bridgebio

hope through
rigorous science

Corporate presentation

December 2021

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BridgeBio Pharma: Hope through rigorous science

Our mission: To **discover**, **create**, **test** and **deliver** transformative medicines to treat patients who suffer from genetic diseases and cancers with clear genetic drivers

Context #1 | Still Day 1 for innovation within genetic medicine

MACROMOLECULES → **MOLECULAR SYSTEMS** → **CLINICAL DIAGNOSIS** → **NEW THERAPEUTIC MODALITIES**

DNA

- gnomAD
- ENCODE3

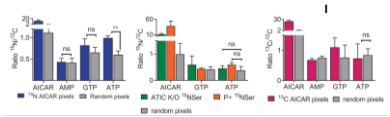
RNA

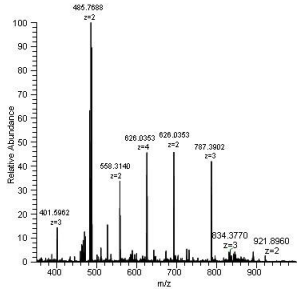
- GTEx
- Single cell sequencing advances

PROTEIN

- CryoEM
- DeepMind

- Mass spectrometry + metabolomics give us 1st snap of purine bio-synthesis





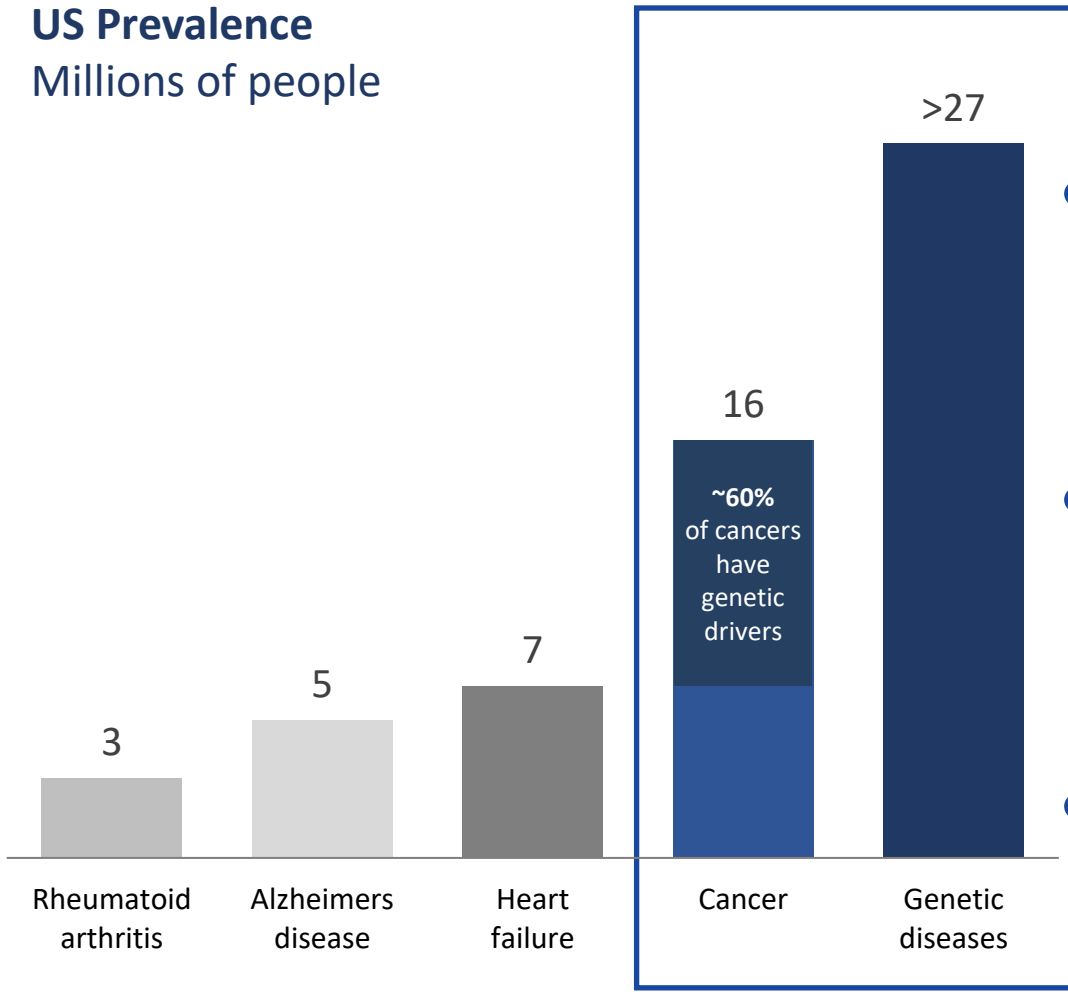
- Whole genome sequencing of rare disease patients in UK Biobank
- Expanded sequencing led to novel causal variants in 28 genetic disorders

- Antisense oligonucleotides coming of age
- Gene therapy continues maturing

16 FDA approvals for drugs targeting rare genetic diseases or genetically defined cancers in 2020

Context #2 | A vast opportunity to help patients…

US Prevalence
Millions of people



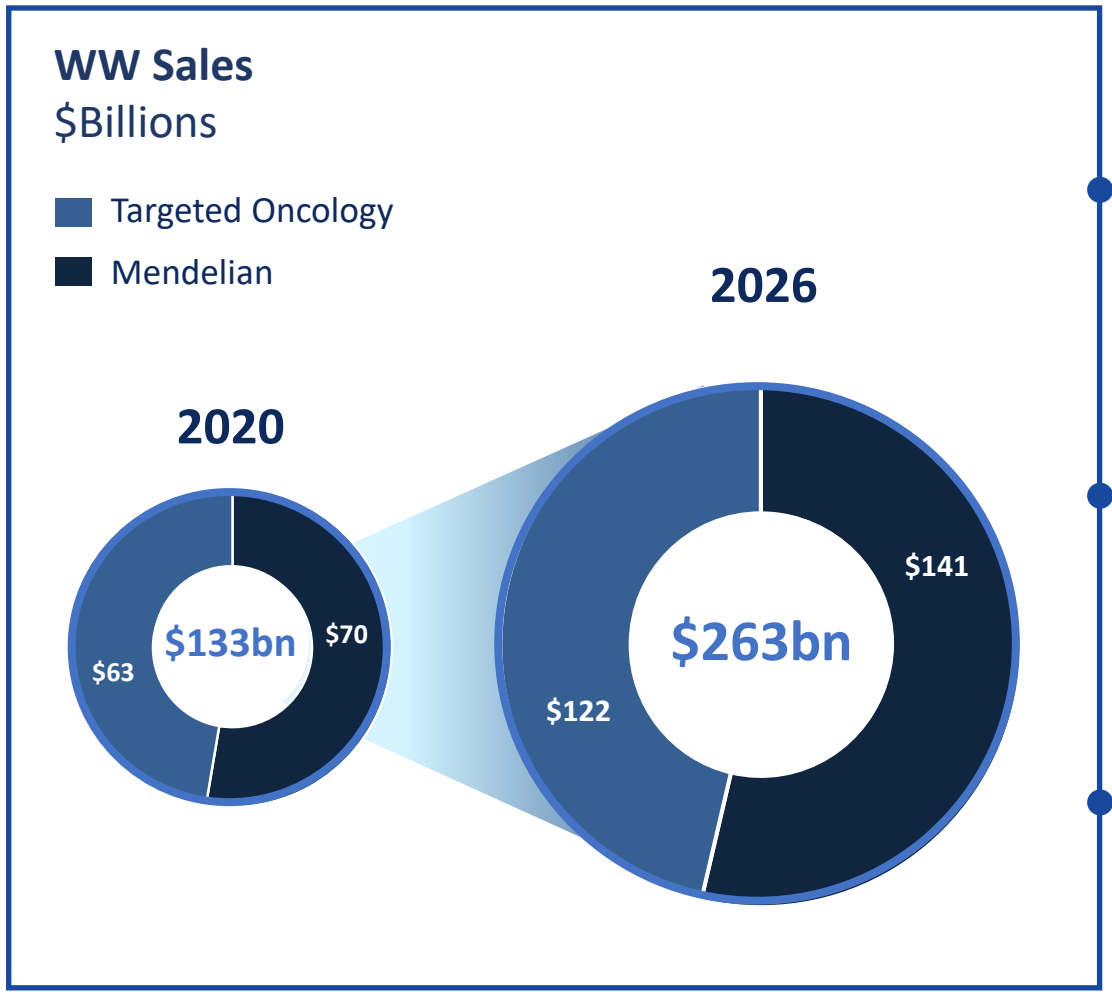
>27 million Americans are living with a genetic disease

50% of people affected are children

Only 5% of these diseases have an approved therapy option

Source: Global Genes, American Cancer Society, American Heart Association, Alzheimer's Association, Arthritis Foundation, Bailey *et al., Cell* 2018

Context #2 (cont'd) | …in several large and growing rare genetic disease markets



$263 billion Projected sales in 2026

12% 2020 – 2026 CAGR

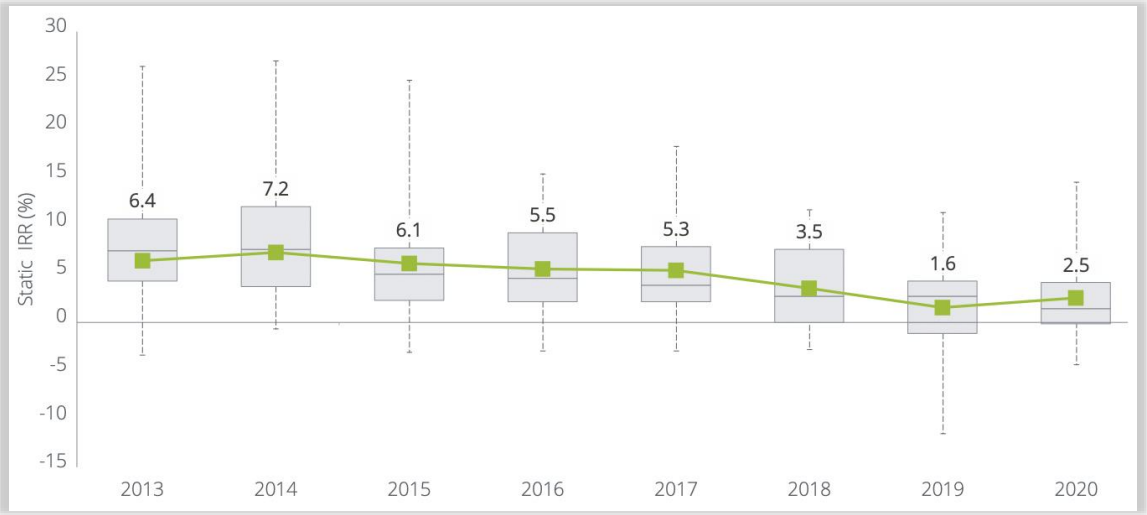
6x Size of projected rheumatoid arthritis, Alzheimer's, heart failure markets

Source: Evaluate Pharma. WW sales include US, EU and Japan. CAGR = Compound annual growth rate.

Context #3 | Currently, few examples of sustainable innovation engines for genetic medicines

Big pharma R&D destroys value in aggregate

Big pharma R&D IRR



- R&D IRR is less than cost of capital for big pharma

Biotech companies have expectations that can't be met

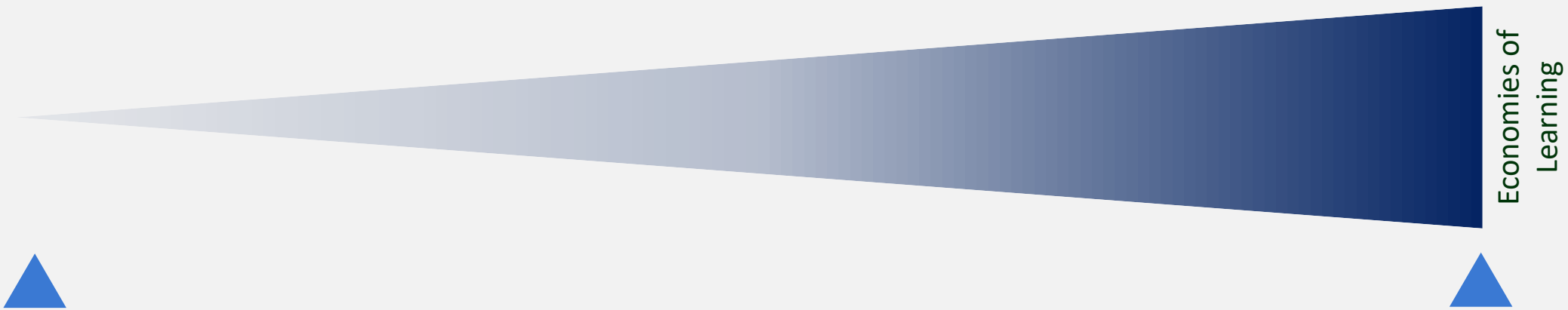
The biotech market requires constant and significant innovation to create long term stable ROIC

- Currently, biotech EV is ~$1.4 Trillion.
  - Assume – One wants to grow market cap by 12% YoY
  - Roughly, capital leaving the system by dividends + M&A = capital raised by IPOs + follow-ons
- If 70% of the value comes from new drugs, biotech would need to generate drugs worth ~$2 Tn over the next 10 years, or approvals with aggregate **~$40 Bn peak year sales every year**

Source: Deloitte, FactSet, Internal BridgeBio analysis

What does a sustainable genetic medicine innovation ecosystem look like? Criteria #1

Criteria #1: Need to solve for diseconomies of scale early, and economies of scale late

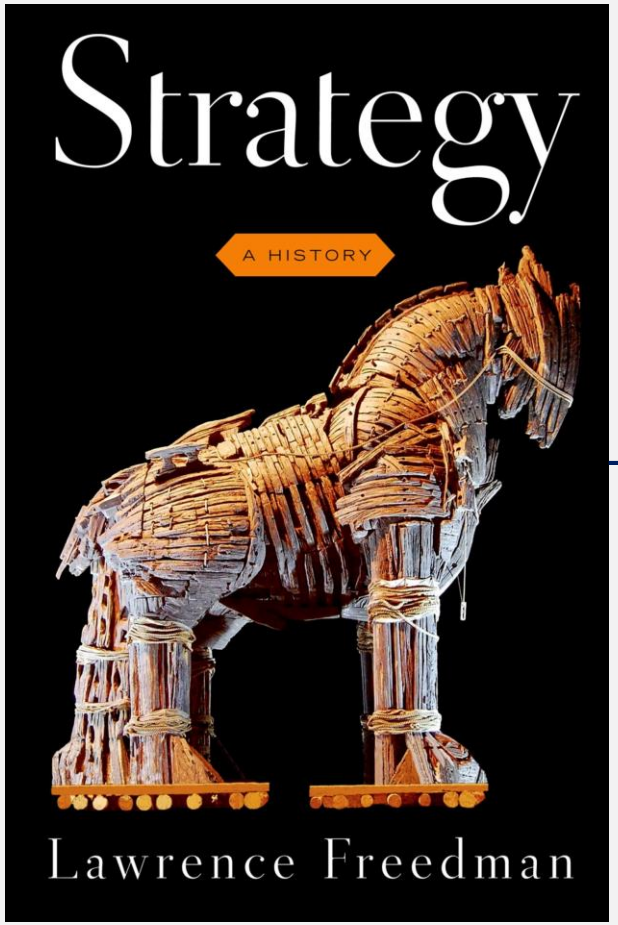


Decentralized R&D enables focus at the level of each asset driving value (Biotech)

Scale and economies of learning drive value in a commercial organization (consolidation)

What does a sustainable genetic medicine innovation ecosystem look like? Criteria #2

Criteria #2: Each program needs to be NPV positive and supported by beautiful science



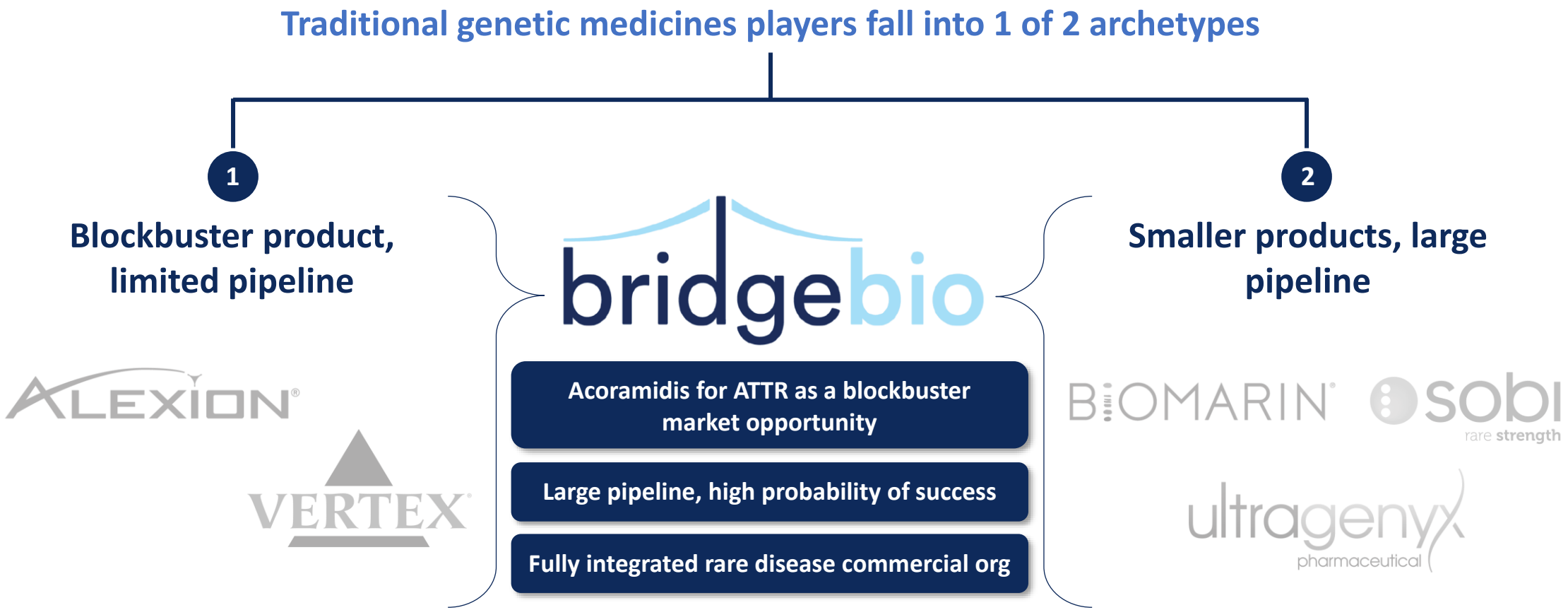
- Each program is NPV positive
  - **Realistic market size estimates**
    Only 2.3% of brands today >$2 Bn
  - **Capital efficient**
    IND cost < $15 Mn for small molecule
- Beautiful science
  - **High POTS programs**
    More like engineering, less biology
  - **Product market fit**
    Therapies which match patient need

Source: Evaluate Pharma

BridgeBio satisfies the criteria of a sustainable genetic medicine innovation engine

Key criteria of a sustainable genetic medicine innovation engine	Key attributes of BridgeBio
Criteria #1	▪ **The willingness and scale to fail** and to re-allocate capital, within a de-centralized company model
	▪ **Focus at the level of individual diseases and assets.** Drug R&D is a game of details
Criteria #2	▪ **Distinctive early-stage asset selection,** based on a deep understanding of clinical unmet need, genetics, and underlying molecular pathophysiology
	▪ **Efficient corporate structure** that cuts no corners on science and medicine, but limits G&A, infrastructure and needless management
People	▪ **Experienced, product-focused R&D leadership** that can define go / no-go's, required product attributes, and can drive programs through the clinic efficiently

We are building the next great genetic medicine company



Fingerprints of hope #1 | BridgeBio is one of the most efficient and productive biotech companies in the genetic medicine space

