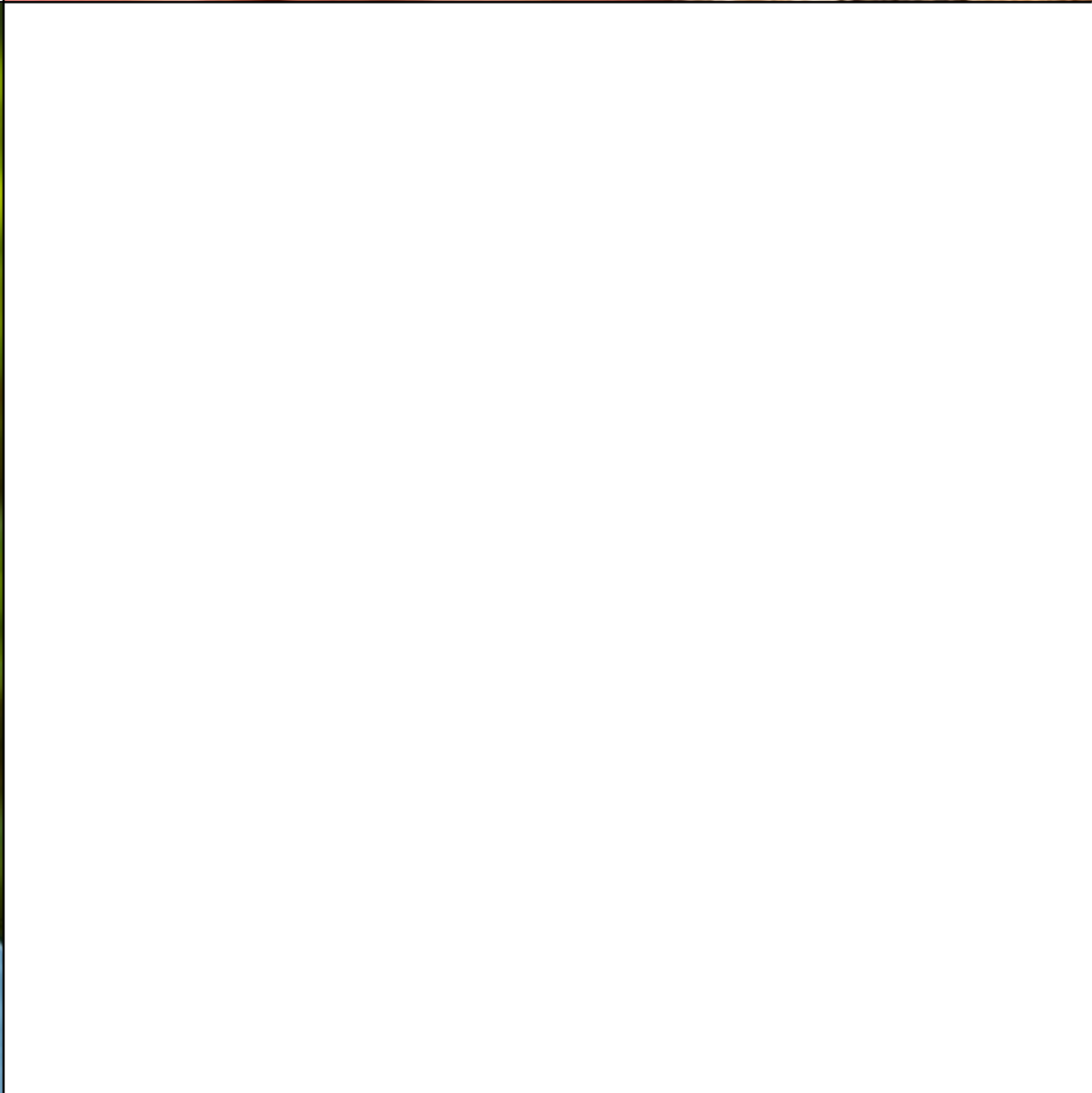




Corporate
Presentation
October 2026



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 advantages of our business model and overall strategy; our speed of creating new and meaningful drugs and related impact on patients, the sustainability and velocity of our engine to deliver medicines, and our value creation potential for patients; our financial position, including our expectations regarding reaching regulatory, financial, and commercial milestones and the sufficiency of our cash position to support three potential product launches; the commercial success of Attruby/Beyonttra (acoramidis) and the safety, efficacy, advantages and market potential of Attruby/Beyonttra; the clinical, therapeutic and market potential of our clinical development programs and our pipeline, including BBP-418, encaleret, infigratinib, and BBP-812; our expected timing for submitting New Drug Applications with the U.S. Food and Drug Administration (“FDA”) and similar submissions with foreign regulatory authorities, receiving U.S. and foreign approvals, and commencing commercial launch for BBP-418, encaleret and infigratinib; our anticipated interactions with and feedback from the FDA and foreign regulatory authorities, including eligibility of Priority Review Vouchers; our expected commercial success of BBP-418, encaleret and infigratinib; our expected timing for initiating trials and releasing data from our trials; the potential of our BBP-717 depleter program as a treatment for ATTR; the potency and safety of our product candidates; and the potential benefits of our products and product candidates, 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 and commercializing 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.

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BridgeBio is a new type of biopharmaceutical company

Clearer Biology

FROM

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



TO

Assets selected to target genetic diseases at the source: higher probability of success from day one

Faster Learning

FROM

Slow and bureaucratic decision making at the portfolio level



TO

Rapid, decentralized R&D decisions: each affiliate operates with scientific focus and autonomy

Smarter Capital

FROM

High fixed costs, limited capital sources, and incentives misaligned at the portfolio level



TO

Variable costs via shared infrastructure, diversified financing, and asset-level incentives that preserve biological focus

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

> 10,000
individuals impacted by
our therapies

Obtained Approval for 3 Medicines

**Attruby**TM
(acoramidis)_{356 mg tablet}

**Nulibry**[®]
(fosdenopterin)
for injection

**TRUSELTIQ**TM
(infigratinib) capsules

3 Positive Phase 3 Results

**fortify**


**CALIBRATE**


**PROPEL3**


**CANaspire**

**ACCEL 2/3**

**RECLAIM-HP**

15
active trials
in ecosystem

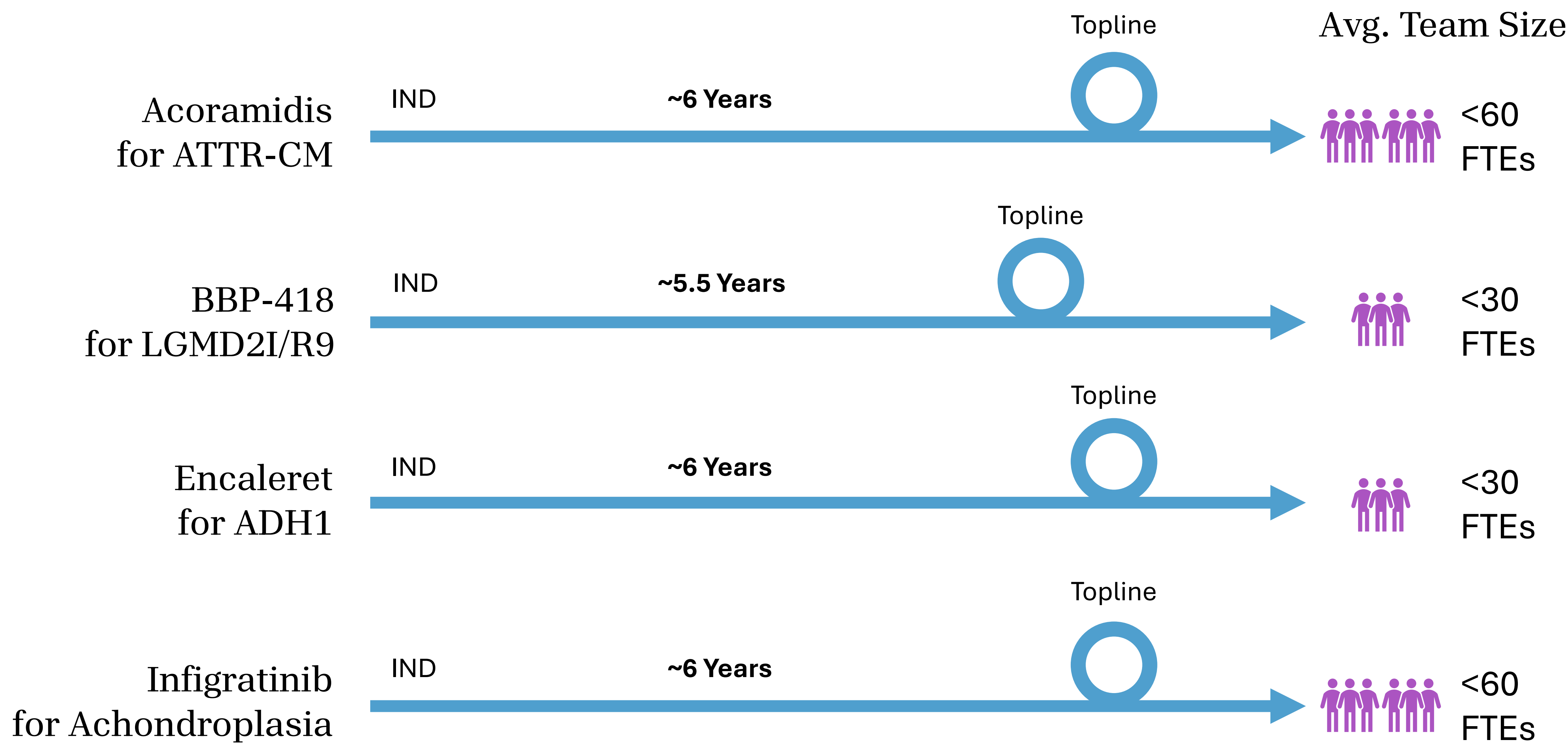
> 70
papers
published

> 35
academic
partnerships

19
INDs
created

< \$40M
spend to proof-
of-concept data

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



A pipeline of products that span 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+
Attruby (acoramidis)	Hereditary Transthyretin Amyloidosis (hATTR-PN and hATTR-CM) Prevention						~50,000	\$1B+
Nulibry (fosdenopterin)	Molybdenum Cofactor Deficiency (MoCD) Type A					✓	<100	Partnered
BBP-418	Limb-Girdle Muscular Dystrophy Type 2I/R9 (LGMD2I/R9)					PDUFA Nov 27 2026	7,000+	\$1B+
Infigratinib	Achondroplasia (ACH)					PDUFA Feb 4 2027	55,000+*	\$3B+
	Hypochondroplasia (HCH)				Observational Run-in Fully Enrolled for Ph. 2		55,000+*	\$2B+
Encaleret	Autosomal Dominant Hypocalcemia Type 1 (ADH1)					PDUFA May 8 2027	25,000+	\$1B+
	Chronic Hypoparathyroidism (CHP)				First patient dosed in Phase 3 RECLAIM-HP trial		200,000+	\$1B+
BBP-812	Canavan Disease				Phase 1/2 Pivotal		1,000	TBD
BBP-717	Transthyretin Amyloidosis (ATTR-CM)						500,000+	\$20B+
GondolaBio	17 Programs (various indications)						Various	Various
EPP	Erythropoietic Protoporphyria						25,000	TBD
ADPKD	Autosomal Dominant Polycystic Kidney Disease						300,000	TBD
AATD	Alpha-1 Antitrypsin Deficiency						200,000	TBD
BridgeBio Oncology Therapeutics	3 Oncology Programs (various indications)						Various	Various

BridgeBio's late stage portfolio at a glance

	Key Data	Peak Sales	Peak Share
ATTR-CM	<ul style="list-style-type: none">▪ Attruby is the first and only near-complete TTR stabilizer, with treatment effect seen as early as Month 1 in ATTRibute-CM▪ Improved real-world clinical outcomes demonstrated by Attruby versus tafamidis in a contemporary patient population of Epic COSMOS EHR data▪ Association with kidney-protective effects in post-hoc analysis of ATTRibute-CM data, with a profile consistent with therapies that act directly on the kidney, including ACE inhibitors, ARBs, and SGLT2 inhibitors	>\$4.0B	30% - 40%
LGMD2I/R9	<ul style="list-style-type: none">▪ All primary and key secondary endpoints showed stat sig improvement: Glycosylated αDG at 3 months (p<0.0001), Glycosylated αDG at 12 months (p<0.0001), Serum CK (p<0.0001), 100MTT (p<0.0001), FVC (p=0.0071)▪ NSAD: clinically and statistically significant improvement vs. PBO*▪ Well-tolerated and oral	>\$1.0B PRV eligible	First and only
ACH	<ul style="list-style-type: none">▪ Treatment effect: 2.10 cm / year (p<0.0001); 5.96 cm/yr absolute AHV on treatment▪ Proportionality: -0.05 vs Pbo in ages < 8 (p<0.05; first stat sig effect in PBO-controlled trial)▪ Clean safety (Only 3 cases (4%) of mild, asymptomatic and transient elevations of phos, no issues with hypotension or hypertrichosis); positioned to be the first oral option	>\$2.0B	~65%
ADH1	<ul style="list-style-type: none">▪ Serum / urine calcium normalization: 76% at Week 24 (p<0.0001)▪ Intact PTH above lower limit of reference range: 91% at Week 24 (p<0.0001)▪ Well-tolerated and oral	>\$1.0B	First and only

\$1.5B in cash to support three potential US launches by mid-2027

BridgeBio currently has **\$1.5B¹** in cash, cash equivalents and marketable securities

1H 2026

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

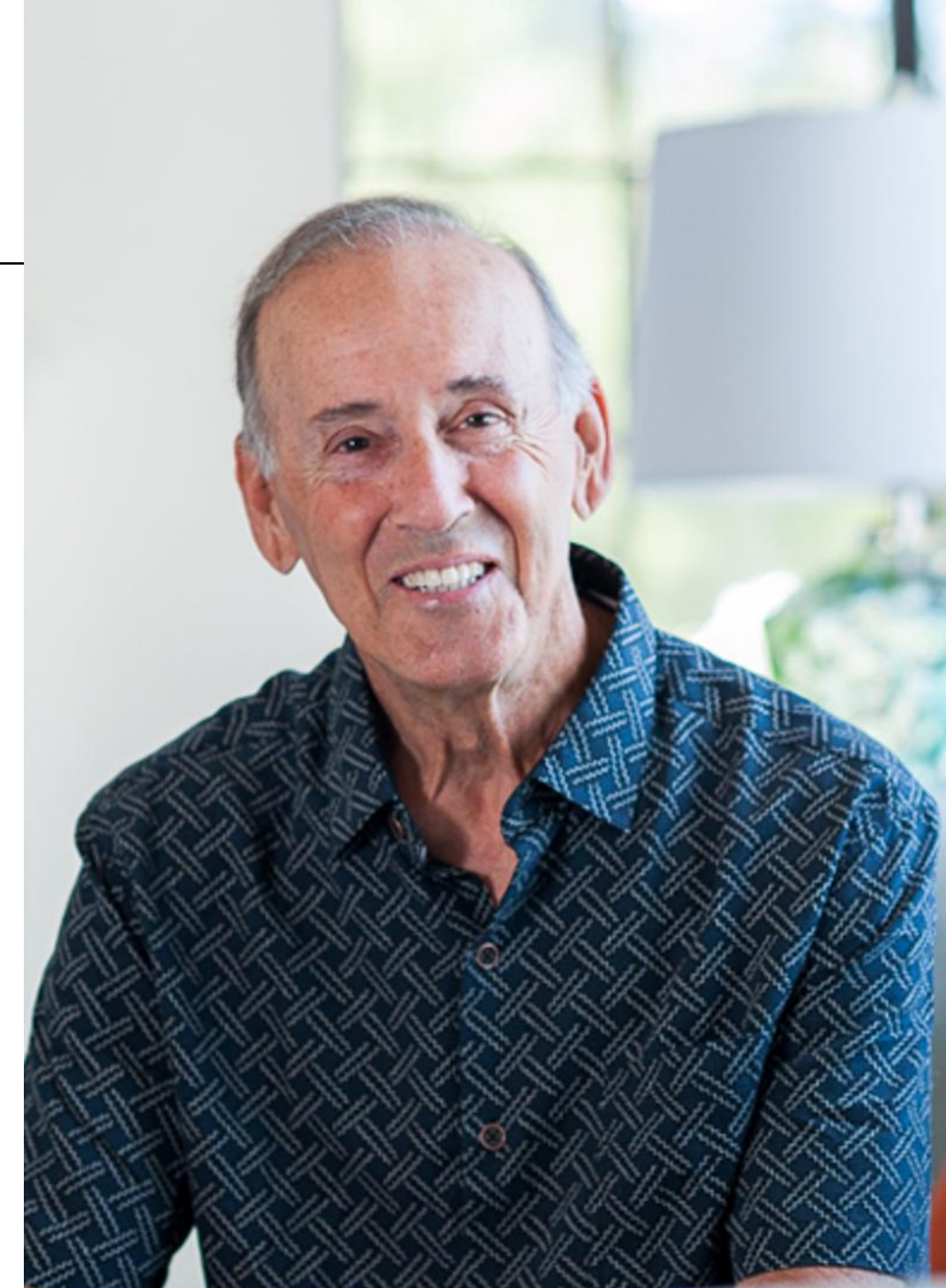
2H 2026

- ✔ **Infigratinib:** NDA submission
- ✔ **Encaleret:** Initiate P3 CHP trial
- ▶ **BBP-418:** PDUFA November 27, 2026 & US launch upon approval

1H 2027

- ▶ **Infigratinib:** PDUFA Feb 4, 2027 & US launch upon approval
- ▶ **Encaleret:** PDUFA May 8, 2027 & US launch upon approval

