bridgebio

hope through
rigorous science

Corporate presentation

May 2021

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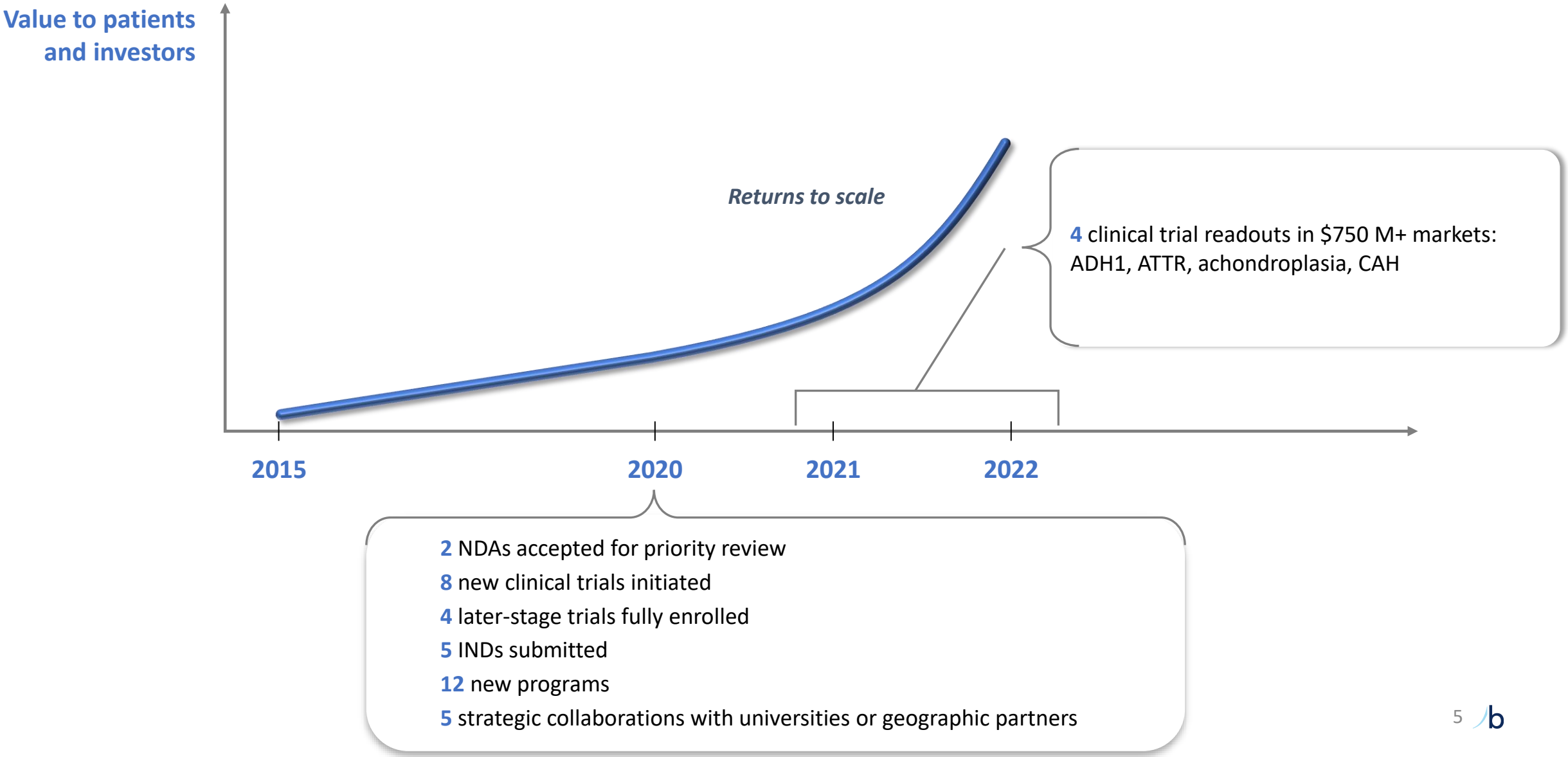
BridgeBio Pharma: Hope through rigorous science