In less than 6 years since inception, BridgeBio has delivered…

2 Nulibry (fosdenopterin) for injection; TRUSELTIQ™ (infigratinib) capsules
APPROVED PRODUCTS

30+
ACTIVE R&D PROGRAMS ACROSS 4 MODALITIES

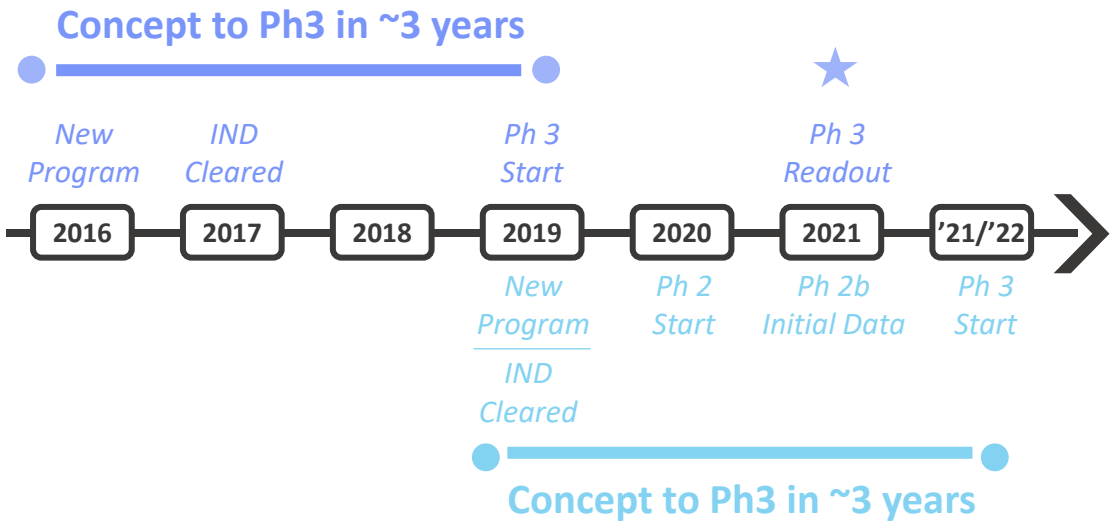
20
ONGOING CLINICAL TRIALS ACROSS >450 SITES

460
FULL-TIME EMPLOYEES

15
INDs (AVERAGE ~3 PER YEAR)

Select Programs:

ATTR



ADH1

…building the framework for efficient, repeatable results

Fingerprints of hope #2 | Leadership team of world-renowned drug hunters

Scientific insight and judgment from industry leaders with a proven track record

Charles Homcy, MD
Founder and Chairman of Pharmaceuticals
MyoKardia GBT

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

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

Len Post, PhD
Advisor
BIOMARIN LEAD THERAPEUTICS

Phil Reilly, MD, JD
Advisor
THIRD ROCK VENTURES bluebirdbio

Experienced team of R&D operators responsible for 100+ INDs and 20+ approved products

Mendelian / Cardio-renal

Uma Sinha, PhD
Chief Scientific Officer
GBT PORTOLA PHARMACEUTICALS

Robert Zamboni, PhD
Chemistry
MERCK FROSST

Jonathan Fox, MD, PhD
Chief Medical Officer, Eidos
MyoKardia AstraZeneca

Oncology

Eli Wallace, PhD
Chief Scientific Officer, Oncology
Peloton Therapeutics ARRAY BIOPHARMA

Pedro Beltran, PhD
SVP, Oncology
AMGEN UNITY BIOTECHNOLOGY

Fingerprints of hope #3 | BridgeBio's product platform

Recent Additions

- 4 new databases
- Bayesian methods for precise disease prevalence estimates
- 14 new university partnerships
- >5000 new rare variants, >100 new causal genes discovered

- NMR spectroscopy for new drug targets
- AI for deciphering new protein structures
- Phenotypic screening for largest genetic diseases
- ASO screens for haploinsufficiency diseases

- 4 new clinical trials
- Activated 62 new sites in 11 countries
- Telperian partnership for ML empowered precision analytics
- Science 37 partnership for agile, decentralized clinical trials

- Two commercial launches (MoCD Type A, 2L CCA)
- 95% of lives covered in 6m of NULIBRY launch
- Established a PAP to provide qualified patient's free access
- European office open, LATAM office upcoming

DISCOVER

Computational genomics, systemic disease mapping, broad network of academic partnerships

CREATE

Molecular dynamics assisted chemistry, gene therapy, therapeutic proteins, antisense oligos

TEST

20 ongoing trials across >450 sites and 26 countries, central operations toolkit and analytics

DELIVER

Global infrastructure, diagnostics, patient support, disease state awareness

Fingerprints of hope #4 | BridgeBio's pipeline, including potential best-in-class candidates

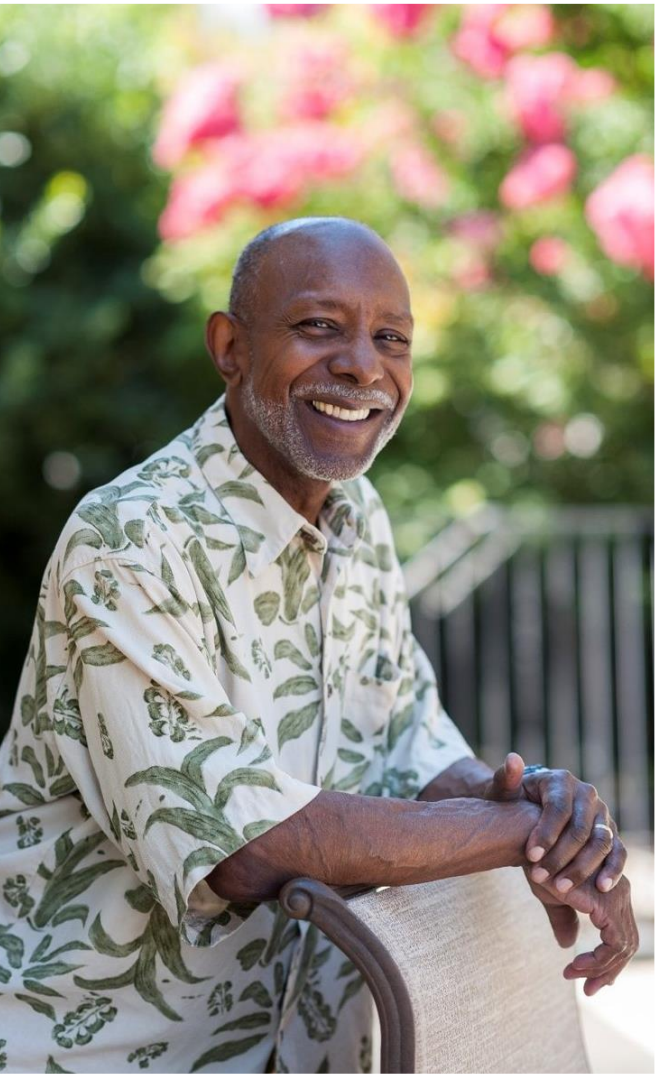
	Indication	Drug Mechanism	Pt. pop. (US+EU)	Discovery	Pre-IND	Phase 1	Phase 2	Phase 3	Approved	Partner
Mendelian	MoCD type A	NULIBRY™ (Synthetic cPMP, fosdenopterin)	100						Nulibry	MEDISON
	Achondroplasia	Low-dose FGFRi (infigratinib)	55k							
	LGMD2i	Glycosylation substrate (ribitol)	7k							
	RDEB	Recombinant COL7 (BBP-589)	2k							
	PKAN / organic acidemia	Pank activator (BBP-671)	7k							
	VM / LM	Topical PI3K inhibitor (BBP-681)	117k							
	Netherton	Topical KLK inhibitor (BBP-561)	11k							
	PTEN autism	PI3Kb inhibitor (BBP-472)	120k							
	4 undisclosed small molecule programs		>500k							
	4 undisclosed antisense oligonucleotide programs		>300k							
Precision Cardiorenal	ATTR amyloidosis	TTR stabilizer (acoramidis)	>400k							AstraZeneca
	ADH1	CaSR antagonist (encaleret)	12k[1]							
	PH1 / frequent stone formers	GO1 inhibitor (BBP-711)	5k / 1.5m							
	Undisclosed DCM small molecule program		>250k							
	Undisclosed DCM AAV gene therapy program									
Precision Oncology	FGFR2+ cholangiocarcinoma (2L)	TRUSELTIQ™ (FGFRi, infigratinib)	4k						TRUSELTIQ	
	FGFR2+ cholangiocarcinoma (1L)	FGFRi (infigratinib)								HELSINN
	FGFR3+ adjuvant urothelial	FGFRi (infigratinib)	21k							
	FGFR1-3+ tumor agnostic	FGFRi (infigratinib)	24k							
	FGFR1-3+ gastric cancer	FGFRi (infigratinib)	41k[2]							
	MAPK / RAS-driven cancer	SHP2i monotherapy (BBP-398)	>500k							
		SHP2i combo therapy (BBP-398)								Bristol Myers Squibb
	KRAS-driven cancer	KRAS G12C dual inhibitor	>500k							
		PI3Kα:RAS Breaker								
		KRAS G12Di								
	Solid tumors	GPX4i	>500k							HELSINN
Gene Therapy	CAH	AAV5 gene therapy (BBP-631)	>75k							
	Canavan	AAV9 gene therapy (BBP-812)	1k							
	TMC1 hearing loss	AAV gene therapy (BBP-815)	2k							
	Galactosemia	AAV gene therapy (BBP-818)	>7k							
	TSC1/2	AAV gene therapy	>100k							
	Cystinuria	AAV gene therapy	20k							
	3 capsid discovery collaborations									

[1]US carriers
[2]China + Japan patient population

BridgeBio's endless summer

MoCD Type A	ATTR-CM/PN	PKAN/OA	KRAS
2L CCA	ADH1	PH1/FSF, VM	ALS, Autism
	Achon	SHP2, UC, RDEB	CF, A1AT, GALT
	CAH	LGMD2i, Canavan	TMC1, TSC1/2

Acoramidis for transthyretin (TTR) amyloidosis (ATTR)



Len
Living with ATTR-CM

Prevalence

400k+

Worldwide

Pathophysiology

Systemic disease most commonly presenting as cardiomyopathy or peripheral neuropathy

Genetic Driver

Destabilized TTR leading to amyloid accumulation

Therapeutic Hypothesis

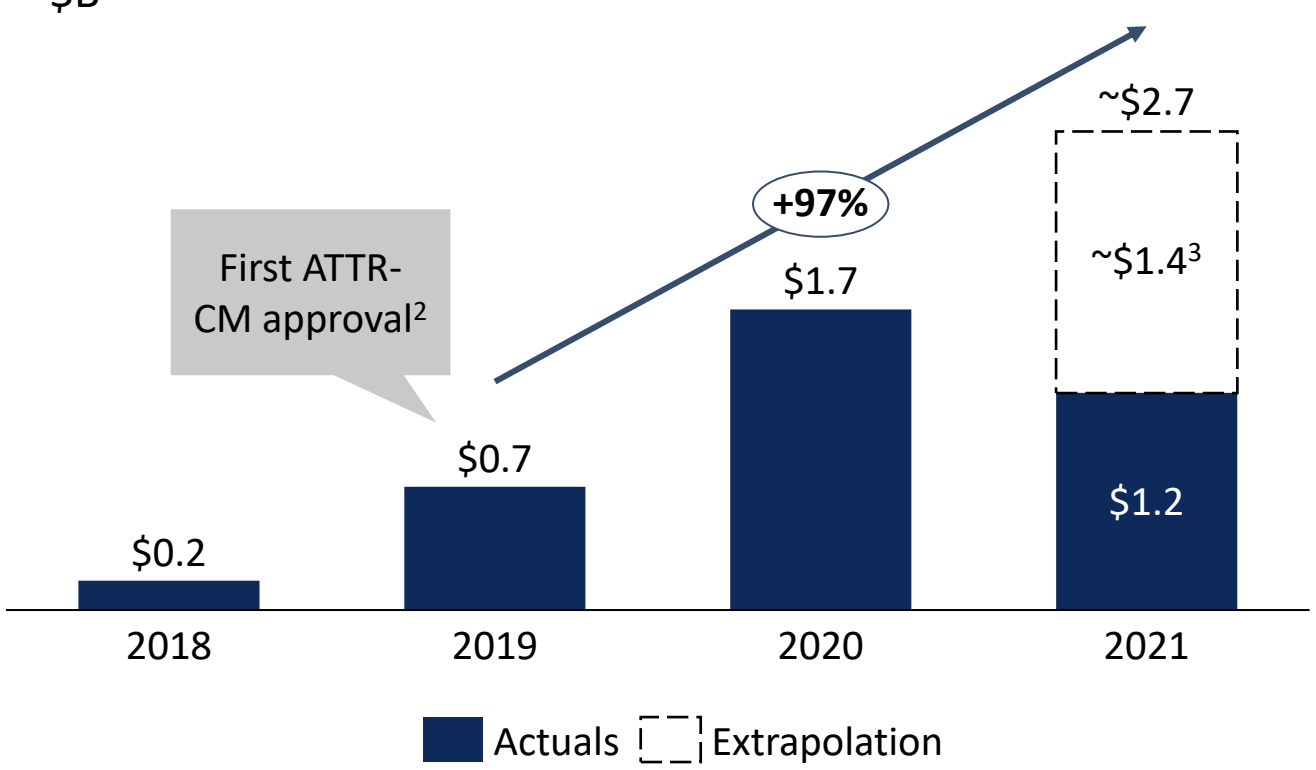
TTR stabilizer designed to mimic protective T119M mutation

Design Criteria for Optimal Therapy

- Near-complete stabilization of TTR
- Preservation of TTR tetramer
- Oral Dosing

Following first ATTR-CM approval in 2019, ATTR has become a $2B+ market with substantial remaining upside

Global annual ATTR market sales[1]



Dramatic ATTR market growth driven by:

- Increasing diagnosis in established geographies *(~27K ATTR-CM US patients currently diagnosed vs. <5k before first approval[4])*
- Launch and patient finding in new geographies

[1]ATTR market includes all approved drugs for ATTR-PN and ATTR-CM
[2]First ATTR-CM sales occurred in Q2 2019
[3]Assumes Q1 '21 – Q2 '21 growth flatlined for 2H 2021
[4]Pfizer press release

Acoramidis was designed to treat ATTR at its source

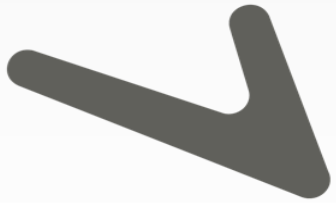
Disease mechanism

Native TTR circulates in blood as a tetramer

~130 known destabilizing mutations

Protective T119M mutation

Dissociation into monomers initiates pathogenesis



Monomers aggregate, causing disease

Therapeutic hypothesis

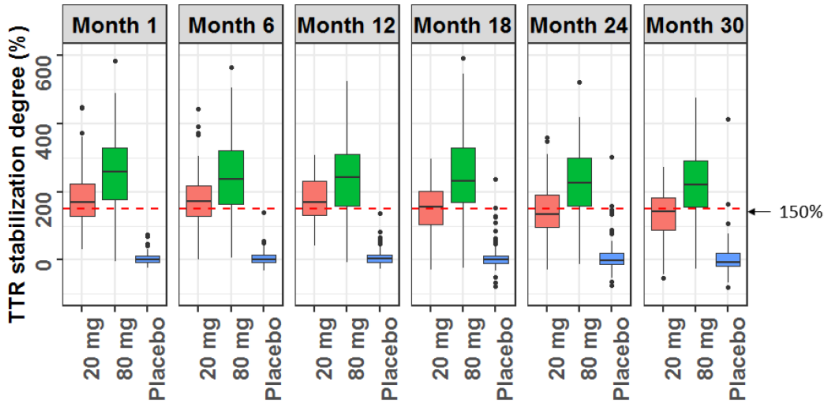
Acoramidis was designed to mimic protective T119M mutation by stabilizing TTR tetramers to slow or halt disease progression

Higher dose of tafamidis demonstrated increased TTR stabilization and greater clinical benefit in ATTR-ACT + LTE

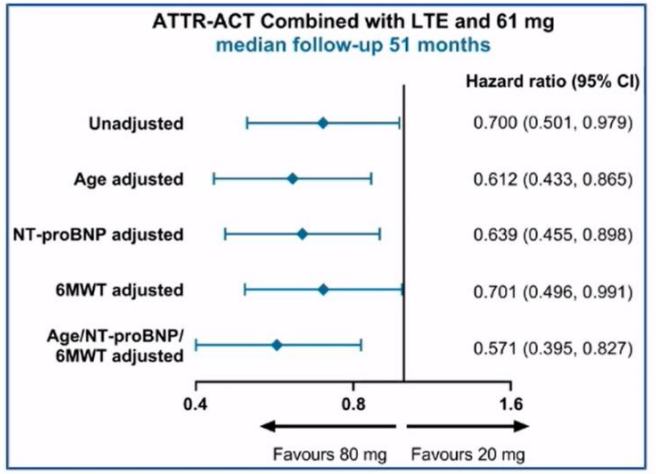
Phase 3 ATTR-ACT study tested two doses of tafamidis (20 mg & 80 mg) vs. placebo

- In an analysis of ATTR-ACT combined with long-term extension (LTE), benefit of tafamidis 80 mg vs. 20 mg was evident on all-cause mortality[1]
- At baseline, ATTR-ACT participants treated with 80 mg of tafamidis were older and had more severe evidence of disease than those treated with 20 mg of tafamidis[1]
- Participants receiving 80 mg of tafamidis (vs. 20 mg) exhibited greater TTR stabilization[2]

TTR stabilization[2]



All-cause mortality[1]



Increased levels of TTR stabilization may translate to improved clinical outcomes in ATTR-CM

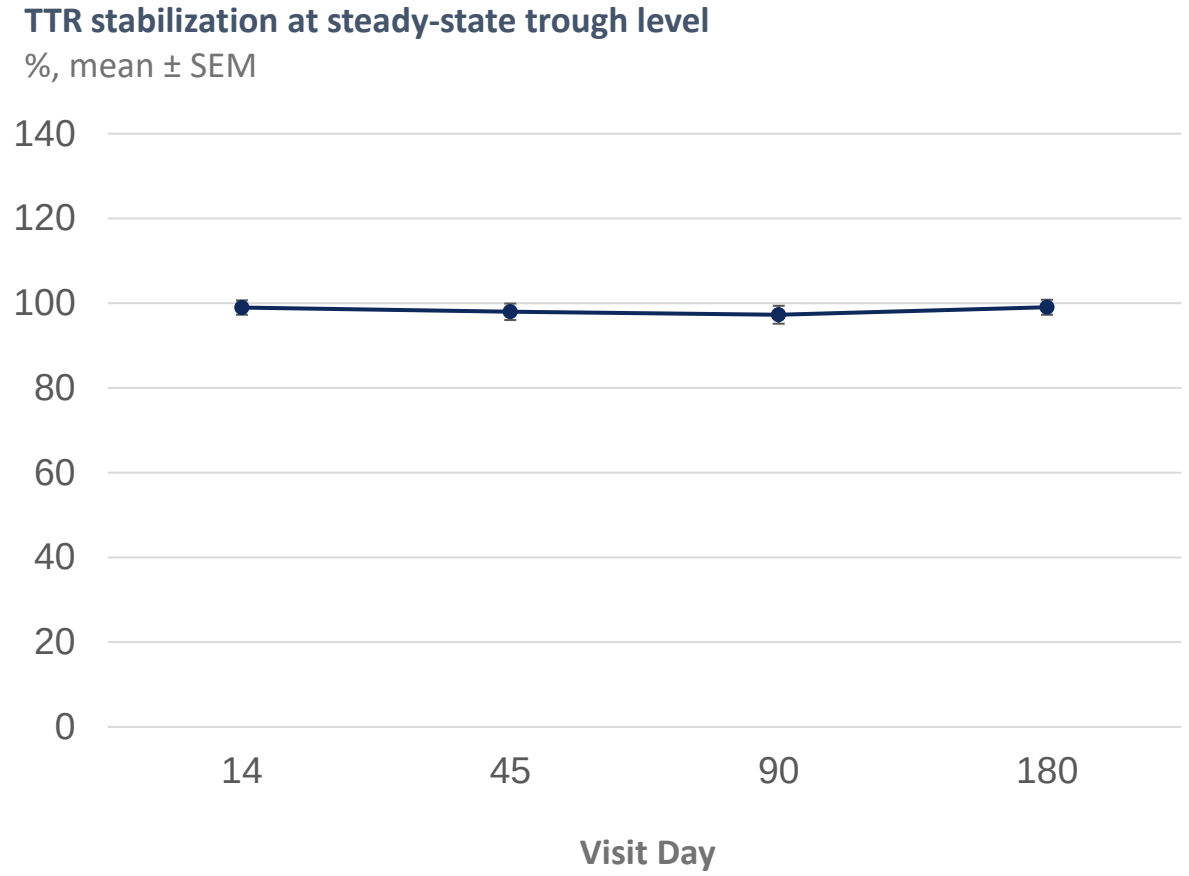
[1]Damy, T., ESC Heart Failure Association Discoveries 2020. "The Tafamidis in Transthyretin Cardiomyopathy Clinical Trial"
[2]FDA CDER Clinical Pharmacology and Biopharmaceutics, Clinical Review (Vyndaqel/Vyndamax), 2019; Fourfold increase in tafamidis dose did not lead to a fourfold increase in TTR stabilization due to non-linear pharmacokinetics

