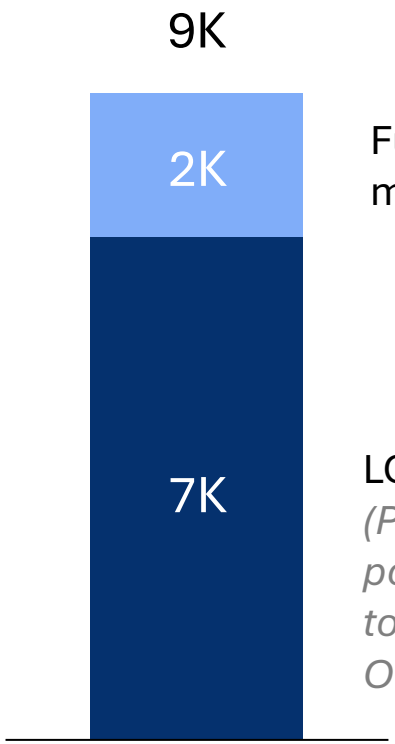
		Competitors		BridgeBio Target Profile	
	Property	Company A	Company B	BridgeBio's Depleter	Why it Matters
1	Improved fibril:tetramer binding ratio	✗	Limited	✓ >10× preferential binding to misfolded TTR fibrils vs. native TTR tetramers	• Maximizes on-target engagement with misfolded TTR fibrils • Minimizes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

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

BBP-418

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



Fukuyama congenital muscular dystrophy *(Japan)*

LGMD2I/R9[1,2] *(US + EU)*
(Phase 3 fully enrolled and positive interim analysis topline announced as of Oct. 2025)

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.

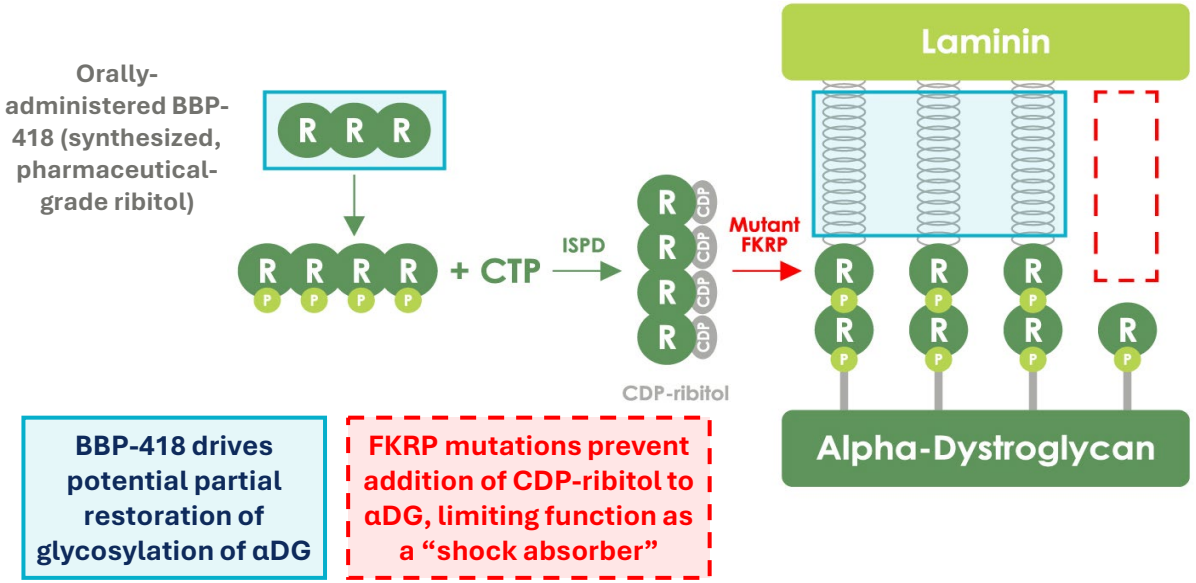
BBP-418 is an oral, disease-modifying therapy that targets LGMD2I/R9 at its source by restoring glycosylation of alpha-dystroglycan

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



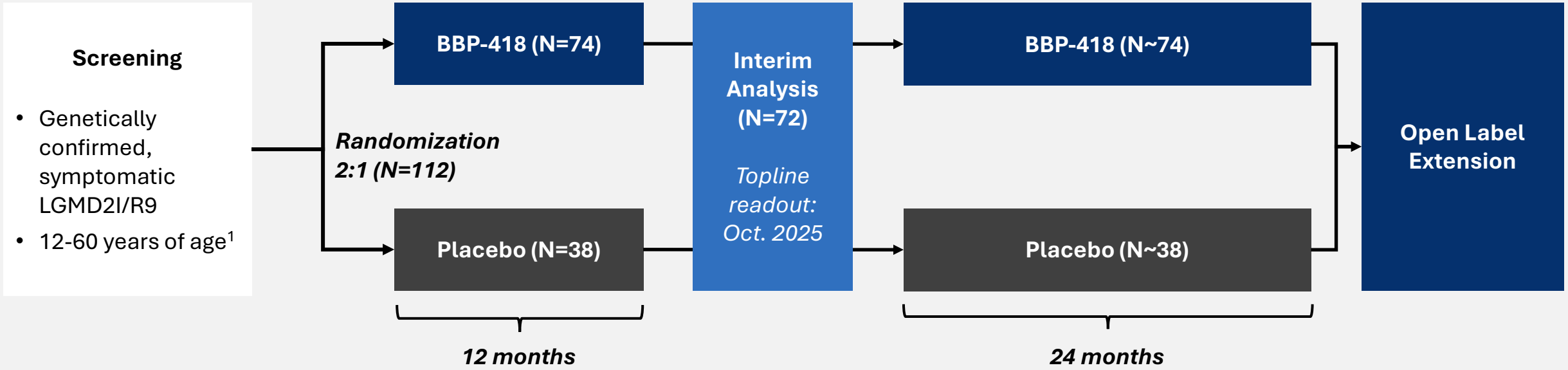
Design Principles

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;
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 months

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

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

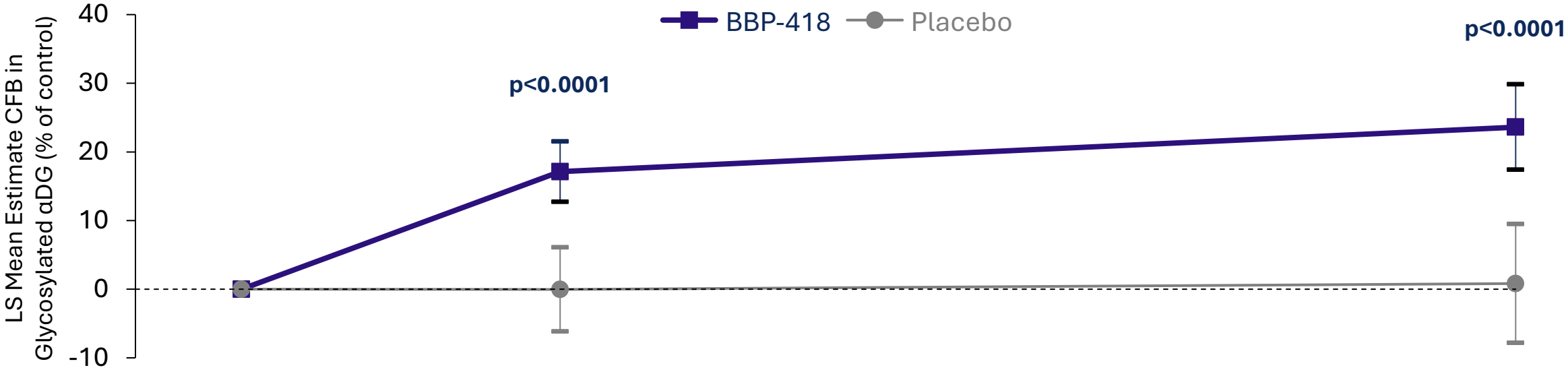
Ph. 3 FORTIFY interim analysis

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
Exploratory *(12 months)*	NSAD	• Trend favoring BBP-418 treated vs. placebo	✓ **Nominally statistically significant***and **clinically meaningful improvement** (2.6 point benefit vs. placebo)
Safety		• Well-tolerated *(consistent with Ph. 2 results)*	✓ Well-tolerated; consistent with Ph. 2 results

Note: * Nominally statistically significant based on exploratory analysis; analysis not part of alpha-controlled hierarchy at interim analysis
CFB = Change From Baseline; αDG = alpha-dystroglycan; 100MTT = 100-Meter Timed Test; FVC = Forced Vital Capacity; NSAD = North Star Assessment for Limb-Girdle Type Muscular Dystrophies

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

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

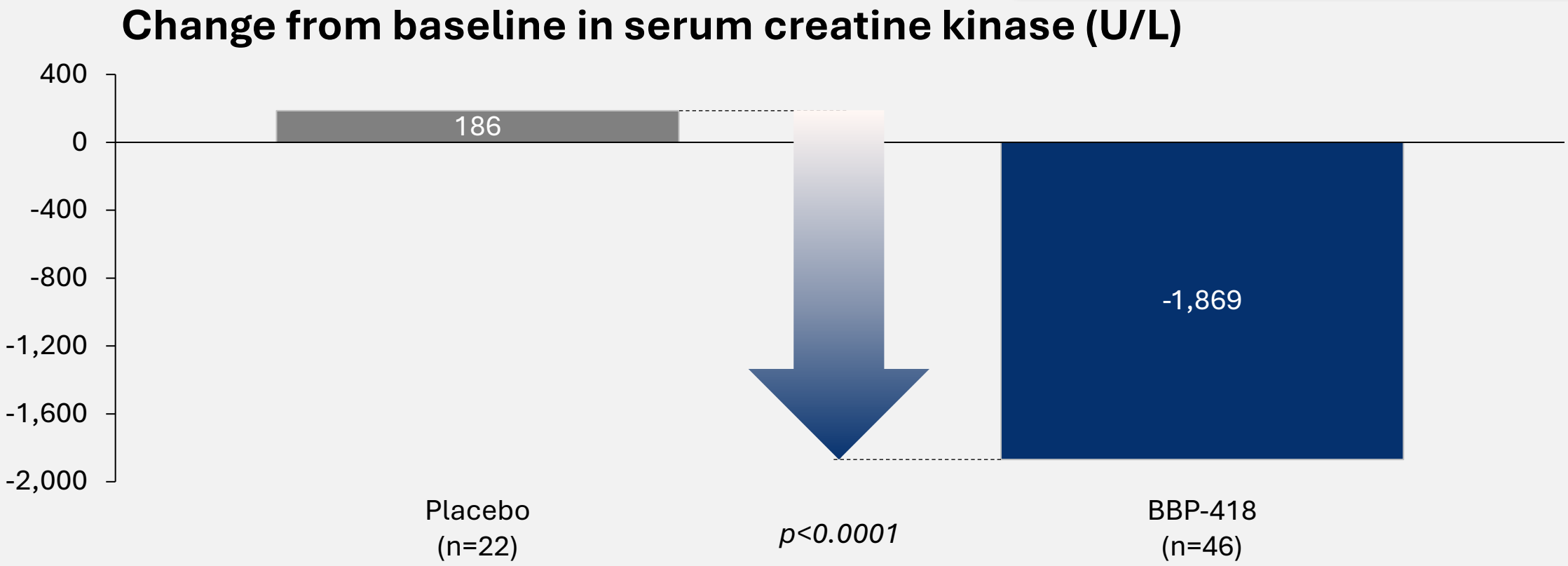


Timepoint	0 mo.	3 mo.	12 mo.
Cohort		All patients	Interim analysis
BBP-418		17.1% (n=71)	23.6% (n=49)
Placebo		-0.04% (n=36)	0.8% (n=22)