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

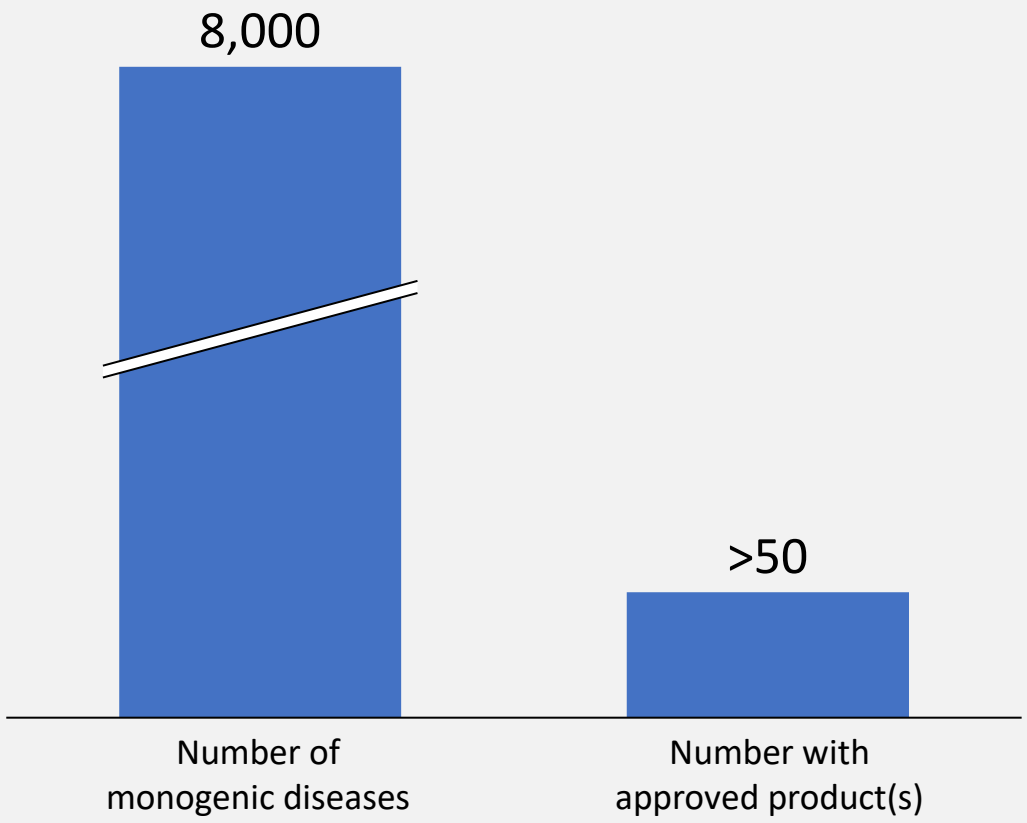
Our 2025 vision – A leading player in genetic medicine

- Multiple best-in-class or first-in-class products in blockbuster markets, with a total of 4+ NDAs on file
- Patient-centric global commercial infrastructure
- World-class drug discovery and development platform
- Broad network of >40 university partnerships
- Multiple therapeutic modalities, many diseases
- Deep pipeline of 30+ R&D programs

Context #1: 2021 is a critical year for BridgeBio



Context #2: The opportunity to help patients remains large



Thousands of distinct diseases requiring novel therapeutic options

8

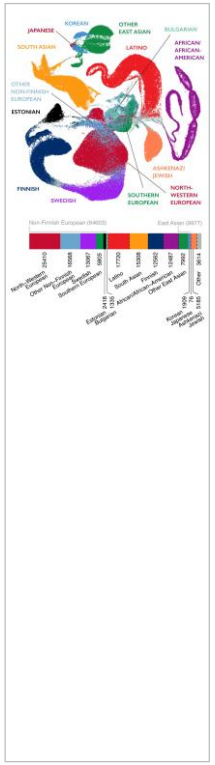
$1B+ opportunities in the pipeline

1) **Acoramidis** for ATTR CM and PN
2) **Low-dose infigratinib** for achondroplasia
3) **AAV5 gene therapy** for congenital adrenal hyperplasia
4) **High-dose infigratinib** for adjuvant urothelial carcinoma
5) **Pan-mutant KRAS inhibitor** for KRAS+ cancer
6) **SHP2 inhibitor** for RAS and kinase mutant cancer
7) **GPX4 inhibitor** for multiple tumor types
8) **GO1 inhibitor** for frequent kidney stone formers