Attruby® Approved for ATTR-CM



Key Attruby[®] (acoramidis) highlights



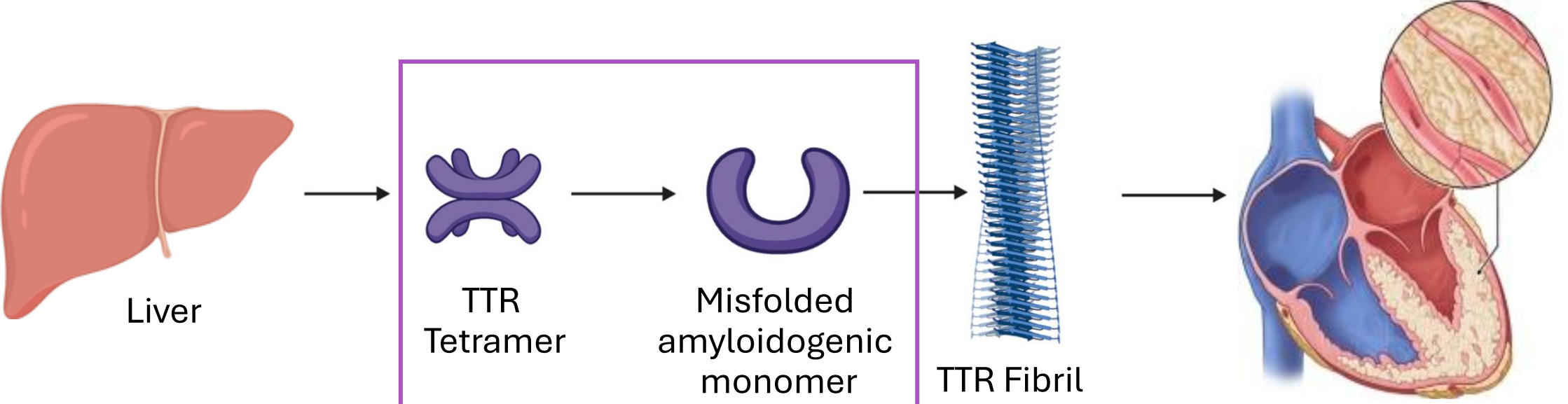
<p>First and only approved product with a label specifying near-complete stabilization of TTR</p>	<p>Effect seen as early as 1 month – reduction in cumulative cardiovascular outcomes within the first month of treatment in patients with ATTR-CM</p>	<p>42% reduction in composite of all-cause mortality and recurrent cardiovascular-related hospitalization events at Month 30</p>	<p>50% reduction in the cumulative frequency of cardiovascular-related hospitalization events at Month 30</p>	<p>43% reduction in frequency of CVH due to atrial fibrillation</p>
<p>\$253.2M Q3 2026 U.S. Net Product Revenue</p>	<p>>25% Estimated Share of NBRx for Attruby</p>	<p>30-40% Potential Peak Market Share for Attruby</p>	<p>\$4B+ Potential Peak Market Sales for Acoramidis</p>	

Attruby is well-positioned for the second stage of launch: accelerating towards orbital velocity

1 Attruby is well positioned as the only near-complete stabilizer	2 Stabilizer monotherapy is firmly anchored as 1L therapy of choice	3 The market is growing and durable despite potential disruptions
<ul style="list-style-type: none">Attruby achieves near-complete stabilization by seeing and binding more target and gluing it together more stronglyThe speed and strength of this near-complete stabilization is shown in clinical and RWE dataData from ATTRibute-CM suggests Attruby has similar qualities as kidney-protective drugs like SGLT2s, ACEis, and ARBs	<ul style="list-style-type: none">Knockdown agents take 1.5+ years to show visual separation from placebo in monotherapy on core clinical endpointsKnockdowns are not differentiated from each other in monotherapyCombination therapy has not shown incremental benefit over partial stabilizer monotherapy	<ul style="list-style-type: none">The ATTR-CM market is large and steadily growing towards \$20B peak salesAttruby has the potential to hold or grow share after the entry of tafamidis generic given Attruby's differentiation and market dynamics

1 Attruby's advantages stem from the fact that it is the only near-complete stabilizer, as more TTR is associated with a longer life

ATTR-CM is a progressive disease of TTR protein instability



Attruby: near-complete stabilizer that prevents dissociation of TTR tetramers into monomers

- ▶ **TTR protein instability:** normally stable TTR tetramers dissociate into unstable monomers
- ▶ **Cardiac infiltration:** Unstable TTR monomers misfold and deposit as insoluble amyloid fibrils in the heart and nerves
- ▶ **Cardiovascular risk:** Increased risk of cardiovascular-related mortality (CVM) and cardiovascular-related hospitalizations (CVH)

Attruby uniquely achieves near-complete stabilization, preserving the important TTR protein



Attruby is the **only** TTR stabilizer with near-complete (≥90%) stabilization reflected in its **approved label**

How Attruby achieves near-complete stabilization

- ✓ **Sees more target** (superior free fraction)
- ✓ **Binds more target** (superior K_{D2})
- ✓ **Glues the target together stronger** (enthalpic binding mode)

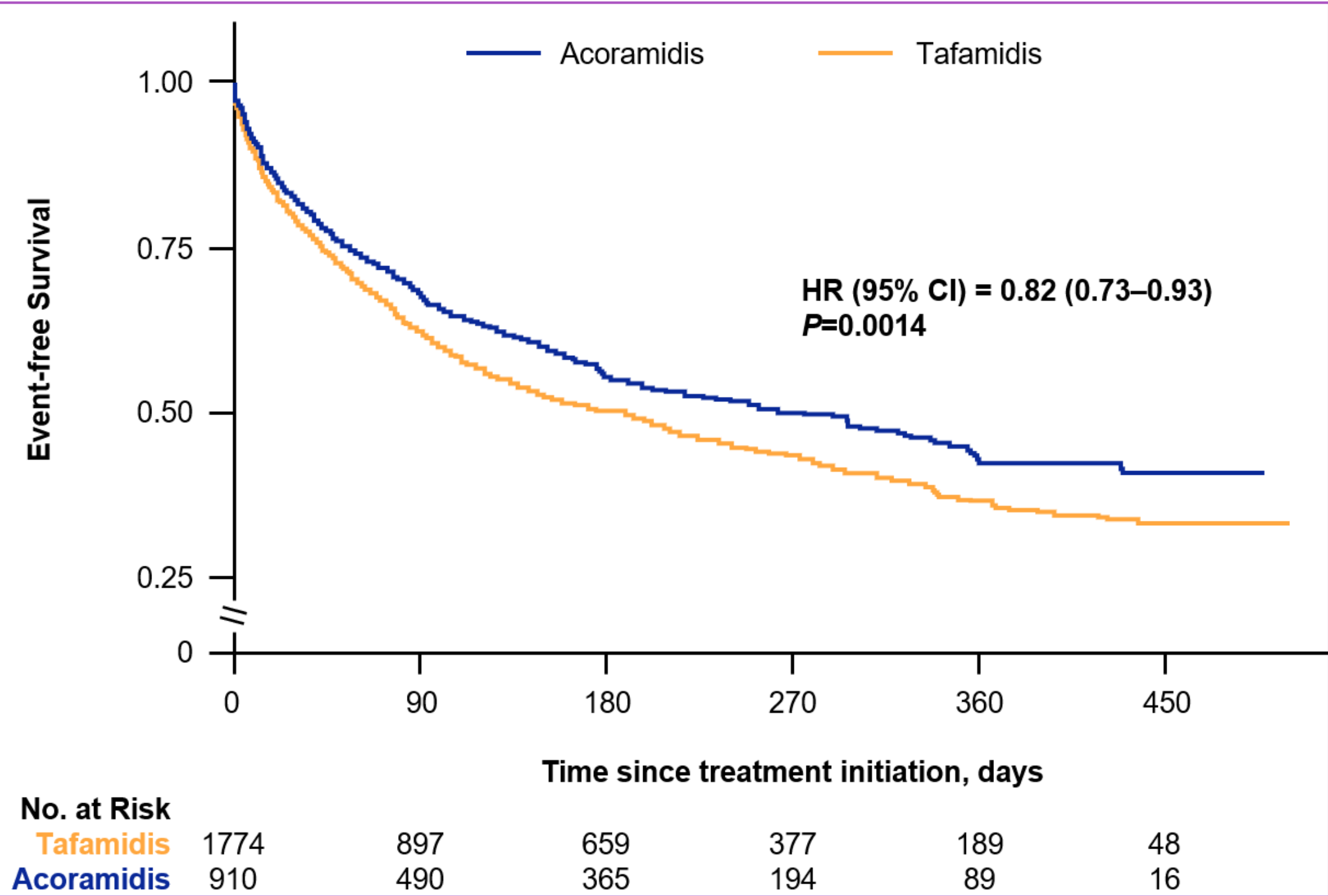
Why near-complete stabilization matters

- ✓ **More TTR, longer life:** Higher serum TTR independently predicts improved survival^{1 2}
- ✓ **Every increment counts:** Each 5 mg/dL rise in serum TTR associated with ~32% lower odds of death by 30 months²

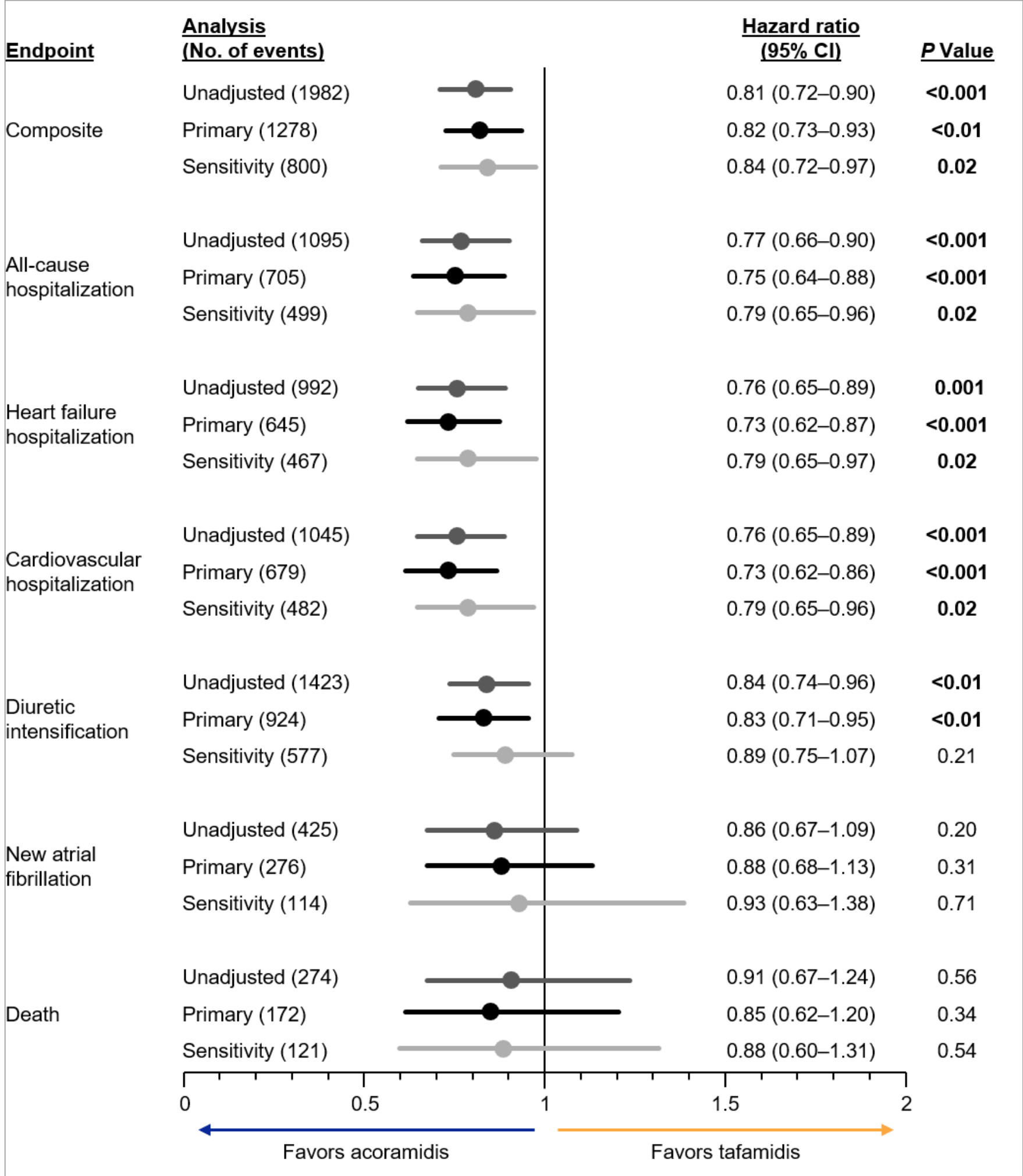
1 Attruby demonstrates improved real-world clinical outcomes vs. tafamidis in EHR Epic COSMOS data in a contemporary patient population

Attruby significantly improved clinical outcomes compared to tafamidis (n=2684 patient study)

Attruby favored across all analyses and endpoints, with strongest effect seen in hospitalizations



Attruby was associated with a statistically significant 18% lower risk of composite events*, with curve separation occurring within one month

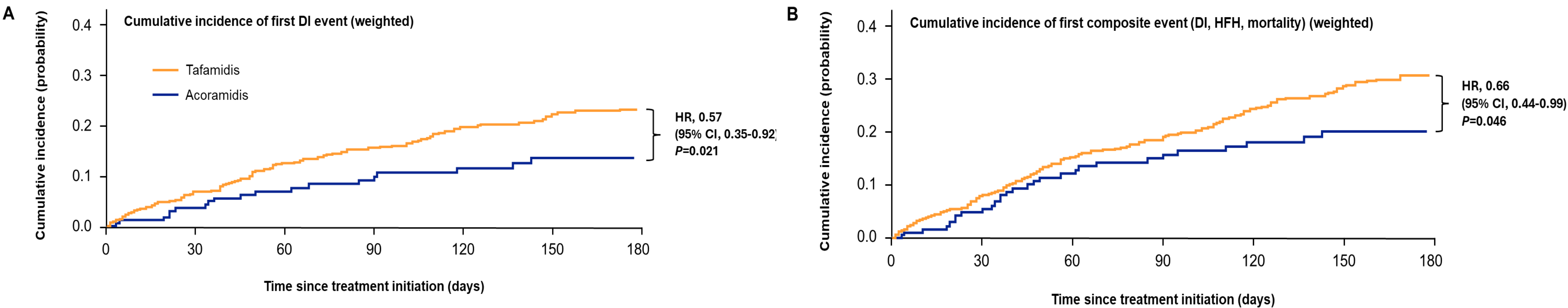


1 Attruby's comparative effectiveness has been replicated in claims data

Real world evidence suggests that Attruby achieves a 43% reduction in diuretic intensification relative to tafamidis

Acoramidis vs. Tafamidis · Propensity Score Weighted Analysis · N=170 vs. ESS=448 · Mean f/u: 140 days · SGLT2i use: ~44%

43%	Reduction in Diuretic Intensification	34%	Reduction in Composite Endpoint	Outcome	Acoramidis (N=170)	Tafamidis (ESS=448)	HR (95% CI)	p-value
				A) Diuretic Intensification (DI)	20 (11.8%)	92 (20.5%)	0.57 (0.35–0.92)	0.021
				B) DI + HF hospitalization + mortality	30 (17.6%)	118 (26.3%)	0.66 (0.44–0.99)	0.046



Published in Cardiology & Therapy on September 23, 2026

Article published in Circulation: Heart Failure suggests the first-ever direct kidney-protective effects in ATTR-CM

Circulation: Heart Failure

New online

<https://doi.org/10.1161/CIRCHEARTFAILURE.126.014656>



ORIGINAL ARTICLE

Effects of Acoramidis on Kidney Function in Transthyretin Amyloid Cardiomyopathy

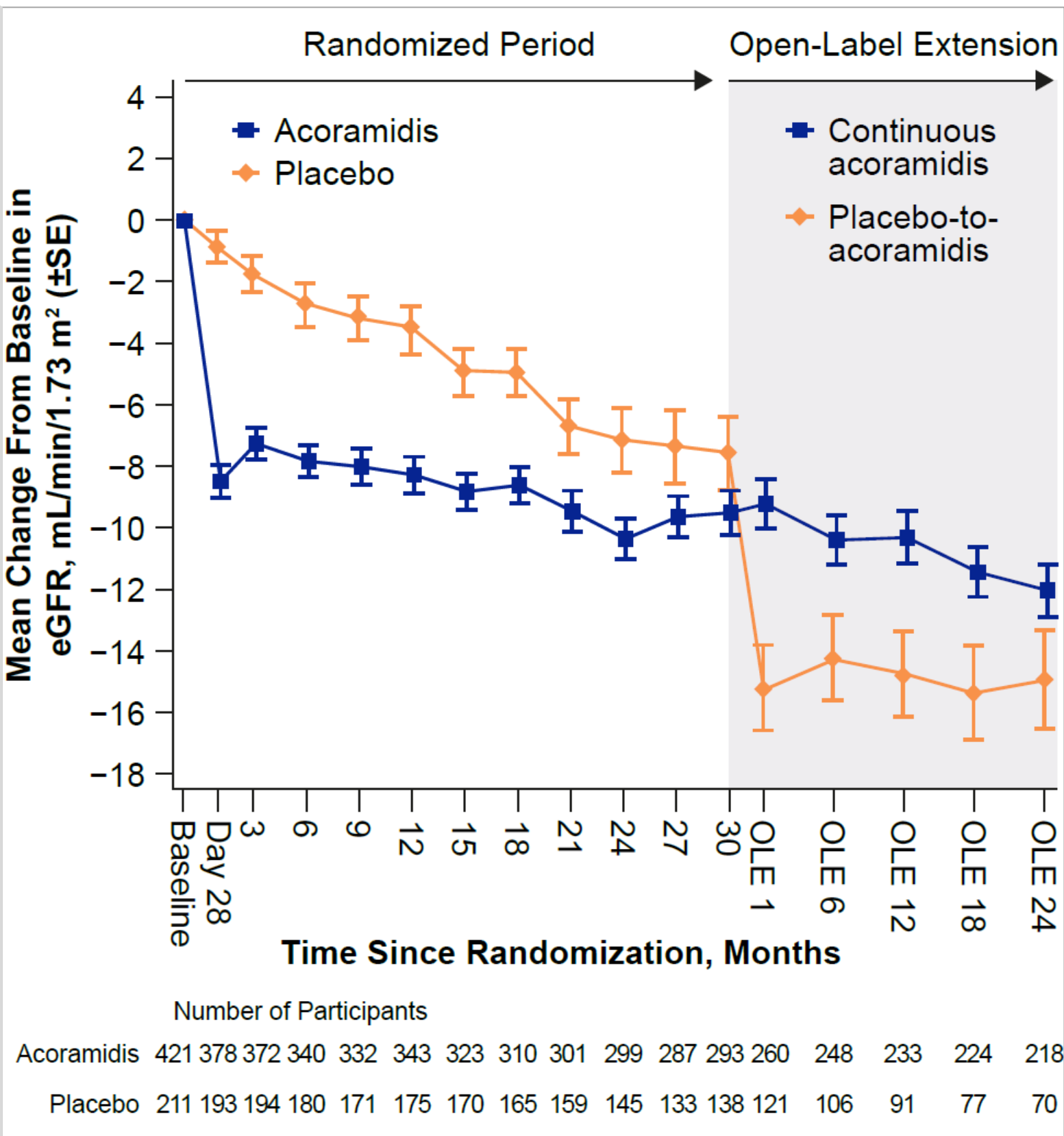
Jeffrey M. Testani, MD, MTR , Daniel P. Judge, MD , Barry A. Borlaug, MD , David Cherney, MD, PhD , Zachary L. Cox, PharmD , Julian D. Gillmore, MD, PhD , Julia B. Lewis, MD , Scott H. Adler, MD , Xiaofan Cao, PhD , Adam Castaño, MD, MS , Jonathan C. Fox, MD, PhD , Leonid Katz, MD, Vandana Mathur, MD, Kuangnan Xiong, PhD , Javed Butler, MD, MPH, MBA , and Ahmad Masri, MD, MS 

Background: Acoramidis achieves near-complete (≥90%) transthyretin stabilization and is approved to reduce cardiovascular-related hospitalization in transthyretin amyloid cardiomyopathy. Its effects on kidney function are not well characterized.