Acoramidis has been well-tolerated and demonstrated near-complete TTR stabilization in preclinical, Phase 1, and Phase 2 studies

Phase 2 safety summary[1]

	Placebo N = 17	Acoramidis (pooled doses) N = 32
Any Adverse Event	**15 (88%)**	**21 (66%)**
Mild	6 (35%)	11 (34%)
Moderate	8 (47%)	9 (28%)
Severe	1 (6%)	1 (3%)
Any Serious Adverse Event	**2 (12%)**	**1 (3%)**
AF and CHF	1 (6%)[1]	0
Leg cellulitis	1 (6%)	0
Dyspnea	0	1 (3%)

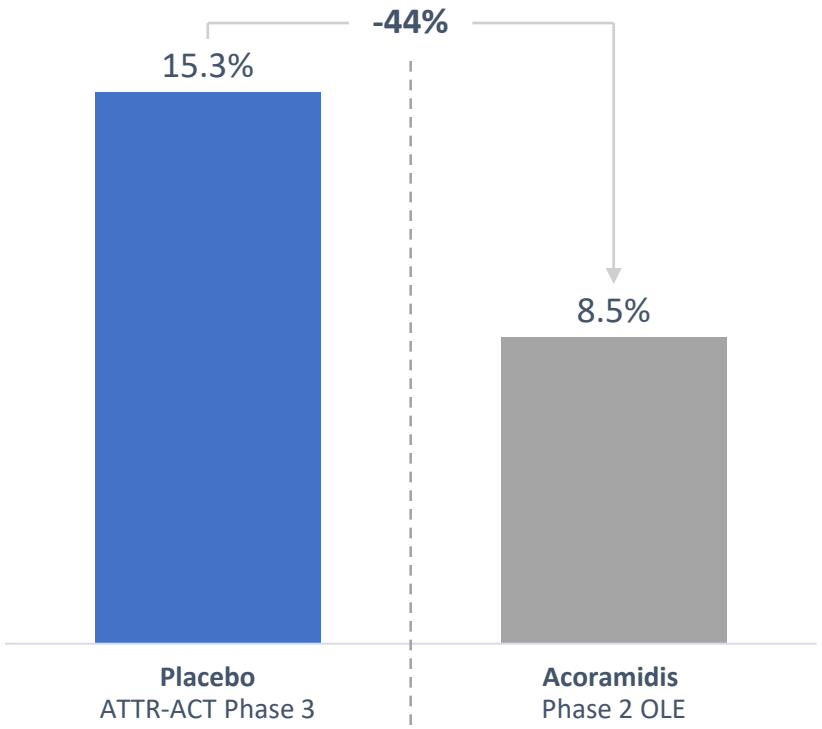
Phase 2 TTR stabilization[2]



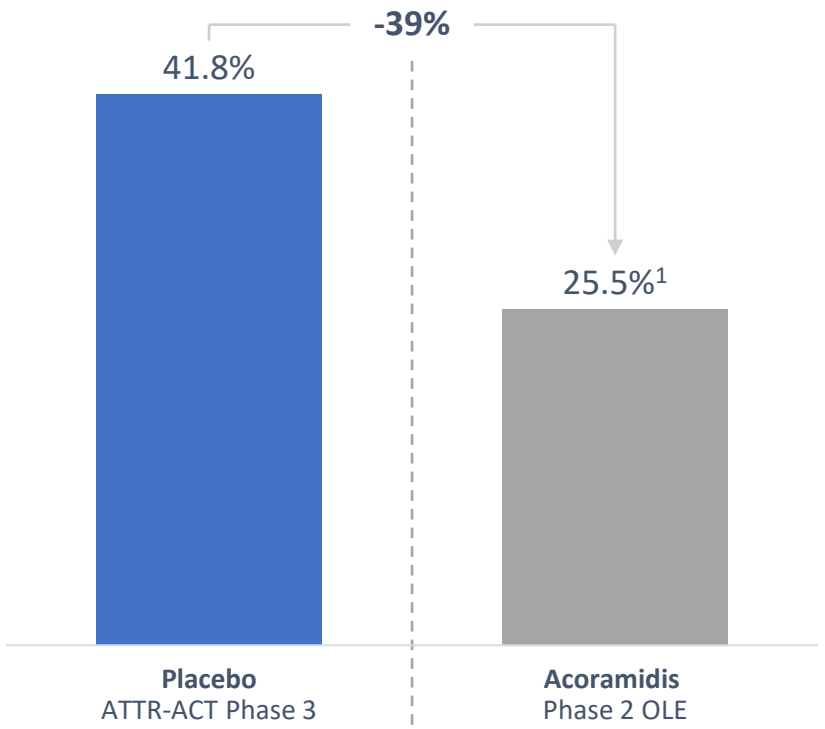
[1] Judge, D.P. et al., JACC Vol. 74, No. 3, 2019:285 – 95
[2] Judge, D.P. et al., American Heart Association 2019

Deaths and CV hospitalizations reported in acoramidis Phase 2 OLE were lower than in placebo-treated ATTR-ACT participants

All-cause mortality at 15 months
Participants died or receiving transplant (%)

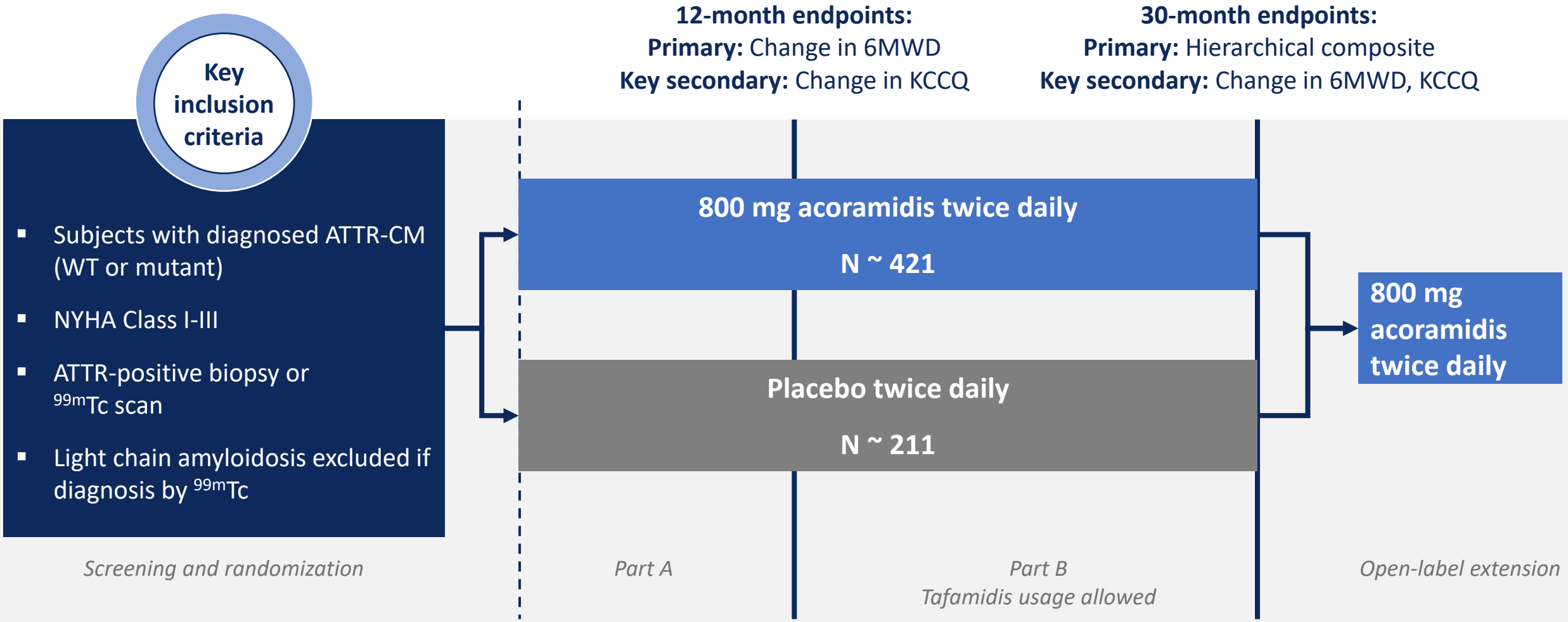


Cardiovascular hospitalizations at 15 months
Participants with ≥1 CV hospitalization (%)



[1]Based on routine adverse event reporting
Note: These data are based on a cross-trial comparison and not a randomized clinical trial. As a result, the values shown may not be directly comparable
Source: Judge, DP et al., American Heart Association Scientific Sessions 2019

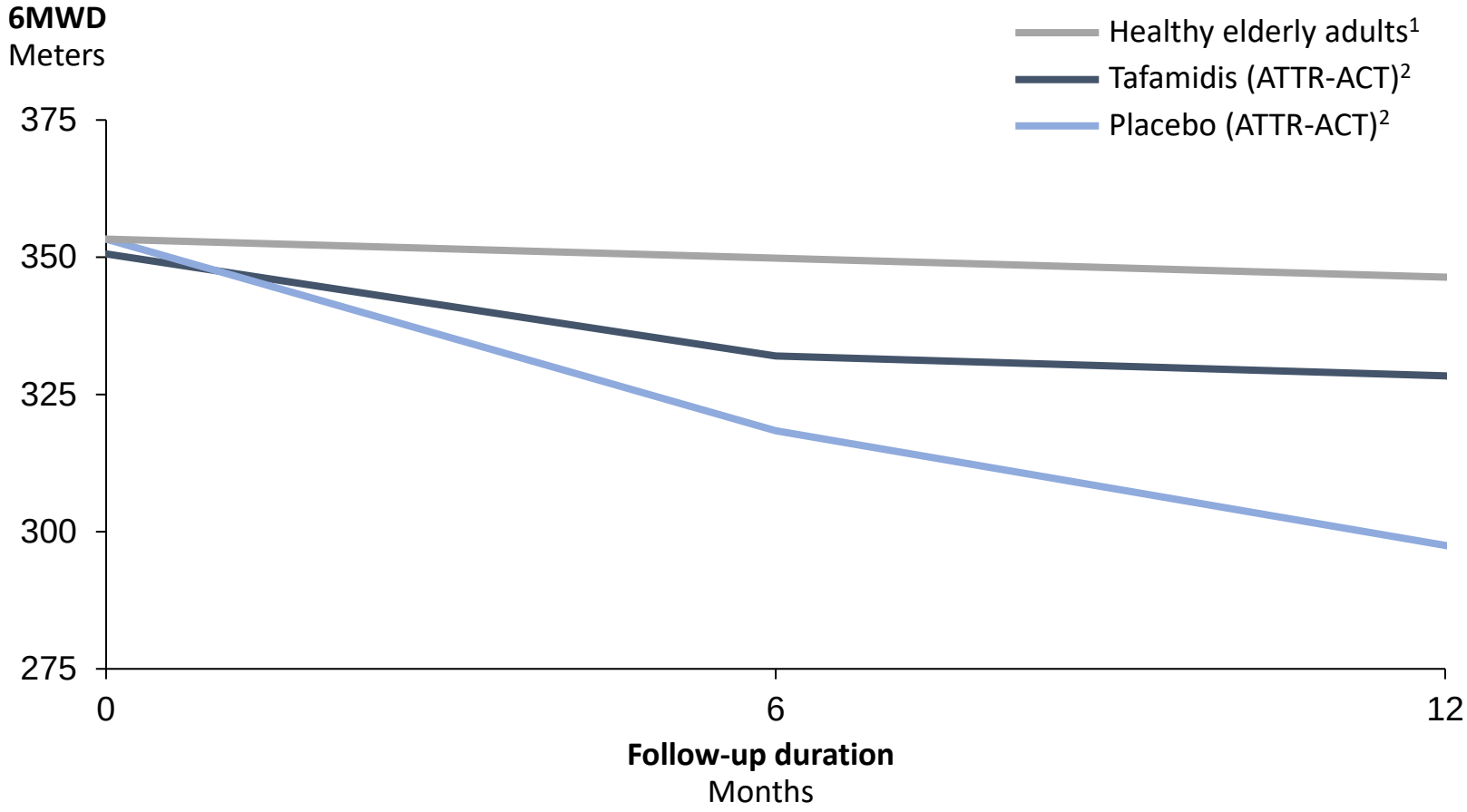
ATTRibute-CM will provide 12-month functional outcome data and 30-month mortality and CV hospitalization data



6MWD = Six-minute walk distance KCCQ = Kansas City Cardiomyopathy Questionnaire NYHA = New York Heart Association
^{99m}Tc = Technetium labeled pyrophosphate (PYP) or bisphosphonate (e.g., DPD)
CV = cardiovascular-related

Rapid functional decline in untreated ATTR-CM patients provides opportunity to demonstrate robust clinical benefit

Summary of six minute walk distance (6MWD) in ATTR-CM and healthy cohorts



Approximate annual decline:

Healthy elderly adult: -7m

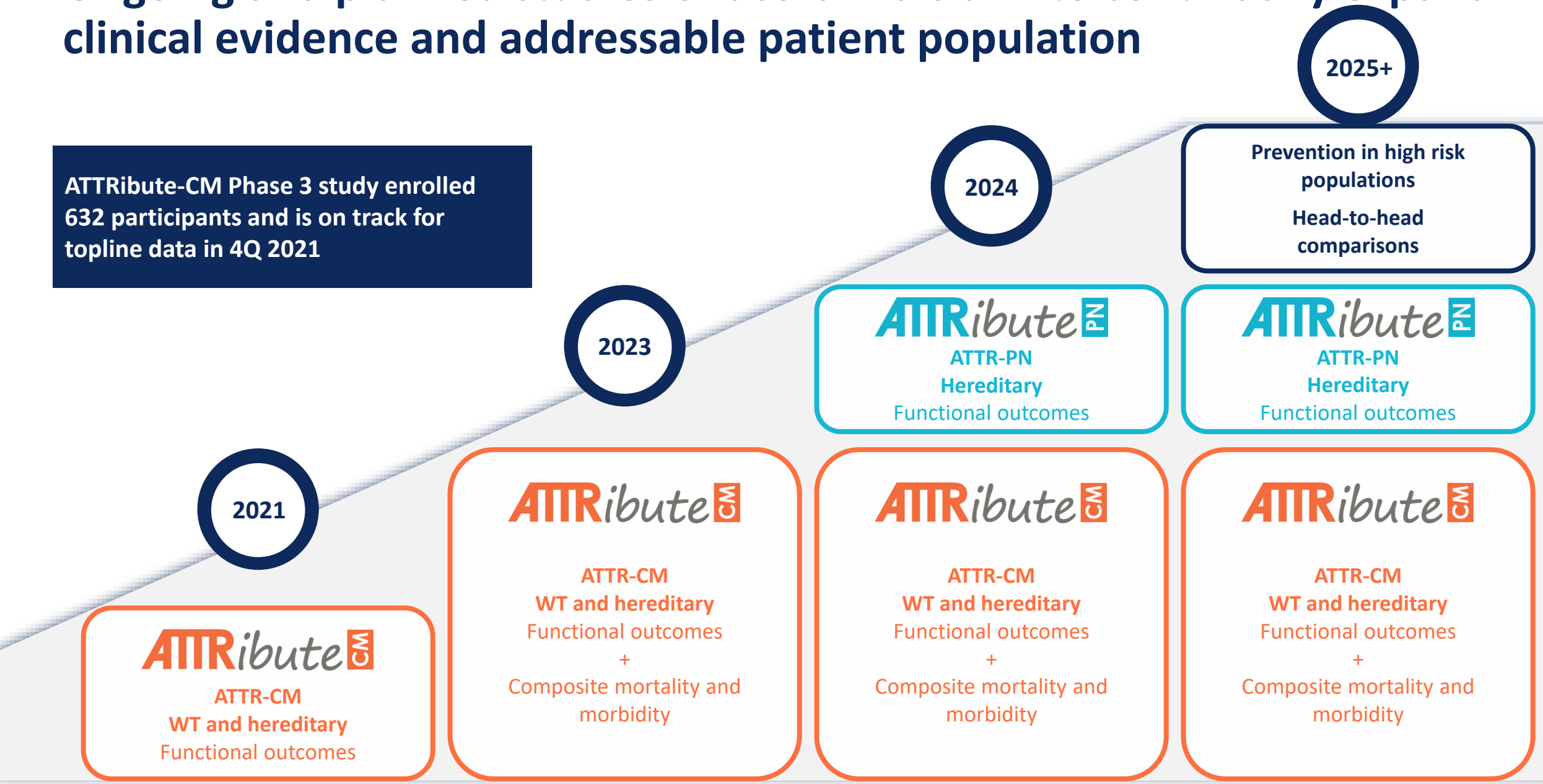
ATTR-ACT (tafamidis): -25m

ATTR-ACT (placebo): -56m

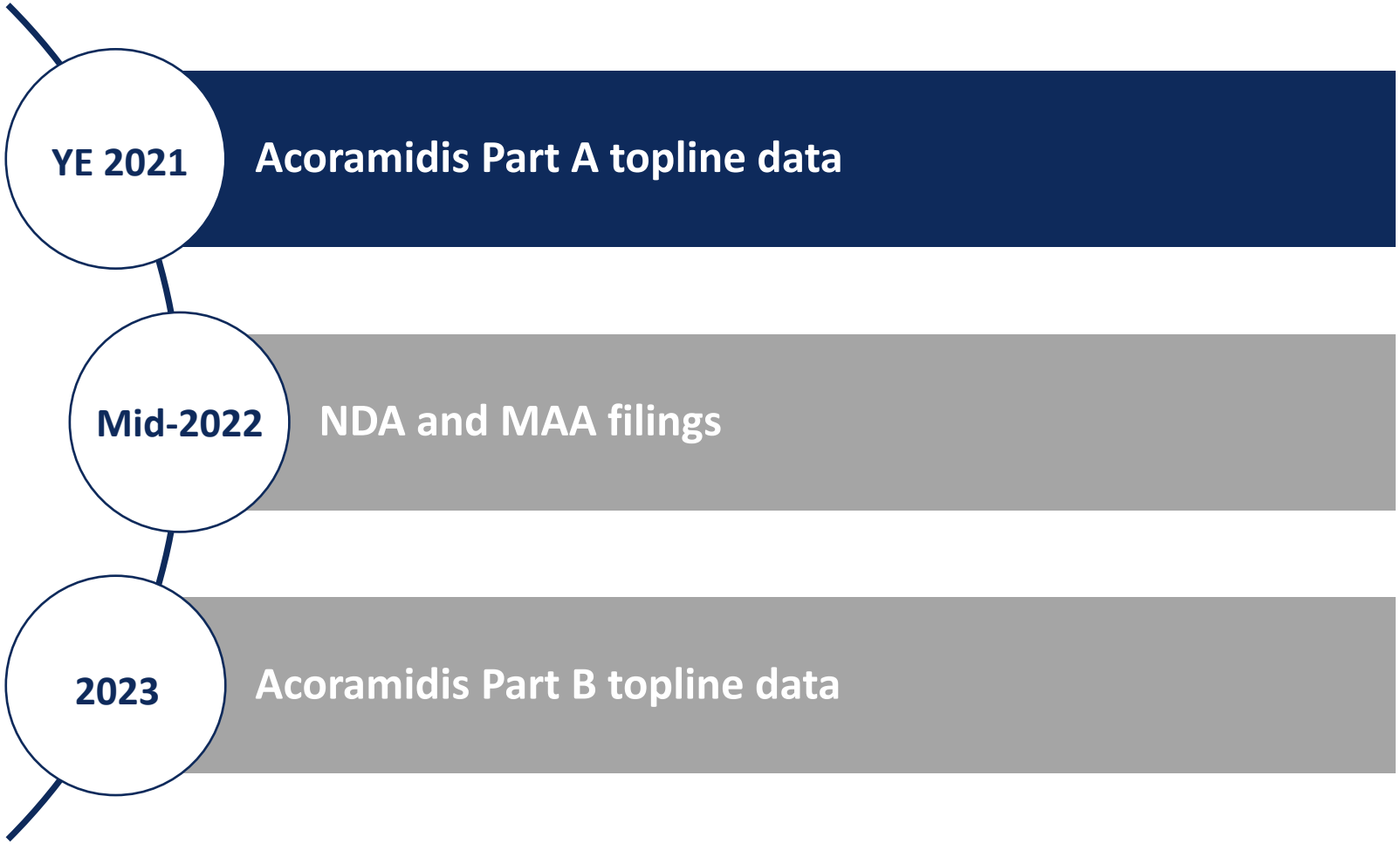
[1]Enright, P.L. et al. Chest 2003. N = 3333 healthy elderly adults, baseline set to match ATTR-ACT placebo group
[2]Maurer, M.S. et al. NEJM 2018. N = 264 (tafamidis), N = 177 (placebo) ATTR-CM trial participants