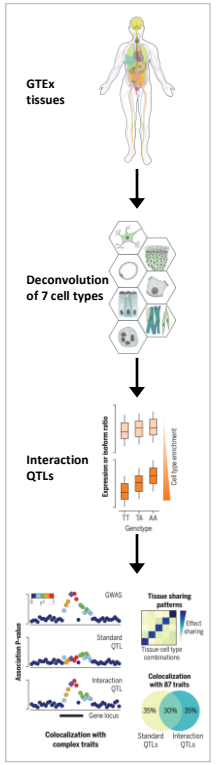
Context #3: Still Day 1 for innovation within genetic medicine

Macromolecules

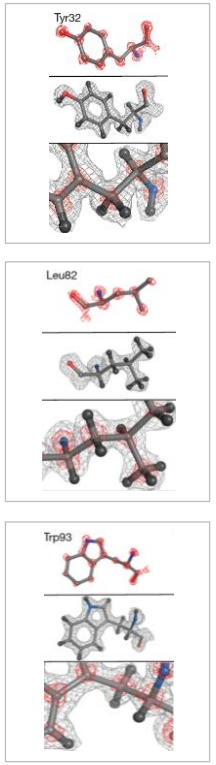
DNA
- gnomAD
- ENCODE3



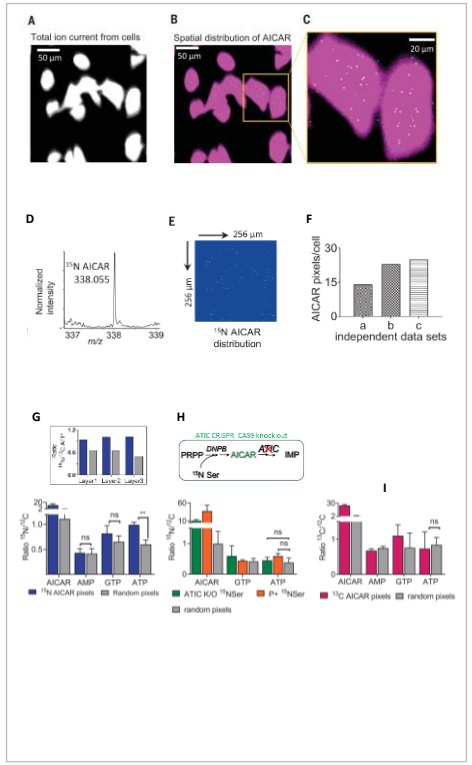
RNA
- GTEx
- Single cell sequencing advances