Methods: Data from randomized phase 2 (N=49) and phase 3 (N=632) studies in transthyretin amyloid cardiomyopathy were included. The estimated glomerular filtration rate (eGFR) slope was generated using a linear spline mixed-effects model. The urinary albumin-to-creatinine ratio was measured longitudinally. Relationships between changes in kidney function and clinical outcomes were explored using Cox proportional hazards models.

Results: Acoramidis initiation resulted in a modest acute dip in eGFR that was dose-dependent, reversible, and not associated with adverse kidney-related events. At Day 28, the mean (±SE) dip in eGFR from baseline with acoramidis was 8.5±0.48 mL/min per 1.73 m²; the placebo-corrected reduction in the urinary albumin-to-creatinine ratio was 15.5% (95% CI, 0.4%–28.4%; *P*=0.044). The rate of decline in kidney function (chronic eGFR slope) was significantly improved with acoramidis versus placebo (−1.01 versus −3.48 mL/min per 1.73 m² per year; *P*<0.001), and the reduction in the urinary albumin-to-creatinine ratio was sustained (13.7% [95% CI, 1.7%–24.2%]; *P*=0.026) over time. Concomitant tafamidis use did not influence the chronic eGFR slope in either arm. Comparing acoramidis versus placebo subgroups with acute eGFR dips ≥ the median (4.89 mL/min per 1.73 m²) favored acoramidis for all-cause mortality or cardiovascular-related hospitalization (hazard ratio, 0.42 [95% CI, 0.22–0.78]; *P*=0.006; *P* interaction=0.043) and cardiovascular-related hospitalization (hazard ratio, 0.34 [95% CI, 0.17–0.66]; *P*=0.002, *P* interaction=0.025). Within the placebo arm, eGFR dips portended worse outcomes.

Conclusions: Acoramidis initiation resulted in an acute dip in eGFR and reductions in both the chronic eGFR slope and urinary albumin-to-creatinine ratio versus placebo without adverse kidney-related events. Acoramidis effects on kidney function may be mediated through direct kidney-protective hemodynamic effects. Importantly, the acute dip in eGFR was associated with a reduced risk of adverse clinical outcomes within the first year.



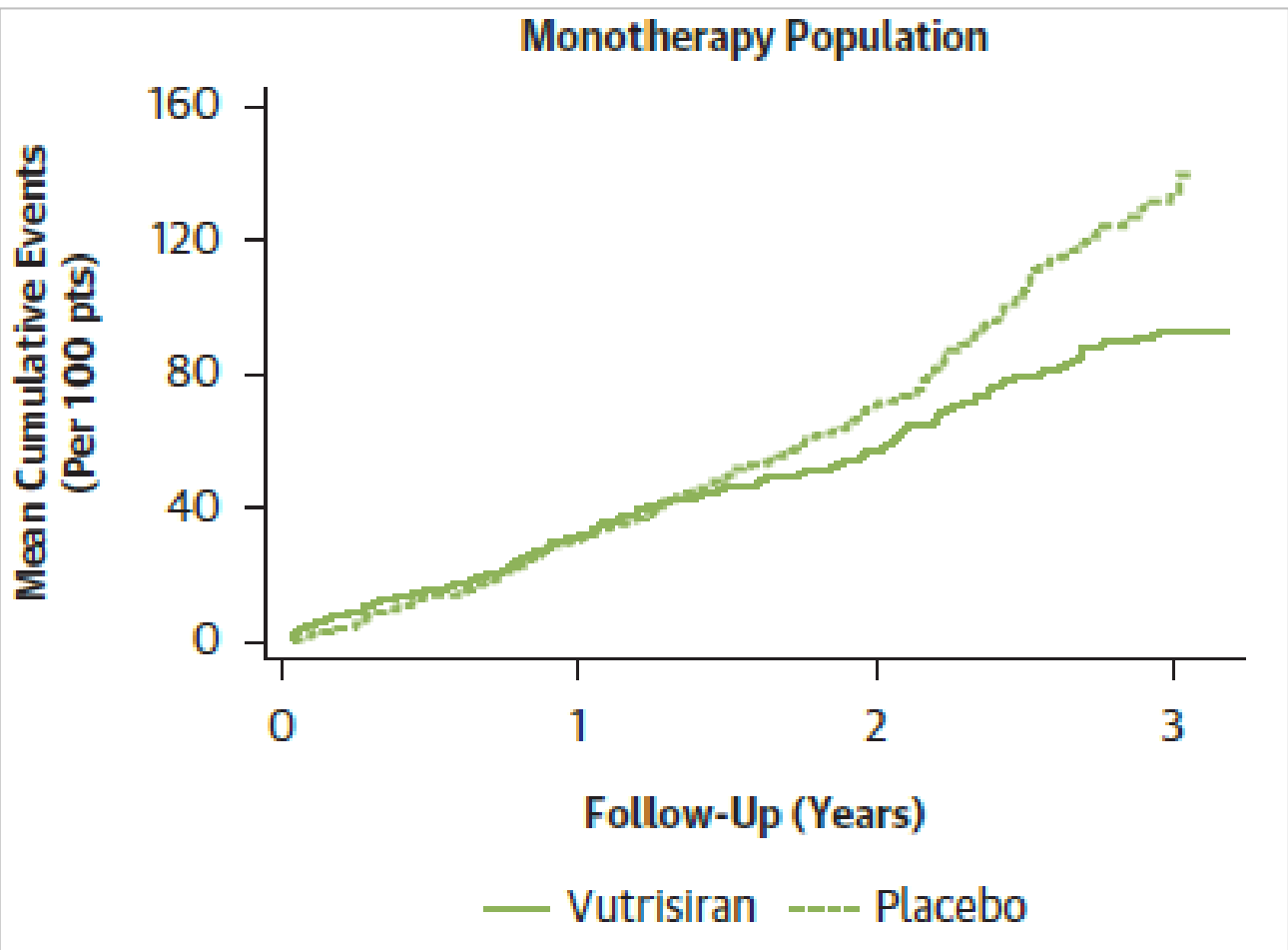
- Key insights associated with Attruby:**
- Early Impact:** hemodynamically mediated, reversible eGFR dip and a 15.5% placebo-corrected reduction in urinary protein to creatinine ratio (UACR; *p*<0.05)
 - Sustained Results:** Over 30 months, significantly improved rate of kidney function decline versus placebo (+2.47 mL/min/1.73m²/year; *p*<0.001) and a sustained reduction in UACR (13.7%; *p*=0.026)
 - Improved Outcomes:** Participants with the largest eGFR dips experienced a 58% lower risk of death or cardiovascular hospitalization and 66% lower risk of hospitalization alone in the first year of the study
 - Continued Differentiation:** This constellation of effects has not been reported with any other approved ATTR-CM therapy

2 Stabilizer monotherapy is firmly anchored as 1L therapy of choice based on the recent data released on knockdown efficacy in mono and combo

Data published at ESC 2026 shows that vutrisiran takes over 1.5 years to visually separate in monotherapy...

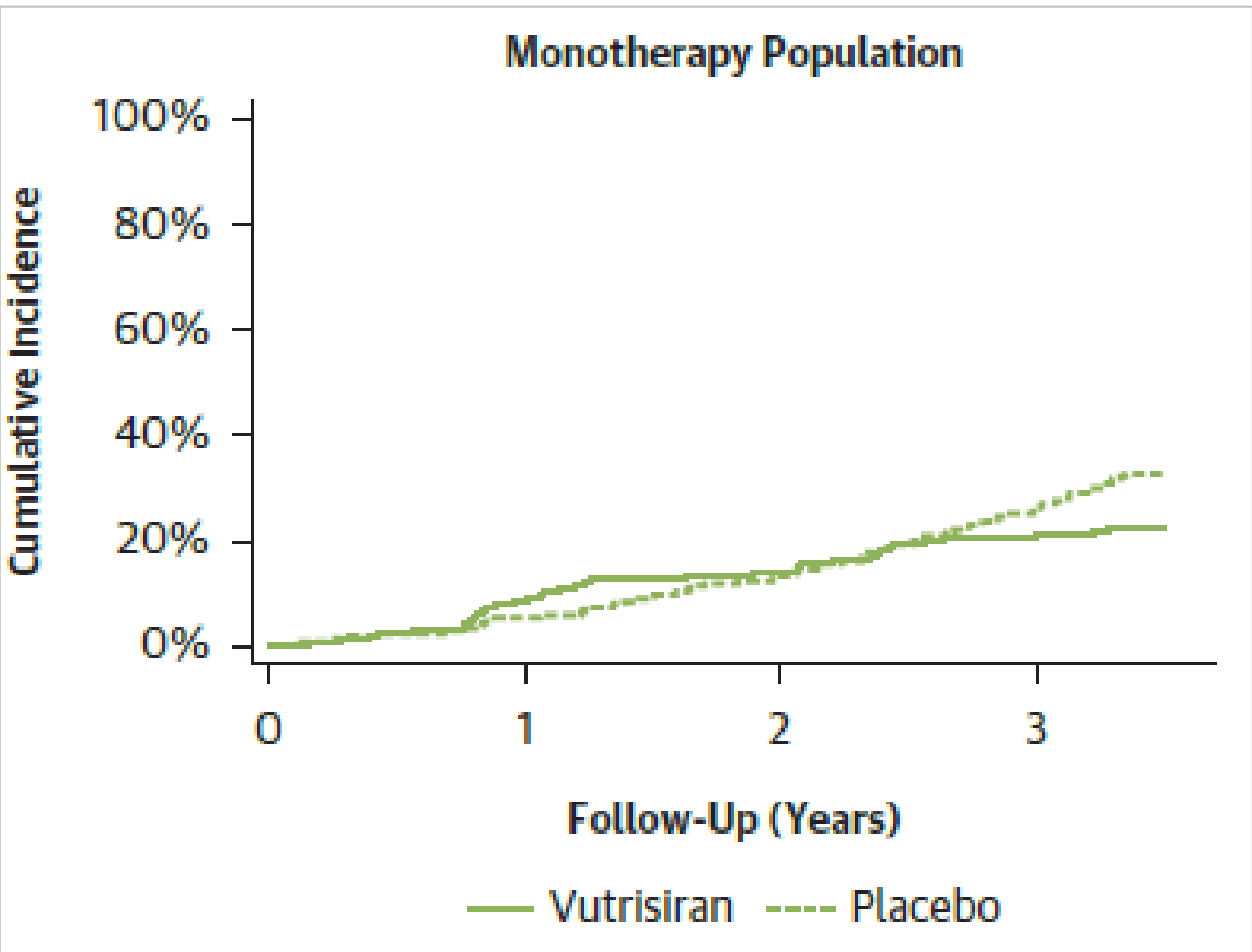
...eplontersen and vutrisiran both failed to show benefit in combo and have similar clinical efficacy in mono

All-cause death and recurrent CV events



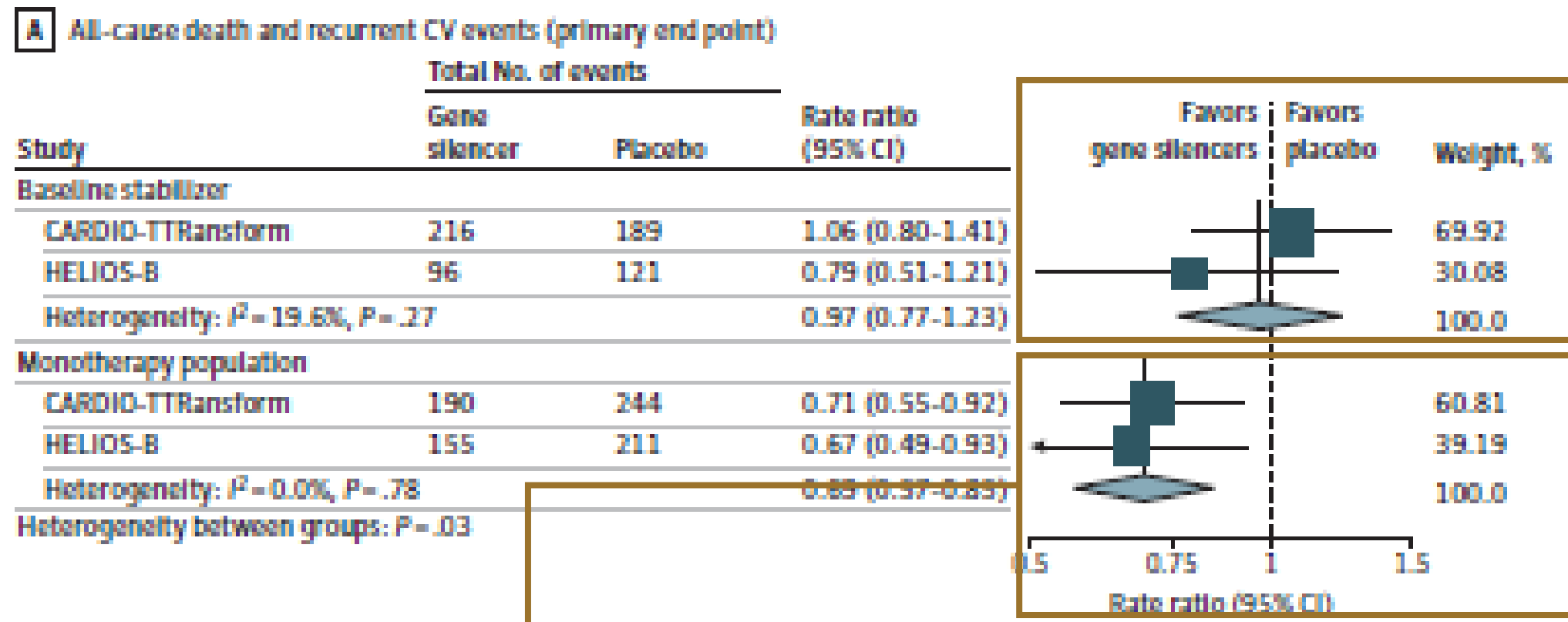
Source: Hamatani, et. al, JACC, 2026

All-cause death up to 42 months



Source: Hamatani, et. al, JACC, 2026

Figure 3. Forest Plot of Treatment Effects on Clinical Outcomes in the Baseline Stabilizer and Monotherapy Population



Source: Gillmore et al, JAMA, August 2026

Monotherapy arms are similar in efficacy on primary endpoint of meta analysis

Both knockdowns failed to show significant benefit in combination with a partial stabilizer

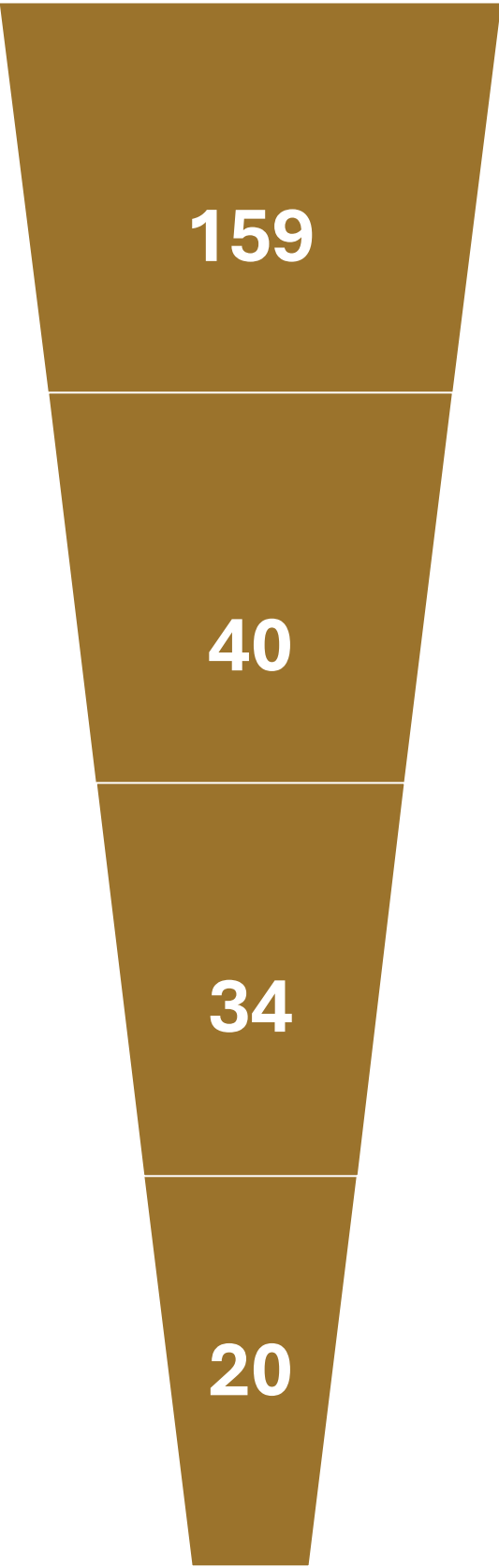


“In patients who were on stabilizer therapy at baseline, no treatment effect [from eplontersen] was observed.”