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

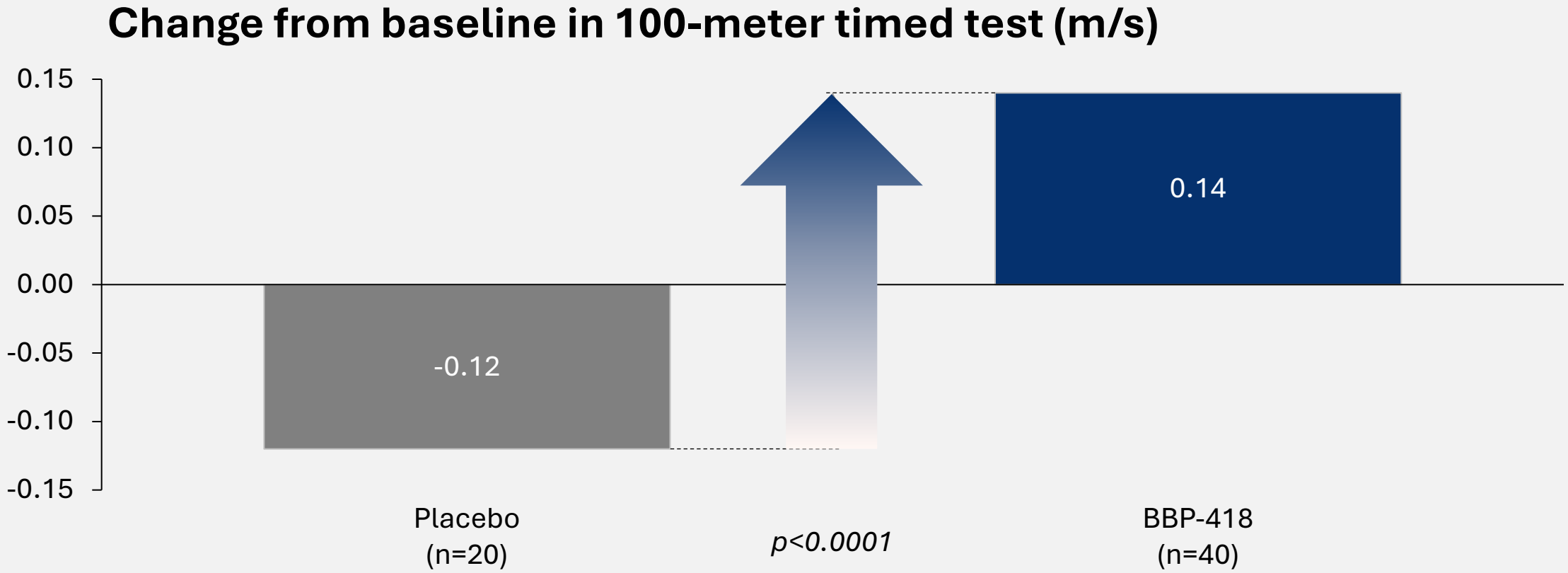
Reduction in muscle damage



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

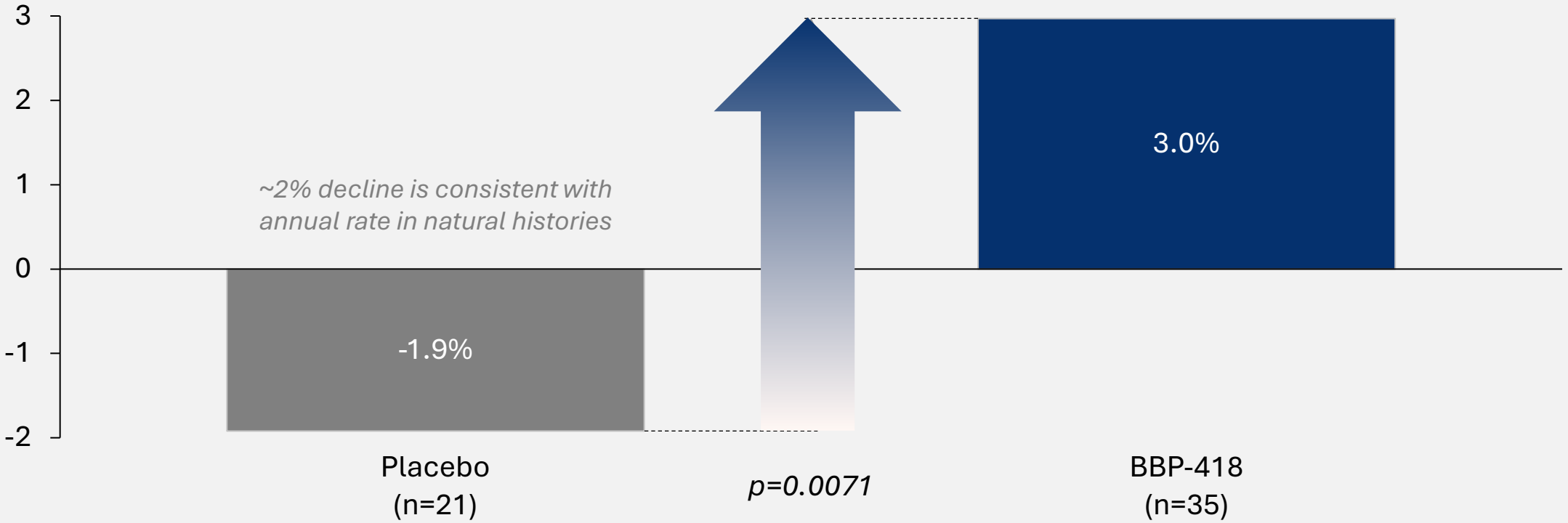


Least-Squares Mean Change from Baseline at 12-month timepoint; Comparison of BBP-418 change from baseline to Placebo change from baseline is based on a linear mixed model for repeated measures; n = number of participants with observed value. Source: Table 14.2.1.3.1.2.2a.2

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)

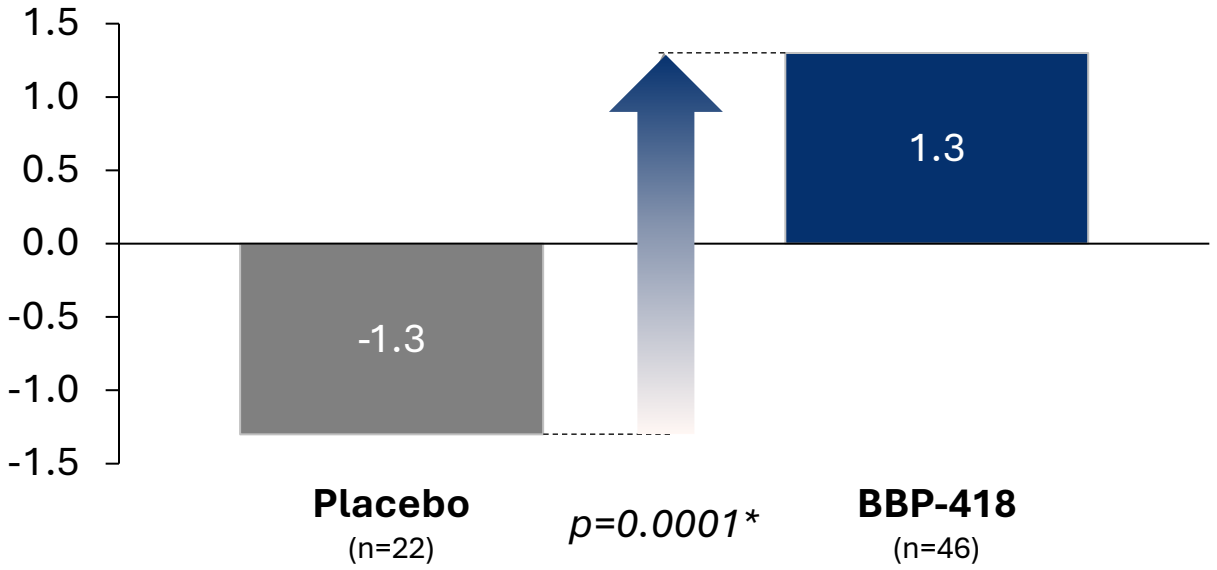


Least-Squares Mean Change from Baseline at 12-month timepoint; Comparison of BBP-418 change from baseline to Placebo change from baseline is based on a linear mixed model for repeated measures; n = number of participants with observed value. Source: Table 14.2.1.4.1.2.2a.2

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

Improved gross motor function

Change from baseline in NSAD (points)



Even a 1-point difference in NSAD can mean…

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

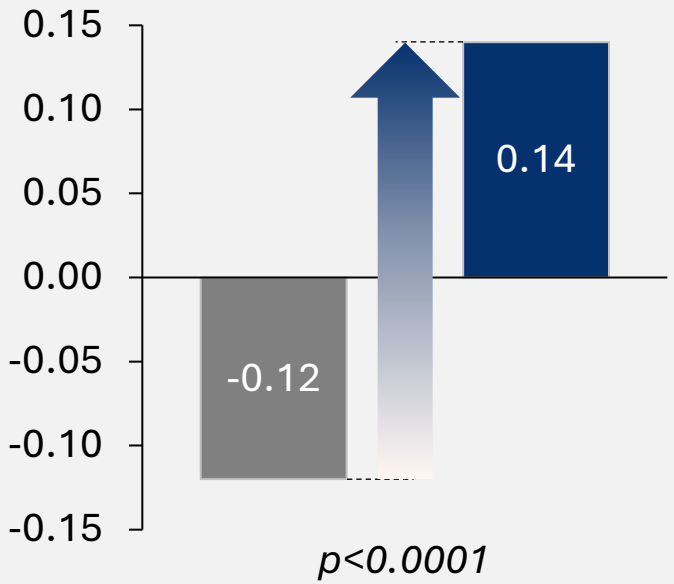
NSAD *(primary endpoint at 36 months)* benefit is highly clinically meaningful even at 12 months

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

NSAD = North Star Assessment for Limb-Girdle Type Muscular Dystrophies; Least-Squares Mean Change from Baseline at 12-month timepoint
Source: Table 14.2.1.5.1.1.1a.2

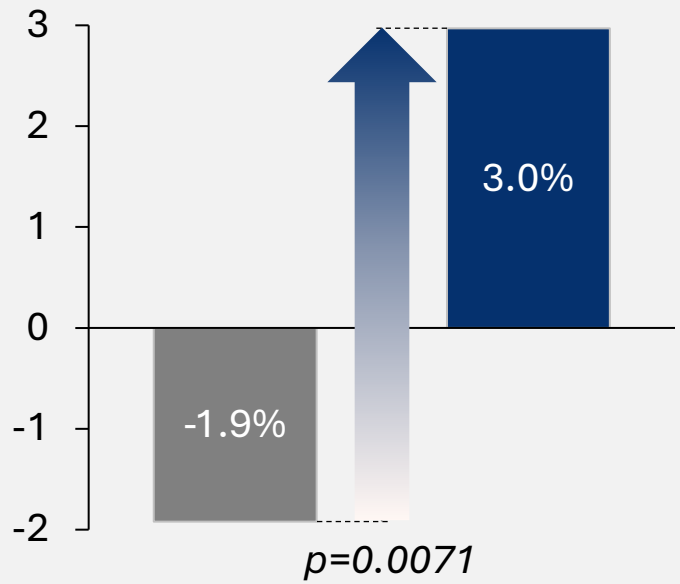
More than stabilization: BBP-418 treated individuals experienced meaningful improvements across clinical endpoints at 12 months

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



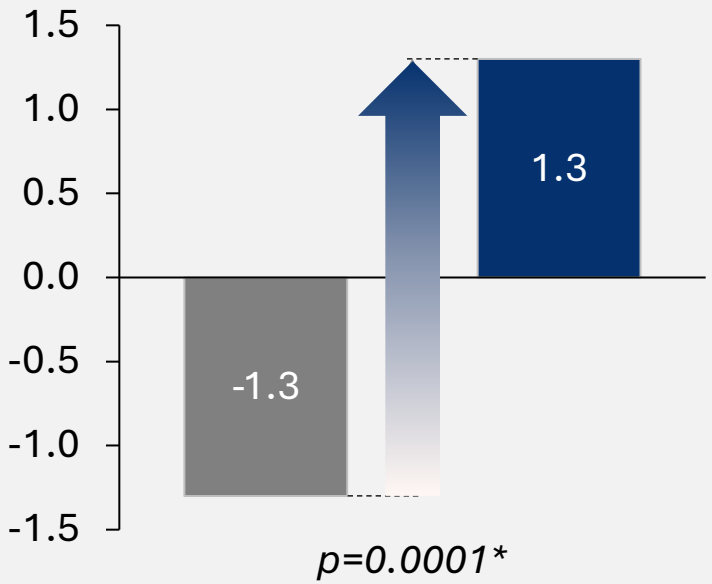
p<0.0001

Improved pulmonary function
CFB in FVC (% predicted)



p=0.0071

Improved gross motor function
CFB in NSAD (points)



*p=0.0001**

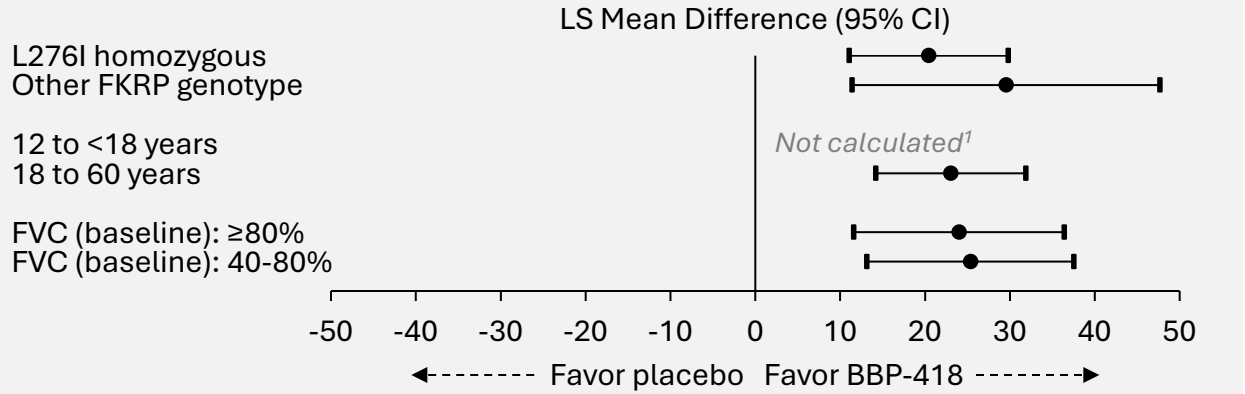
Key: Placebo BBP-418

CFB = Change From Baseline. Note: * Nominally statistically significant based on exploratory analysis; analysis not part of alpha-controlled hierarchy at interim analysis. 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; Table 14.2.1.5.1.1.1a.2

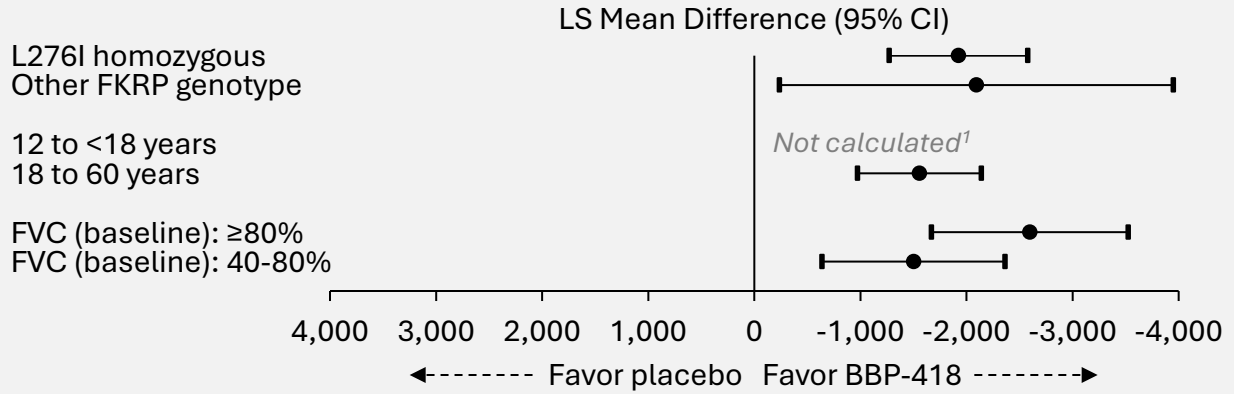
Planned subgroup analyses show consistent benefit of BBP-418 vs. placebo in all subgroups across α-controlled endpoints at 12 months

Biomarker

Glycosylated αDG (% of control)