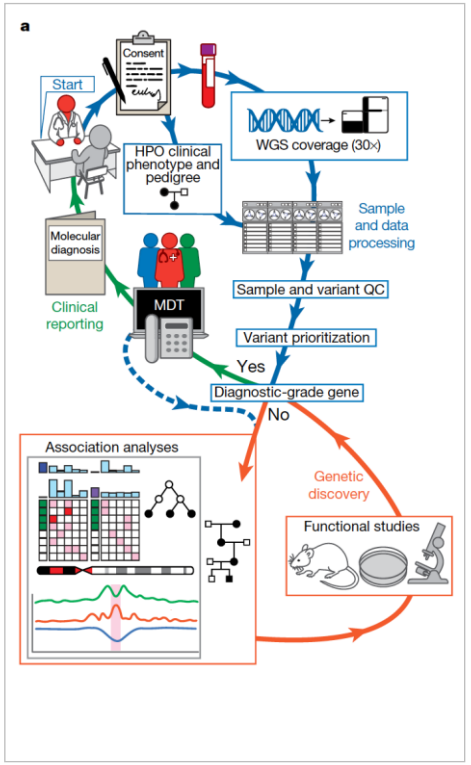
Protein
- CryoEM
- DeepMind



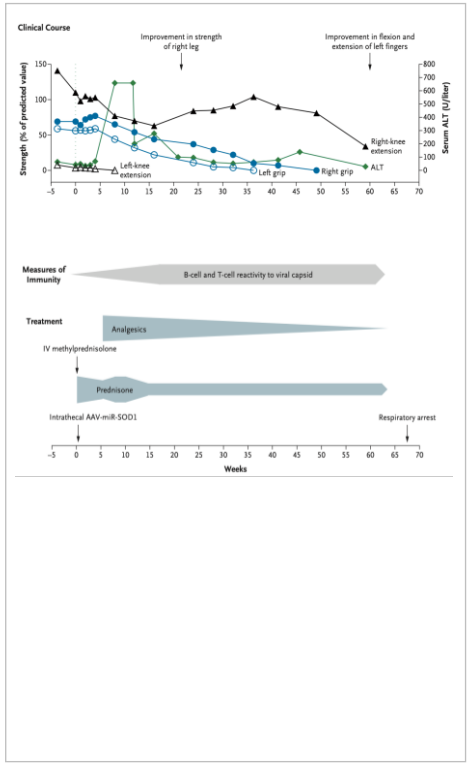
Molecular Systems
- Mass spectrometry + metabolomics give us 1st snap of purine bio-synthesis



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



New Therapeutic Modalities
- Antisense oligonucleotides coming of age
- Gene therapy continues maturing



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

Product platform: Our drug engineering platform leverages and efficiently translates innovation to therapies that matter

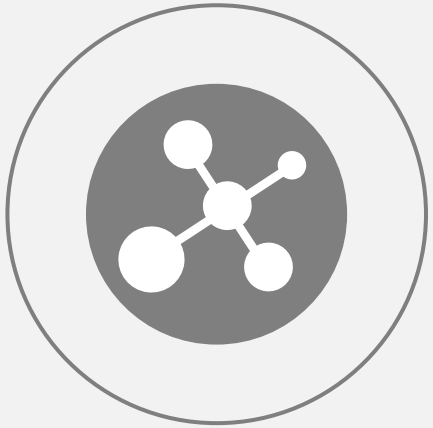
Discover

Novel genetic disease targets

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

Create

Medicines with industry-leading research capabilities



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

Test

Our drugs through global development footprint

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

Deliver

Our products to patients through commercial infrastructure

Global infrastructure, diagnostics, patient support, disease state awareness

Product platform: BridgeBio is a people and a process

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

Discover

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

Create

Mendelian

Uma Sinha, PhD
Chief Scientific Officer
GBT PORTOLA PHARMACEUTICALS

Robert Zamboni, PhD
Chemistry
MERCK FROSST

Oncology

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

Pedro Beltran, PhD
SVP, Oncology
AMGEN UNITY BIOTECHNOLOGY

Susan Moran, MD
Chief Medical Officer, QED Therapeutics
PURA PPD genzyme

Our pipeline spans multiple therapeutic areas with numerous upside opportunities

Small molecule | Topical small molecule | Biologics | Antisense oligo | Gene therapy