Ionis press release for CARDIO-TTTransform results, July 9, 2026

3 Attruby has the potential to hold or grow share after the entry of tafamidis generic given Attruby’s differentiation and market dynamics

Analog funnel, # groups



Universe: Branded drug with expiry post 2000

Scale: Anchor drug has >\$750m in revenue

Competitive dynamics: Non-anchor drug has >\$300M in revenue

Structural similarity: Products that are only pharmacy benefit

Outcomes for top 20 analogs

Analog Drug	LoE Drug (Year)	Revenue outcome (CAGR, %) ¹
Sprycel	Gleevec (2016)	7%
Opsumit	Letairis (2019)	14%
Xarelto	Pradaxa (2022)	-2% ³
Xtandi	Zytiga (2018)	18%
Prezista	Reyataz (2017)	13%
Crestor	Lipitor (2011)	-2%
Benicar	Cozaar (2010)	-3%
Altace	Zestril (2002)	7%
Jardiance	Farxiga (2024)	15% ⁴
Yupelri	Spiriva (2023)	8% ³
Protonix	Prilosec (2002)	16%
Bystolic	Coreg (2007)	188%
Skyrizi	Stelara (2025)	51% ²
Abilify	Risperdal (2008)	3%
Trulicity	Victoza (2024)	-21% ²
Paxil	Prozac (2001)	18% ³
Lunesta	Ambien (2007)	2%
NovoRapid	Humalog (2019)	-12%
Topamax	Neurontin (2004)	22%
Enbrel	Humira (2023)	-22% ⁵

Key insights associated with Attruby:

- After a comprehensive review of all ~160 examples of branded drugs expiring (post 2000); 20 analogs surface that can provide useful guidance
- The results show that the second player (“Analog drug”) can maintain or grow revenue in the 3 years following LOE of a competitor
- While each example has nuance, the takeaway should remain that the majority of the 20 examples show that the analog drug can continue to grow.

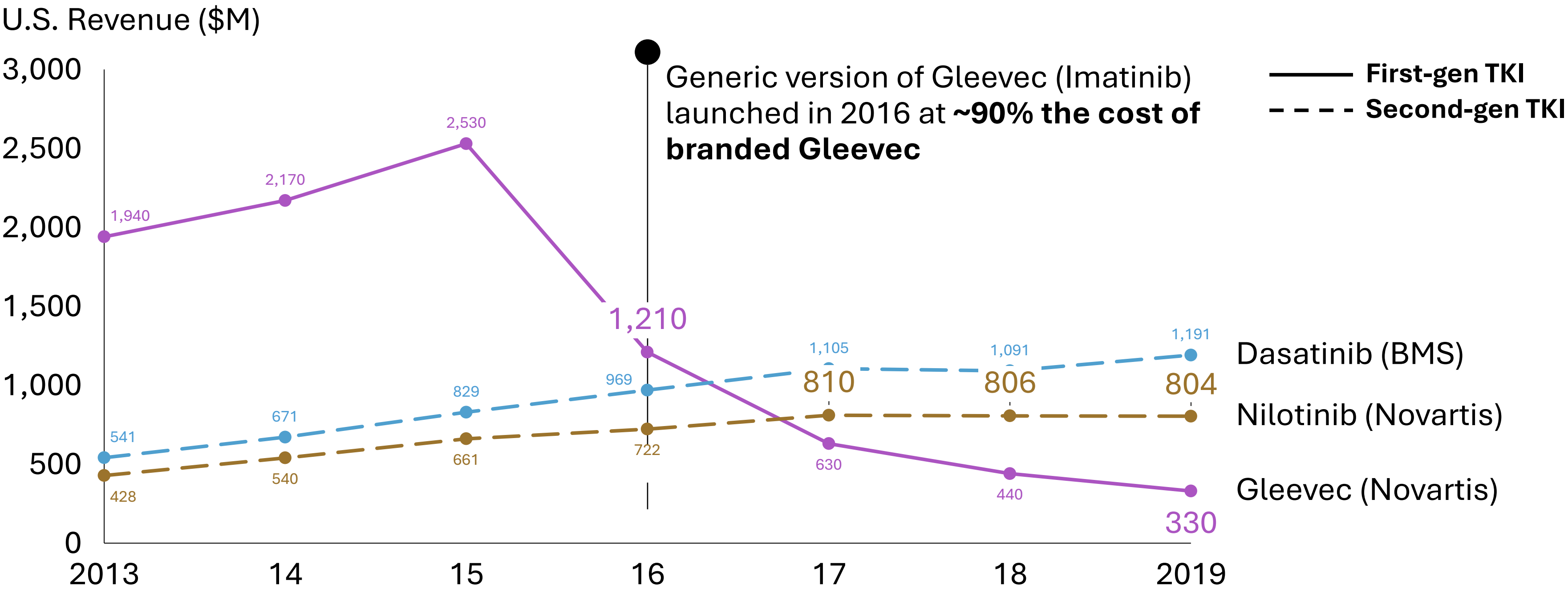
1. Increased: >5% CAGR; Steady: between -5 and 5% CAGR; Decreased: <-5% CAGR; CAGR is assessed for the analog drug in the 3 years following the first LoE event; 2. CAGR reported for only one or two years if LoE drug went generic in 2024 or 2023, respectively; 3. CAGR reported for one or two years if anchor and analog LoE dates are 3 or 2 years apart; 4. LoE year of analog is not considered; CAGR reported for one year as sales data is only available up until 2025. CAGR reported for only one or two years if LoE drug went generic in 2024 or 2023, respectively

3 Case Study: Sales of a clinically differentiated next-gen TKIs for CML continued to grow despite the launch of a generic

- 1 Differentiation
- 3 LoE defense

U.S revenue of select first- and second-generation TKIs for CML

Similarity to Attruby’s case



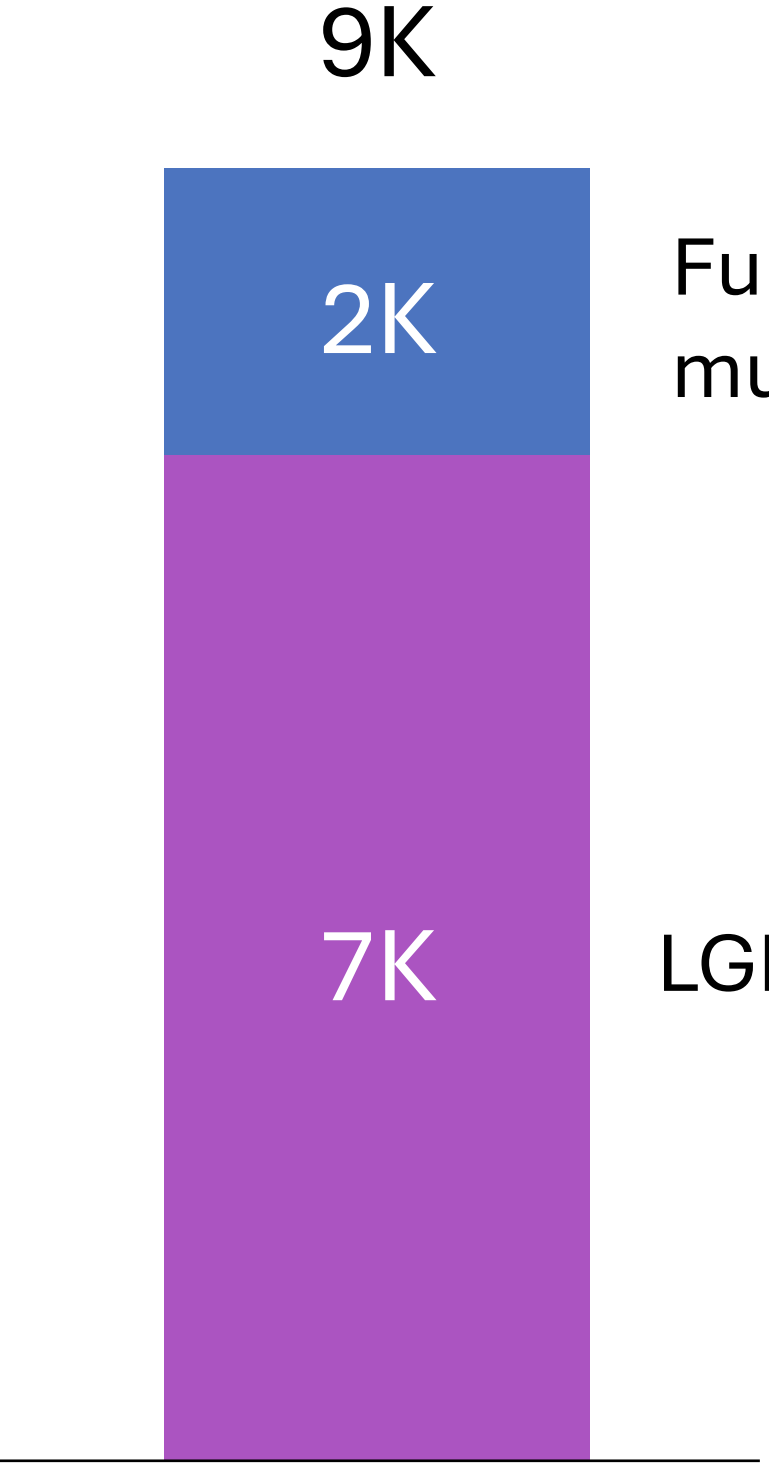
- **Dasatinib achieves 46% major molecular response** vs. 28% with imatinib at 12 months with faster time to response
- **Nilotinib achieves complete molecular response in 21% of patients** vs. 10% with imatinib at 24 months
- As Gleevec’s LoE approached, **Novartis ran marketing campaigns designed to shift oncologist prescribing away from Gleevec** and towards the clinically superior and patent-protected Nilotinib

- Rare disease ✓
- Cardiology ✗
- Small Molecule ✓
- Analog is differentiated ✓
- ~3 generics launched within a year ✓
- Specialty pharmacy ✓
- High proportion Medicare ✓
- Exp with LoE management ✓
- 1-2 other branded competitors ✓

BBP-418



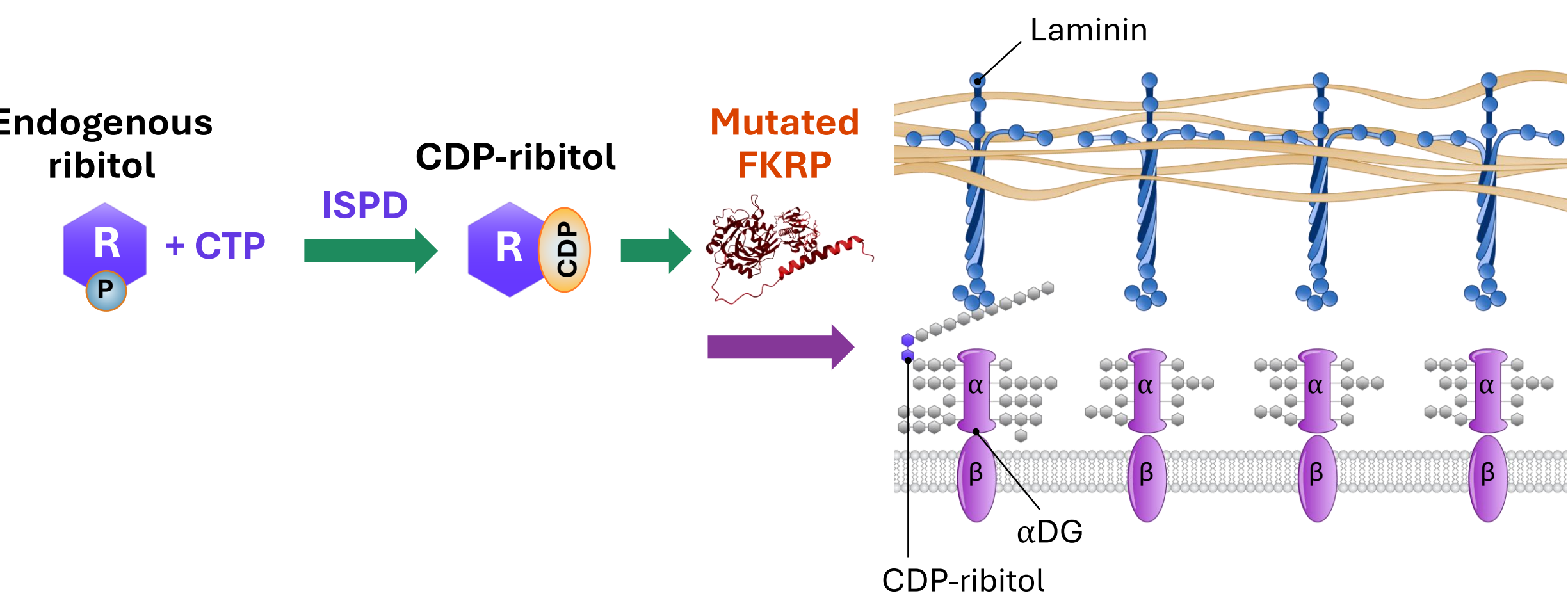
LGMD2I/R9 is a high unmet need muscular dystrophy with no approved therapies, representing a >\$1B market opportunity in the US and EU

Addressable patients by indication		Unmet need	
 <p>9K</p> <p>2K</p> <p>Fukuyama congenital muscular dystrophy (<i>Japan</i>)</p> <p>7K</p> <p>LGMD2I/R9^{1,2} (<i>US + EU</i>)</p>		LGMD2I/R9 is an inherited neuromuscular disorder characterized by lower-limb weakness and loss of ambulation as well as respiratory decline and cardiac dysfunction	 <p>Market opportunity \$1B+ <i>program</i> <i>PRV eligible</i></p>
		No approved therapies for LGMD2I/R9	
		Current standard of care is aimed at symptom management and includes physical therapy, cardiac and pulmonary function surveillance, and pain management	
		Standard of care does not prevent continuous and progressive decline in LGMD2I/R9 patients	

BBP-418 is a first-in-class small molecule therapy that targets LGMD2I/R9 at its source through restoration of αDG glycosylation

LGMD2I/R9 Disease Mechanism¹⁻³

- ✓ FKRPs glycosylates αDG, stabilizing muscle cells by binding extracellular ligands to act as a “shock absorber” for muscle fibers
- ✗ Partial loss-of-function mutation in FKRPs results in dysfunctional, hypoglycosylated αDG in muscle cells, increasing cell susceptibility to damage

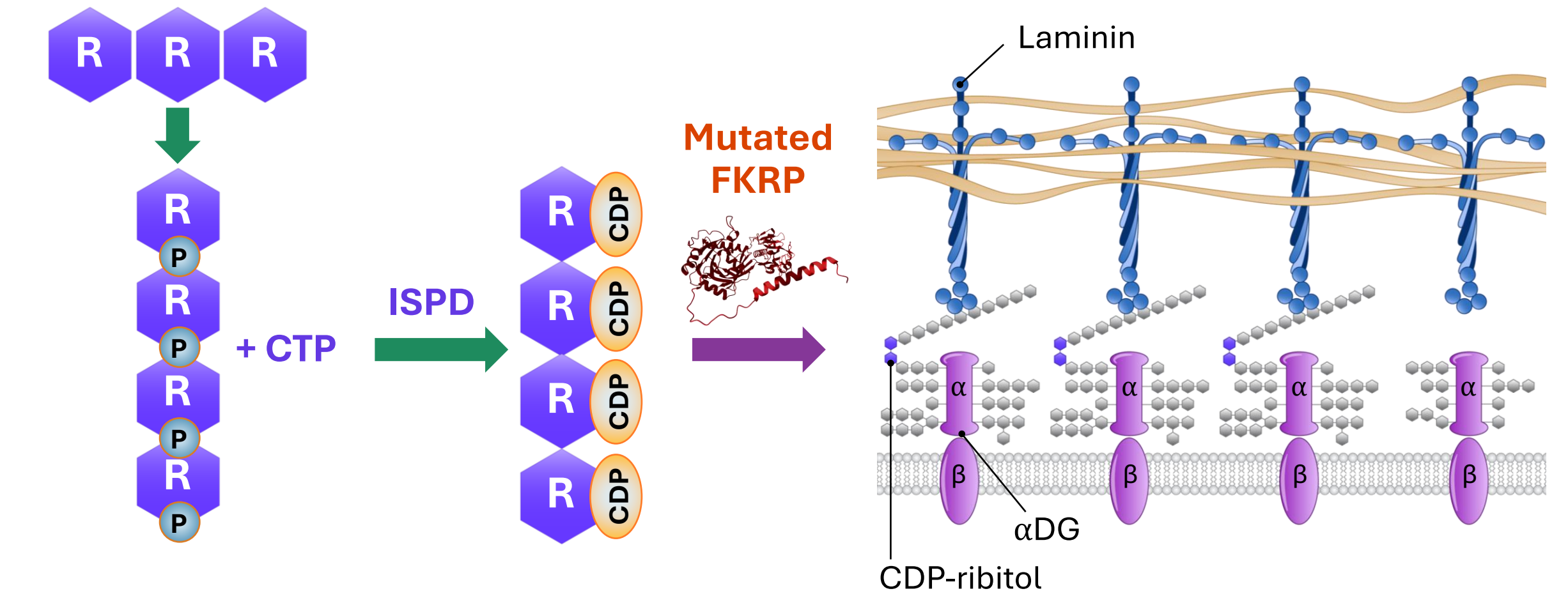


Mutations in *FKRP* prevent addition of CDP-ribitol to αDG, limiting αDG’s ability to function as a “shock absorber” for muscle fibers¹⁻³

BBP-418 Therapeutic Approach

Supply supraphysiological levels of synthesized, pharmaceutical-grade ribitol upstream aiming to drive residual activity of mutant FKRPs enzyme and increase αDG glycosylation levels⁴