Ongoing and planned studies of acoramidis aim to continually expand clinical evidence and addressable patient population

ATTRibute-CM Phase 3 study enrolled 632 participants and is on track for topline data in 4Q 2021



Timeline of upcoming milestones



Encaleret for autosomal dominant hypocalcemia type 1 (ADH1)

Alexis and Jackson
Living with ADH1

Prevalence

12k+

US

Pathophysiology

Decreased blood calcium, elevated urine calcium, and lower parathyroid hormone secretion

Genetic Driver

Hyperactivation of calcium-sensing receptor (CaSR)

Therapeutic Hypothesis

Selectively antagonize CaSR to normalize downstream effects

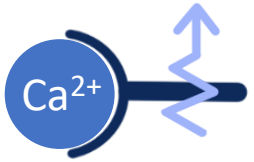
Design Criteria for Optimal Therapy

- Directly target CaSR to potentially resolve key symptoms
- Phase 2 data suggests potential to normalize blood Ca and urine Ca
- Oral Dosing

ADH1-causing variants hyperactivate the CaSR and disrupt calcium homeostasis leading to potentially life-threatening symptoms

Disease Mechanism

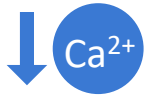
Normal CaSR senses and regulates serum Ca levels to maintain calcium homeostasis



ADH1 CaSR is hyperactive

Hyperactive CaSR causes dysregulation of calcium homeostasis

Decreased *PTH secretion*



Decreased *serum calcium*

Increased *urine calcium*

Clinical Manifestation

Presenting symptoms

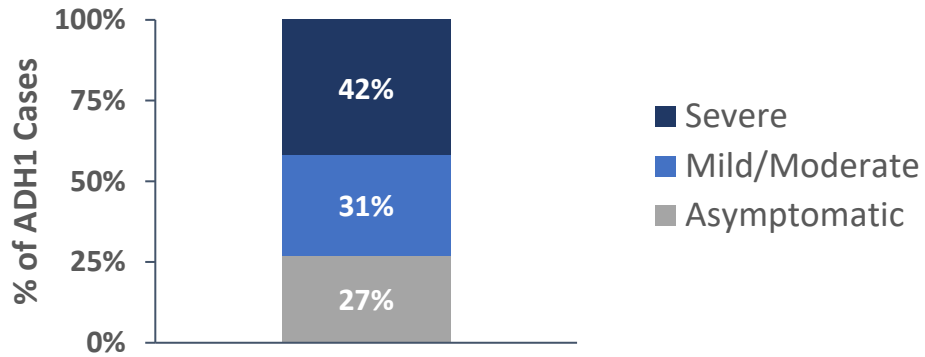
- Hypocalcemic seizures
- Paresthesia
- Tetany
- Muscle cramps

Long-term complications

- Nephrocalcinosis
- Nephrolithiasis

Median age of ADH1 dx[1]: 25 (0-77) years

Symptom presentation[1]

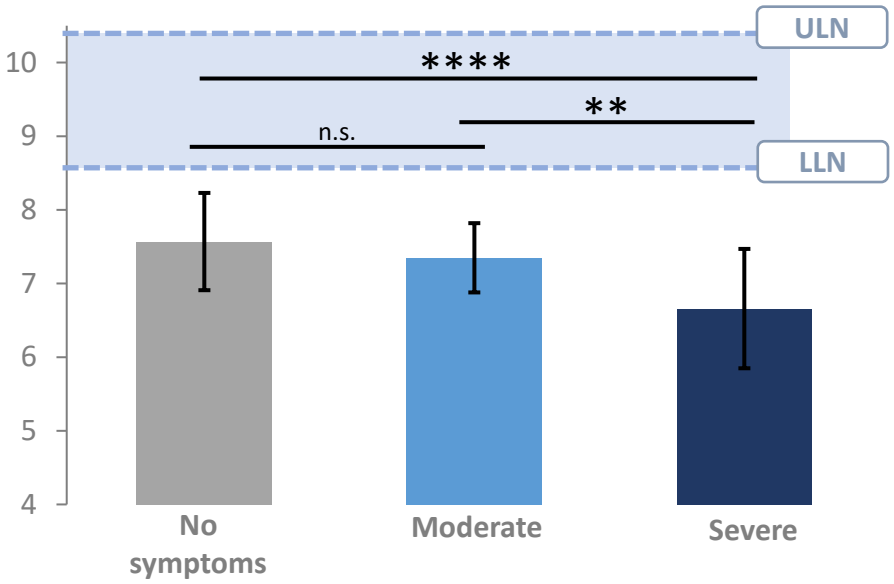


[1]Roszko, et al., ASBMR Annual Meeting, 2021. Abbreviations: dx = diagnosis. Age of dx presented as median (range)

ADH1 symptom severity is associated with blood calcium levels and current treatment inadequately addresses symptom burden

Blood calcium at clinical presentation

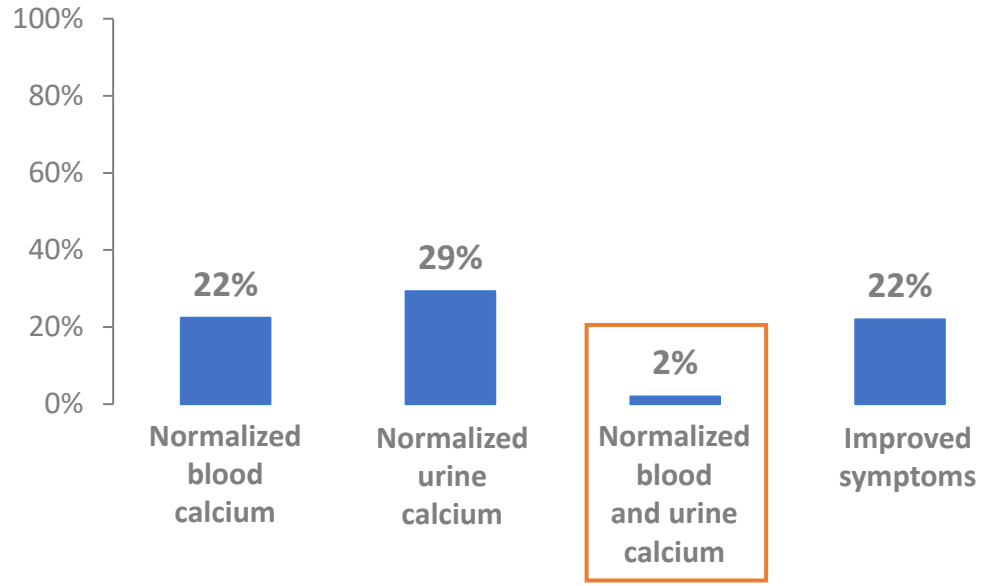
Blood corrected calcium
mg/dL, mean



Severely symptomatic individuals exhibited significantly lower blood calcium compared to asymptomatic and moderately symptomatic[1]

ADH1 medical intervention

Individuals on calcium and/or active vitamin D
%

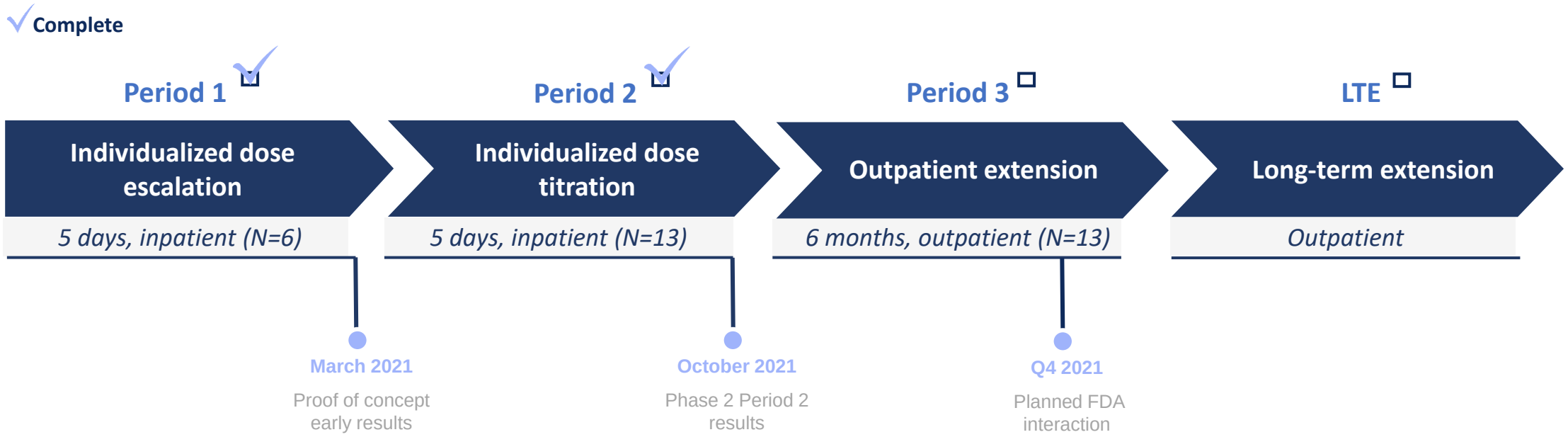


Only 2% of individuals normalized both blood and urine calcium, and only 22% reported symptom improvement on-treatment[1]

ULN = upper limit of normal, LLN = lower limit of normal. ** p-value <0.01. **** p-value < 0.0001. n.s. = not statistically significant

[1]Roszko, et al., ASBMR Annual Meeting, 2021

Encaleret Phase 2 study design



Key study objectives:

- Safety and tolerability
- Blood calcium concentration
- Urine calcium concentration
- Intact parathyroid hormone concentration

Additional measures:

- Blood 1,25-$(OH)_2$ Vitamin D, magnesium, and phosphate
- Urine creatinine, cAMP, citrate, phosphate, sodium, magnesium
- Bone turnover markers (serum collagen C-telopeptide, serum procollagen Type 1 N-propeptide)

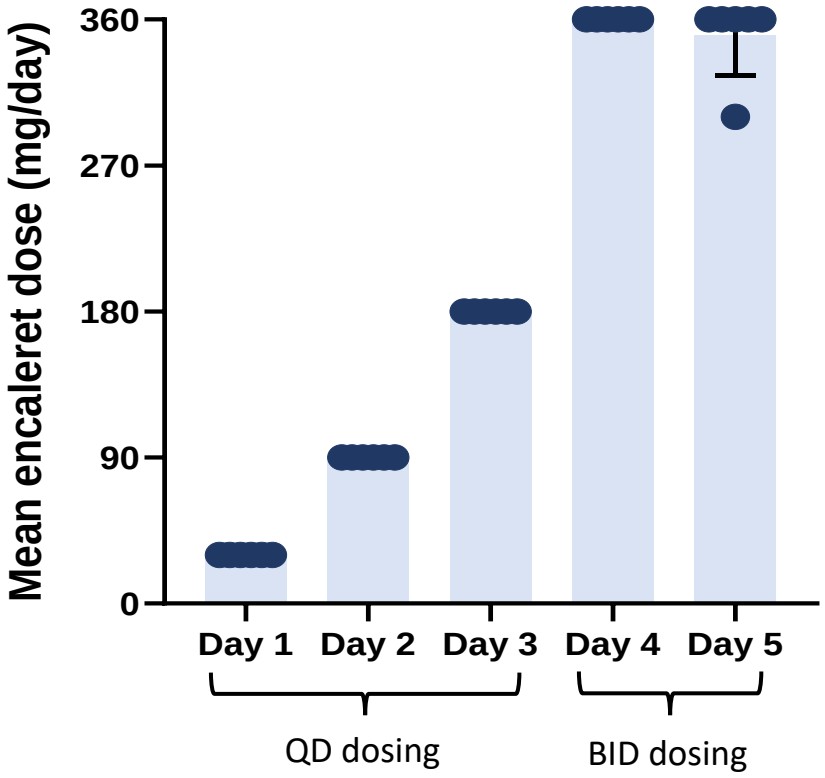
Period 2 individualized dose titration phase resulted in a lower Day 5 mean encaleret dose as compared to Period 1

Period 1 and Period 2 encaleret dosing summary

Period 1 Dosing

Defined dose escalation

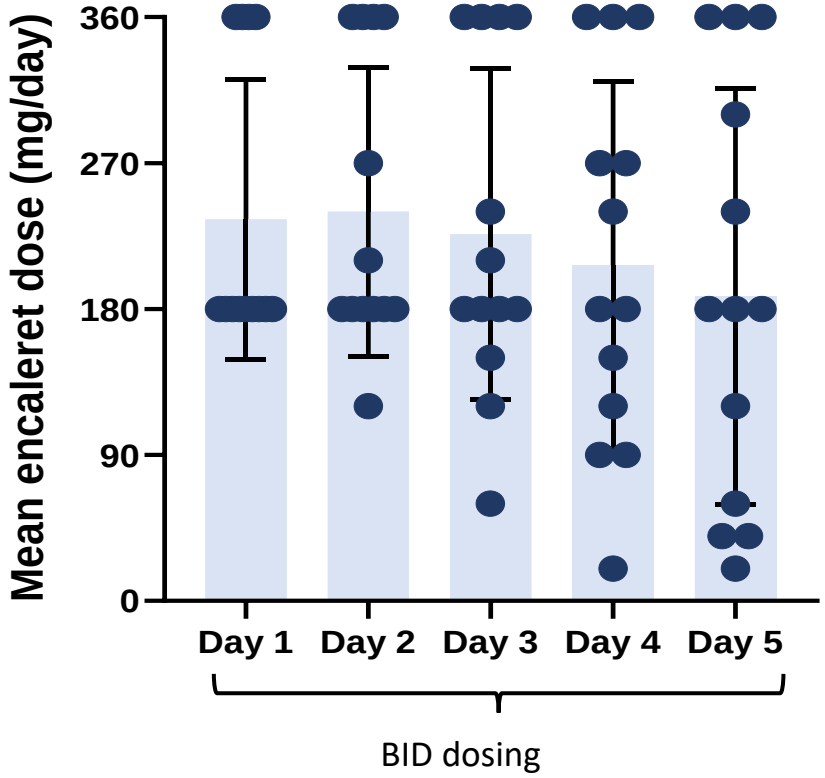
Day 5 Mean: 350.0±22.4 mg/day



Period 2 Dosing

Individualized dose titration

Day 5 Mean: 187.7±128.2 mg/day



Data reported as mean±SD.

Study participants exhibited hypocalcemia, elevated urine calcium, suppressed PTH, and elevated phosphate at baseline

Characteristic	Study Population N = 13	*Normal Range*
Age, mean, yr (range)	39 (22-60)	
Female, n (%)	8 (62%)	
Nephrocalcinosis, n (%)	10 (77%)	
ECG QT_cB (msec)	452 ± 16	*< 440*
Corrected Calcium (mg/dL)*	8.0 ± 0.7	*8.4 –10.2*
Intact PTH (pg/mL)*	2.8 ± 3.4	*15 – 65*
Phosphate (mg/dL)*	5.1 ± 1.1	*2.3 – 4.7*
Magnesium (mg/dL)*	1.8 ± 0.1	*1.6 – 2.6*
24h Urine Calcium (mg/24h)	441 ± 258	*< 250-300*
Supplements		
Elemental Calcium (mg/day) [mean (range)]	2628 (750-4800)	
Calcitriol (µg/day) [mean (range)]	0.8 (0.2-2.0)	
***CASR* Variants**	C131Y (2), P221L (2), E604K (1), A840V (3), F788C (1), T151M (1), Q245R (1), I692F (1), E228K (1)	

Data reported as mean±SD. ECG QTcB = electrocardiogram Bazett-corrected Q-T interval. *Measurements taken pre-dose Day 1 in Period 1 or Period 2

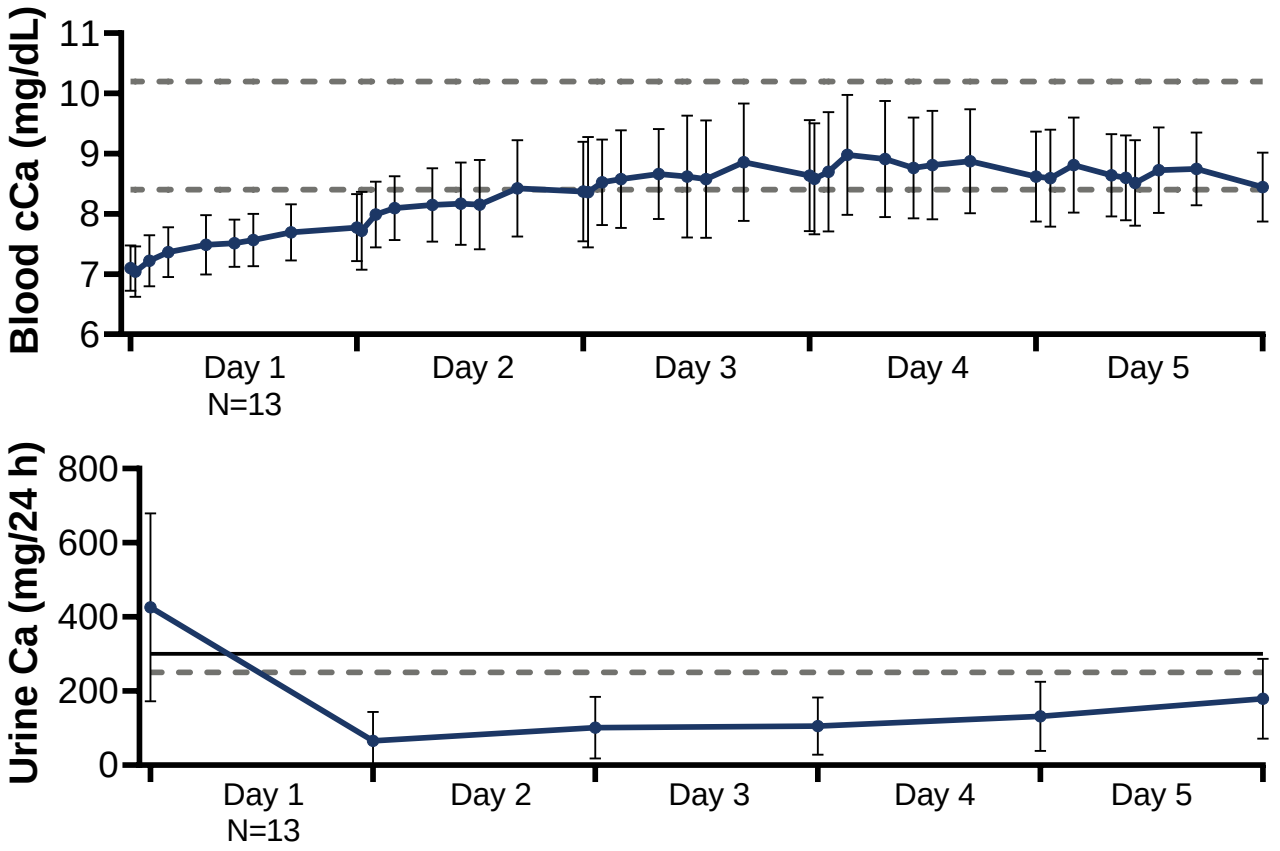
Encaleret continues to be generally well-tolerated with no serious adverse events reported[1]