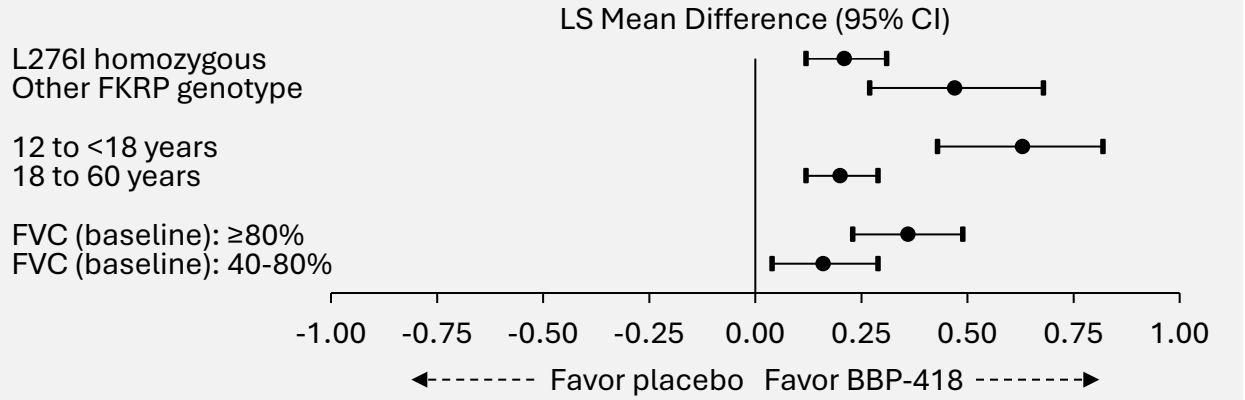


Serum creatine kinase (U/L)

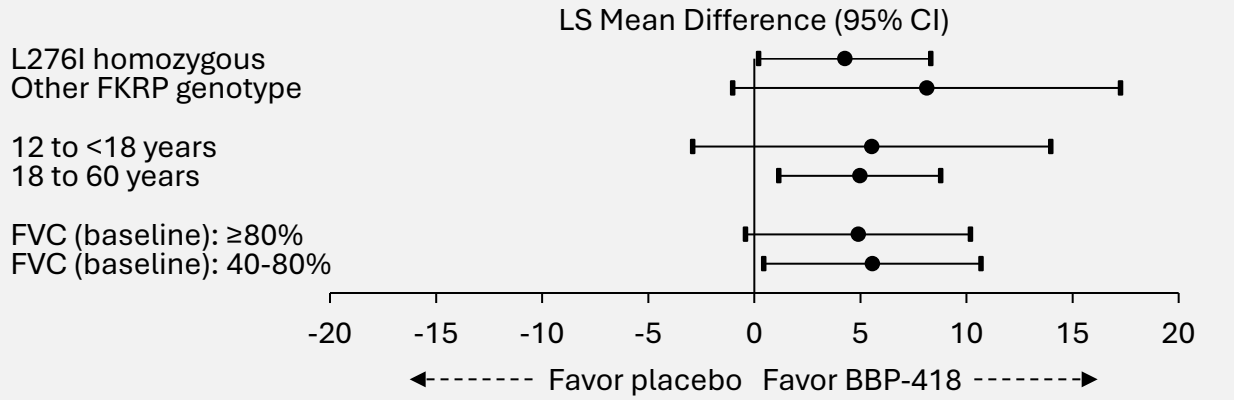


Functional

100-meter timed test (m/s)



Forced vital capacity (% predicted)



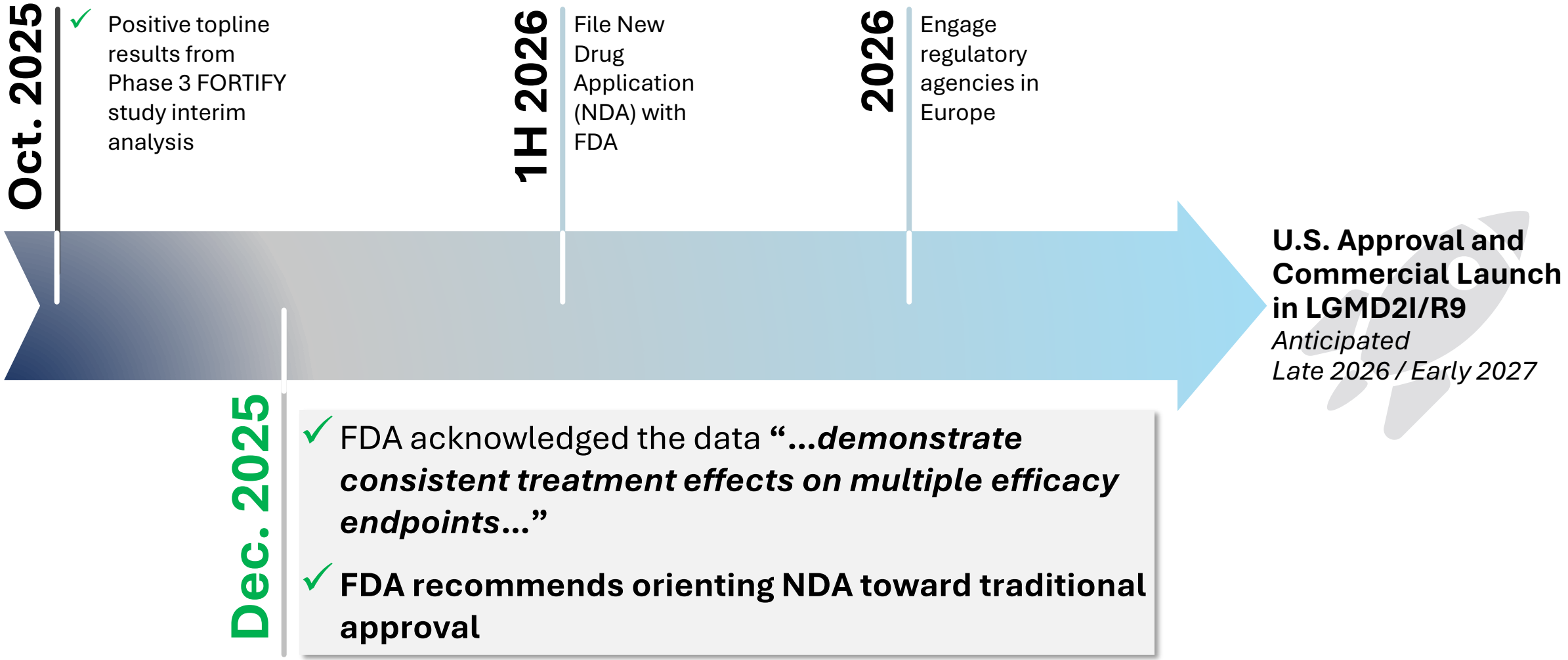
Note: [1] Pre-specified inferential statistics not performed for subgroups representing <15% of all participants in relevant analysis set; αDG=alpha dystroglycan; PBO=placebo; FKRP= Fukutin Related Protein; FVC=forced vital capacity. Source: Figure 14.2.1.3.1.0.2a.4; Figure 14.2.1.4.1.0.2a.4; Figure 14.2.1.1.1.0.3a.4; Figure 14.2.1.2.1.0.1a.4

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

We completed a successful meeting with FDA post interim analysis, and they recommend orienting our NDA toward traditional approval



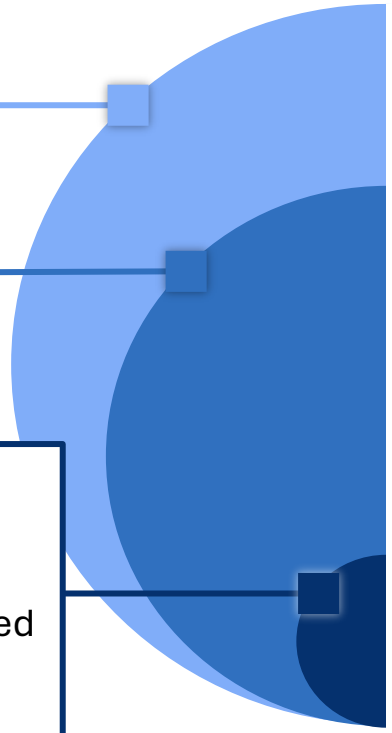
Encaleret

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]

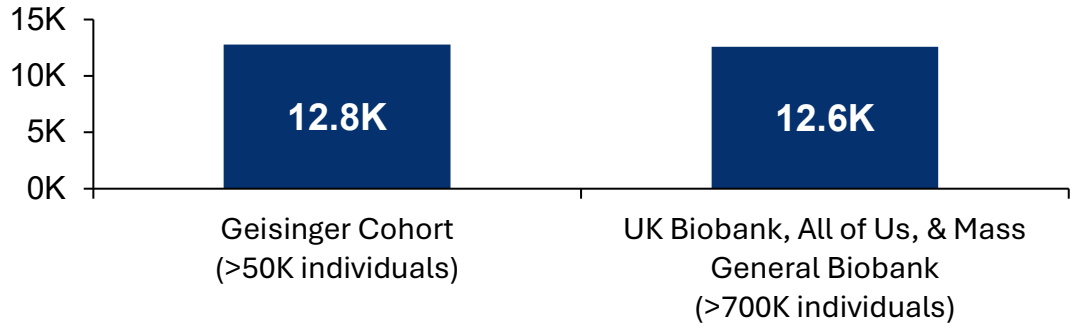
3K – 5K Currently Addressable
40-60% of symptomatic ADH1 patients are estimated to be diagnosed today; anticipate increase



An analogous ADH1 market is XLH

	XLH	ADH1
Prevalence (US)	12K[4]	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+[5]	$1B+

ADH1 variant frequency estimates in literature[1-2]



[1]Dershem, et al. Am J Hum Genet., 2020. [2] Chang, et al. Am J Hum Genet., 2025. [3]Roszko, et al. JBMR. 2022. [4]Dahir, et al. Jour Endo Soc., 2020. [5]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

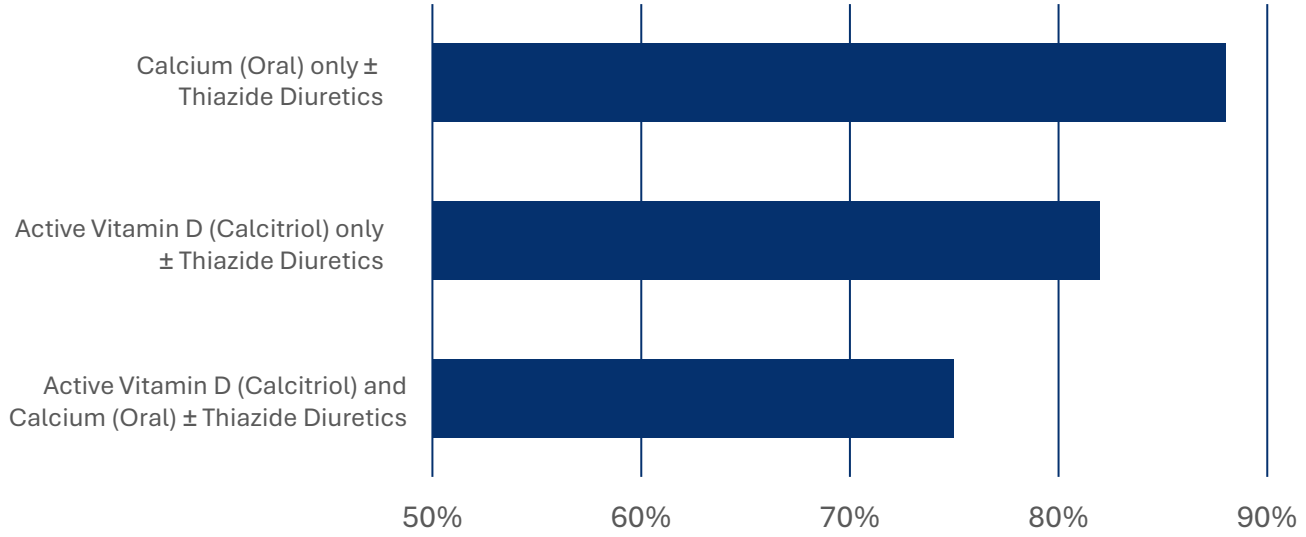
CaSR = Calcium-sensing Receptor; PTH = Parathyroid Hormone; Ca = Calcium.

Endocrinologists & nephrologists report low satisfaction with conventional therapy, leading to strong interest in novel investigational agents like encaleret

HCPs are not satisfied with conventional therapy

Dissatisfaction with Currently Available Treatment Options

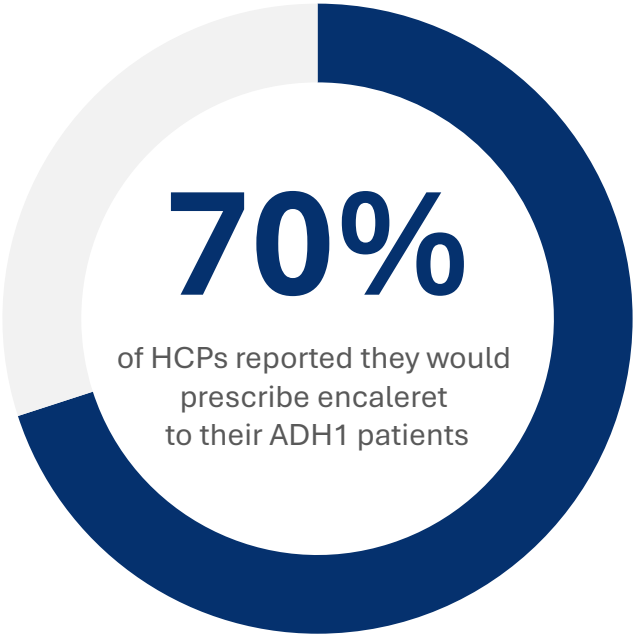
(N=44, % of HCPs indicating not highly satisfied with treatment option)



Survey of N=44 HCPs managing nonsurgical hypoparathyroidism and ADH1 patients

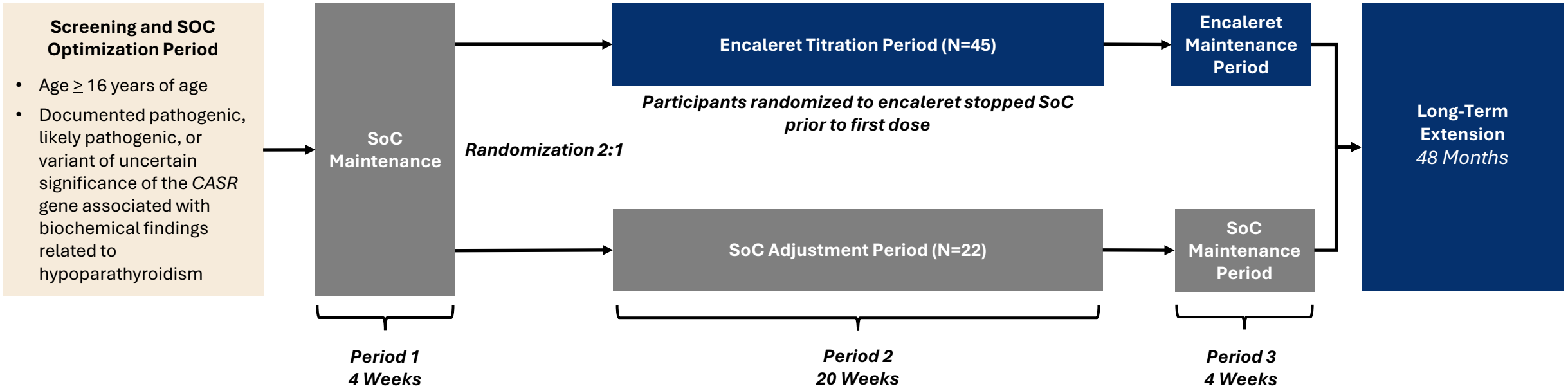
80% of HCPs indicated a product's ability to normalize urine calcium levels is the top attribute influencing treatment decisions for ADH1 patients

High likelihood HCPs would prescribe encaleret



Source: BridgeBio Market Research (N=44), December 2025.