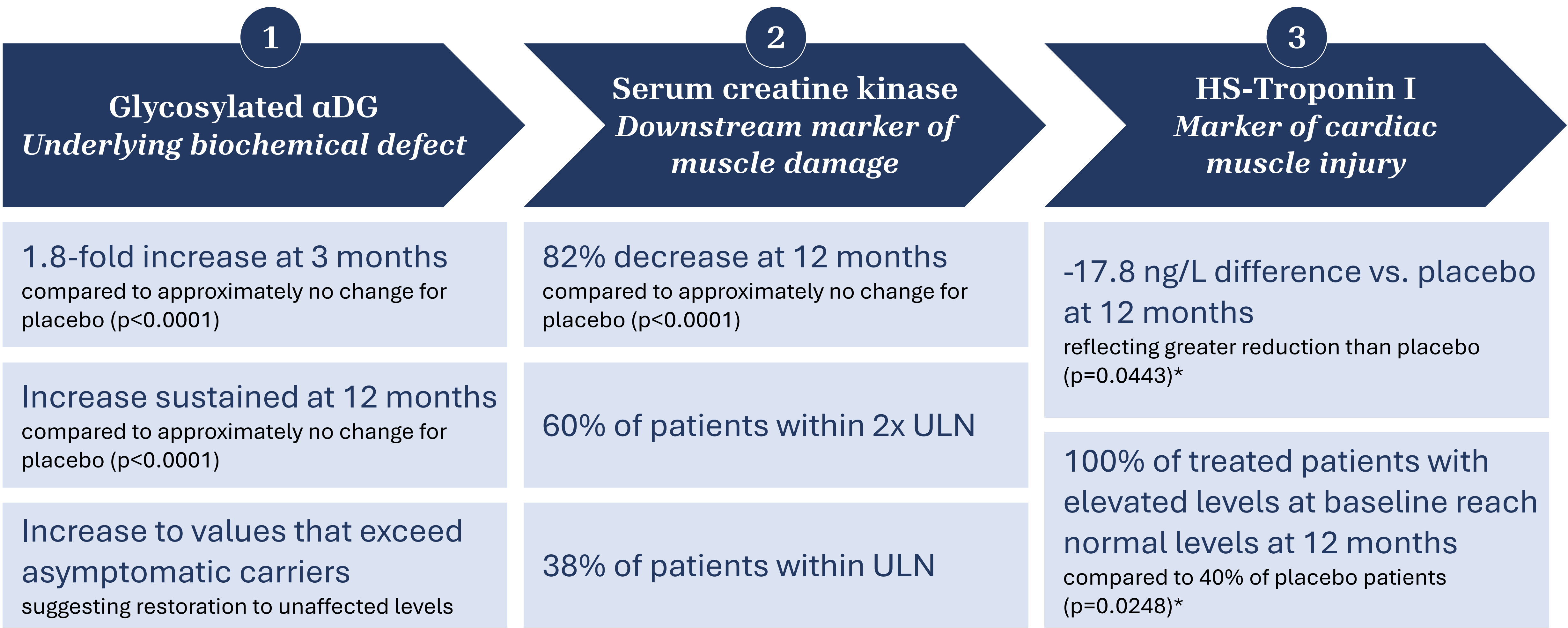
Orally administered BBP-418 (synthesized, pharmaceutical-grade ribitol)⁵



Potential partial restoration of αDG glycosylation⁴

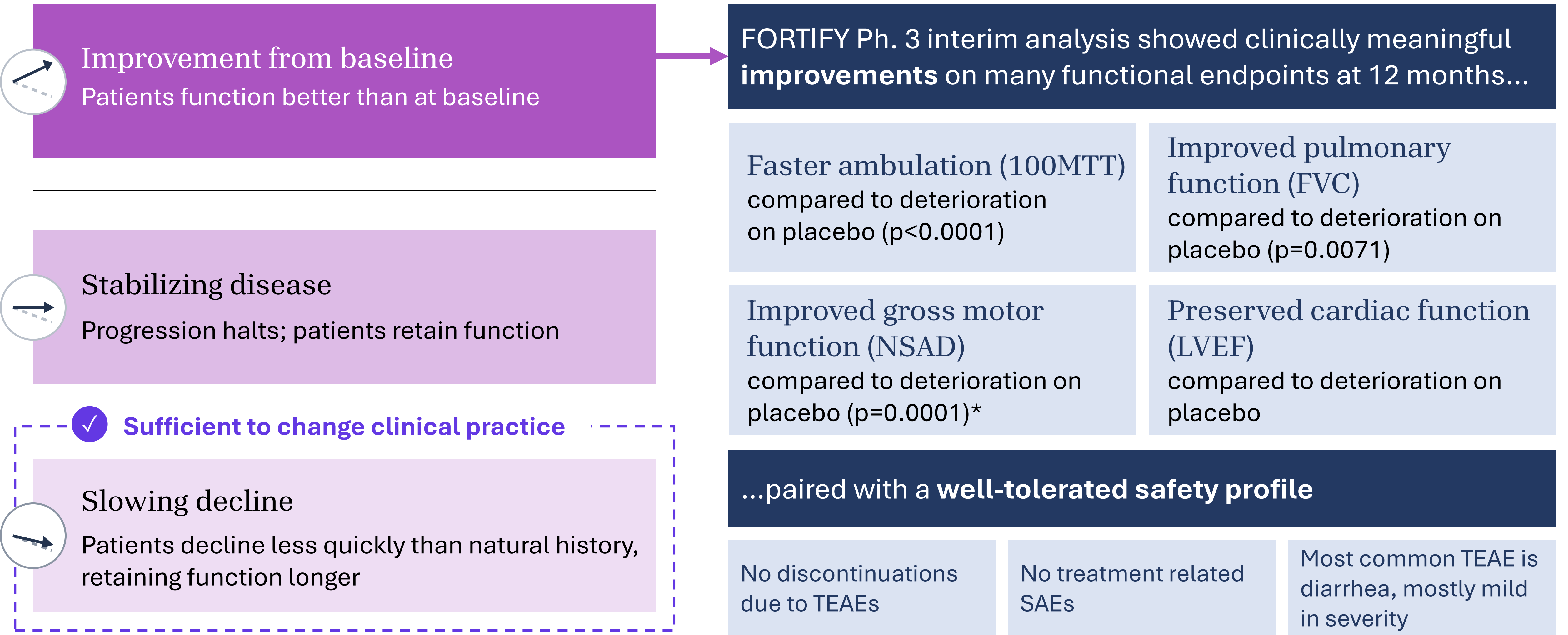
Targeting the disease at its source creates potential for a therapeutic cure, as reflected by some patients achieving normal levels on key biomarkers

Ph. 3 FORTIFY interim analysis: Key biomarker results

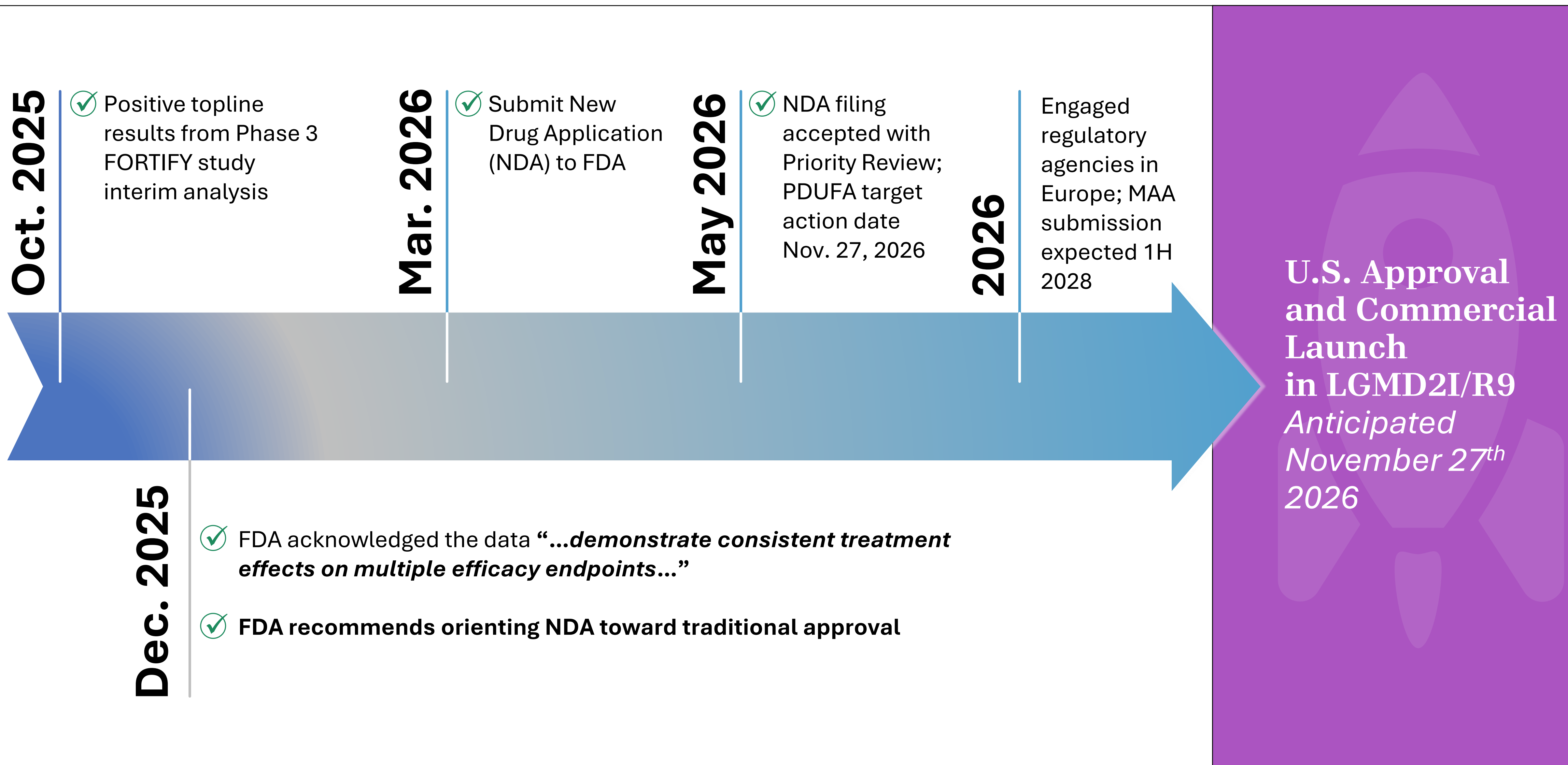


In a debilitating disease with no options, slowing decline is an important win, but by targeting the disease at its source, BBP-418 may deliver much more

Ph. 3 FORTIFY interim analysis: Key functional outcome and safety results



Reflecting the strength of the results, FDA recommended orienting our NDA toward traditional approval and granted priority review for Nov. 2026 PDUFA



If approved, we are prepared to launch BBP-418 as the first and only therapy for LGMD2I/R9, which we expect to be adopted broadly as standard of care

Built on the foundation of Attruby’s commercial success and tailored to the unique LGMD2I/R9 market opportunity

Established commercial infrastructure

Dedicated commercial and medical teams onboarded and in field; field team sized consistent with comparable rare neuromuscular launches, focused on the concentrated prescriber base at MDA care centers

Proven launch playbook

PDUFA target action date November 27, 2026; U.S. launch anticipated upon approval

Ensure broad access

Leverage first-in-class data to secure broad coverage; if approved, BBP-418 would be the first approved therapy for any form of LGMD

We will commercialize and launch BBP-418

Our commercial strategy combines proven rare disease launch capabilities with engagement tailored to neuromuscular specialists and the LGMD2I/R9 community with a field team sized to match the concentrated prescriber base at MDA centers	Strategic area		Objective
		Position BBP-418 as the standard of care	FORTIFY met all primary and secondary endpoints at interim; dataset presented at MDA 2026; PRO data presented at ICNMD 2026 and exploratory cardiac data at WMS 2026
		Systematic patient identification	~500 genetically confirmed LGMD2I/R9 patients identified through account profiling, genetic testing databases, registry data, and clinical site mapping
		Drive awareness and accelerate diagnosis	Focused on concentrated prescriber base at MDA care centers; actively uncovering undiagnosed patients within broader LGMD and Becker MD populations
		Build urgency for early diagnosis and initiation	KOL engagement ongoing: European centers see higher patient concentration; U.S. care more distributed, reinforcing targeted field deployment model

Infigratinib



Infigratinib: A potential best-in-class oral therapy positioned to both grow the treated population and redefine treatment goals beyond height

55,000

addressable individuals with achondroplasia globally

7,000 to 10,000

achondroplasia individuals with open growth plates in the US+EU

\$5B+

potential global market

~30%

of the addressable population in the US is treated today

~70%

For the remainder, the status quo is not good enough

Infigratinib is well positioned to both convert the existing treated base, and significantly grow the market by having delivered on three key priorities

1

More impact on AHV

2

No more injections: Daily or weekly

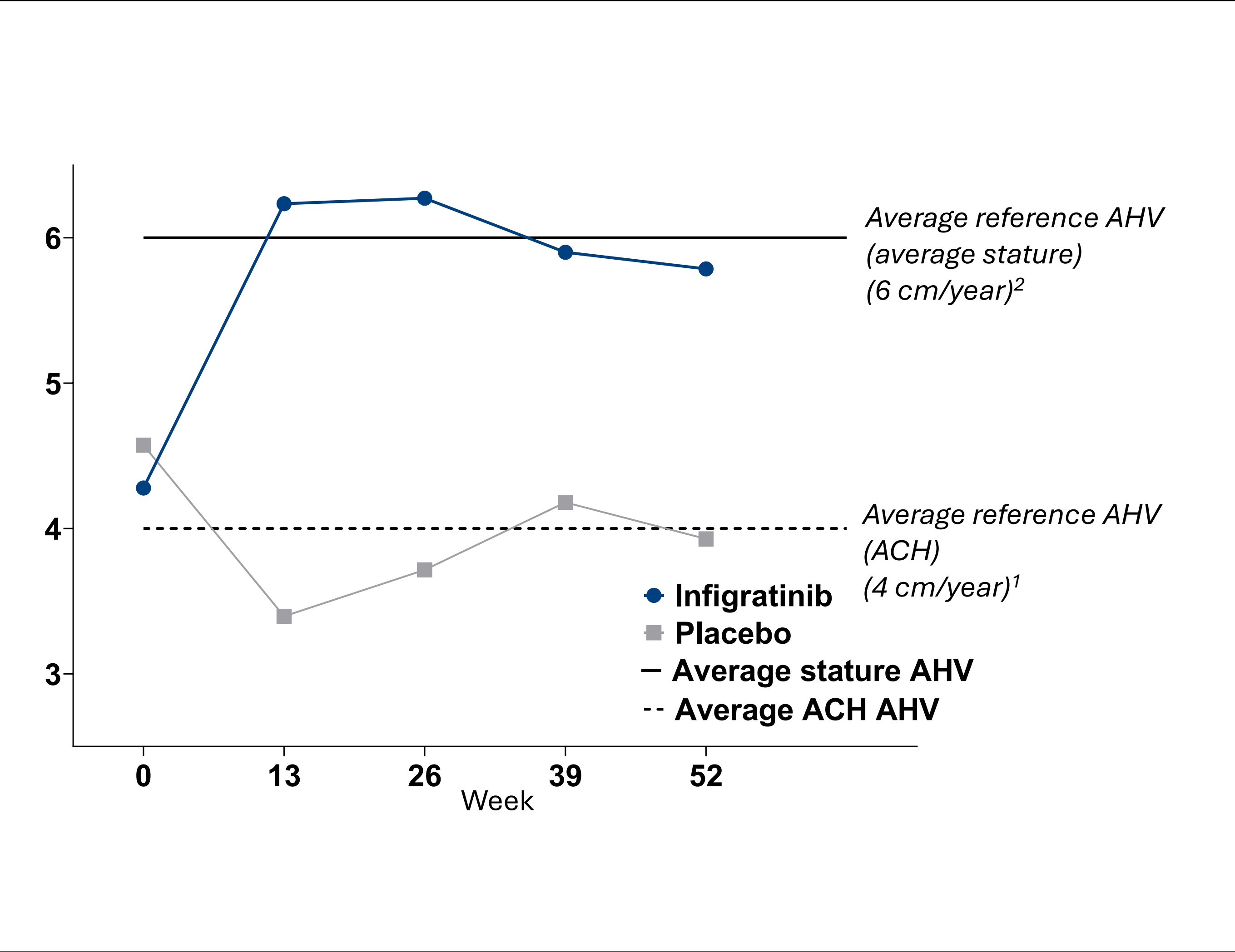
3

Impact on endpoints beyond height/growth

1 Infigratinib improved growth to average stature levels, with a best-in-class change from baseline of +2.1 cm/yr

Mean annualized height velocity in PROPEL 3, infigratinib vs placebo (cm/yr, mean)