	Period 1 N = 6	Period 2 N=13
Number of subjects experiencing any Serious Adverse Event	**0 (0%)**	**0 (0%)**
Number of subjects experiencing any Adverse Event	**6 (100%)**	**10 (77%)**
Mild	6 (100%)	10 (77%)
Moderate	1 (17%)	0 (0%)
Severe	0 (0%)	0 (0%)
Number of Adverse Events Reported	**19**	**12**
Mild	18 (95%)	12 (100%)
Moderate	1 (5%)	0 (0%)
Severe	0 (0%)	0 (0%)
Treatment-related Adverse Events[2]	**3 (16%)**	**8 (67%)**
Hypocalcemia	1 (33%)	0 (0%)
Hypophosphatemia	2 (67%)	7 (88%)
Hypercalcemia	0 (0%)	1 (12%)

[1]Data as of September 3, 2021. [2]Treatment-related adverse events were transient and resolved with dose-adjustment. Treatment-related AEs were counted as the number of events per period and are presented as a percentage of the total number of AEs. The most common AEs (≥ 2 subjects) were hypophosphatemia, hypocalcemia, and headache

Encaleret treatment normalized mean blood and urine calcium during Period 2

Mean responses on Day 1 through Day 5 in Period 2 (N=13)

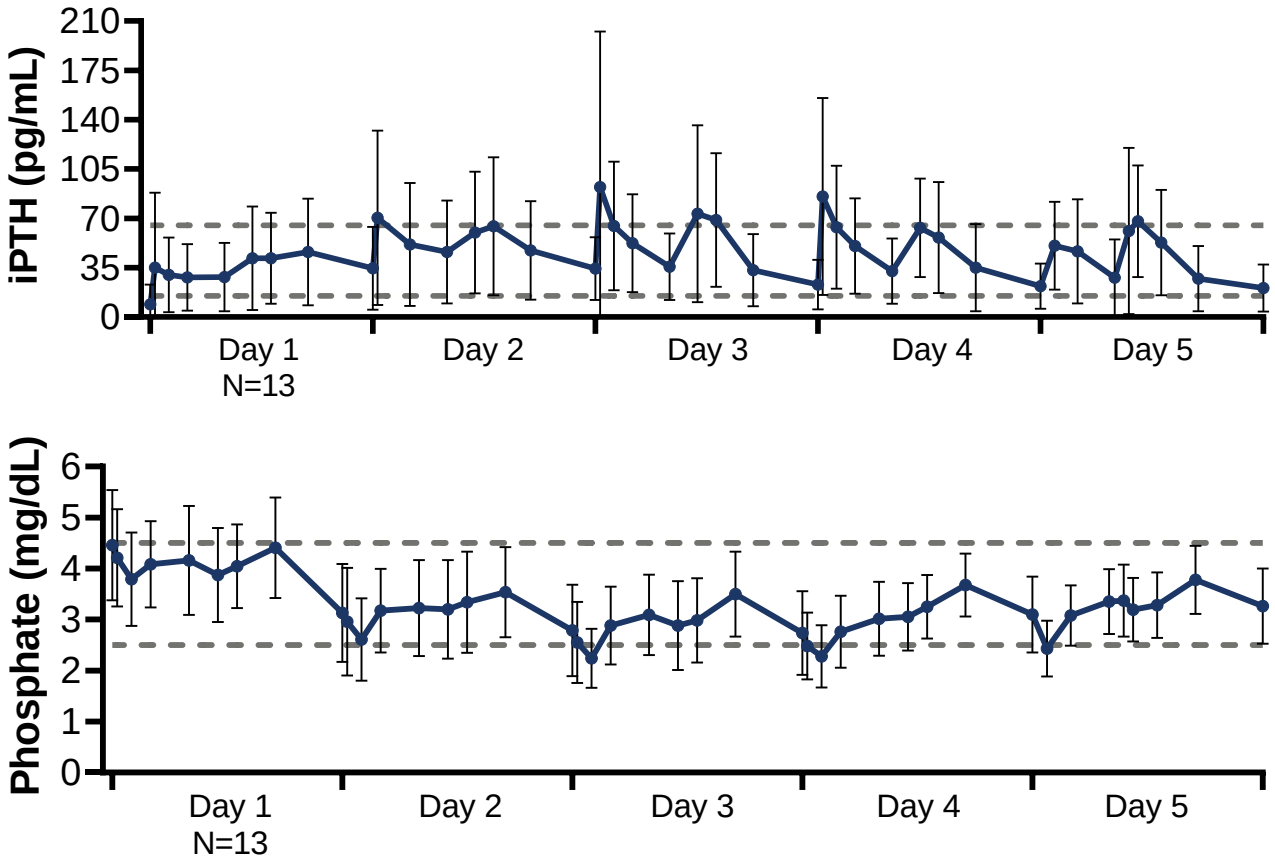


Increasing urine calcium is likely due to both increasing corrected calcium and decreasing encaleret dose

Data reported as mean±SD. Values below limit of assay quantitation recorded as "0". Gray shading reflects normal range. Solid line for urine calcium reflects the upper limit for men and dashed line reflects upper limit for women

Encaleret increased PTH and decreased mean blood phosphate during Period 2

Mean responses on Day 1 through Day 5 in Period 2 (N=13)



Data reported as mean±SD. Values below limit of assay quantitation recorded as "0". Gray shading reflects normal range

Summary reported Phase 2 data and next steps

Summary of encaleret development program

- ☑ In 13 participants, encaleret normalized mean blood calcium and 24-hour urine calcium excretion, increased PTH, and decreased phosphate into the normal range during both Periods 1 and 2
- ☑ Individualized BID dosing in Period 2 resulted in a decrease in the mean Day 5 encaleret dose as compared to Period 1
- ☑ Encaleret was well-tolerated when administered once or twice daily over 5 days, with no serious adverse events reported
- ☑ Consistent improvements in mineral homeostasis suggest encaleret may become an effective treatment for ADH1
- ☑ Granted Fast Track Designation and Orphan Drug Designation by the FDA

Next 12 months

- ☐ Interact with FDA
- ☐ Present complete Phase 2 data
- ☐ Initiate Phase 3 registrational study

Planned activities

- Pediatric development program in ADH1
- Evaluation of encaleret in non-genetic hypoparathyroidism

Low-dose FGFR inhibitor (infigratinib) for achondroplasia

Miguel
Living with achondroplasia

Prevalence

55k+

US & EU

Pathophysiology

Up-regulation of STAT1 and MAPK in the growth plate cause cranial, spinal, and stature symptoms

Genetic Driver

Gain of function of FGFR3

Therapeutic Hypothesis

Low dose inhibition of FGFR3

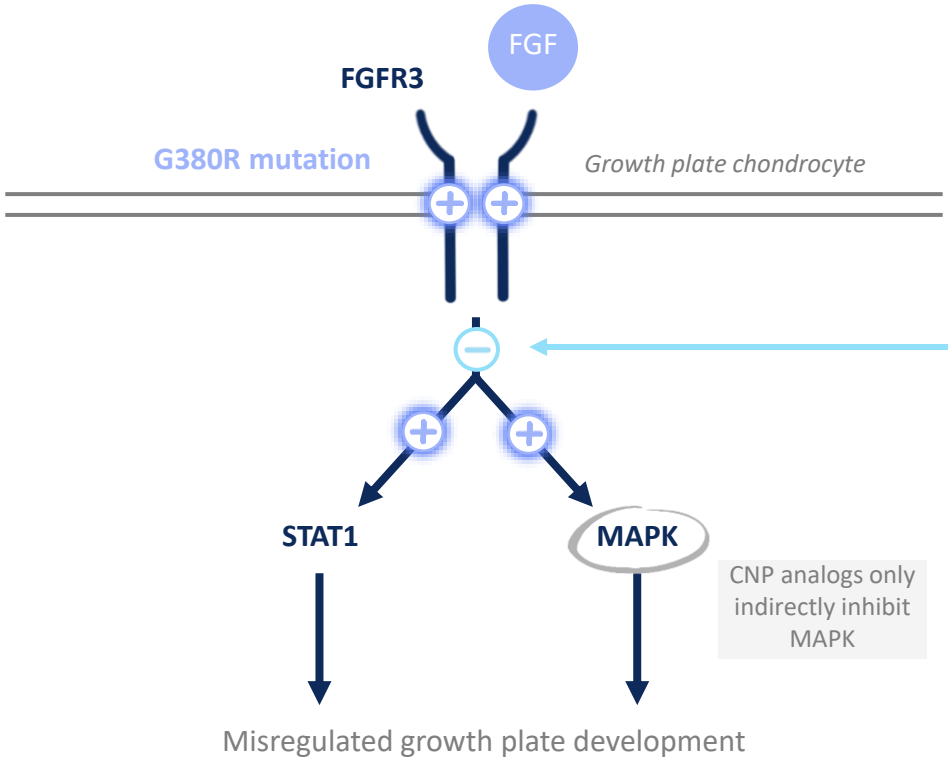
Key Differentiation

- Directly target FGFR3 to normalize both STAT1 and MAPK pathways
- Differentiated pre-clinical efficacy in mouse model
- Oral Dosing

Low-dose infigratinib is designed to treat achon directly at its genetic source

Disease Mechanism

- **ACH FGFR3** gain-of-function mutation causes 2-3x overactivation of the receptor



Symptoms

- Disproportionate short stature
- Narrowed foramen magnum
- Spinal stenosis

Therapeutic Hypothesis

Low-dose infigratinib has the potential to:

- Directly inhibit the causal gain-of-function mutation in FGFR3
- Normalize both the STAT1 and MAPK signaling pathways
- Reverse all key drivers of symptoms

Source: Ornitz DM et al., Developmental Dynamic 2017, Richette Joint Bone Spine 2007, Unger Curr Osteoporos Rep 2017, Hoover-Fong Am J Gen Med 2017

Improved all the key drivers of clinical symptomology in validated ACH mouse model

FGFR3 WT
No treatment

FGFR3$^{Y367C/+}$
No treatment

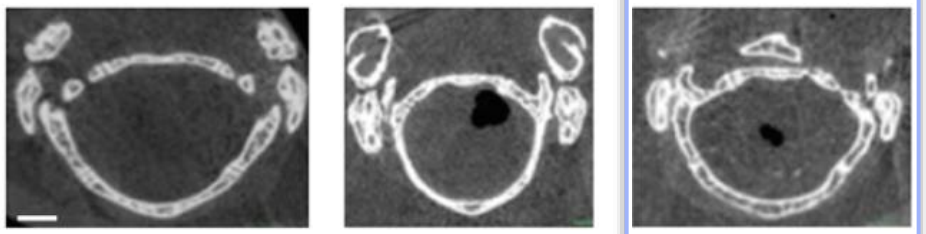
FGFR3$^{Y367C/+}$
Infigratinib tx

1 Cranial bone issues

17% increase in FM area

6% increase in AP skull length

May lead to **decrease in foramen magnum stenosis** and fewer surgeries

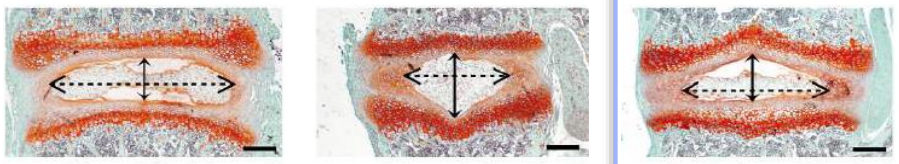


2 Disorders of the spine

12% increase in L4-L6 length

73% increase in disc width

May lead to **decrease** in **spinal stenosis**, possibly **reducing need for surgery**

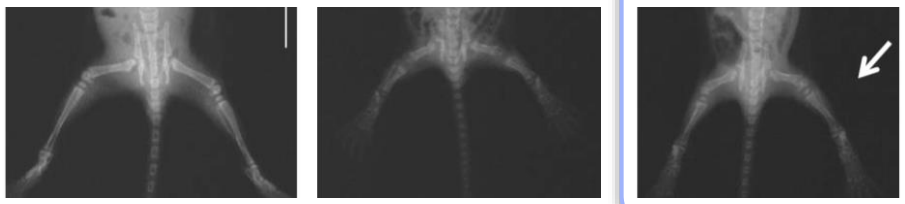


3 Disproportionate short stature

21% increase in femur length

33% increase in tibia length

May lead to **increased stature** and **proportionality**



Source: Komla-Ebri et al., J Clin Inv 2016
Note: percent increase compared to vehicle treated FGFR3Y367C/+ mouse, infigratinib treatment with 2mg/kg subcutaneous dose

Potential best in-class preclinical profile in validated ACH mouse model

Preclinical data from infigratinib and other investigational achondroplasia therapies

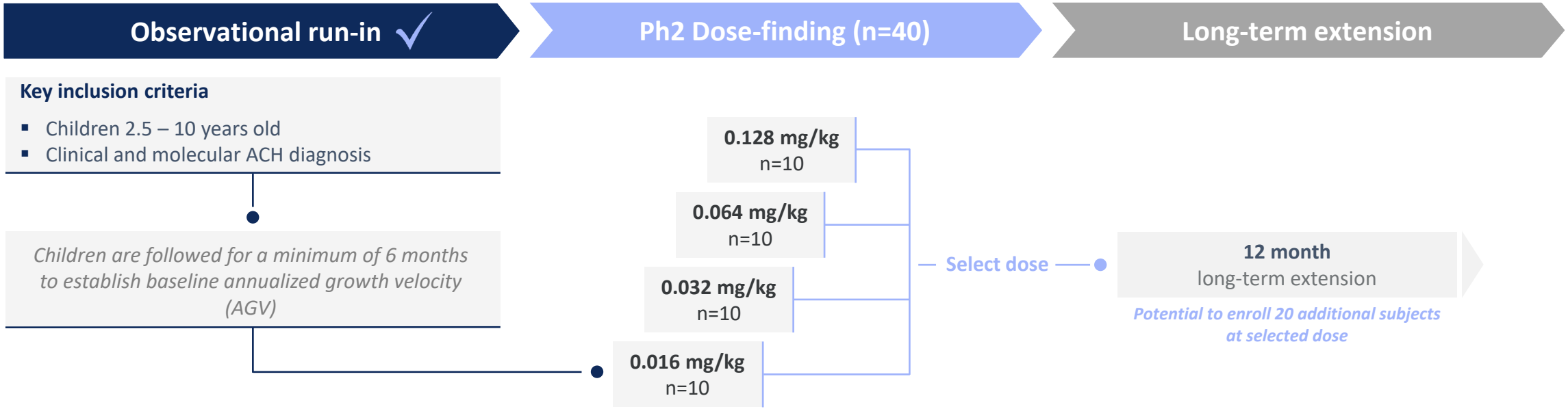
Company/ Asset	MOA	Route	Status	Mouse model	Tibia length	Femur	Foramen magnum area	L4-L6 height
bridgebio *Infigratinib*	**Selective FGFR1-3i**	**Oral**	**Ph2**	**FGFR3$^{Y367C/+}$**	32.6%	20.9%	17.0%	12.1%
BIOMARIN *Vosoritide (BMN111)*	CNP analogue	Daily SQ	Pivotal (NDA filed)	FGFR3$^{Y367C/+}$	6.6%	5.2%	**No known publicly available data**	3.3%
ascendis pharma TransCon CNP[1]	CNP analogue	Weekly SQ	Ph2	FGFR3$^{Y367C/+}$	12.3%			
Pfizer Reifercept (TA-46)	FGFR3 decoy	Weekly SQ	Ph2	FGFR3^{ACH}	8.6%	6.2%		

Percent increase compared to non-treated mouse

Source: Komla-Ebri et al., J Clin Inv 2016, Lorget et al., Am J Hum Genet 2012, Garcia et al., Science Trans Med 2013, Breinholt ENDO 2017
Note: subcutaneous doses, percent increase compared to vehicle treated FGFR3$^{Y367C/+}$, FGFR3$^{ACH/+}$ mouse as noted in "Mouse model" columns
Infigratinib treatment with 2mg/kg subcutaneous dose [1]Based on vosoritide continuous infusion; *Value estimated using DigitizeIt.

The PROPEL clinical program is enrolling with data expected in 1H 2022

✓ Complete



Primary objectives

- Baseline annualized growth velocity (AGV)

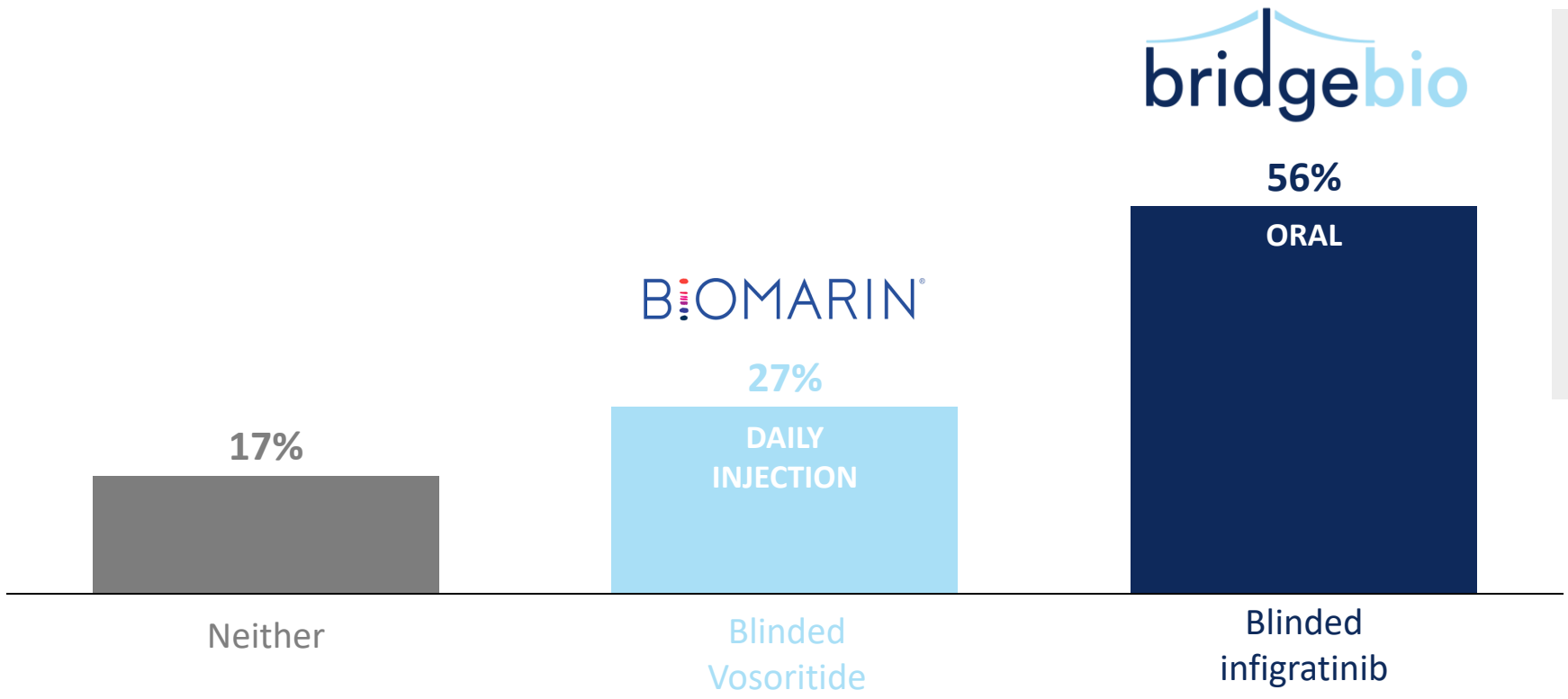
- Identify safe therapeutic dose for expansion / pivotal study
- Safety and tolerability
- Change from baseline in AGV

- Long-term safety and efficacy

HCP survey suggests oral route of administration with efficacy equivalent to vosoritide takes majority market share

Vosoritide vs. low-dose infigratinib showing equivalent efficacy

% of children with achondroplasia who would receive each product[1]



Clear share preference for an **oral solution** for achondroplasia vs. daily injection for children

Source: US market research testing blinded product profiles for vosoritide and infigratinib among HCPs who treat children with achondroplasia; responses weighted by specialty (31 endos, 23 geneticists).
1 Question text: Imagine that Product A [blinded vosoritide] has been on the market for some time and Product B [blinded infigratinib with equivalent efficacy] has just now been approved. Consider the children you manage with ACH not already receiving therapeutic treatment: what percentage of these children would receive each product?