Portfolio segment	Program	Drug mechanism	Diseases	Patient pop. (US+EU)	Modality	Preclinical: Discovery / IND-enabling	Preclinical: Phase1	Clinical: Phase 2	Clinical: Phase 3
Mendelian	**Acoramidis**	TTR stabilizer	ATTR-CM	>400K	Small molecule				
	NULIBRY (fosdenopterin)	cPMP replacement	MoCD type A	100	Small molecule				Approved
	Infigratinib	Low-dose FGFR1-3i	Achondroplasia	55K	Small molecule				
	Encaleret	CaSR antagonist	ADH1 / HP	12K[1] / 200K	Small molecule				
	BBP-418	Glycosylation substrate	LGMD2i	7K	Small molecule				
	BBP-671	PanK activator	PKAN / OA	7K	Small molecule				
	BBP-711	GO1 inhibitor	PH1 / FSF	5K / 1.5M	Small molecule				
	BBP-472	PI3Kβi	PTEN autism	120K	Small molecule				
	4 undisclosed small molecule programs			>500K	Small molecule				
	4 undisclosed antisense oligonucleotide programs			>300K	Antisense oligo				
Genetic Derm	**BBP-589**	Recombinant COL7	RDEB	1.5K	Biologics				
	BBP-681	Topical PI3Kai	VM / LM	117K	Topical small molecule				
	BBP-561	Topical KLK 5/7i	Netherton	11K	Topical small molecule				
Targeted Oncology	**Infigratinib**	FGFR1-3i	3 FGFR+ tumor programs	37K	Small molecule				NDA filed
	BBP-398	SHP2i	Multiple tumors	>500K	Small molecule				
	BBP-454	Pan-mutant KRASi	3 KRAS+ tumors programs	>500K	Small molecule				
	BBP-954	GPX4i	Multiple tumors	>500K	Small molecule				
Gene Therapy	**BBP-631**	21-OH gene therapy	CAH	>75K	Gene therapy				
	BBP-812	ASPA gene therapy	Canavan	1K	Gene therapy				
	BBP-815	TMC1 gene therapy	Genetic hearing loss	10K	Gene therapy				
	4 undisclosed AAV gene therapy programs			150K	Gene therapy				

1 US carriers

Product pipeline: Layers of de-risking and upside

Future pipeline catalysts and long-term growth

Targeted oncology (FGFR3 in UC, SHP2, KRAS)

Common mendelian (LGMD2i, RDEB, PKAN, VM)

Validation of asset picking and execution

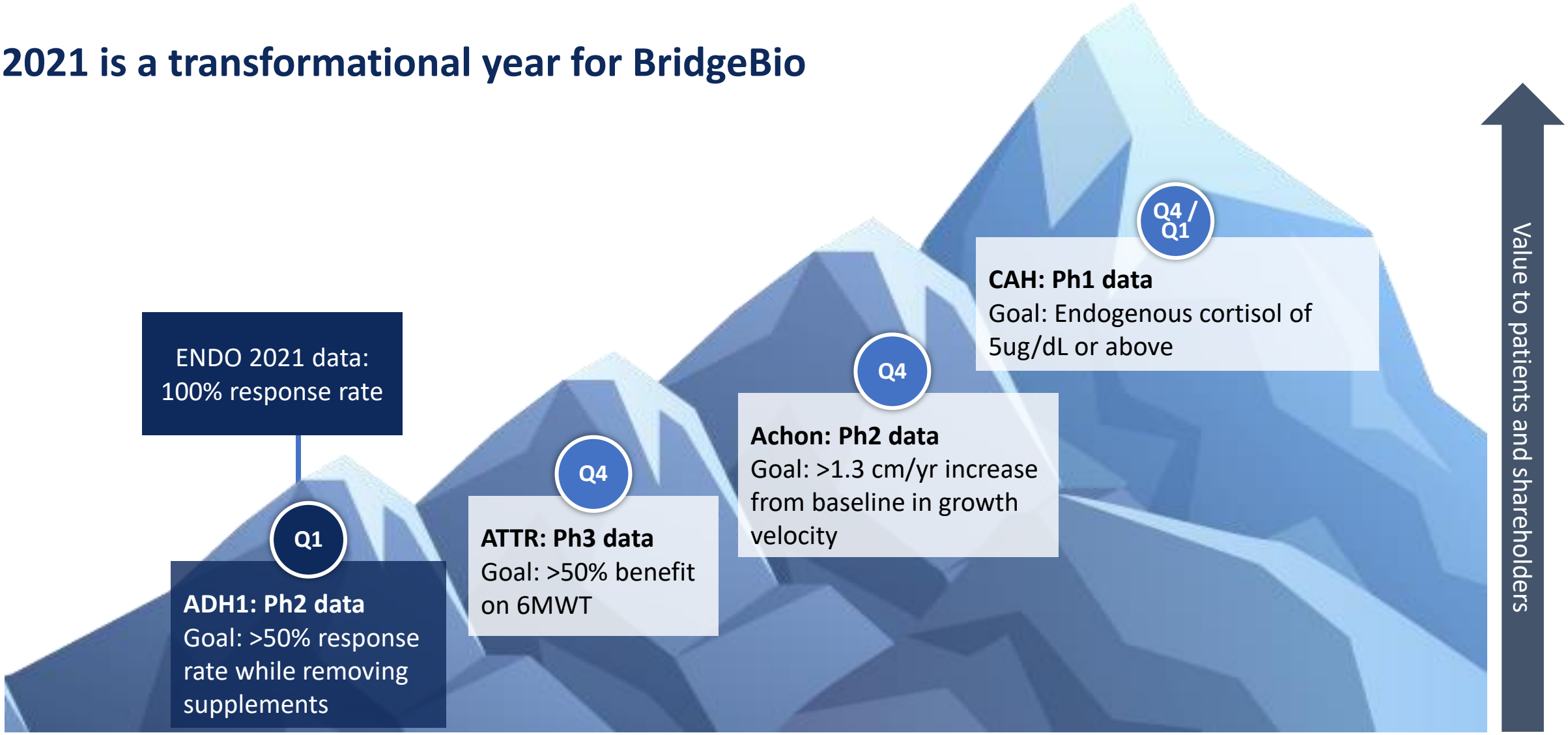
Near-term major catalysts from 4 core value drivers (ATTR, ADH1, achondroplasia, CAH)