The only Ph3 trial in ACH published in NEJM



The NEW ENGLAND JOURNAL of MEDICINE

EDITORIAL

Infigratinib in Achondroplasia — A Potentially Giant Step Forward

2 An oral therapeutic option is required to fully unlock the market opportunity

Daily Injection
Once-daily SC injection

365
Injections per year

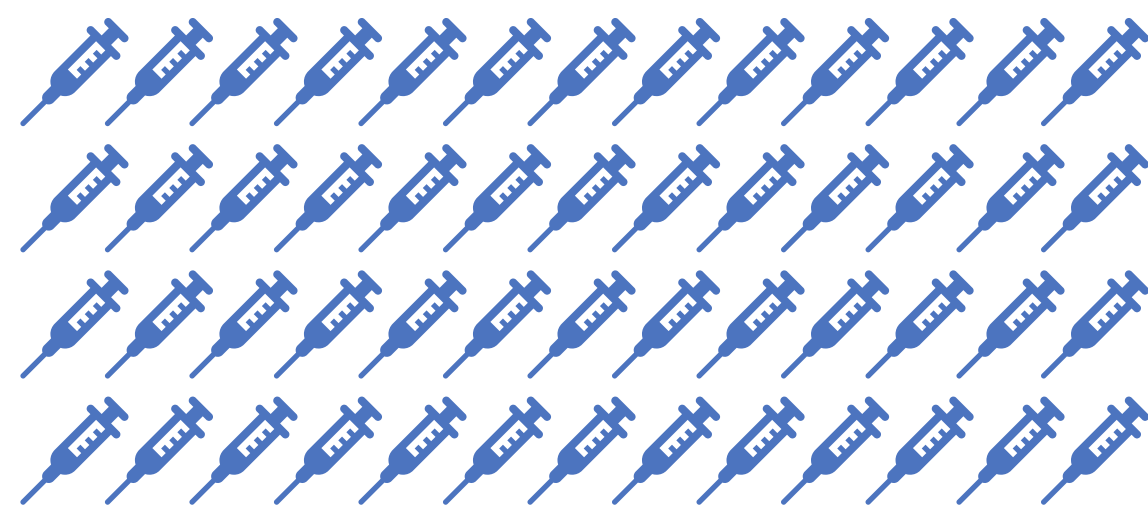
7 per week



Weekly Injection
Once-weekly SC injection

52
Injections per year

1 per week



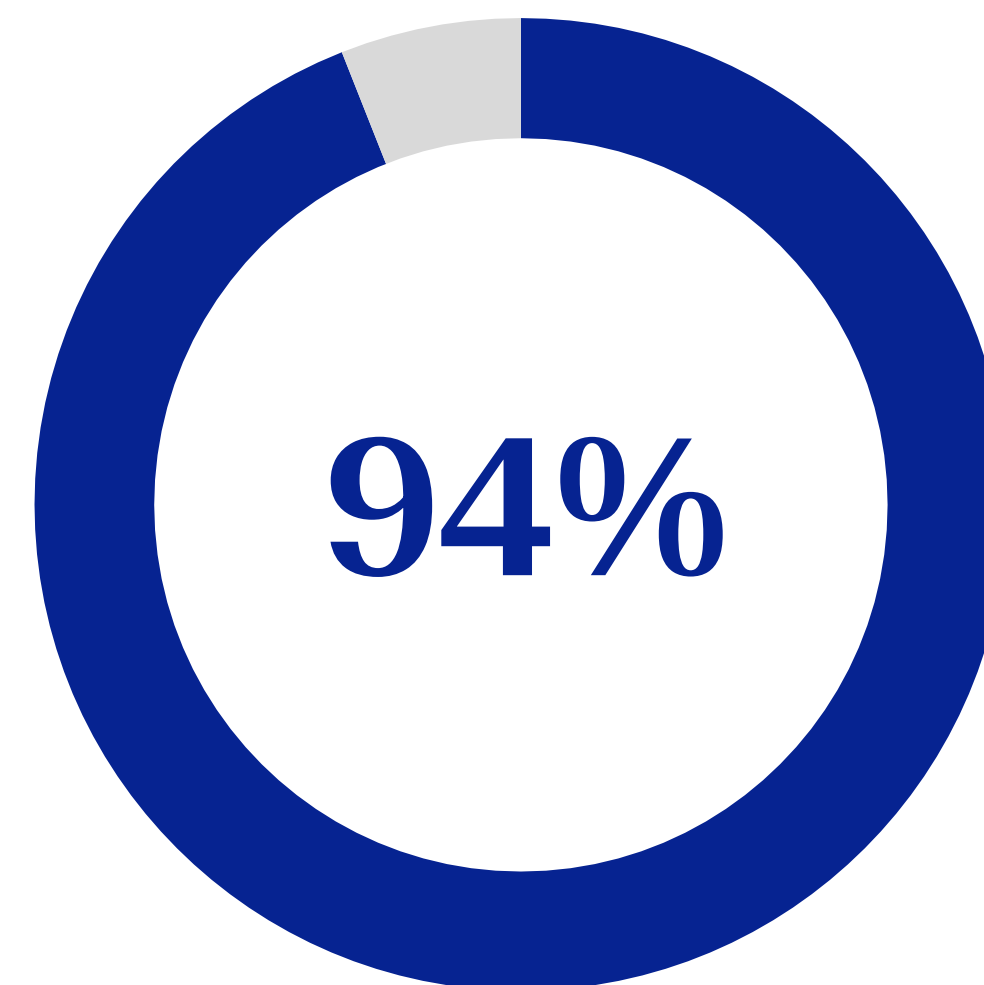
Oral administration
Once-daily oral dosing

0
Injections per year

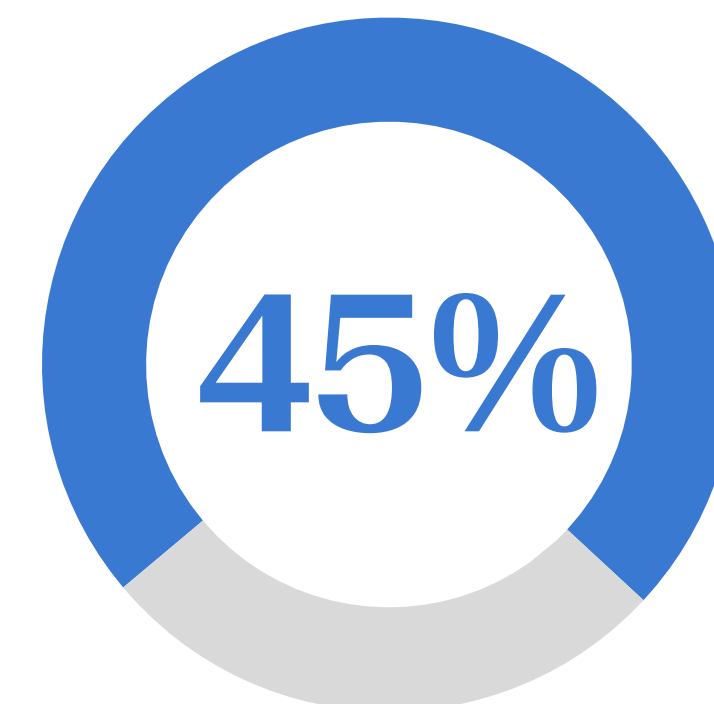
No injections



Physicians indicate injection-related burden and tolerability as the primary barrier towards adoption of current therapies



include injection-related burden and tolerability in their **top 3** reasons for lack of uptake

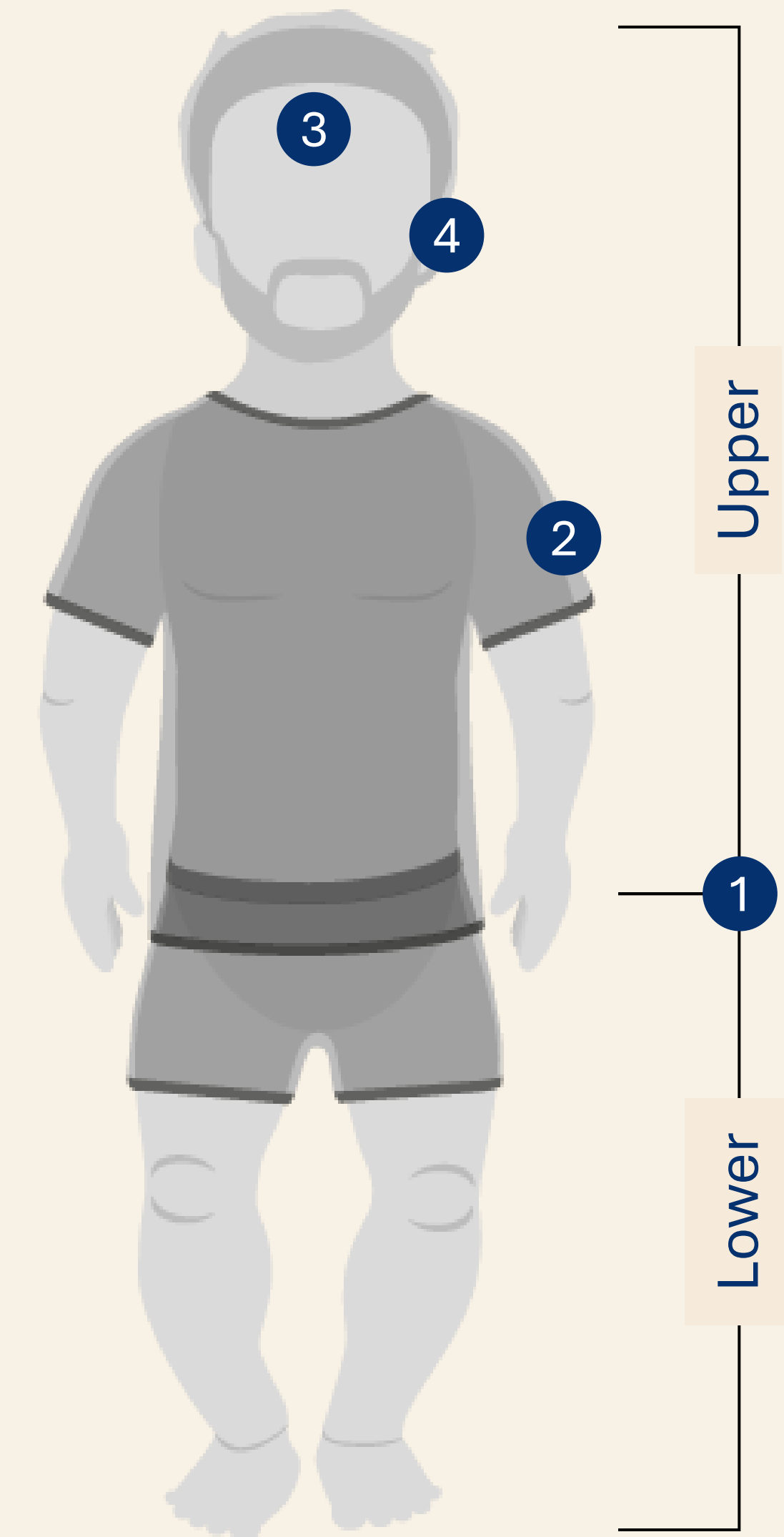


cite injection-related burden and tolerability as the **primary** barrier to adoption

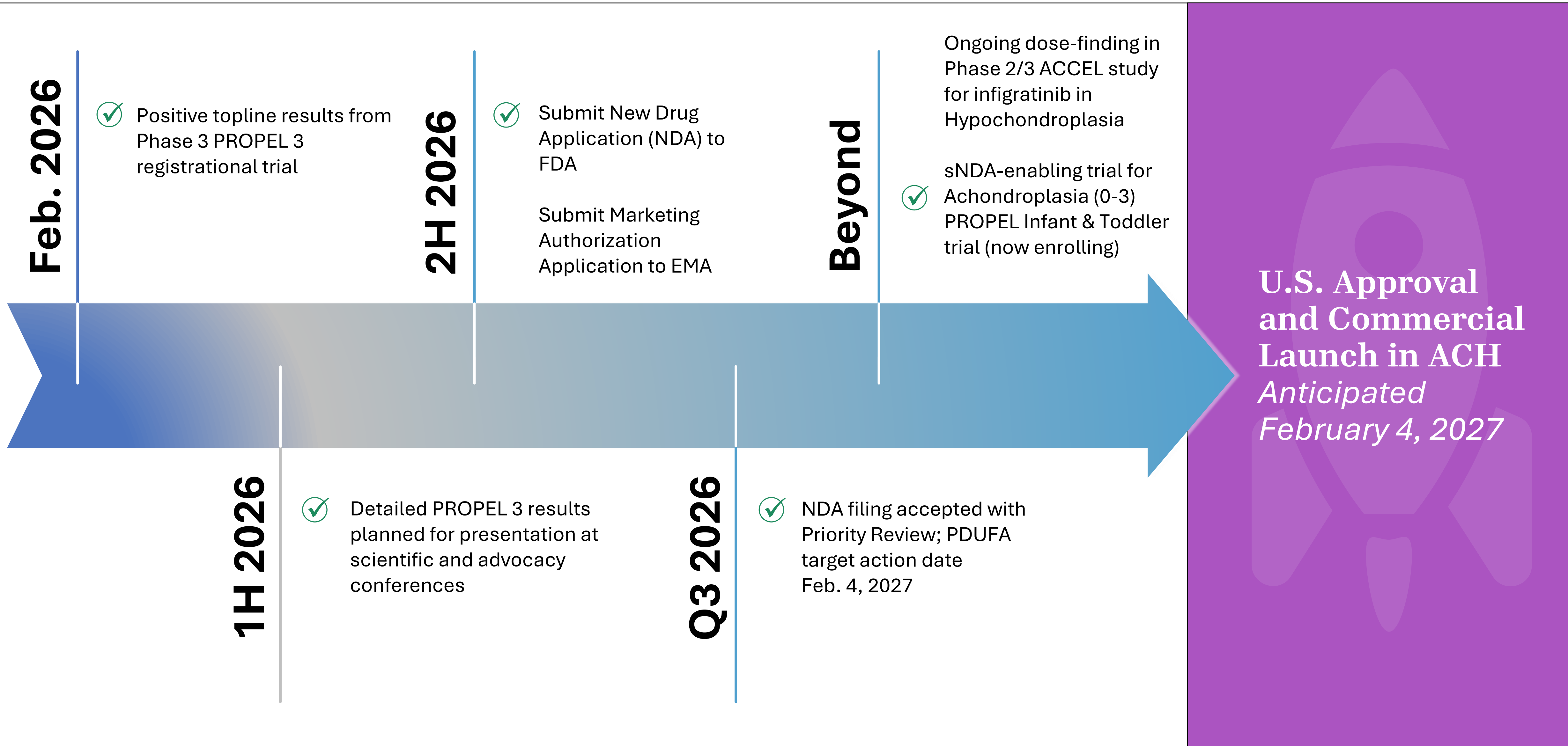
3 Directly targeting FGFR3 enables infigratinib to uniquely show impact on clinically meaningful endpoints beyond height

Infigratinib is the only drug for ACH to show in a 52-week PBO, controlled trial:

- 1 Statistical significance on proportionality (Children <8 yrs of age)
- 2 Statistical significance on arm span (+0.37 SD vs. PBO)
- 3 Favorable trend on sleep apnea measures (In children <8 yrs of age, no change in total AHI vs. 63% increase in PBO)
- 4 Favorable trend on ear infections (In children <8 yrs of age, 47% lower annualized rate of otitis media vs. PBO)



FDA has provided a PDUFA date of February 4, 2027



Encaleret

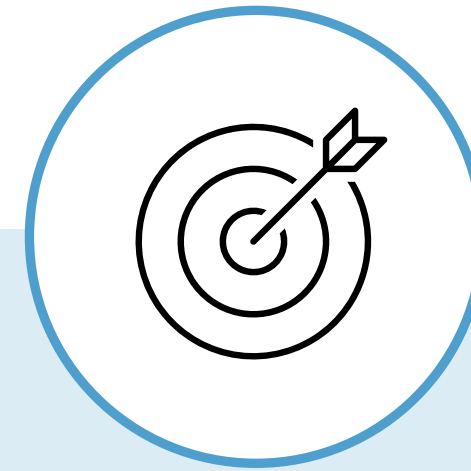


Encaleret provides a first-in-category disease-modifying oral option that targets the root cause of ADH1, and near-term market expansion opportunity



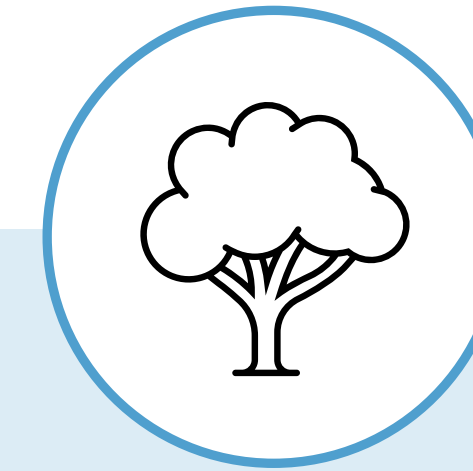
First-in-Category Launch...

ADH1 represents a \$1B market, with an estimated prevalence of ~12K individuals harboring causative *CASR* variants in the US alone



Targeting the Root Cause...