Encaleret phase 3 registrational study design



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.

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

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

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

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

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.

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

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

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

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

Expanded availability of testing through sponsored genetic testing program

Detect Hypopara

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

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

E20.810

Billable ICD-10 code associated with ADH1 & ADH2

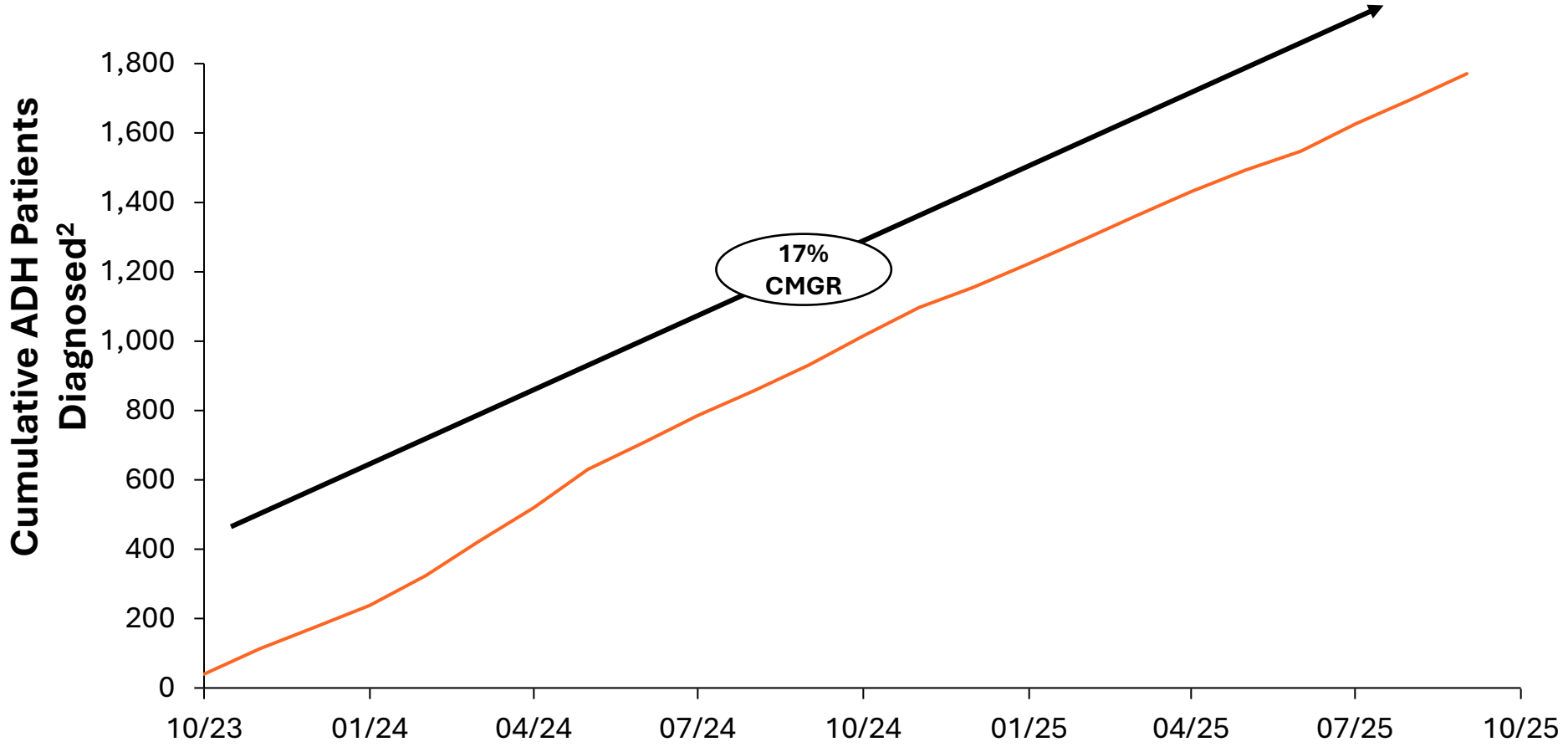
Enabled update to treatment guidelines specifying need for genetic testing[1]

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

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

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

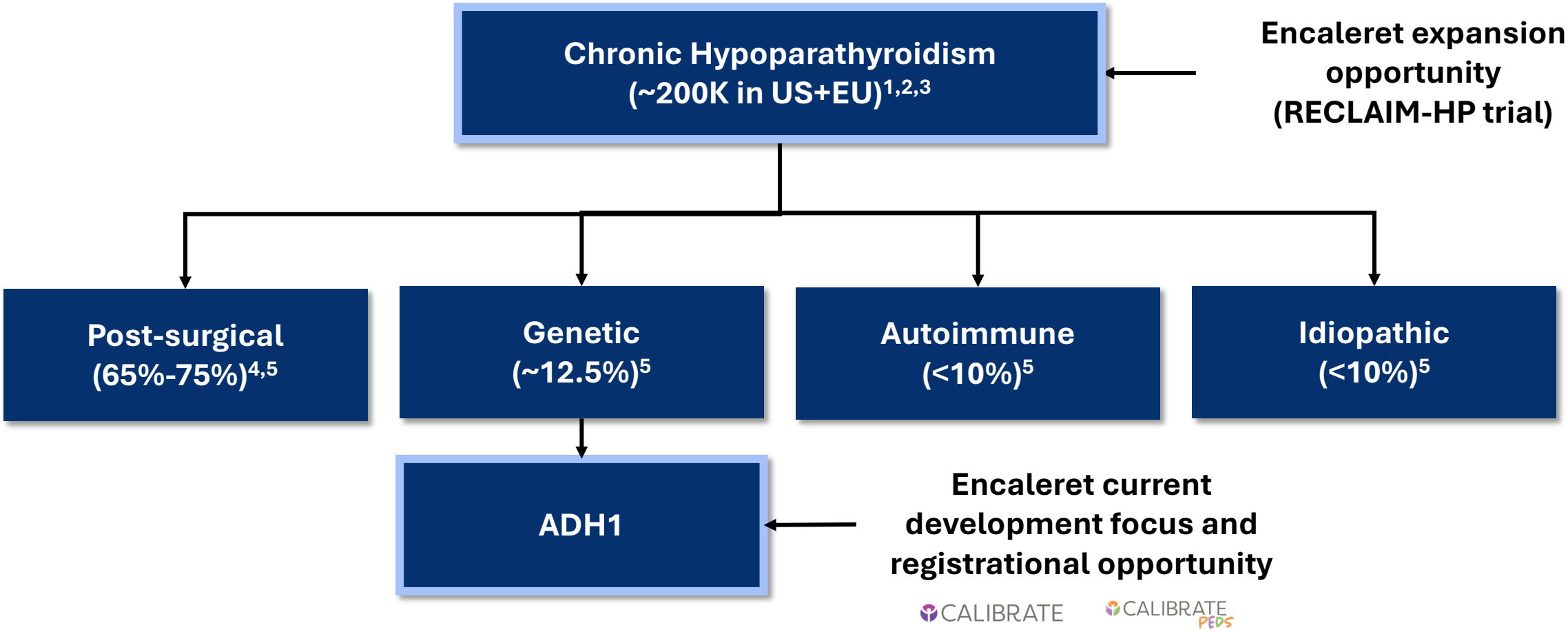
Over a 24-month period, >1,700 unique patients were diagnosed with ADH in the US



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

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

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

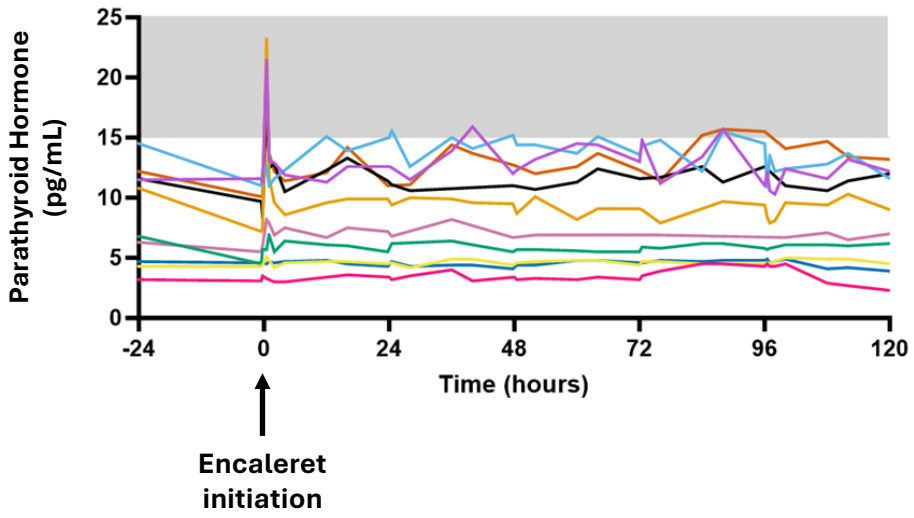


[1]Powers et al., J. Bone Miner. Res., 2013. [2]Clarke et al., J. Clin. Endocrinol. Metab., 2016. [3]Underbjerg et al., J. Bone Miner. Res., 2013. [4]Mannstadt et al., Nat. Rev. Dis. Primers., 2017.
[5]Kiam et al., Clin. Endocr., 2022.

Normalization of calcium homeostasis in post-surgical hypoparathyroidism (PSH) patients is indicative of encaleret's broader potential

Effect on PTH in PSH patients[1,2]

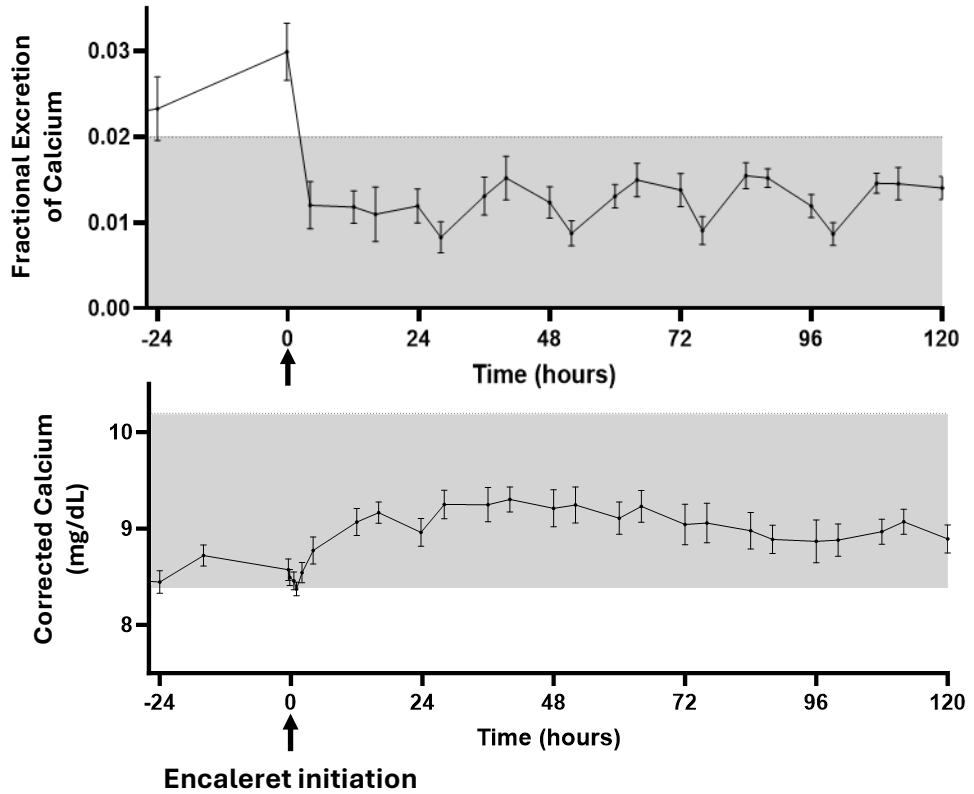
(N = 10)



Encaleret did not restore PTH levels in PSH patients

Effect on calcium homeostasis in PSH patients[1,2]

(N = 10)



Encaleret normalized blood and urine calcium through PTH independent pathways, demonstrating its potential to modulate urine and serum calcium levels through renal CaSRs for a broader set of chronic HPT patients

[1]Phase 2a trial (N=10) in post-surgical hypoparathyroidism patients (Hartley I.R. et al., presented at ASBMR 2025 Annual Meeting.)
[2]Gray shading indicates the normal range.