Proving ground and revenue

2 FDA approvals in 2021 (MoCD Type A, 2L+ CCA)

Assumes achievement of anticipated milestones

2021 is a transformational year for BridgeBio



Growth potential this year:

- Positive pivotal data in a multi-billion market
- Positive POC data in multiple blockbuster indications
- Transition to commercial-stage biopharma company

Assumes achievement of anticipated milestones

Alexis and Jackson
ADH1 patients

Encaleret for autosomal dominant hypocalcemia type 1 (ADH1) overview

ADH1 overview

Prevalence

12K individuals harboring variants in US[1]

Genetic driver

Calcium-sensing receptor (CaSR) hyperactivation

Pathophysiology

Decreased blood calcium, elevated urine calcium, and lower parathyroid hormone secretion [2]

Features of a potential best-in-class medicine for ADH1

Direct targeting of CaSR

Normalization of all downstream effects of CaSR hyperactivity

Potential to address most common symptoms

arising from altered calcium and parathyroid hormone dysregulation

Oral dosing, the first targeted therapy for ADH1 in a convenient form for patients and families

1 Dershem et al., Amer Jour of Hum Genetics, 2020; 2 Lienhardt, et al., JCEM, 2001

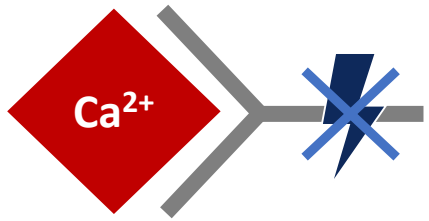
Encaleret is designed to treat ADH1 at its source by normalizing CaSR sensitivity

ADH1 disease mechanism

Therapeutic hypothesis

CaSR senses and regulates serum Ca levels

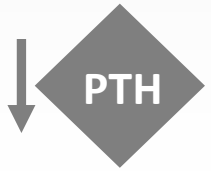
ADH1-causing mutations hyperactivate CaSR