CALIBRATE Phase 3 study demonstrated a rapid, robust, and durable effect of encaleret with 76% of participants achieving normalization of both blood and 24-hour urine calcium



With Ongoing Development to Enable Indication Expansion

RECLAIM-HP Phase 3 study in chronic hypoparathyroidism is ongoing; topline results anticipated in early 2028

There are no therapies currently indicated to treat ADH1, a serious and rare genetic condition in a >\$1B market

U.S. Market Opportunity

12K Individuals with ADH1

~12K ADH1 prevalent population in the US based on 4 population databases¹⁻²

9K Symptomatic

73% of patients with ADH1 are symptomatic³

3K – 5K Currently Addressable

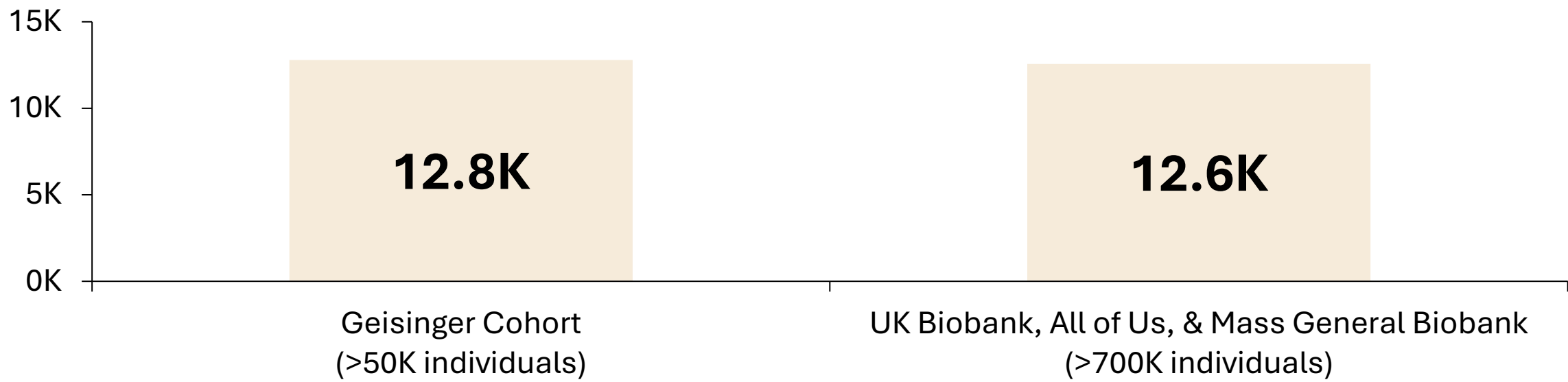
40-60% of symptomatic ADH1 patients are estimated to be diagnosed today; anticipate increase

>600 genetically confirmed patients identified

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 literature¹⁻²

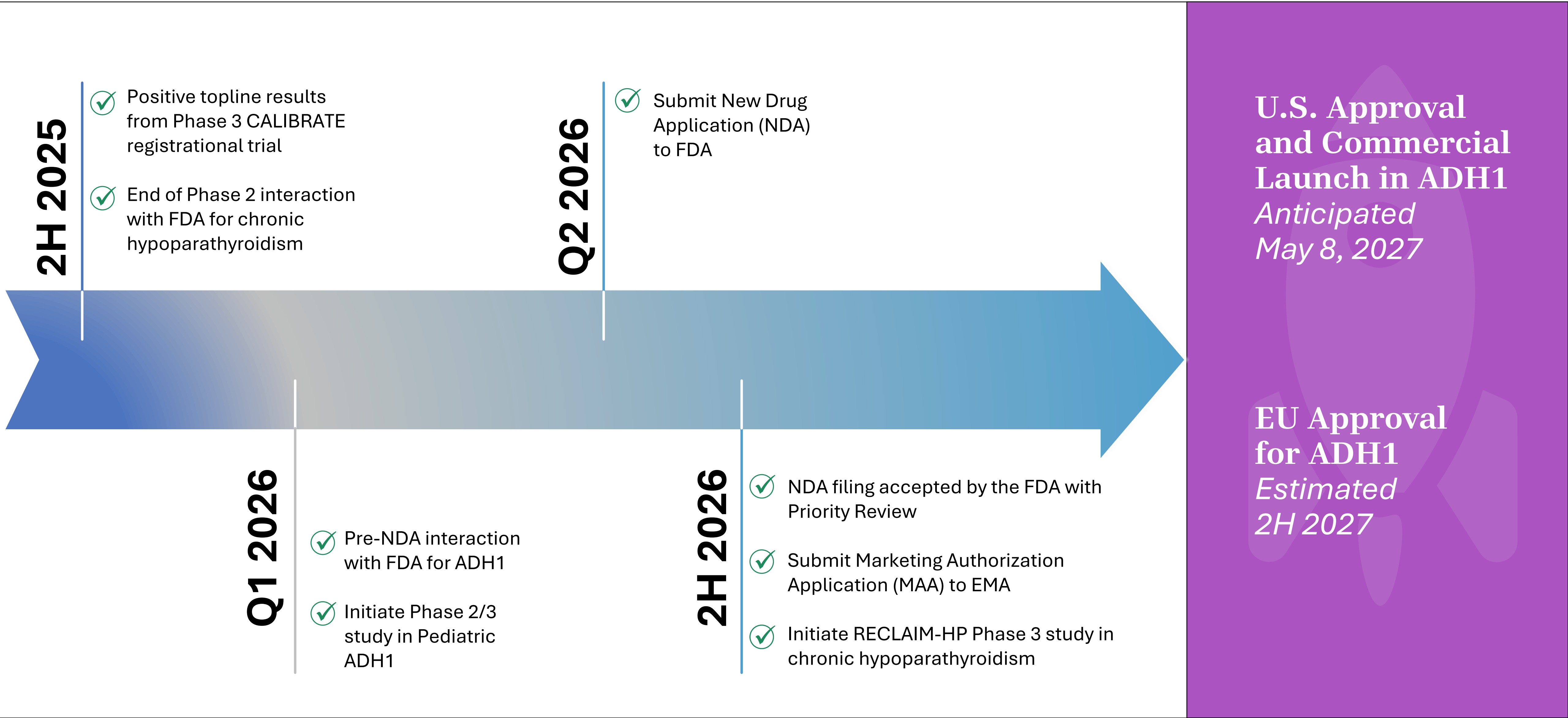


Encaleret directly targets ADH1 at its source

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

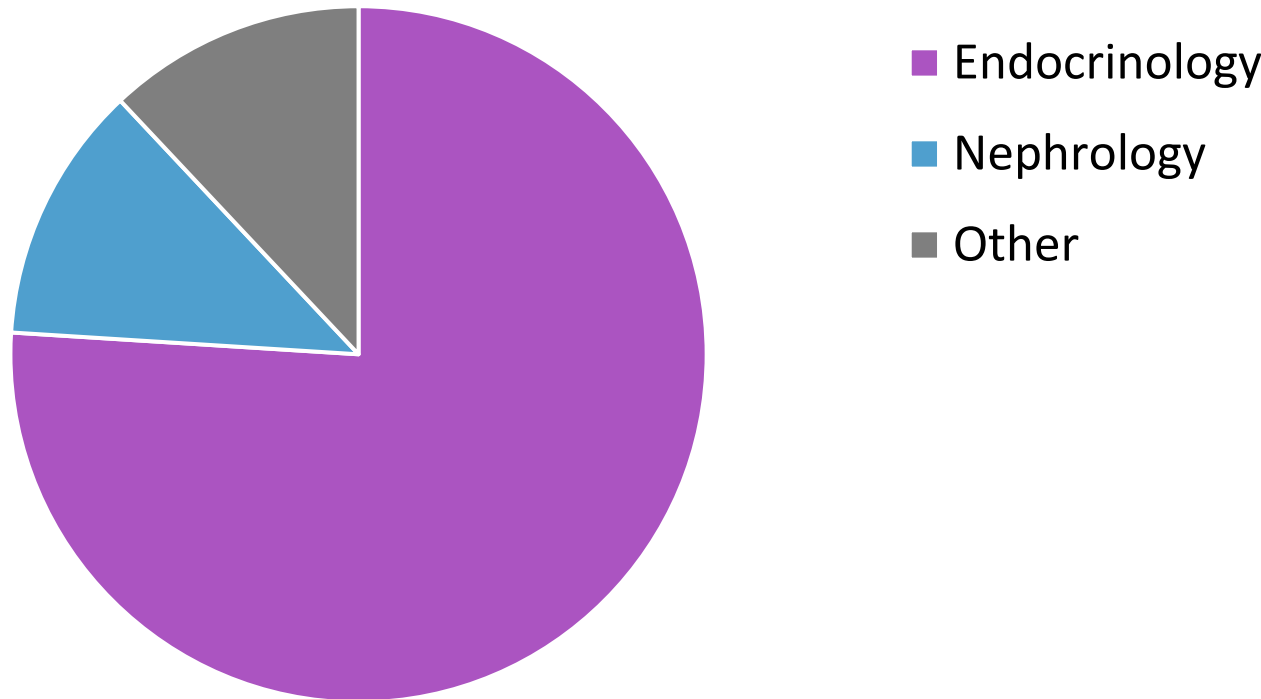
FDA has provided a PDUFA date of May 8, 2027 and our MAA has been submitted to the EMA with potential approval in 2H 2027



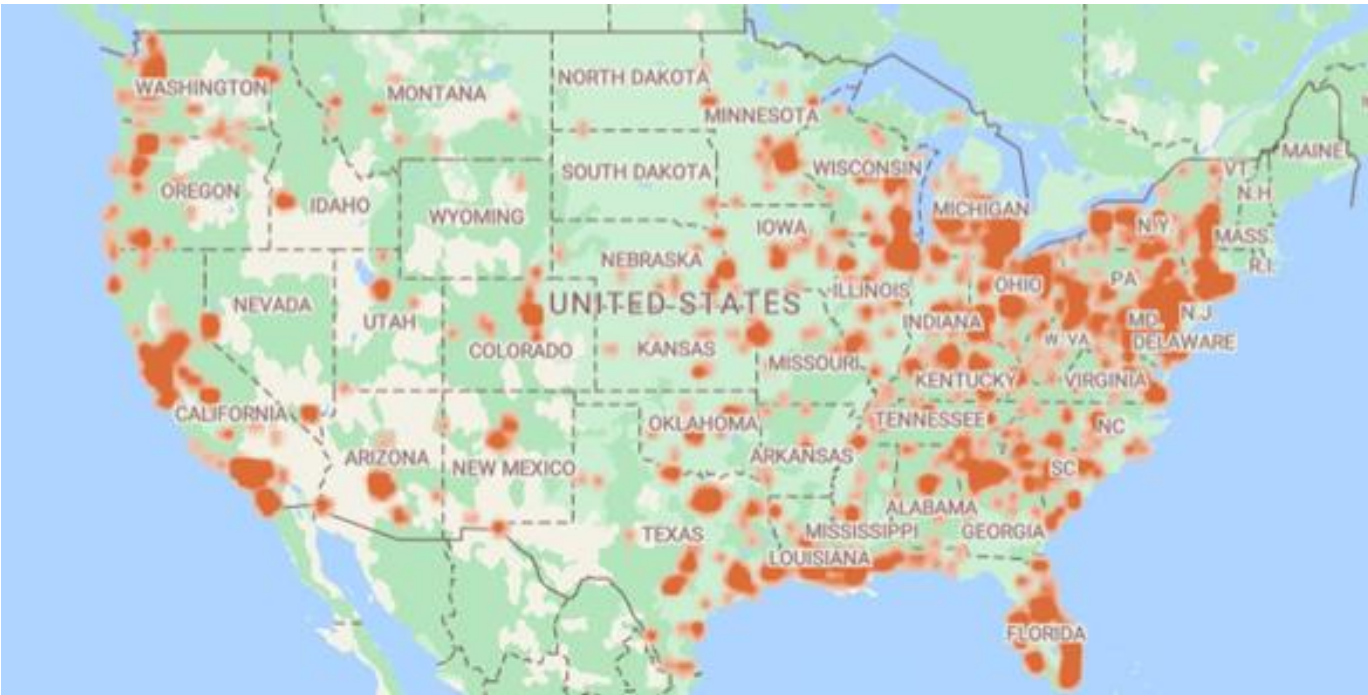
The majority of ADH1 patients are cared for by endocrinologists and/or nephrologists, with coastal concentration

Current State of Market

ADH1 patients predominantly managed by endocrinologists



Concentration of HCPs Managing Patients



How we will capitalize on this first-in-category opportunity



Leverage existing data to prioritize and activate top endocrinologists and nephrologists

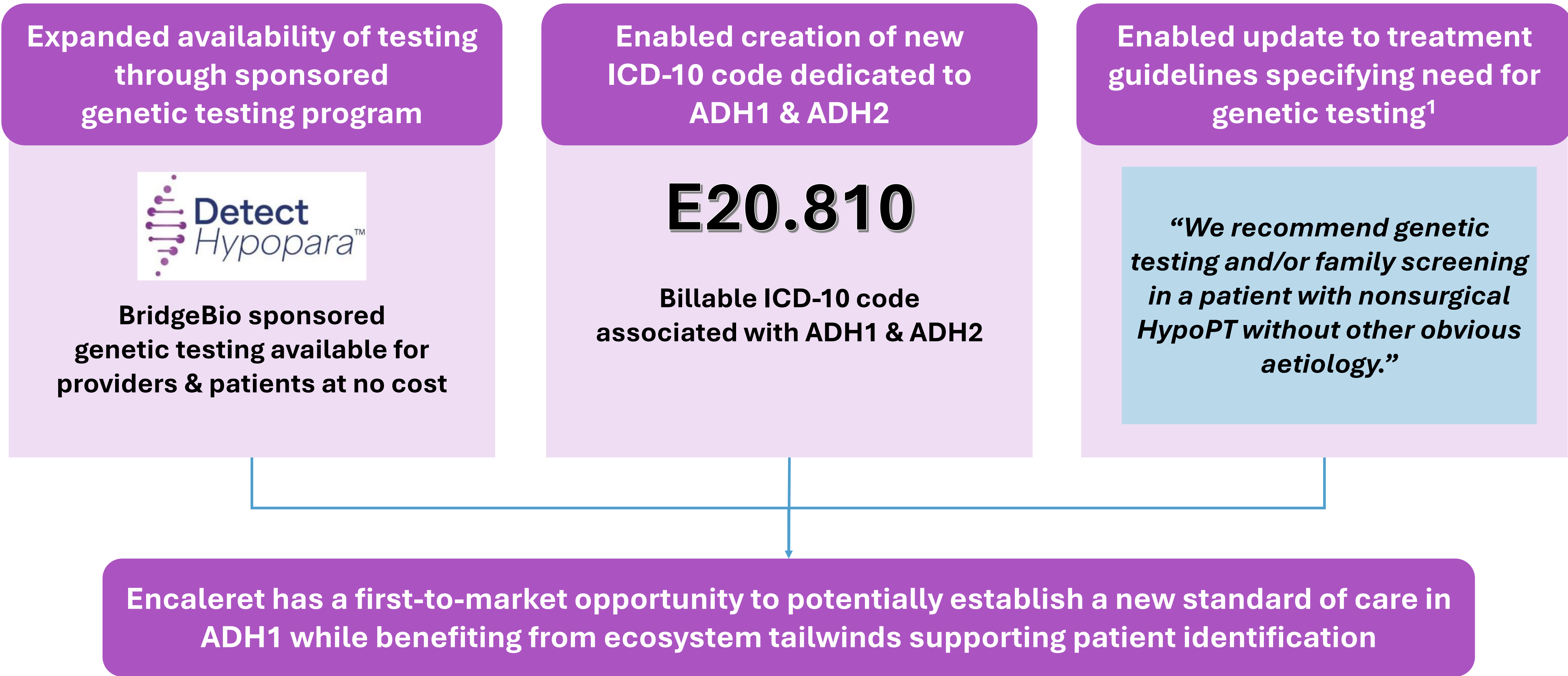


Employ AI/ML analytics to identify undiagnosed patients and managing HCPs

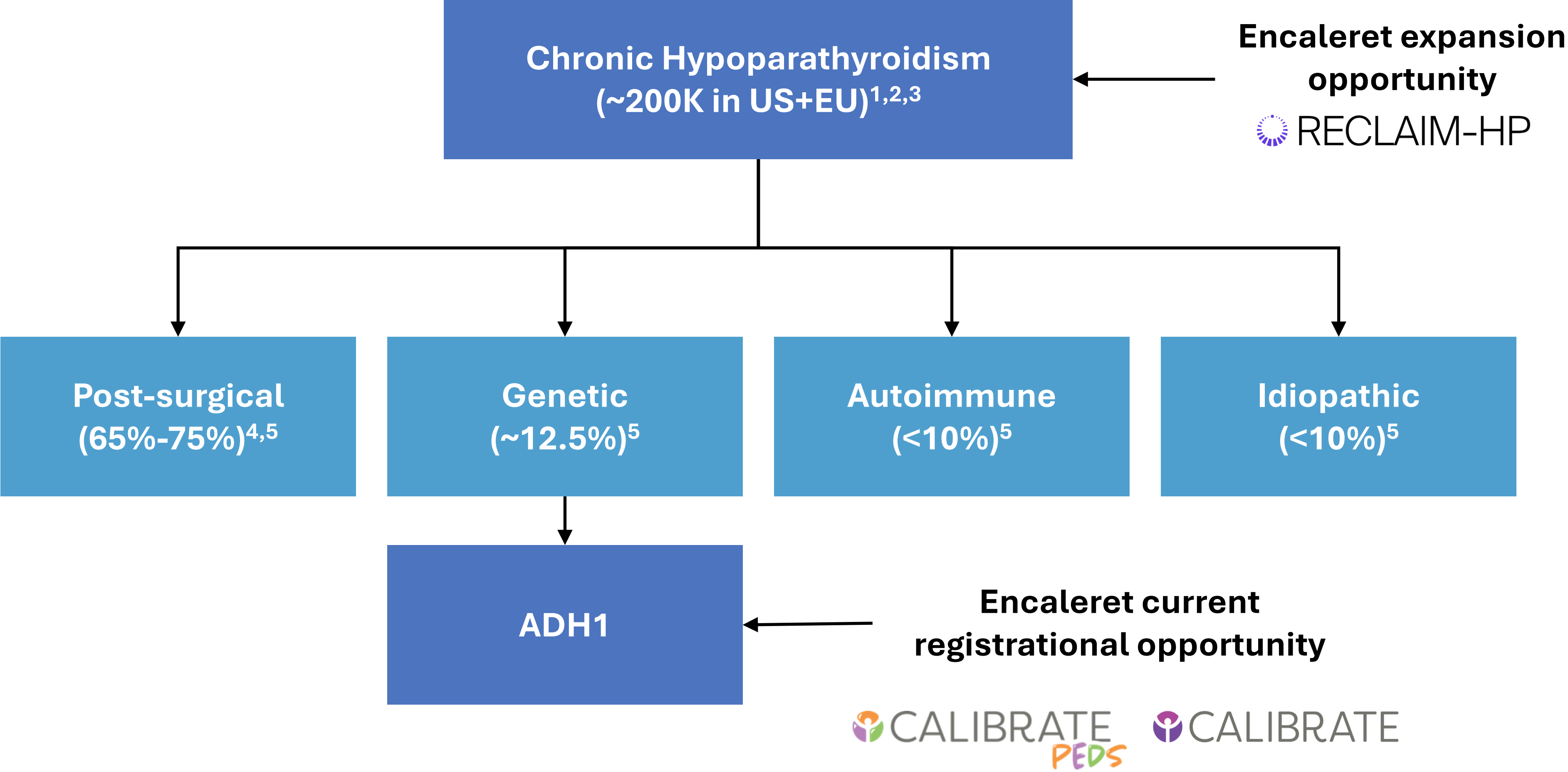


Attract patients to diagnosis opportunities via familial screening and digital targeting