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

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

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

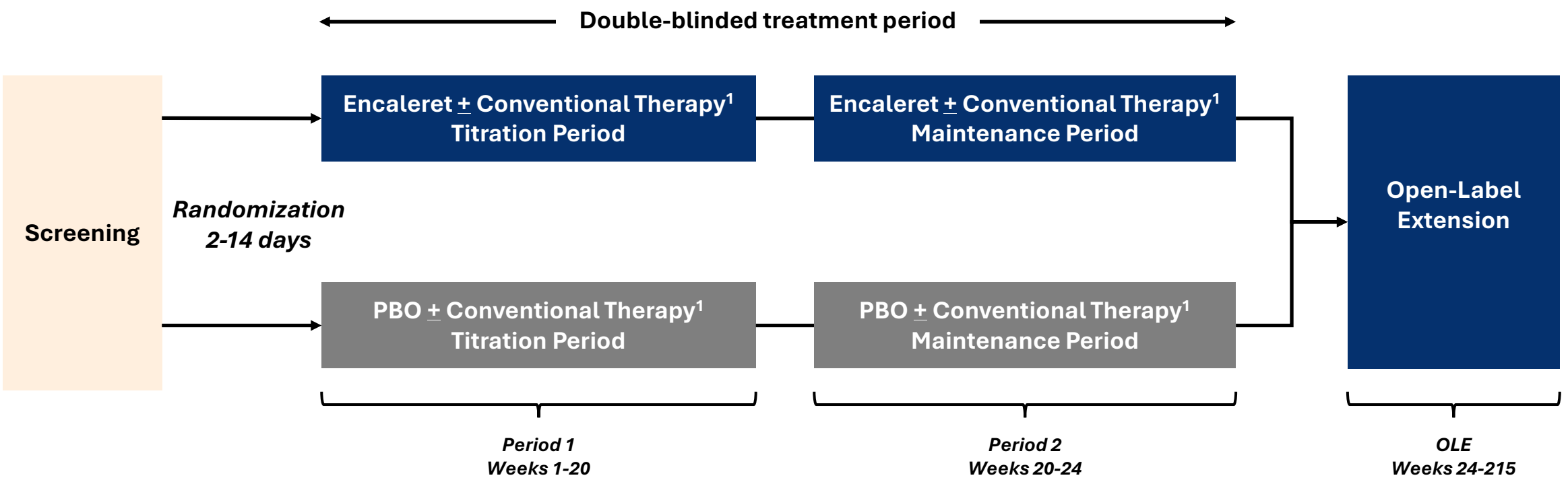
Announcing the RECLAIM-HP Phase 3 Study of Encaleret in CHP *Initiating in summer 2026*

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

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

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

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



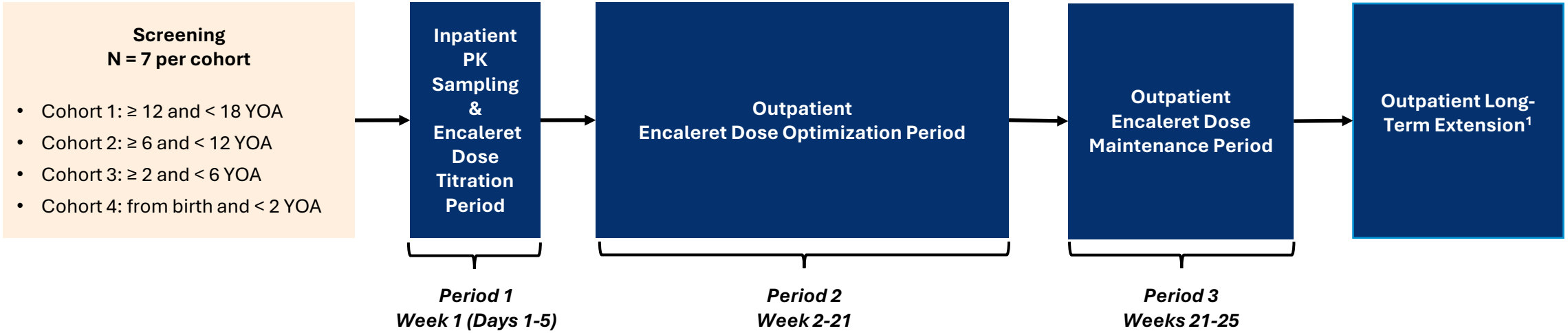
Primary Endpoint Proportion of participants achieving albumin-corrected blood and urine calcium within target range

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

CALIBRATE-PEDs Phase 2/3 registrational trial has initiated to study encaleret in pediatric ADH1 patients

NEW INFORMATION

CALIBRATE-PEDS: Global, multi-center, open-label, single-arm study



Primary Endpoint:

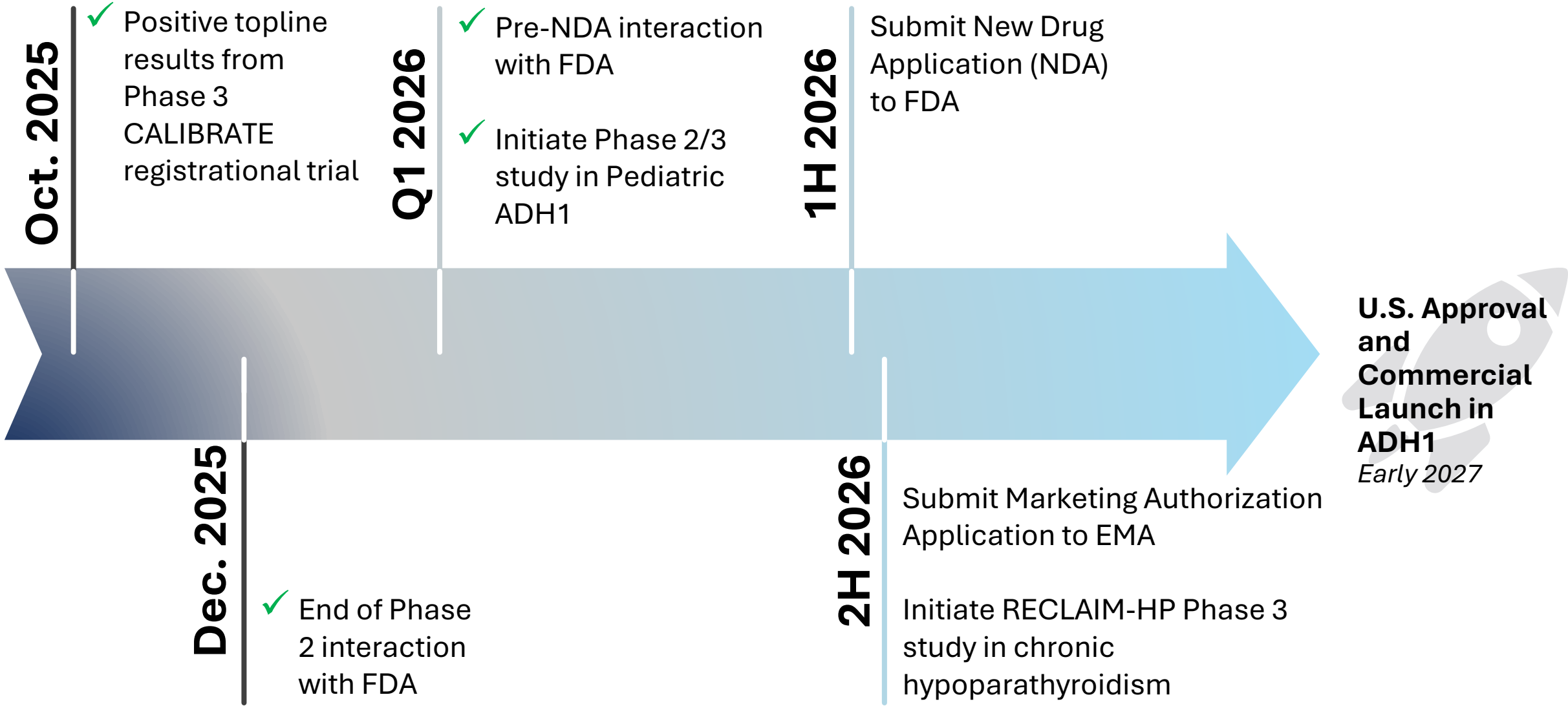
- PK parameters following encaleret administration in Period 1
- Proportion of participants achieving at Week 25:
  - Blood cCa within the target range **AND**
  - 24-hour urine Ca/Spot urine Ca/Cr within the reference range

Select Secondary and Exploratory Endpoints:

- Blood iPTH, 1,25-$(OH)_2$ Vitamin D, magnesium, and phosphate
- Safety assessment
- Bone turnover markers and bone mineral density
- Renal ultrasound and renal function
- Quality of life (SF-10) in children ≥ 6 YOA

[1]Long term extension is approximately 24 months in the EU and 48 months ROW

NDA submission for ADH1 planned in the first half of 2026 with two additional registrational studies to initiate this year



Infigratinib

PROPEL3

Infigratinib for Achondroplasia

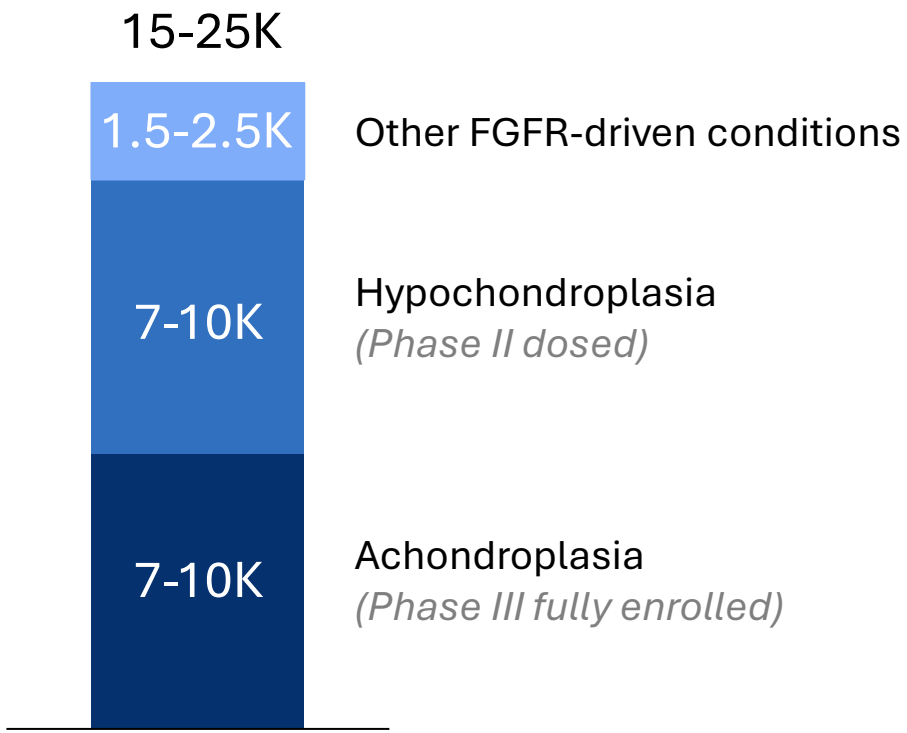
THANK YOU to the study participants, their families, investigators, clinical research staff, advocates and collaborating research partners

"Taken together, these best-in-class results highlight the transformative potential for infigratinib to address aspects of achondroplasia beyond linear height, and with a product administered orally."

Ravi Savarirayan, M.D., Ph.D. of Murdoch Children's Research Institute in Melbourne, Australia

There remains a significant unmet need for many children with skeletal dysplasias; this represents a large and rapidly growing market

Addressable people by indication in US/EU[1]
(current population eligible for treatment)



55,000 individuals with achondroplasia globally

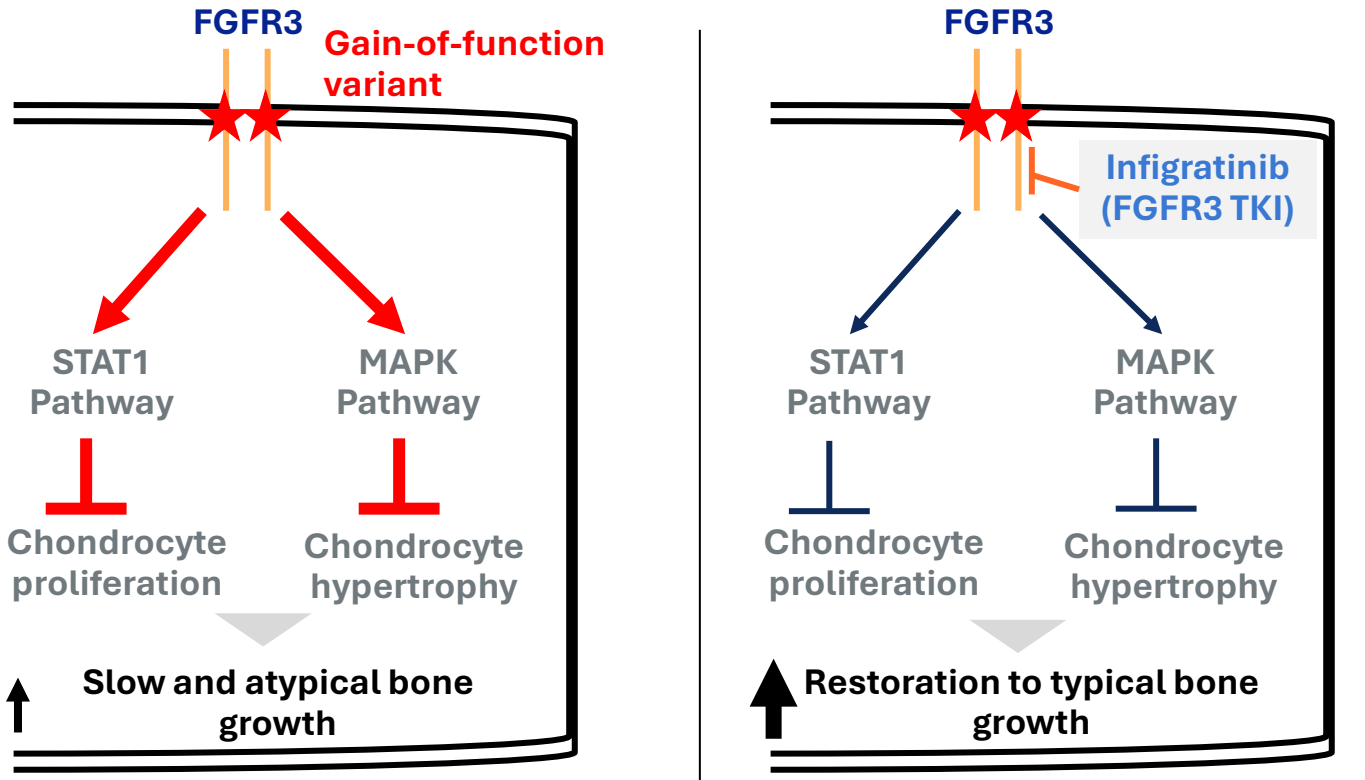
Represents diagnosed and addressable ACH population with open growth plates