BBP-631: AAV5 gene therapy for congenital adrenal hyperplasia (CAH)

Maddie
Living with CAH

Prevalence

>75k

US & EU

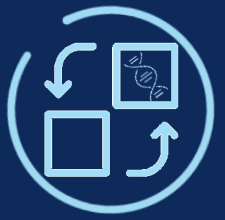
Pathophysiology

Inability to produce cortisol causes need for supraphysiologic doses of synthetic steroids, 3-4x increase in mortality risk, hirsutism, Cushingoid symptoms

Genetic Driver

Loss of function of 21-hydroxylase (21-OH)

Therapeutic Hypothesis



AAV5 gene therapy to provide 21-OH

Design Criteria for Optimal Therapy

- Only approach designed to induce endogenous cortisol and mineralocorticoid production
- Durable transgene delivery to the adrenal gland of NHPs
- Low threshold to correct phenotype

Research and manufacturing capabilities

Facility | 20,000 sq ft lab space in Raleigh, NC

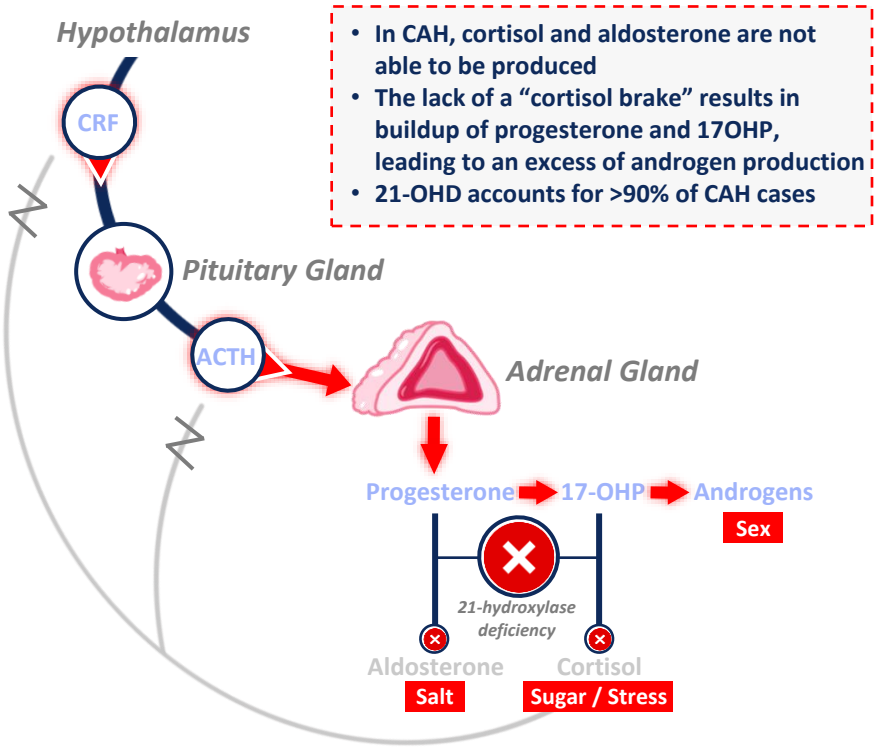
People | 60+ gene therapy employees (>50% in research or CMC)

Capabilities | Vector development, optimization, analytical development, and production (200L)

External Manufacturing | Dedicated GMP manufacturing suite at Catalent

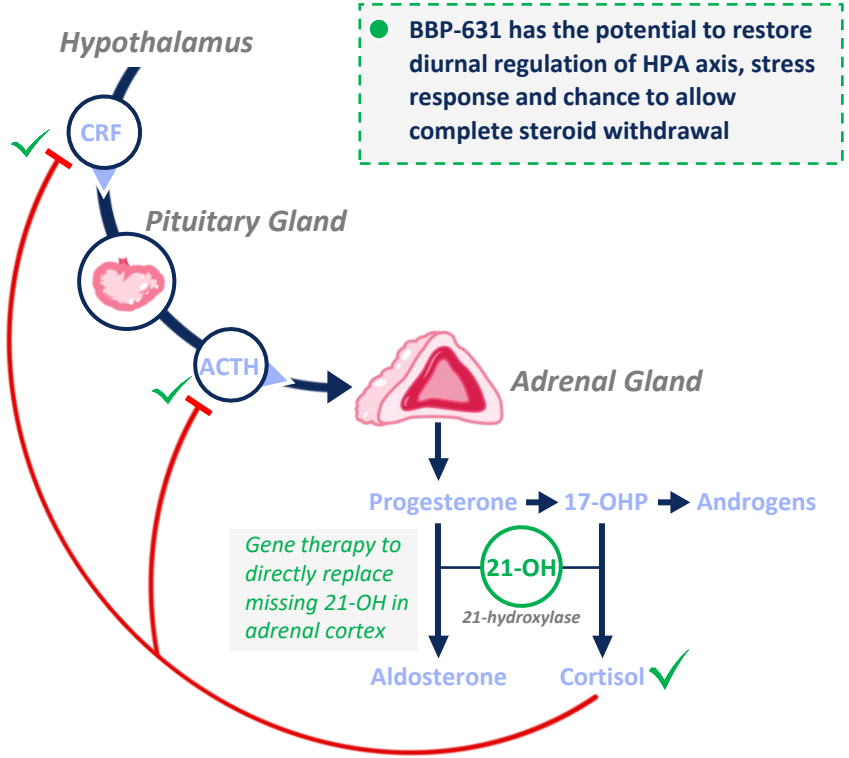
Gene therapy is the only modality designed to treat CAH at its source and allow for production of endogenous cortisol

Hormonal dysregulation in HPA Axis due to 21-Hydroxylase Deficiency (21-OHD)



- In CAH, cortisol and aldosterone are not able to be produced
- The lack of a "cortisol brake" results in buildup of progesterone and 17OHP, leading to an excess of androgen production
- 21-OHD accounts for >90% of CAH cases

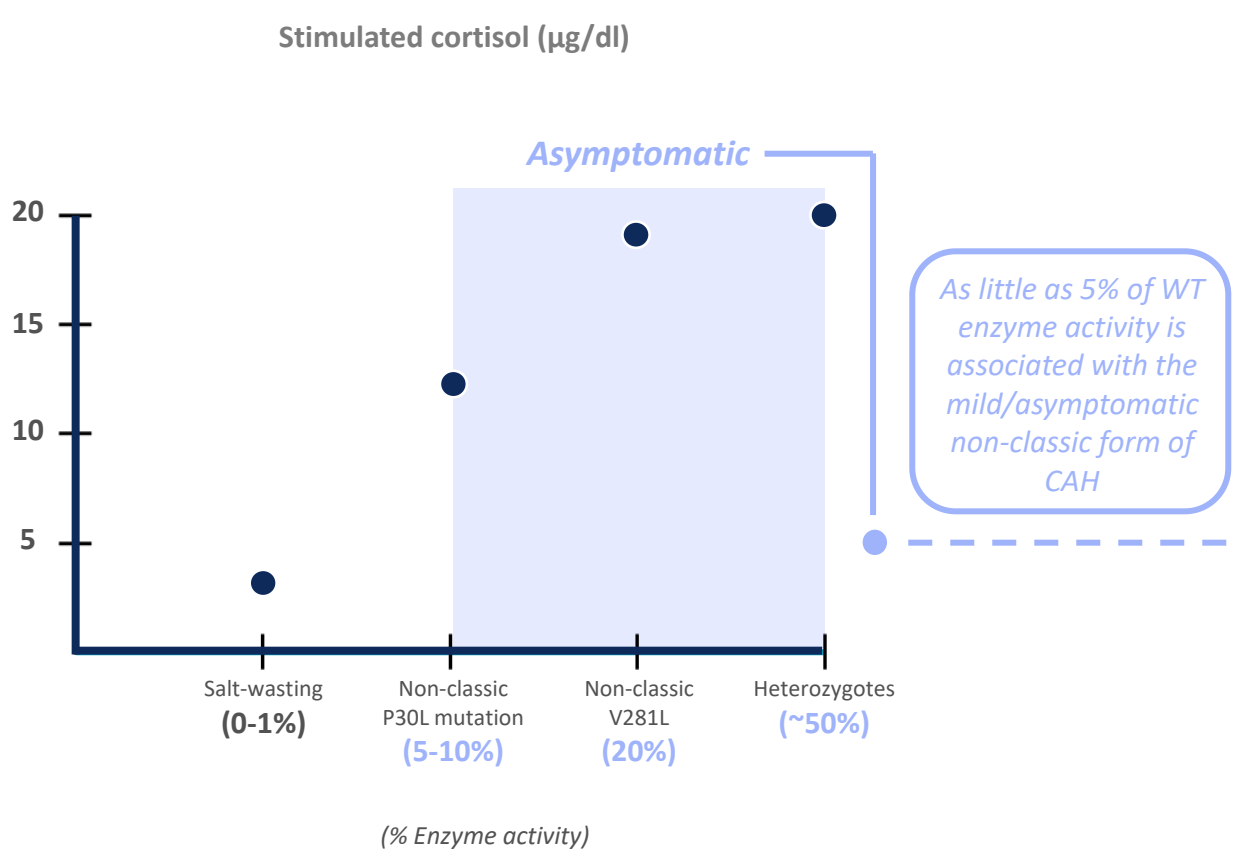
BBP-631 is the only agent designed to restore endogenous cortisol production



- BBP-631 has the potential to restore diurnal regulation of HPA axis, stress response and chance to allow complete steroid withdrawal

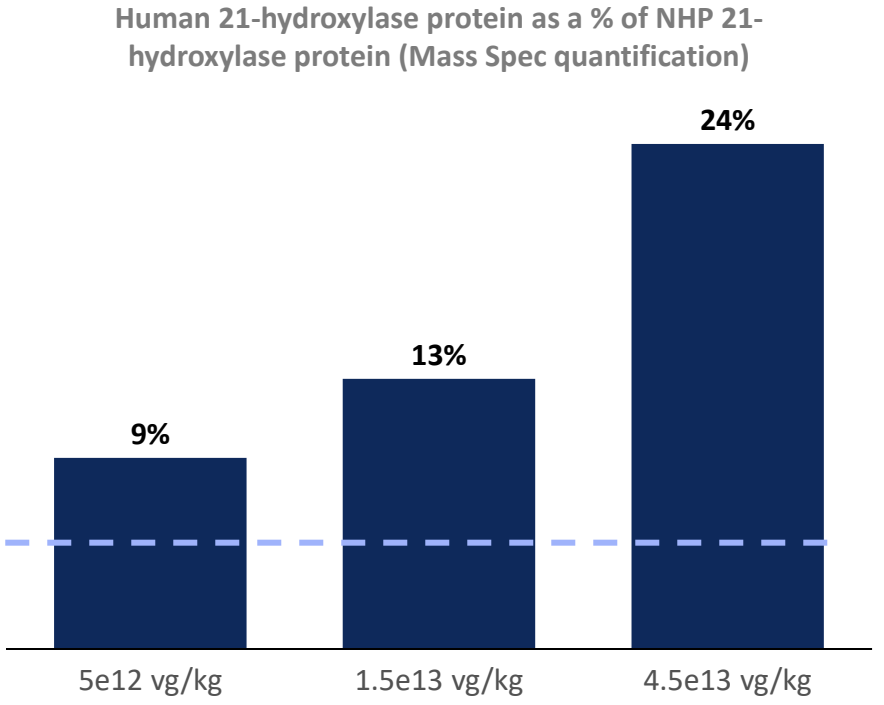
5-10% of WT enzyme may be sufficient for clinical impact

Genotype-phenotype studies show that >5-10% of enzyme activity results in nonclassical CAH



As little as 5% of WT enzyme activity is associated with the mild/asymptomatic non-classic form of CAH

NHP protein data suggests potentially therapeutic levels of 21-hydroxylase enzyme



- Mass-spec methods to quantify protein expression by identifying differential peptides between human and NHP 21-OH
- Data suggest dose-dependent enzyme expression in the adrenal cortex from 9%-24% of WT levels

Source: Perdomini, Gene Therapy 2017; ESGCT 2019; data on file

Phase 1/2 first-in-human trial design

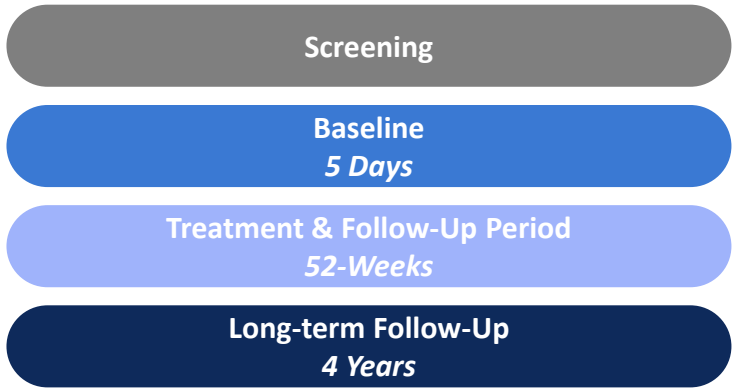
Status

- Trial enrollment underway

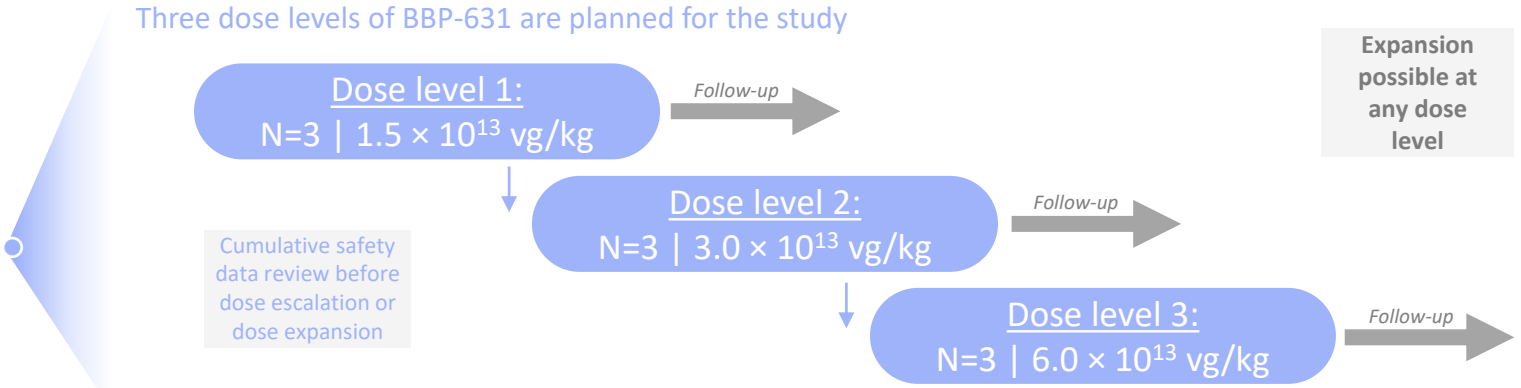
Eligibility

- Age >18 years with classic CAH (simple virilizing or salt-wasting) due to 21-Hydroxylase Deficiency (21-OHD)
- Screening/baseline 17-OHP levels > 5-10 × ULN

FIH Trial Design



Dose Escalation Design



Primary Objectives

- Evaluate safety
- Levels of endogenous cortisol (pre- and post-ACTH stimulation)
- Quality-of-life assessment

KRAS mutant-driven cancers

Basia

Living with pancreatic cancer (>90% KRAS-driven)

Prevalence

>500k

US & EU

Pathophysiology

RAS is the most frequently mutated oncogene, leading to abnormal cell proliferation and survival

Program Highlights

G12C dual inhibitor

MOA: first to directly bind and inhibit both GTP (active) and GDP (inactive) states of $KRAS^{G12C}$

PI3Kα:RAS Breaker

MOA: first to block RAS-driven PI3Kα activation with the potential to avoid adverse effects on glucose metabolism

G12D inhibitor

MOA: directly bind and inhibit $KRAS^{G12D}$ - the single most prevalent KRAS mutant

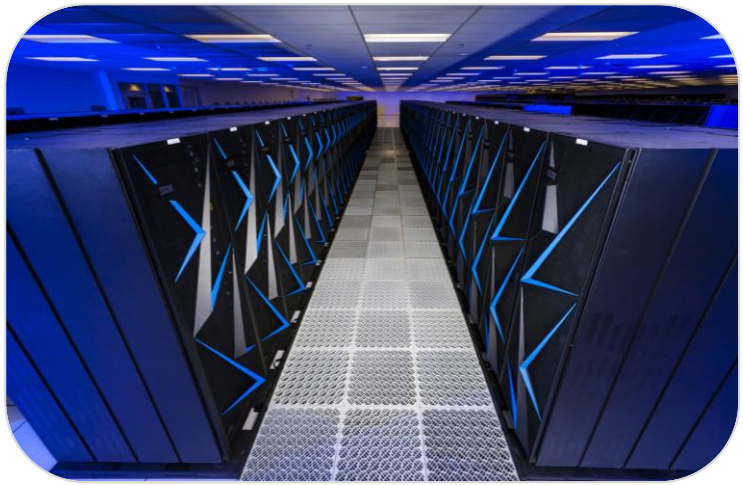
MOA = mechanism of action

Partnerships afford us exceptional collaborators and resources

NIH NATIONAL CANCER INSTITUTE

- Partnership with the National RAS Initiative, including **60 of the world's foremost academic RAS researchers**
- Cutting edge RAS **structural biology expertise**
- Utilization of **cutting-edge instrumentation and techniques**, as well as the **expertise** to lead experiments