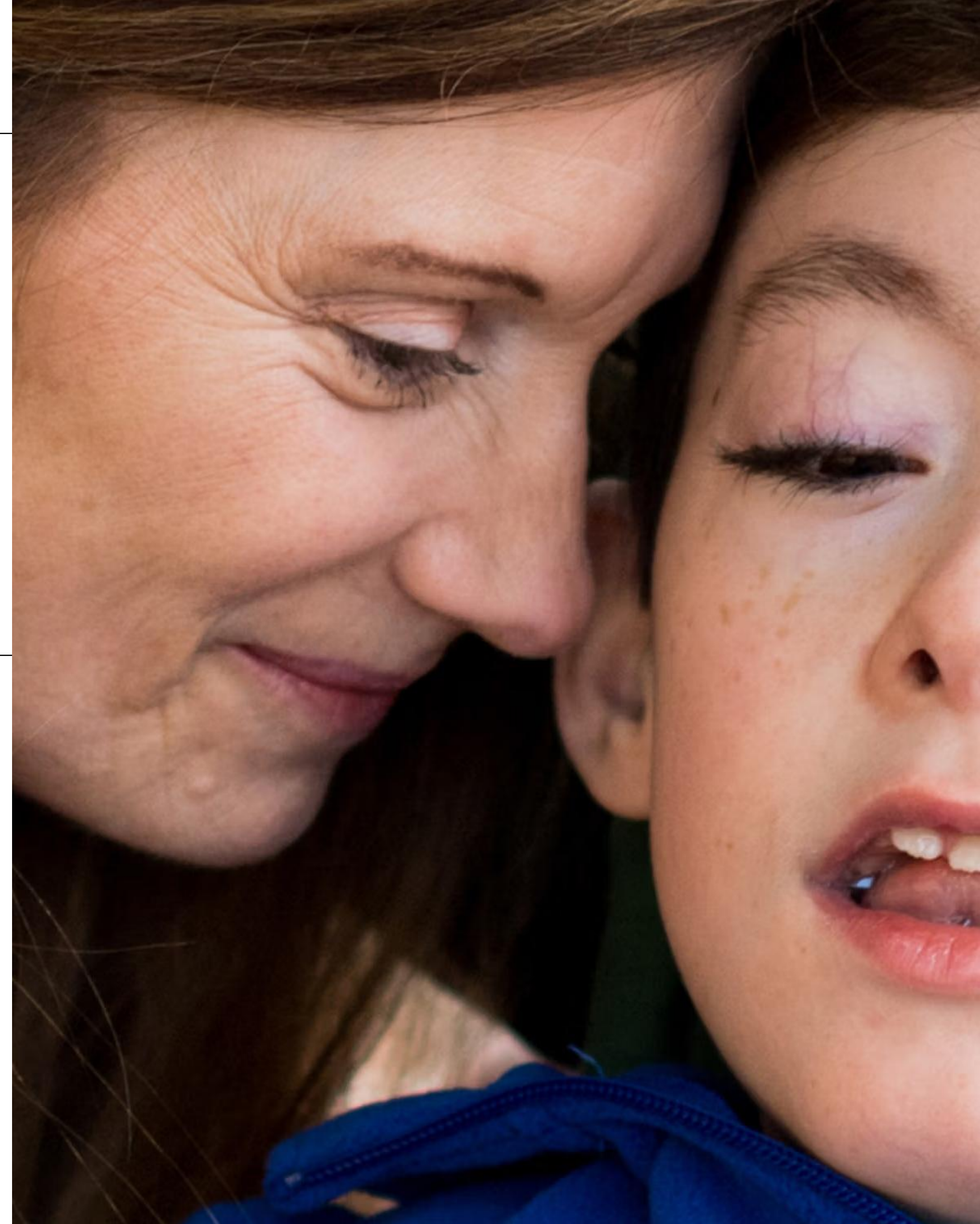
BridgeBio is pioneering efforts to enable the successful launch of encaleret, potentially the first molecule in the class of calcilytics to be approved for any condition



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



BBP-812



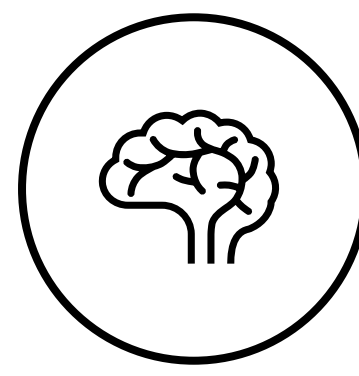
Canavan disease (CD) is a severe, fatal, and ultra-rare neurodegenerative pediatric disease with no approved therapies

CD is a devastating disease with significant unmet need

~1,000 patients across the US and EU

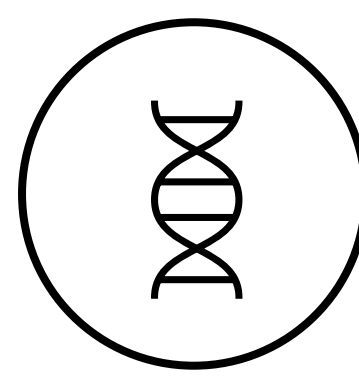
- Canavan disease (CD) is an ultra-rare neurodegenerative disease, usually fatal within the first two decades of life, and >25% of patients die by the age of 10 years
- Children with CD exhibit global and severe cognitive, motor, and language impairment, missing or regressing on most developmental milestones
- 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 approved disease-modifying therapies available for Canavan disease

We designed a therapy to target the disease at its source



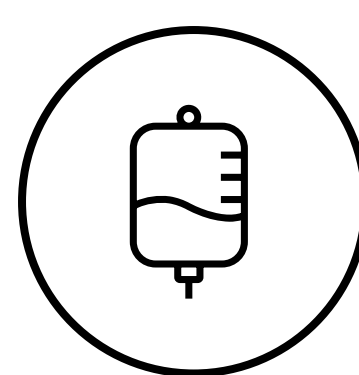
Provide first disease-modifying therapy

Target the condition directly at the source by directly replacing the mutated ASPA gene; 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

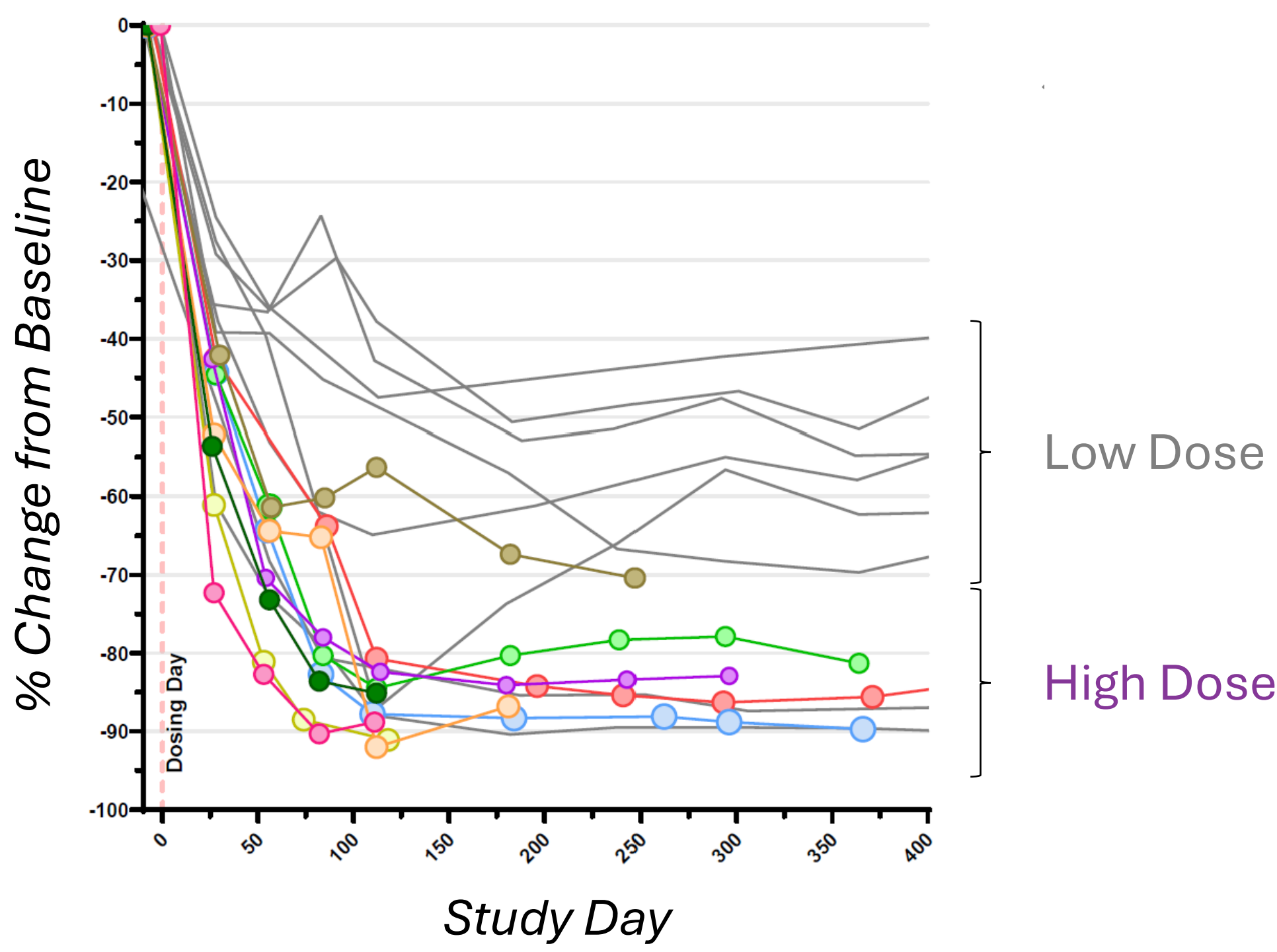
The program is eligible for Priority Review Voucher (PRV) upon approval

BBP-812 has shown dose dependent reductions in urine NAA, the primary disease biomarker, along with significant motor function improvements

Decrease in Urine N-acetylaspartic acid (NAA) levels to mild disease levels

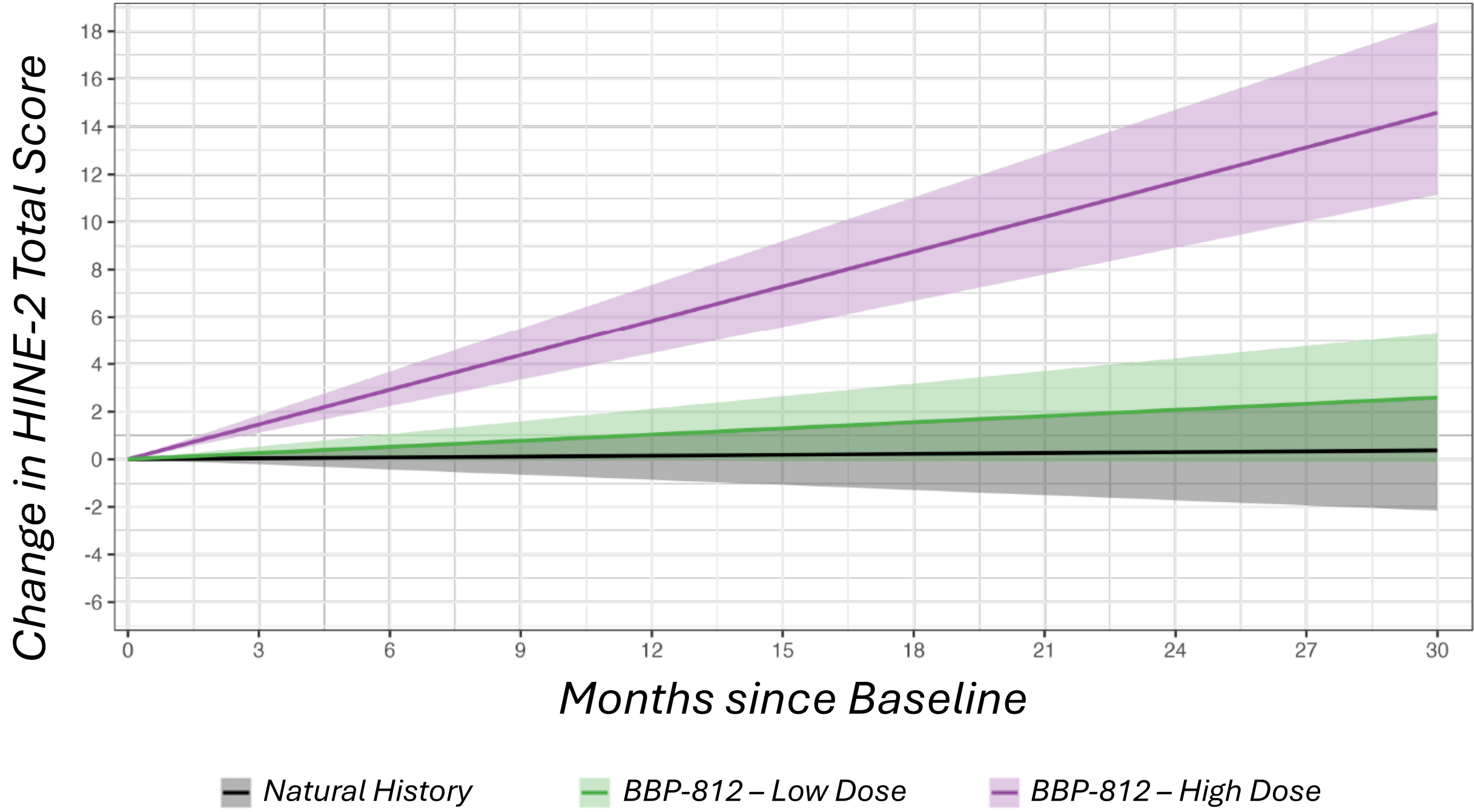
Improvement in Hammersmith Infant Neurological Examination (HINE-2) Trajectory

% Change from Baseline Urine NAA Over Time



- **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

Change in HINE-2 Total Score Over Time



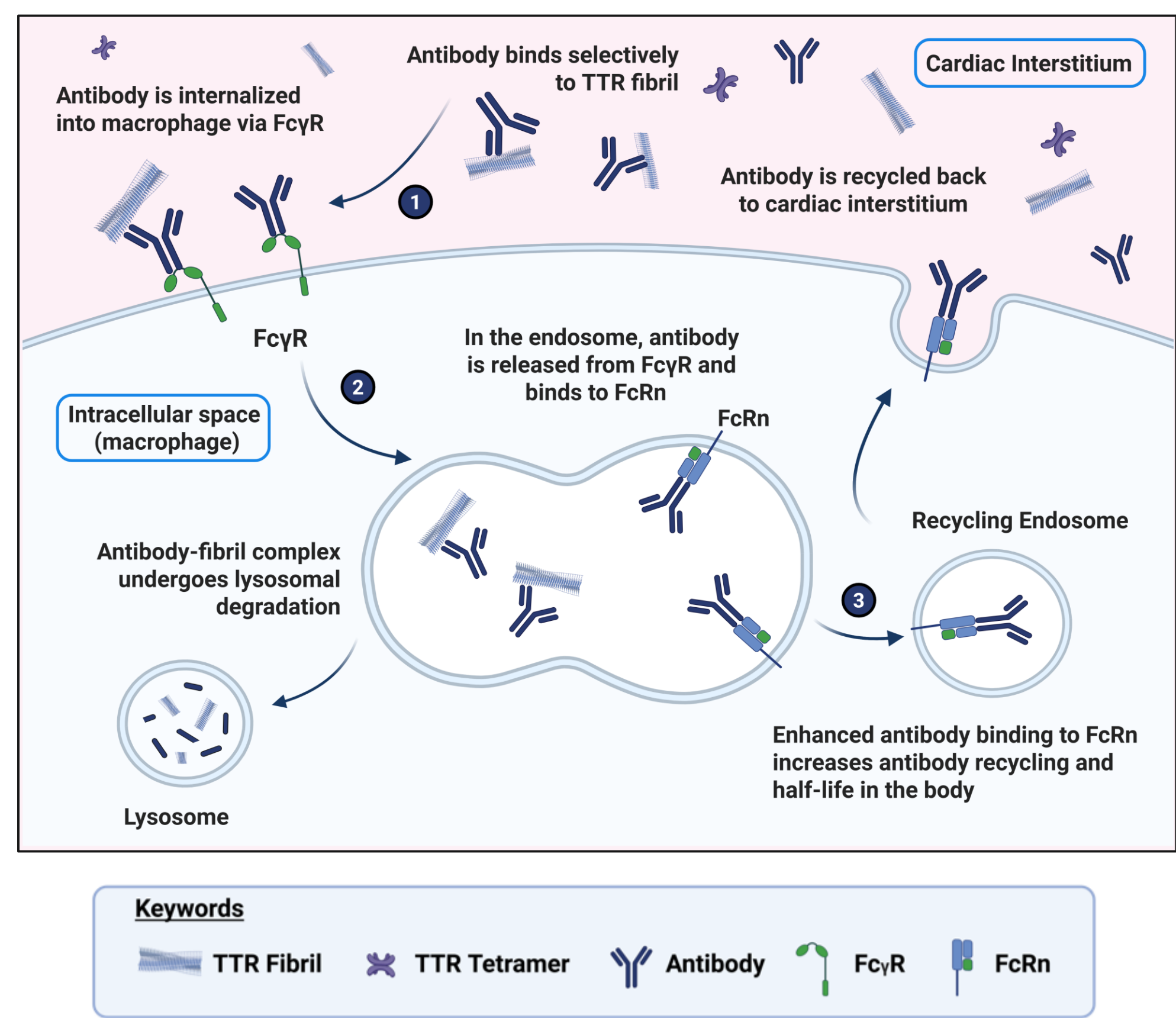
- Trajectory analysis shows **clear, dose-dependent separation in HINE-2 total score** with BBP-812 vs. natural history study
- Children are also showing **improvement on key motor metrics such as sitting, head control, and reaching / grasping**

BBP-717



BBP-717 is a next-generation depleter engineered for differentiation on clinical efficacy and dosing convenience

Depleter Mechanism of Action (MOA)



BBP-717's Differentiated Target Properties

- 1 High-affinity binding for misfolded TTR fibril**
 - No detectable binding to native TTR tetramers
 - ➔ **Potent, selective binding**
- 2 Enhanced macrophage phagocytosis through FcγR activation**
 - First depleter engineered for enhanced Fcγ receptor binding to boost macrophage activity
 - ➔ **Clears more target**
- 3 Improved dosing convenience through antibody half-life extension**
 - First depleter engineered for enhanced FcRn binding
 - ➔ **Extends antibody half-life**

Depleter program expected to advance into the clinic in 2027–2028

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](https://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.