$5B+ potential global market

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

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

Infigratinib is a sprinkle capsule that can be taken intact whole, or sprinkled

Capsules
(17 mm long*)

Granules
(2 mm long)

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

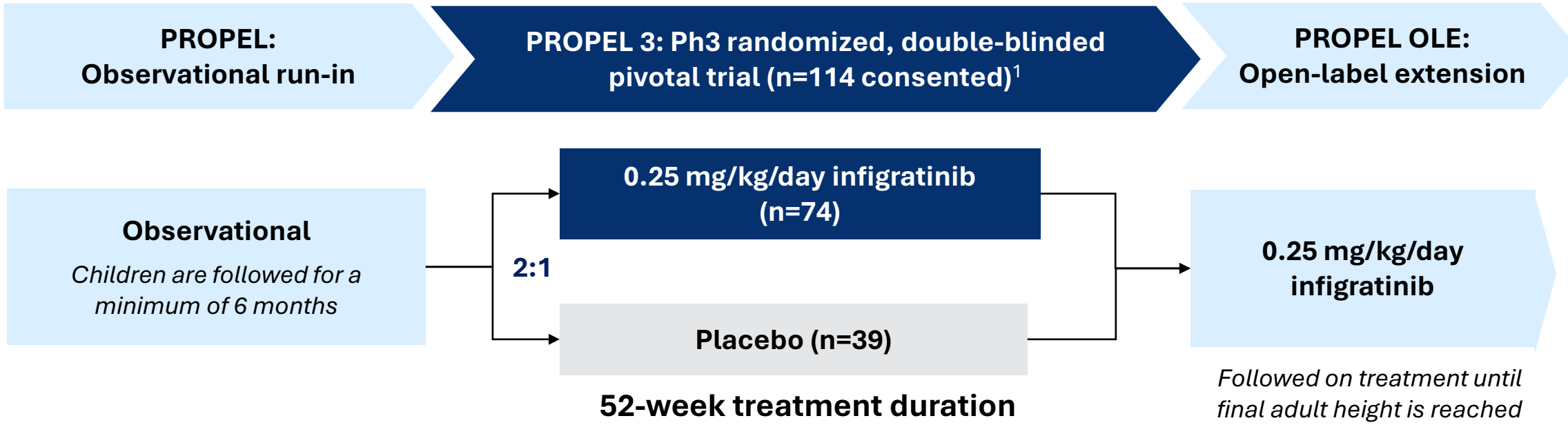
[1]Savarirayan R, et al. N Engl J Med. 2024;392(9):865–874. [2]BridgeBio data on file.
*Infigratinib is an investigational agent that is not approved for use by any regulatory authority. Size 2 capsules are shown in photo.

Infigratinib exceeded the bar for a potential best-in-class therapeutic option, in the broadest age range studied across any achondroplasia trial

Target Clinical Profile for Commercial Success	Outcomes
CFBL in AHV: More than +1.5 cm/yr against placebo at Week 52	**Met the primary endpoint** with mean difference against placebo of **+2.10 cm/yr** (p<0.0001), and a LS mean difference against placebo of **+1.74 cm/yr** (p<0.0001), the largest change observed in a randomized trial for ACH.
CFBL in height Z-score (ACH charts): More than +0.3 SD on treatment arm at Week 52	The LS mean improvement on the tx arm was +0.41 SD, the largest improvement observed in a RCT in ACH. LS mean difference against placebo was +0.32 SD (p<0.0001), the largest difference observed in a RCT in ACH.
Proportionality: More than 0.05 decrease in upper to lower body ratio on treatment arm	In a pre-specified exploratory analysis of children <8yrs of age (>50% of trial), there was a statistically significant LS mean change from baseline against placebo of -0.05 (p<0.05). In the overall population, infigratinib achieved a LS mean decrease of -0.05 on the tx arm, with a favorable LS treatment difference of -0.02 versus placebo at Week 52 (p=0.1849)
Safety: No symptomatic hypotension. Less than 10% low-grade hyperphosphatemia rate.	Well-tolerated safety profile, **consistent with no inhibition of FGFR1 and FGFR2**. ▪ No discontinuations related to study drug ▪ No serious adverse events related to study drug ▪ 3 cases of hyperphosphatemia (4%), all mild, asymptomatic, transient and did not require dose reduction or discontinuation ▪ No FGFR1 or FGFR2 associated AEs (E.g., retinal or corneal) ▪ No AEs associated with CNP analogues: symptomatic hypotension, ISRs, or hypertrichosis

BBIO data on file; Tx : treatment; pbo : placebo; ISR : injection site reactions; AEs : adverse events; RCT : randomized controlled trial. *Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

PROPEL 3 - Phase 3 registrational study design



Key inclusion criteria

- Children 3 – <18 years old with open epiphyses

Primary endpoint:

- Change from baseline in annualized height velocity (AHV) at week 52 compared to placebo

Key secondary endpoints:

- Change from baseline in height z-score (in relation to ACH tables)
- Change from baseline in upper body : lower body segment ratio

Other secondary endpoints:

- Change in physical functioning; HRQoL, cognitive function, participant and caregiver evaluation of treatment benefit (qualitative interview)

1-One participant withdrew consent before dosing, Clinicaltrials.gov ID: NCT06164951, HRQoL: health-related quality of life, ACH: achondroplasia.
*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Demographic and baseline characteristics

Baseline demographics	Infigratinib (n=74)	Placebo (n=39)	All Participants (n=113)
Age (yrs), mean (SD) (min, max)	7.92 (2.7) (3.7 – 14.4)	7.74 (2.5) (3.9 – 14.9)	7.86 (2.7) (3.7 – 14.9)
Age group, n (%)			
3 – <5	10 (13.5)	4 (10.3)	14 (12.4)
5 – <11	50 (67.6)	31 (79.5)	81 (71.7)
11 – <18	14 (18.9)	4 (10.3)	18 (15.9)
Sex			
Male, n (%)	42 (56.8)	23 (59.0)	65 (57.5)
Female, n (%)	32 (43.2)	16 (41.0)	48 (42.5)
Baseline AHV (cm/year), mean (SD)	4.28 (1.39)	4.57 (1.45)	4.38 (1.41)
Height z-score (ACH reference), mean (SD)	0.09 (0.87)	-0.00 (1.00)	0.05 (0.91)

The study population was adequate to evaluate the study objectives, with well balanced treatment arms

BBIO data on file; SD : standard deviation; AHV : annualized height velocity.
*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Primary endpoint: Change from baseline to Week 52 in AHV

	Infigratinib (n=74)	Placebo (n=39)
Baseline AHV (cm/yr), Mean (SE)	4.28 (0.16)	4.57 (0.23)
Week 52 AHV (cm/yr), Mean (SE)	5.75 (0.15)	3.95 (0.14)
CFBL AHV at Week 52, Mean (SE)	1.48 (0.22)	-0.62 (0.29)
Infigratinib vs. placebo difference, Mean (95% CI) **P-value[1]**	**2.10 (1.38, 2.81)** **p < 0.0001**	
CFBL AHV at Week 52, LS Mean (SE)	1.58 (0.19)	-0.16 (0.23)
Primary Endpoint: Infigratinib vs. placebo difference, LS Mean (95% CI) **P-value[2]**	**1.74 (1.31, 2.17)** **p < 0.0001**	

Infigratinib has demonstrated the largest CFBL in AHV at Week 52 in a randomized trial

BBIO data on file; 1- Nominal; 2- ANCOVA model; CFBL : change from baseline; AHV : annualized height velocity.
*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

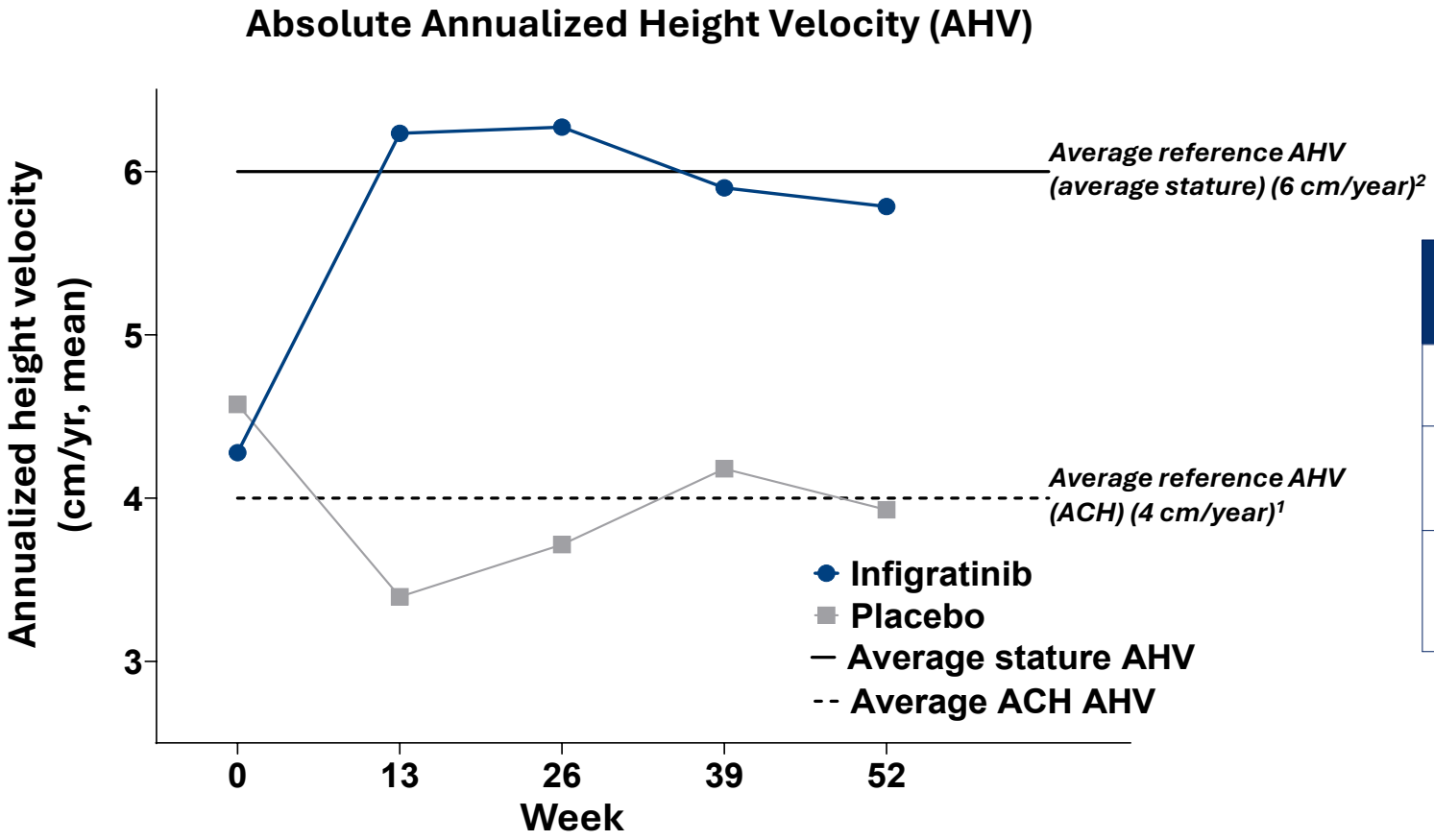
Subgroup analyses: Change from baseline to Week 52 in AHV by age group

Age subgroups	3 to < 5 years		5 to < 11 years		11 to < 18 years	
	Infigratinib (n=10)	Placebo (n=4)	Infigratinib (n=50)	Placebo (n=31)	Infigratinib (n=14)	Placebo (n=4)
Infigratinib vs. placebo difference, Mean	2.30		1.93		2.78	
Infigratinib vs. placebo difference, LS Mean	1.47		1.84		1.40	

Across all of the above age subgroups, infigratinib demonstrated the largest CFBL in AHV at Week 52 in a randomized trial for ACH

BBIO data on file; AHV : annualized height velocity; CFBL : Change from Baseline.
*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Secondary endpoint: Absolute AHV at Week 52

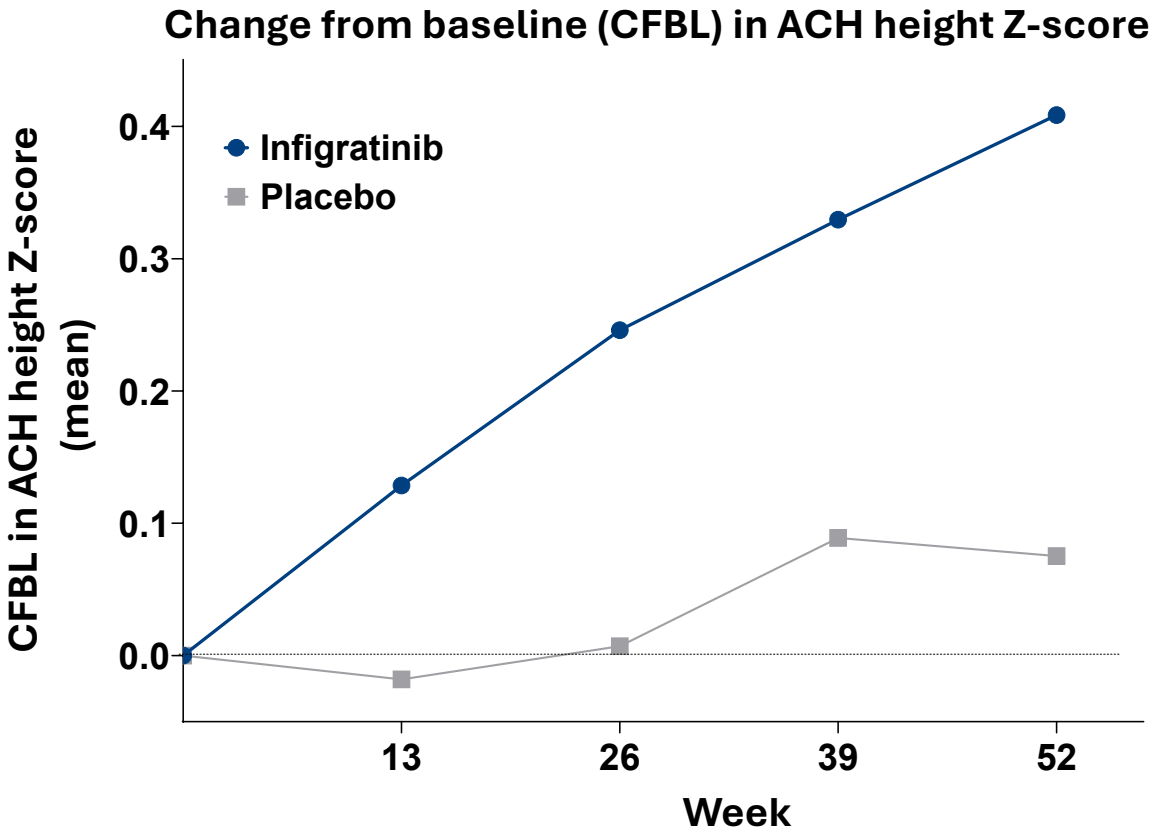


Absolute AHV at Week 52	Infigratinib (n=74)	Placebo (n=39)
LS Mean	**5.96**	**4.22**
Infigratinib vs. placebo difference, LS mean	1.74	
P-value	p<0.0001	