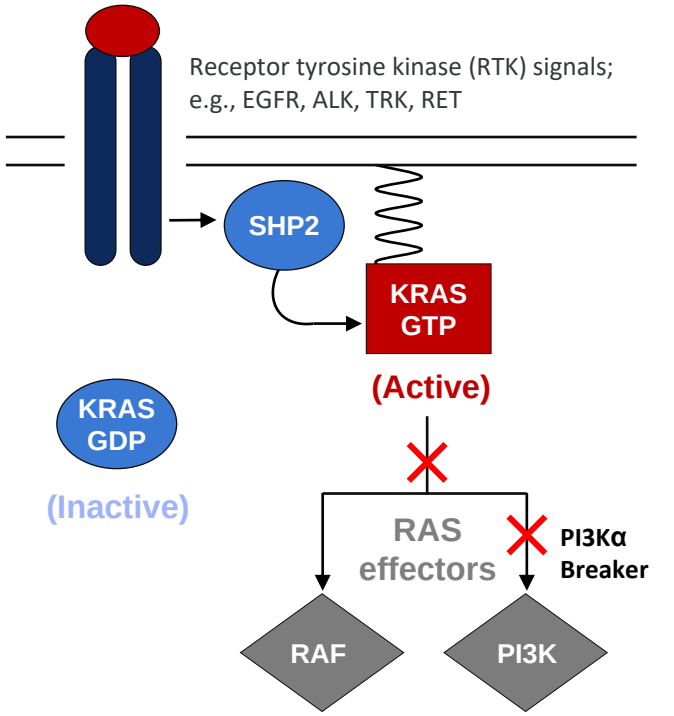
Lawrence Livermore National Laboratory



- Home to Sierra: the **world's 3rd fastest computing system**
- Enables **multi-microsecond molecular dynamics simulations** of protein complexes, and highly efficient in silico docking simulations
- This computing power, combined with RAS structural biology expertise at the NCI, delivers **unique insights that fuel our drug design**

KRAS mutations are implicated in 30% of all cancers, and we have multiple approaches against the target

KRAS Pathway in Cancer

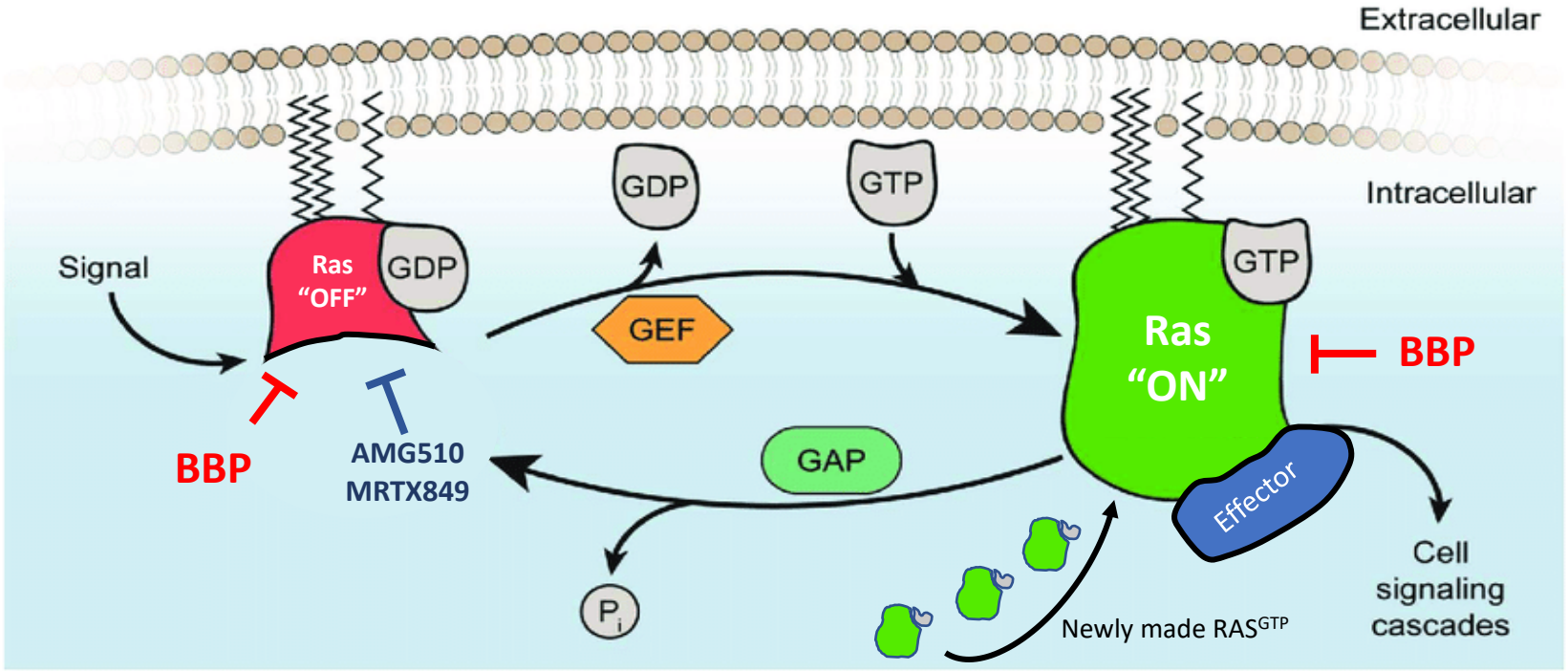


Tumor cell proliferation and survival

RAS Portfolio

Program	Mechanism of Action	Crystal Structure	Stage
KRAS^{G12C}	▪ Inhibits both KRAS^{G12C} GTP (active) and GDP (inactive) states; directly binds KRAS ▪ Differentiates from KRAS^{G12C} GDP (inactive)-only inhibitors	✓	Lead Optimization
PI3Kα Breaker	▪ Blocks specific interaction between RAS and PI3Ka ▪ RAS driver agnostic ▪ Blocks PI3K / AKT effector signaling	✓	Lead Optimization
KRAS^{G12D}	▪ Potent and selective KRAS^{G12D} inhibitor ▪ Directly binds KRAS	✓	Lead Optimization

We hypothesize that a compound that inhibits both GTP (active) and GDP (inactive) forms of KRAS^{G12C} will be superior to one that only inhibits the latter



		GTP (active) / GDP (inactive) dual inhibitor e.g. BBP compounds	GDP (inactive) inhibitors e.g. AMG510, MRTX849
1	Blocks oncogenic signaling from KRAS^{G12C} GTP (active)	✓	
2	Prevents KRAS^{G12C} GDP (inactive) from cycling to KRAS^{G12C} GTP (active)	✓	✓
3	Prevents resistance from residual KRAS^{G12C} GTP (active) signaling	✓	

Source: Adapted from Schoneborn & Heumann, IJMS, 2018. Note: Conclusions based on preclinical models

BridgeBio G12C inhibitors modify both GTP (active) and GDP (inactive) forms of $KRAS^{G12C}$

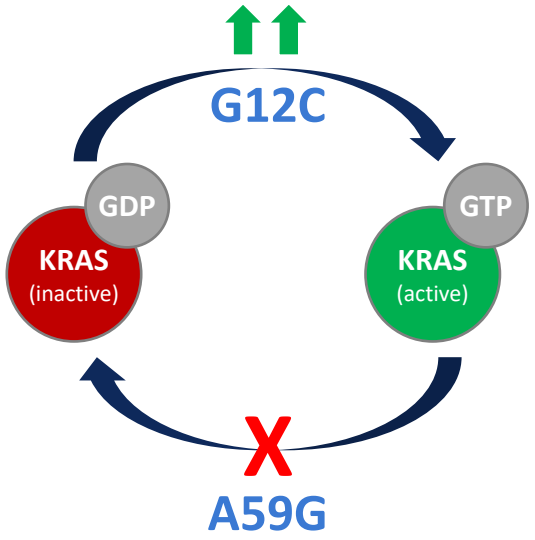
			BBP	AMG510	MRTX849
% modified	$KRAS^{G12C}$ GTP (active)	15’	100	0	0
		120’	100	0	0
	$KRAS^{G12C}$ GDP (inactive)	15’	100	80	73
		120’	100	83	80
$KRAS^{G12C}$: RAF1 Effector Binding IC_{50} (nM)			35	>100,000	20,000
H358 pERK IC_{50} @ 30’ (nM)			8	50	310

Multiple series of dual inhibitors progressing to identify development candidate

Note: Conclusions based on preclinical models

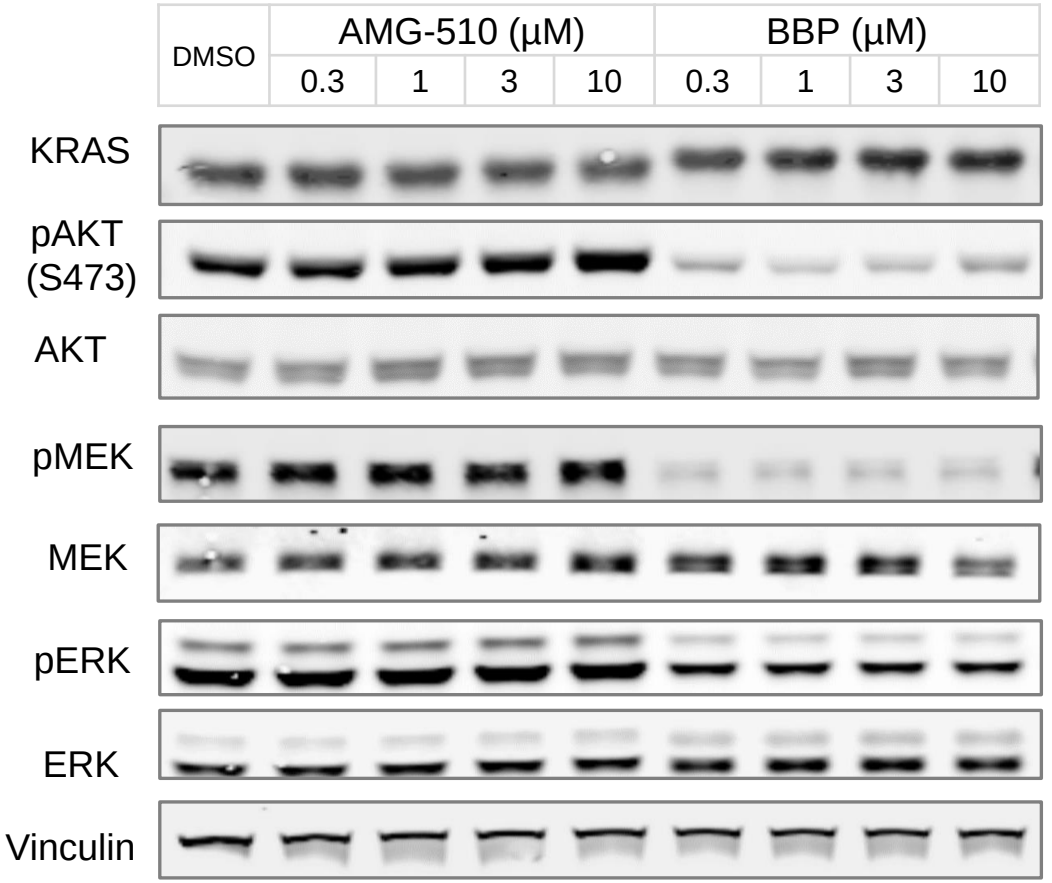
RAS-GTP “locked” mutant A59G, provides strong evidence for cellular GTP-state inhibitor activity

Impact of KRAS Mutations on Nucleotide Turnover



A59G is a 'transition state' mutant that abrogates GTPase activity and locks KRAS in GTP-state

$KRAS^{G12C/A59G}$

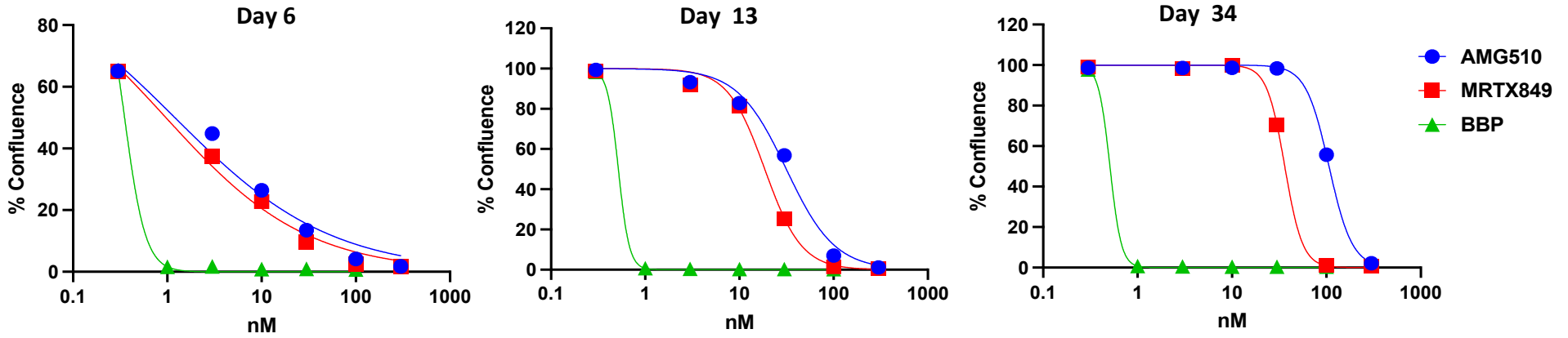


Strong pAKT, pMek and pERK inhibition observed with BBP KRAS-GTP/GDP dual inhibitor

Note: Conclusions based on preclinical models

BridgeBio G12C dual inhibitors are more potent and retain activity compared to inhibitors that only target the GDP (inactive) form

Clonogenic Assay



	% Confluence (IC_{50}, nM)		
	BBP	AMG510	MRTX849
Day 6	< 1	7	5
Day 13	< 1	32	19
Day 34	< 1	107	36

GTP/GDP dual inhibitors:

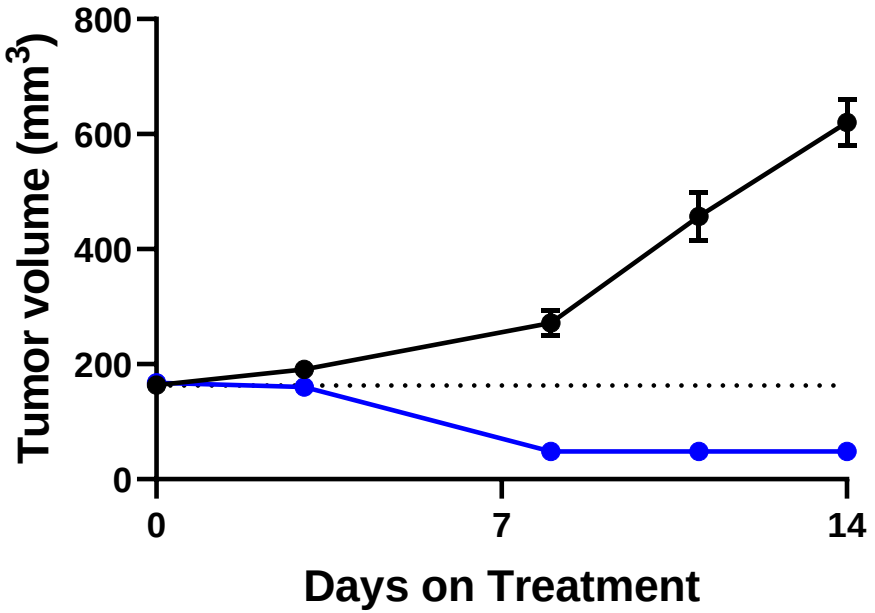
- ✓ Potently inhibit colony formation
- ✓ Retain potent activity suggesting that inhibiting both states of mutant KRAS reduces or delays development of resistance

Note: Conclusions based on preclinical models

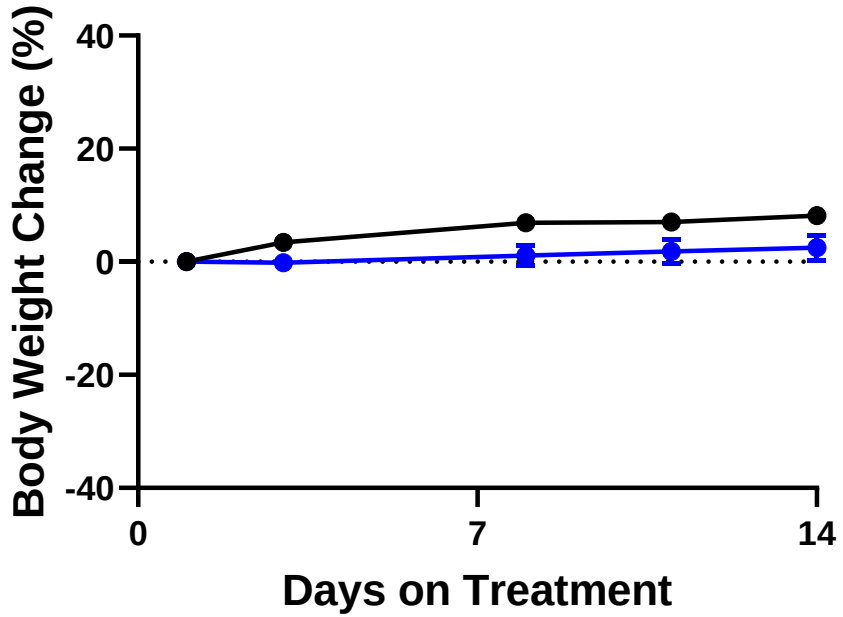
BBP induces tumor regressions and is well tolerated in the MIA PaCa-2 CDX model

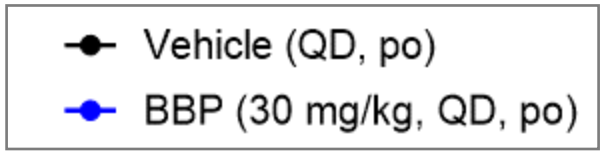
MIA PaCa-2 pancreatic CDX ($KRAS^{G12C}$)

Tumor volume



Body weight % change



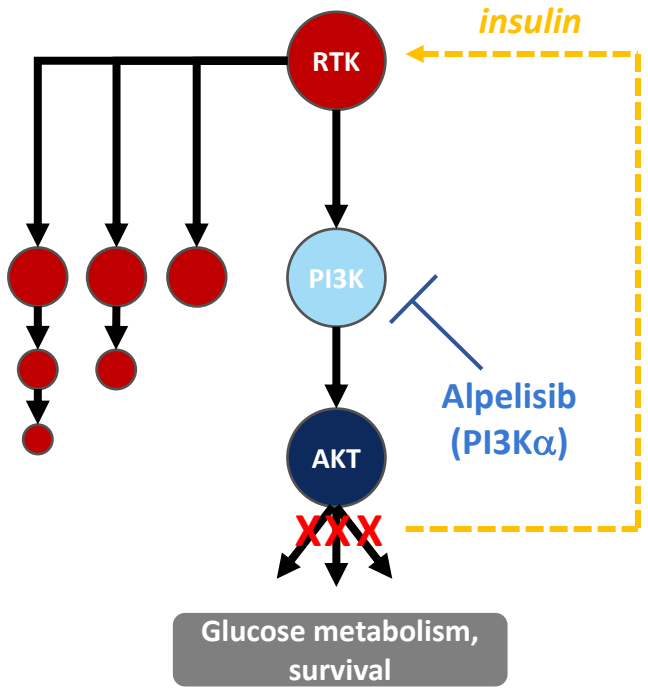


Note: Conclusions based on preclinical models

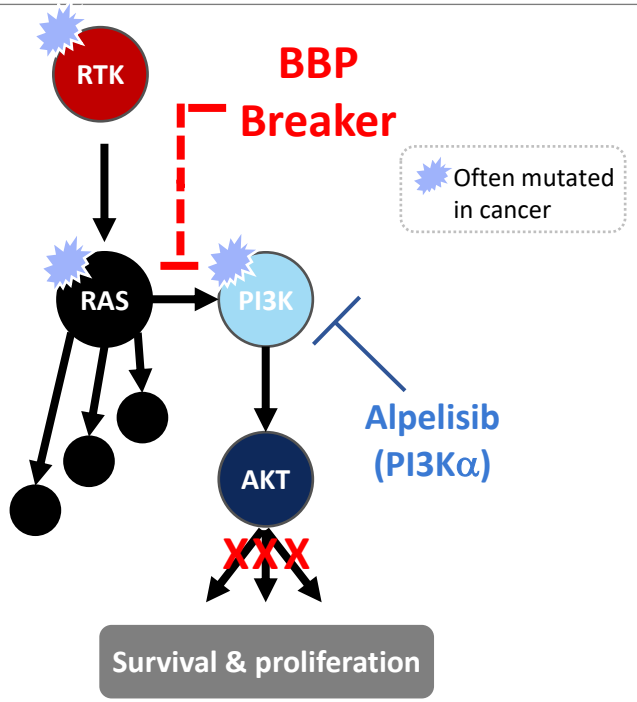
Novel approach to target PI3Kα is tumor cell specific and differentiates from kinase inhibitors