Infigratinib has demonstrated the largest absolute AHV (LS mean) at Week 52 in any randomized trial

BBIO data on file; ACH: achondroplasia; 1-Hoover-Fong et al., 2021; 2-Sperling, 2020.
*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Key secondary endpoint: Change from baseline in height Z-score in relation to an achondroplasia population over 52 weeks

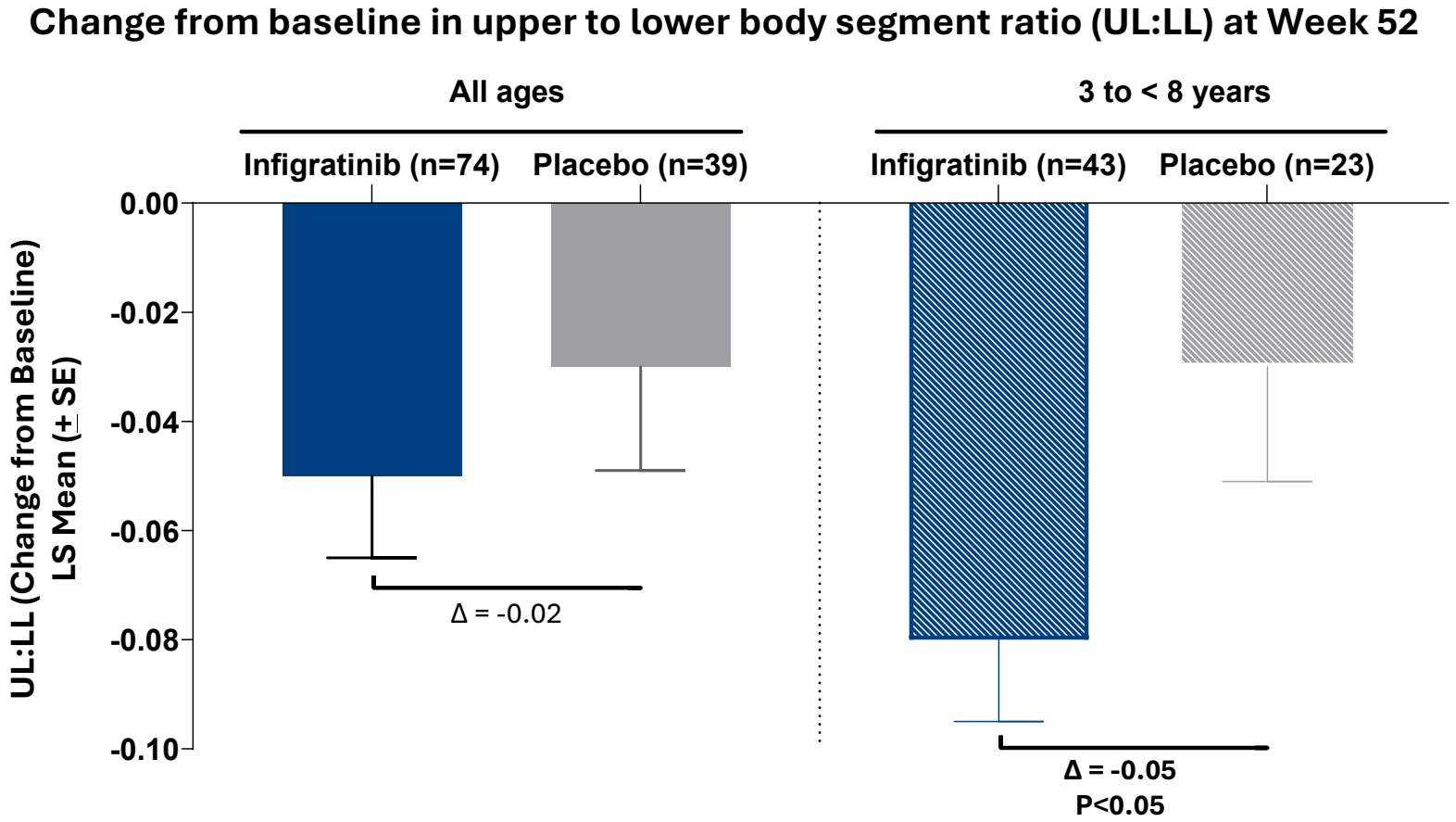


ACH Height Z-score at Week 52	Infigratinib (n=74)	Placebo (n=39)
LS Mean	0.41	0.09
Infigratinib vs. placebo difference, LS mean	0.32	
P-value	p<0.0001	

Infigratinib significantly increased the height Z-score in relation to an ACH population over 52 weeks by 0.32SDs (p<0.0001)

BBIO data on file.
*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Key secondary endpoint: Change from baseline to Week 52 in upper-to-lower body segment ratio



Infigratinib is the first therapeutic option to demonstrate a statistically significantly result on proportionality against placebo in a RCT for ACH

BBIO data on file; Exploratory analysis pre-specified in the SAP; RCT : randomized controlled trial.
*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Infigratinib at 0.25 mg/kg was well-tolerated, with no safety signal indicating inhibition of FGFR1 or 2

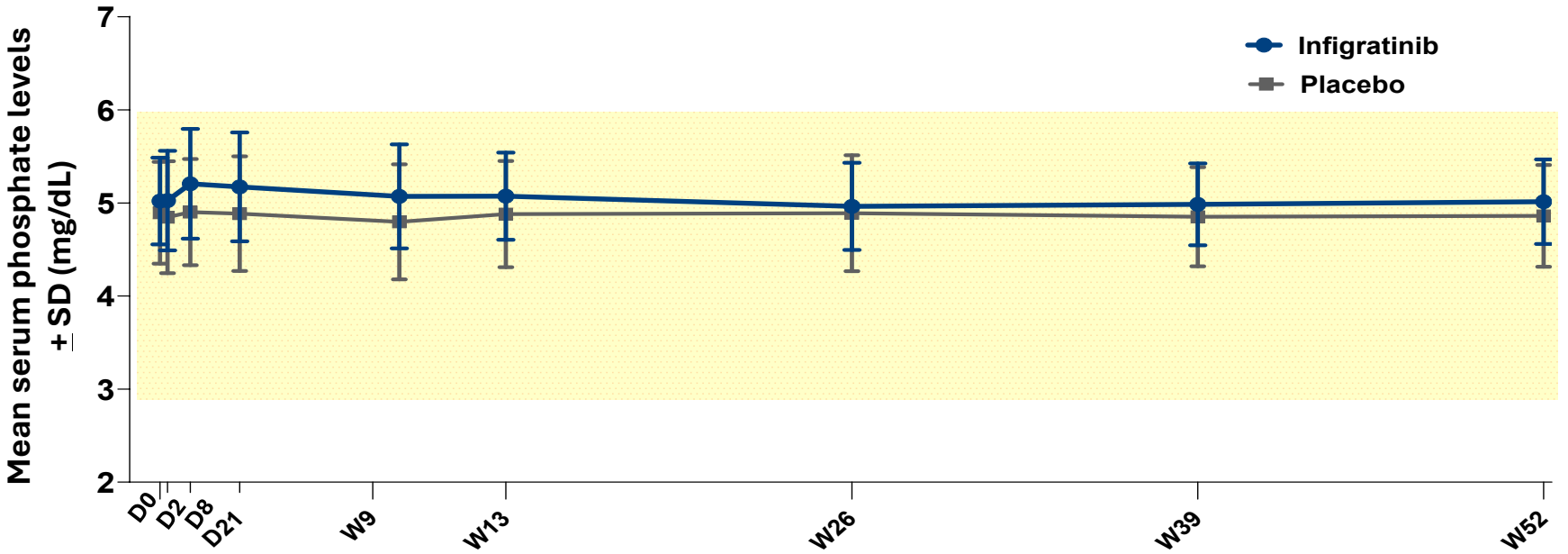
- The majority of treatment-emergent adverse events (TEAEs) were grade 1 or 2 in severity and typical for children of these ages
- TEAEs were balanced between the treatment and placebo arms
- No serious adverse events related to study drug
- No discontinuations due to AEs related to study drug

Areas of interest

- 3 cases of hyperphosphatemia, all mild, asymptomatic, and transient; no cases required a dose reduction or discontinuation
- No AEs associated with the inhibition of FGFR1 or FGFR2 (e.g. retinal or corneal)

Mean serum phosphate levels were comparable across infigratinib and placebo at all measured time points

Mean Serum Phosphate Levels



For all participants, the maximal change in phosphate levels was within the typical intra-subject variability in the pediatric population[1]

Mean phosphate levels (mg/dL)	Baseline	Day 2	Day 8	Day 21	Week 9	Week 13	Week 26	Week 39	Week 52
Infigratinib	*5.0*	*5.0*	*5.2*	*5.2*	*5.1*	*5.1*	*5.0*	*5.0*	*5.0*
Placebo	*4.9*	*4.9*	*4.9*	*4.9*	*4.8*	*4.9*	*4.9*	*4.9*	*4.9*

Mean phosphate levels at all measured time points were similar and within normal ranges

Shaded area show reference range of serum phosphorus for study central lab reference ranges for ages 3 to 13 years old;
BBIO data on file; D:day; W : week; 1 – Hanudel (2020). *Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

NDA and MAA submissions expected in 2H 2026

Advance infigratinib towards registration in achondroplasia

Submit New Drug Application to FDA
2H 2026

Submit Marketing Authorization Application to EMA
2H 2026

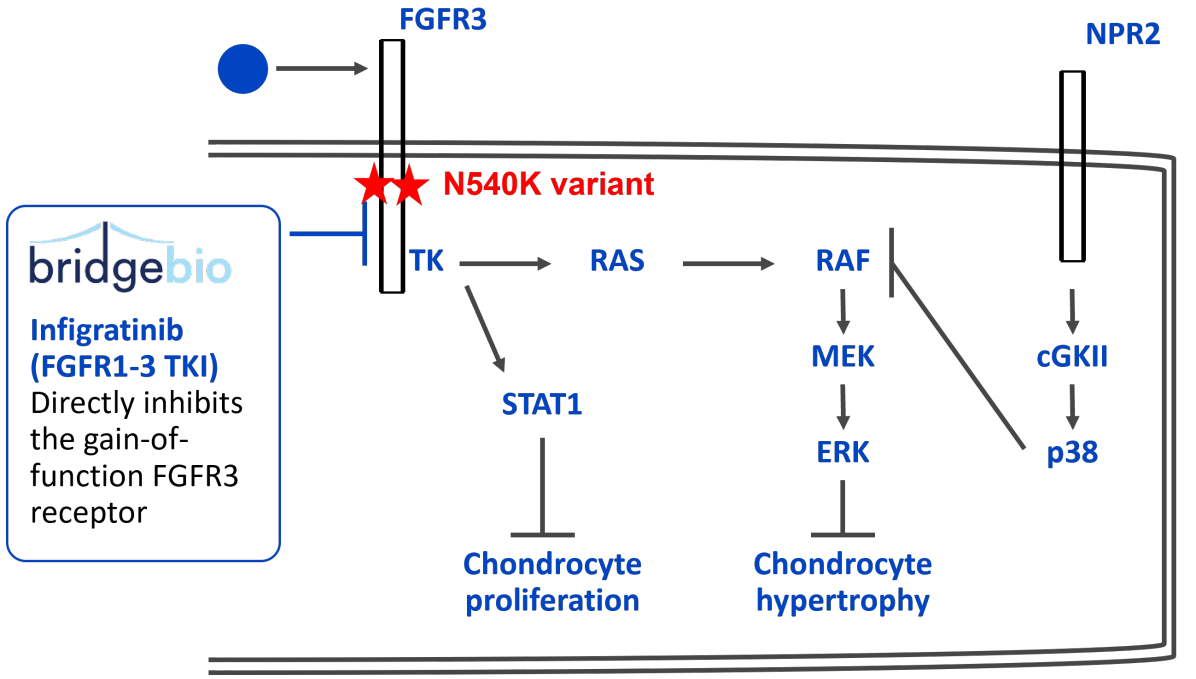
Detailed PROPEL 3 results planned for presentation at scientific and advocacy conferences
2026

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

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

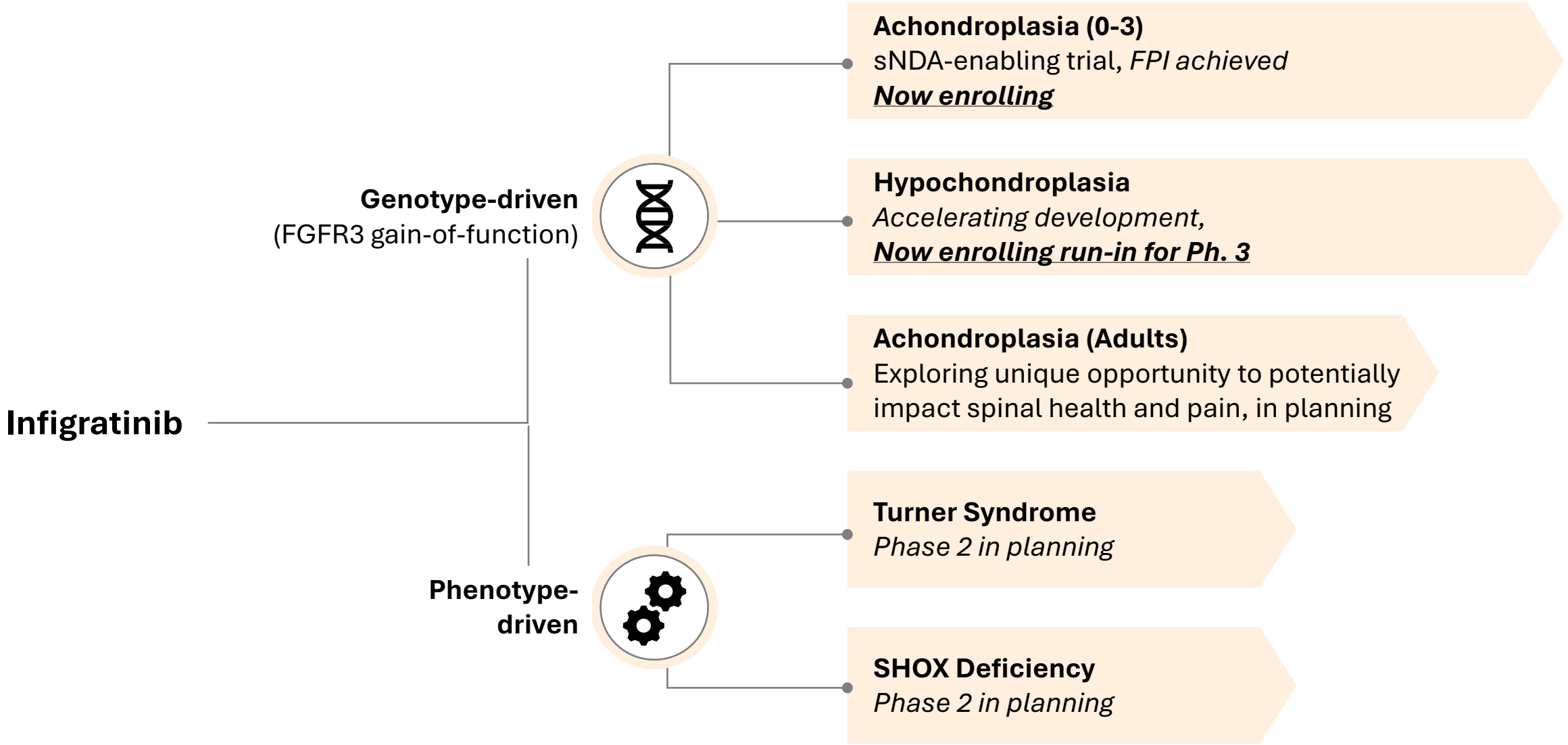
- **Directly targets the underlying cause of hypochondroplasia, FGFR3 overactivity[1,2]**
- **Single-digit nM potency against multiple FGFR3 variants associated with hypochondroplasia, including in vivo improvement of bone growth in an HCH mouse model[1,2]**
- **Ability for infigratinib to cross the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

Recent progress and upcoming milestones

- **The observational run-in study for the Phase 3 is now enrolling**
- **Based on totality of data in ACH, we are now accelerating our HCH program**
- **Phase 2 results 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

We are on Day 1 of the opportunity for infigratinib



BBP-812

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

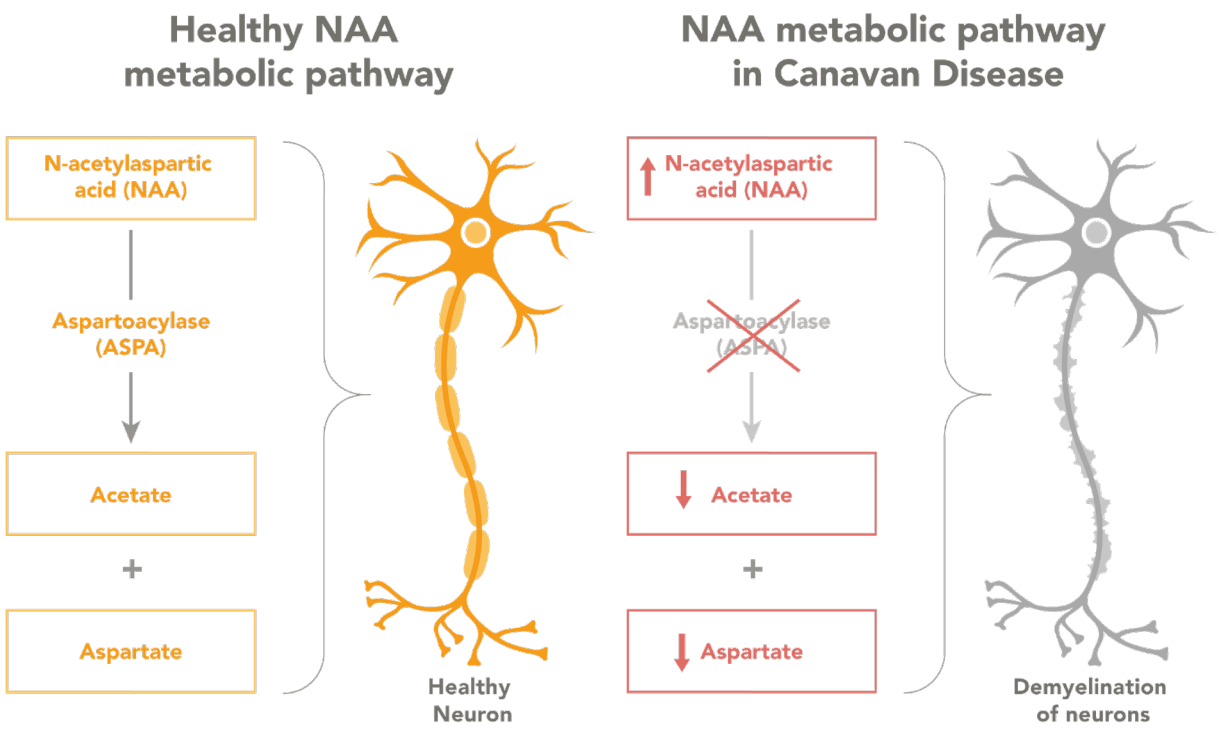
Unmet need

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

[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

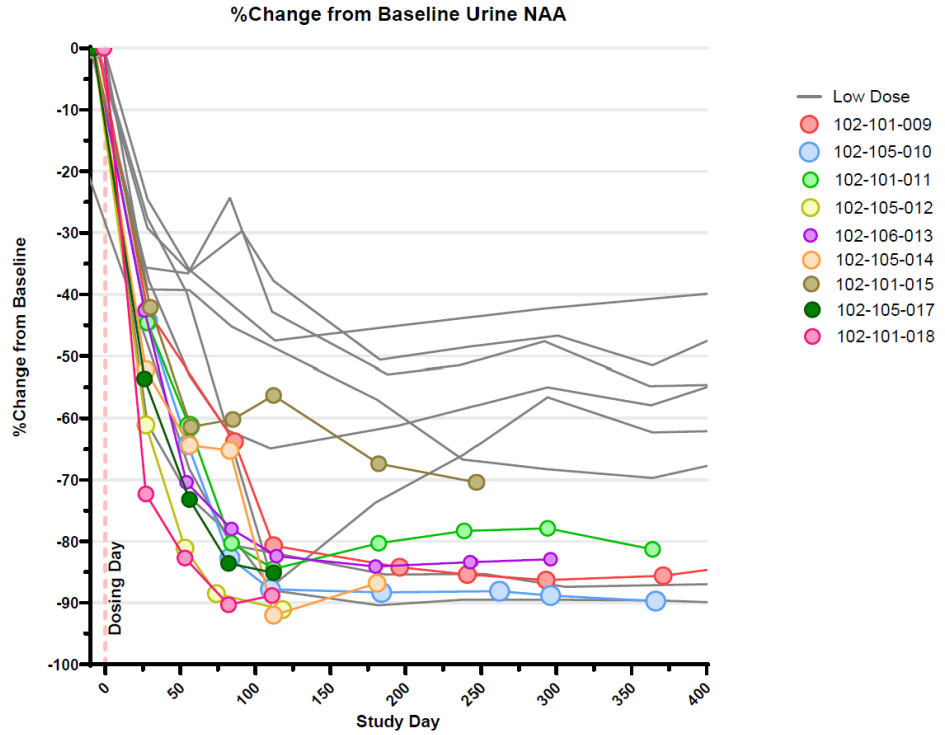
Provide first disease-modifying therapy
Target the condition directly at the source, utilize single registrational study & biomarker for accelerated approval

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

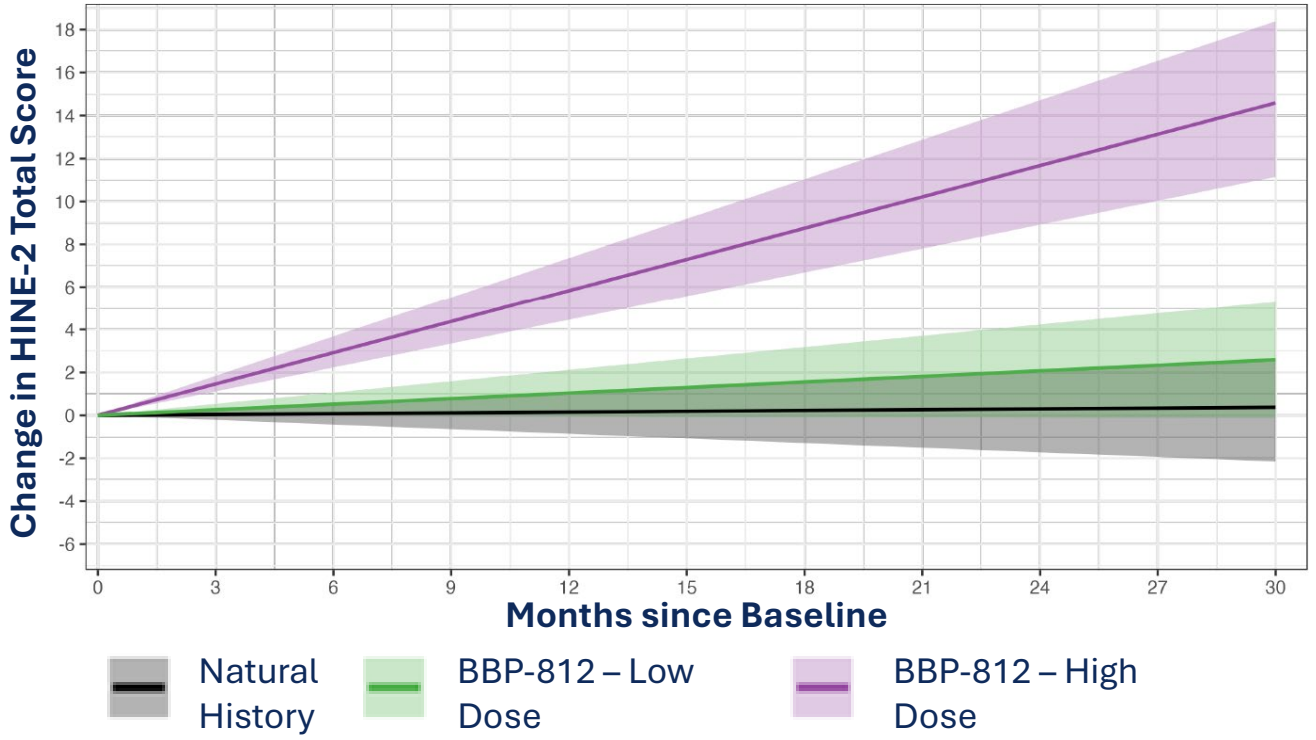
Current path to a potential BLA filing in 2027 based on reductions in urine NAA (surrogate endpoint) supported by motor function improvements

Urine N-acetylaspartic acid (NAA) levels



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

Hammersmith Infant Neurological Examination (HINE-2) Trajectory



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

Source: 10 December 2025 data cut, N = 18

BBP-812 is changing the lives of children with Canavan and the lives of their families

Study Investigator Quotes

Patient is showing "*lots of improvement at 3-month visit, **emotional presence, visual tracking, reaching for things***"

Dr. Alexander Fay, UCSF Benioff Children's Hospital

"*One patient in particular is **sitting independently** and **taking steps and walking**, and that is certainly something I've never seen with Canavan disease.*"

Dr. Florian Eichler, Mass General Hospital for Children

"*It is **unheard of for a child with typical Canavan disease to be able to walk independently**. None of the 23 individuals with Canavan disease described in the natural history by Bley et al (2021) were able to walk independently. **In our experience, this is a remarkable finding**.*"

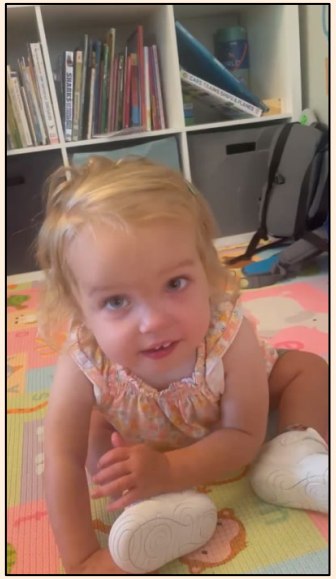
Dr. Amanda Nagy, Mass General Hospital for Children

Post-Treatment Patient Progression

+ gene therapy (22 months old)

12 months old (pre-treatment)

24 months old



30 months old

48 months old

BridgeBio Ecosystem Highlights

BBOTi
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

BBO-8520

First direct inhibitor
of $KRAS^{G12C}$ ON/OFF with effector blockade function

- ✓ Promising monotherapy data with **65% ORR, 66% 6-mo. PFS, and 83% of patients remaining on treatment** for ≥6mo follow-up; **differentiated tolerability**
- ✓ **Differentiated pembro combination safety** observed at **optimally active dose level** with **favorable liver safety profile compared to pembro alone**
- ✓ Promising early efficacy signals in combination with pembro
- ✓ Encouraging early efficacy signals in patients **with STK11/KEAP1 co-mutations**

BBO-11818

Potent, direct inhibitor
of panKRAS ON/OFF

- ✓ **Partial response (PR) observed in PDAC**
- ✓ Anti-tumor activity across dose levels and tumor types with tumor reductions at higher dose levels
- ✓ **Differentiated safety profile** observed in dose escalation
- ✓ **PK exposure approximately dose proportional**

BBO-10203

Novel RAS:PI3Kα Breaker
specifically blocks RAS activation of PI3Ka

- ✓ Potentially **differentiated safety** profile
- ✓ **No observed events of hyperglycemia with no enrollment restrictions on HbA1c and glucose levels**
- ✓ Achieved **target systemic exposure and rapid full target engagement**
- ✓ Recommended dose for expansion has been determined **and combo cohorts are open**

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

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

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

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.