- PI3Kα kinase inhibitors ***block normal cell signaling*** as well as RAS-driven PI3Kα pathway activation in tumor cells, resulting in ***dose-limiting hyperglycemia and insulin-driven resistance***
- Our novel approach of inhibiting PI3Kα:RAS PPI with a ***"PI3Kα Breaker"*** should avoid hyperglycemia and insulin-driven resistance by specifically targeting tumor cells and may provide multiple therapeutic opportunities:
  - ***Tumors with RAS or PI3Kα helical mutations and RTK mutant/amplified drivers***
  - ***Potential combination with ERK pathway inhibition (BRAFi, MEKi, ERKi, $KRAS^{G12C}$i)***

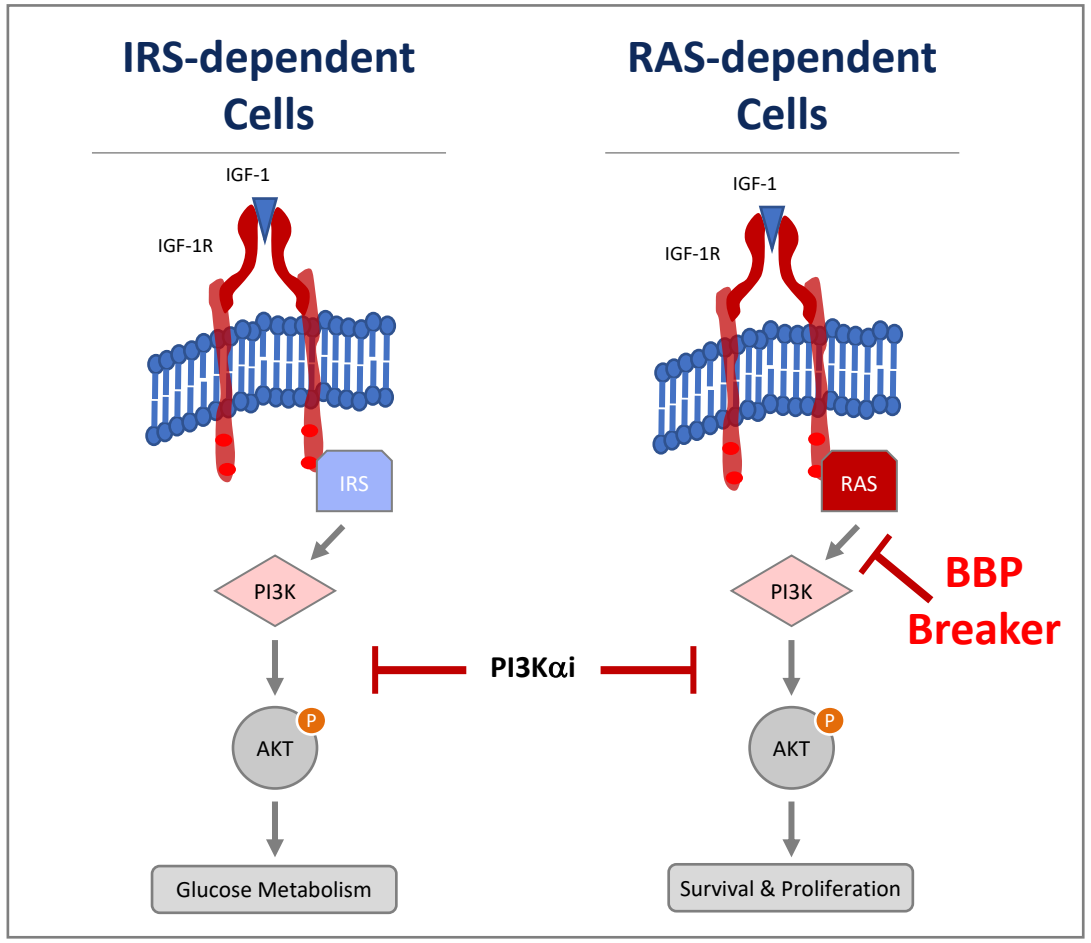
Normal Cells



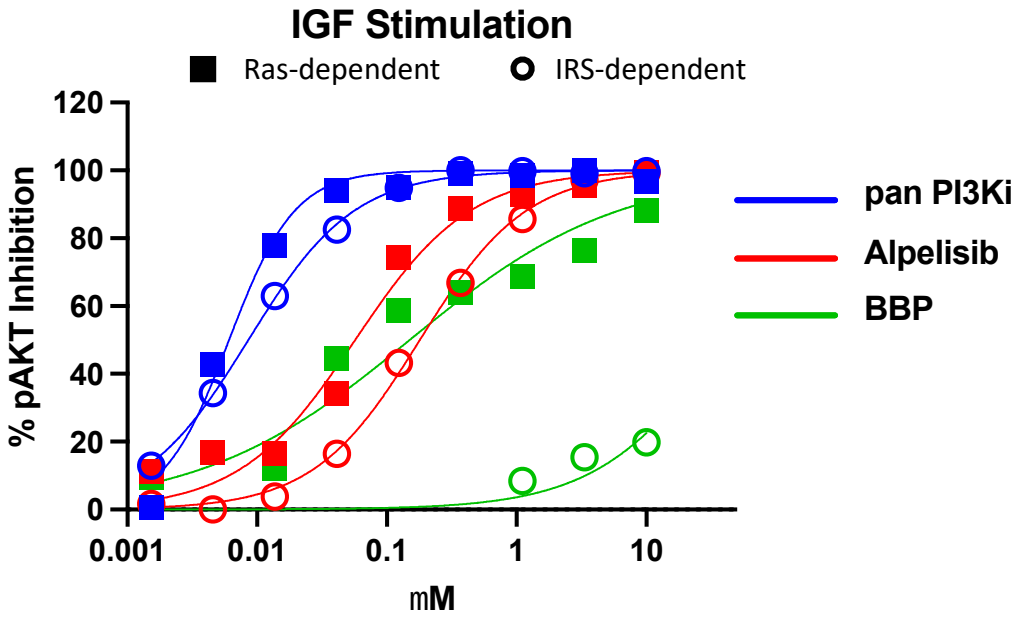
Tumor Cells



Cellular experiments show that only PI3Kα breaker differentiates between RAS and IRS-driven pAKT activation



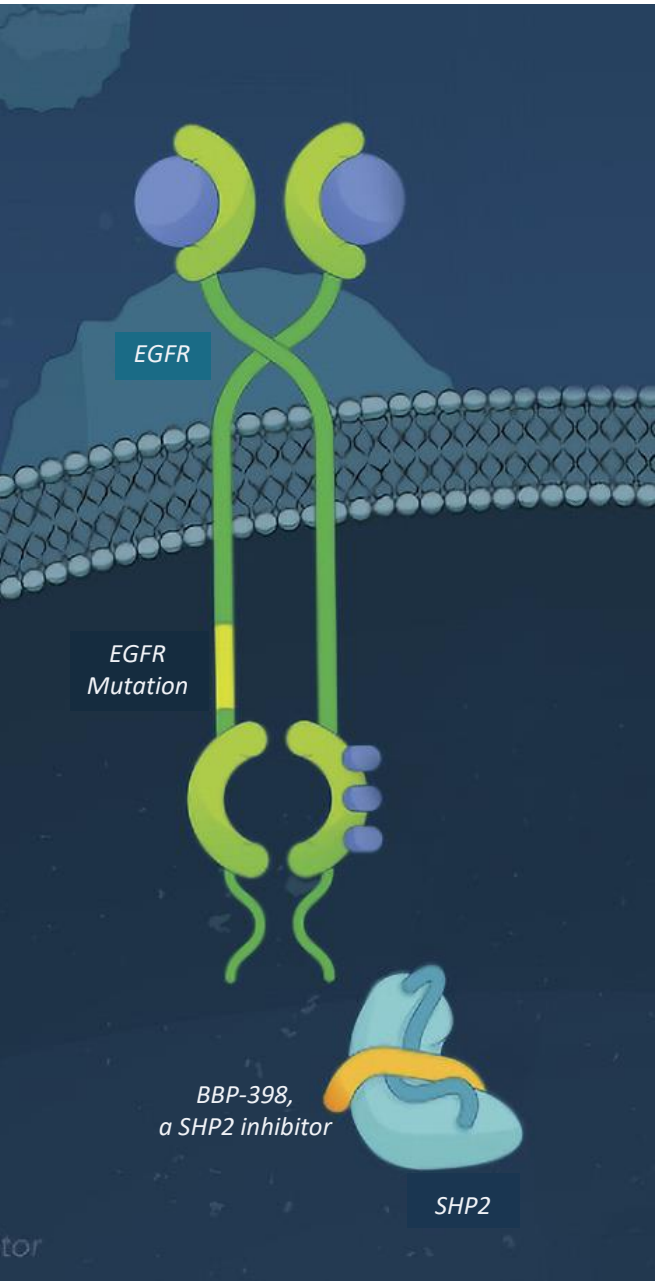
% AKT inhibition in cells treated with IGF



These data suggest that PI3Kα breakers may avoid the on-target hyperglycemia associated with PI3Kα kinase inhibitors

IRS – insulin receptor substrate; IGF-1 – insulin-like growth factor-1

BBP-398: SHP2 inhibitor for treatment resistant cancer



Prevalence

>500k

US & EU

Pathophysiology

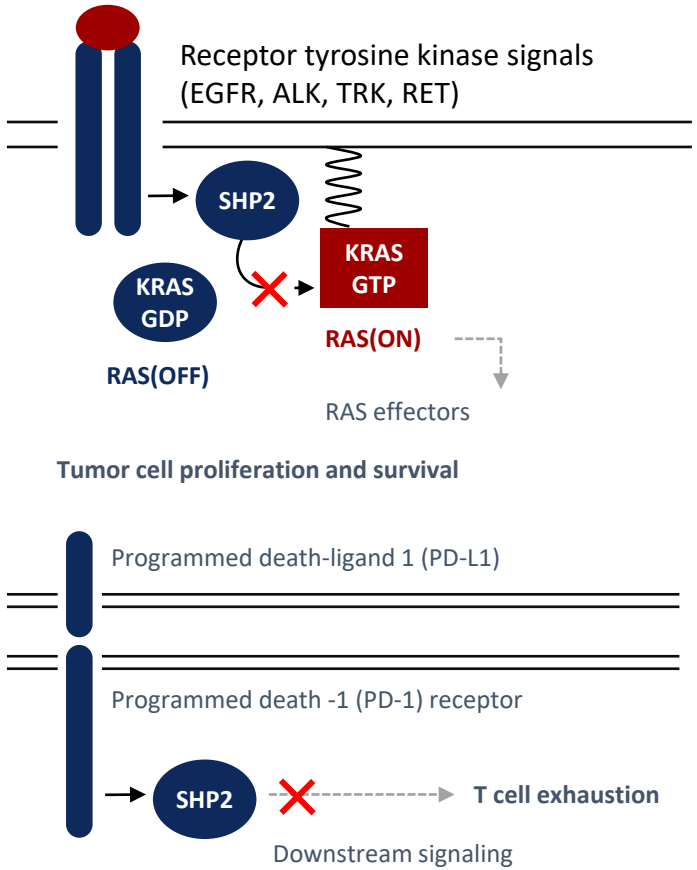
SHP2 acts upstream of RAS/ERK in RTK and cytokine signaling to regulate cell proliferation, survival, adhesion, and migration

Program Highlights

- BBP-398 is a selective, orally bioavailable, allosteric SHP2 inhibitor
- Potential to be best-in-class based on optimal PK profile that may enable tolerable once-daily dosing
- Monotherapy dose escalation is ongoing with plans to initiate combination studies next year

BBP-398 shows best-in-class potential in a large cancer market

Our SHP2i blocks downstream MAPK signaling and abrogates T cell exhaustion



We believe BBP-398 has the ideal properties for combination with a multitude of other therapeutic classes

- ✓ **Human half life: ~10-15 hours**
- ✓ **Optimal PK profile which may enable better tolerability in combination**

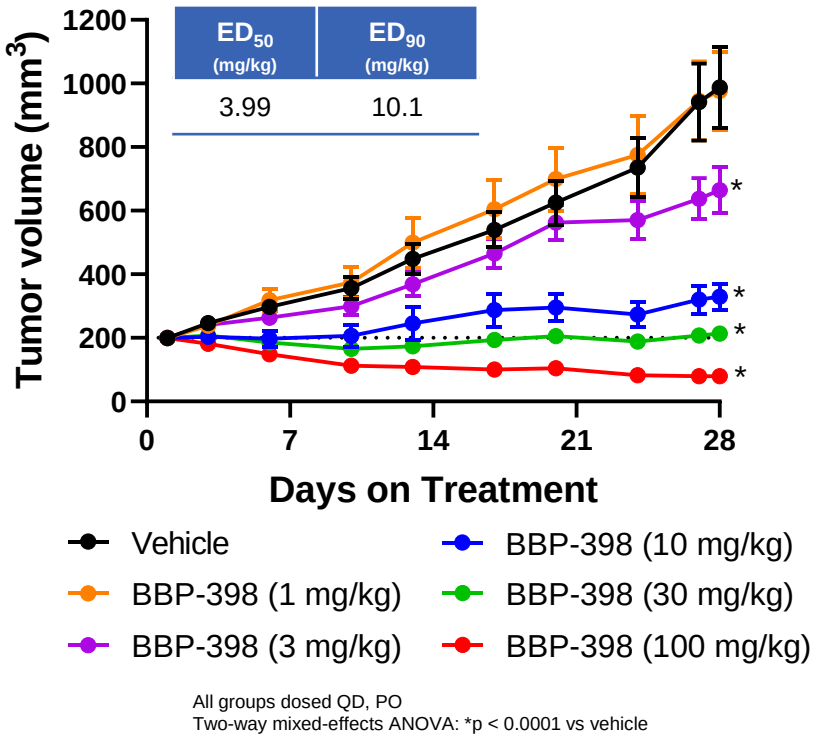
Initial clinical combinations of focus based on SHP2i preclinical data

Combination Agent	Patient Population[1]
KRAS G12Ci	70,000
EGFRi (LianBio)	150,000
PD-1 Bristol Myers Squibb™	700,000

[1]US incidence estimated from SEER, TCGA; all scaled for WW incidence

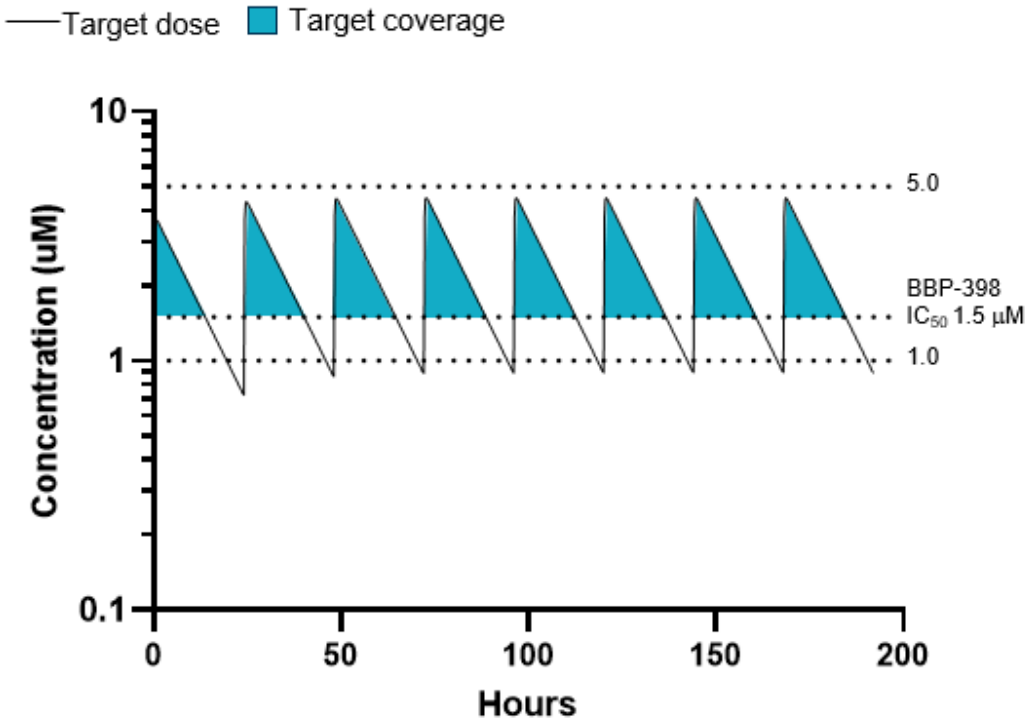
Predicted BBP-398 pharmacokinetics support once daily oral administration to achieve target coverage

HCC827 ($EGFR^{ex19del}$ & $EGFR^{amp}$) - NSCLC CDX



All groups dosed QD, PO
Two-way mixed-effects ANOVA: *p < 0.0001 vs vehicle

BBP-398 steady-state PK simulation for optimal efficacy



Predicted clinical exposure supports coverage of efficacy target in patients may be achieved with continuous once daily dosing

Precision oncology summary

BridgeBio Oncology

- ☑ Infigratinib approved for 2nd line FGFR2 fusion cholangiocarcinoma with multiple late-stage studies ongoing
- ☑ Identified multiple series of differentiated novel $KRAS^{G12C}$ GTP/GDP inhibitors
- ☑ Identified multiple series of differentiated novel PI3Kα:RAS Breakers
- ☑ Progressing potentially best-in-class SHP2 inhibitor BBP-398 with differentiated pharmacokinetic profile that may enable once-daily dosing in combination studies

2022 Targets

- ☐ RAS development candidate
- ☐ Present BBP-398 Phase 1 monotherapy data
- ☐ Initiate BBP-398 combination studies (KRAS G12Ci, IO, EGFRi)

Major catalysts across the pipeline anticipated over the next 12 months

ANTICIPATED

Execution in 2021

- ☑ **BBIO / EIDX merger closure:** Completed January 26th
- ☑ **Four new INDs cleared**
- ☑ **NULIBRY™ (fosdenopterin) for MoCD type A:** FDA approval
- ☑ **TRUSELTIQ™ (high-dose infigratinib) for second-line cholangiocarcinoma:** FDA approval
- ☑ **Secured up to $750m in non-dilutive debt financing:** Completed November 18th

4 core value drivers

Encaleret (CaSRi) for ADH1:
- ☑ Initial Ph2 proof-of-concept (1Q21)
- ☑ Complete in-patient Ph2 data (4Q21)

- ☐ **Acoramidis (ATTR stabilizer) for ATTR-CM:** Ph3 topline data (4Q21)
- ☐ **Low-dose infigratinib (FGFRi) for achondroplasia:** Ph2 proof-of-concept data (1H22)
- ☐ **AAV5 gene therapy for CAH:** Initial data from Ph1/2 study (mid-22)
- ☐ **Acoramidis (ATTR stabilizer) for ATTR-CM:** NDA submission (mid-22)

Pipeline upside

- ☐ **COL7 replacement for RDEB:** Data from Ph2 study (early ’22)
- ☐ **GO inhibitor for hyperoxaluria:** Data from Ph1 study (2022)
- ☐ **SHP2 inhibitor for RAS and RTK driven cancer:** Monotherapy Phase 2 dose selection (2022)
- ☐ **Ribitol for LGMD2i:** Ph2 proof-of-concept data (2022)
- ☐ **KRAS inhibitor program:** Clinical candidate selection (2022)

$1.2bn in cash and equivalents as of November 2021 anticipated to provide runway into 2024