Hyperactive CaSR causes dysregulation of Ca homeostasis

Increased urinary calcium

Decreased serum calcium



Decreased parathyroid hormone secretion

Acute symptoms and long-term complications

Presenting symptoms

Hypocalcemic seizures

Paresthesia

Tetany

Muscle cramps

Long-term complications

Nephrocalcinosis

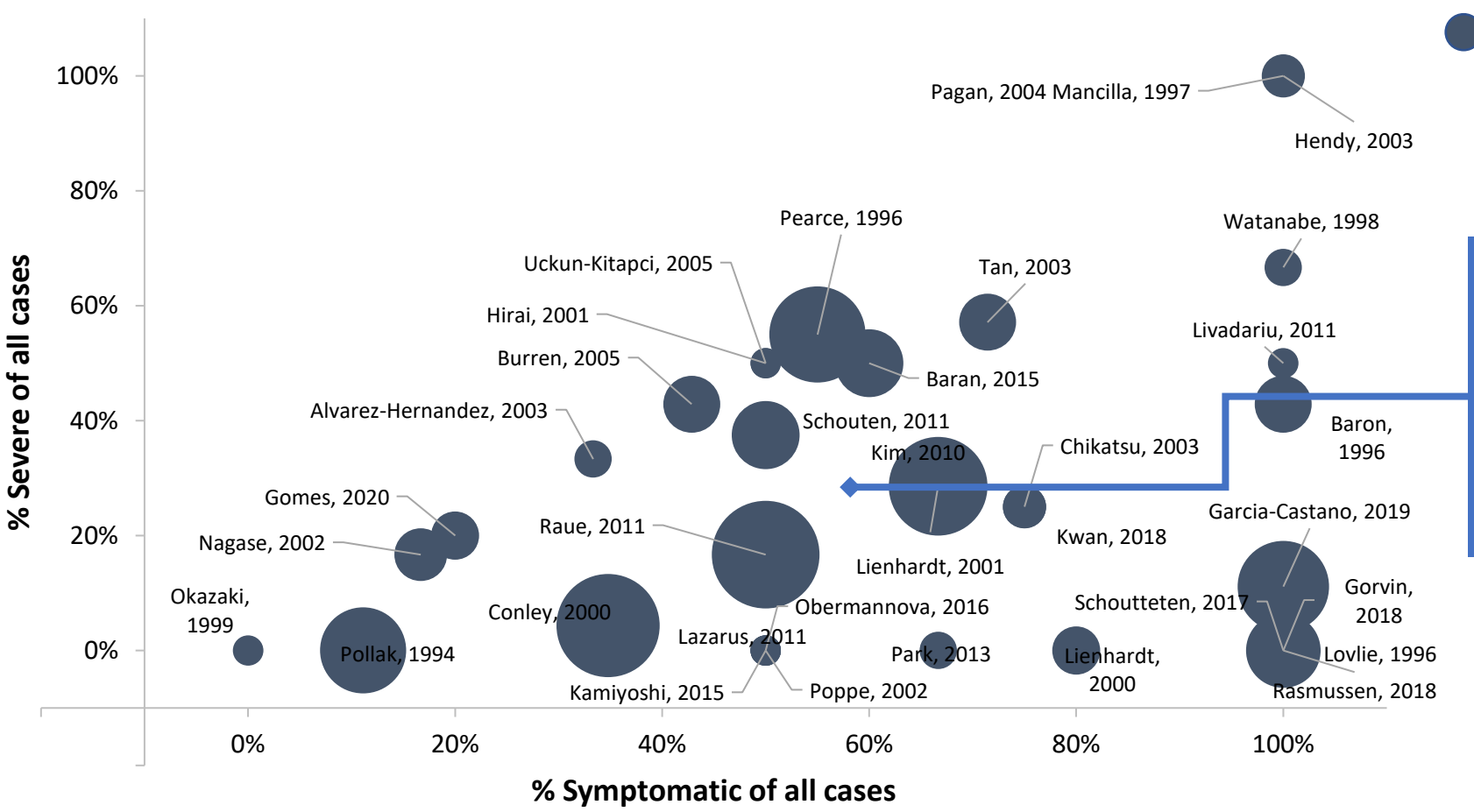
Nephrolithiasis

Chronic kidney disease

Encaleret is a CaSR inhibitor designed to normalize PTH, serum Ca and urine Ca levels, potentially resolving key symptoms of disease

Majority of ADH1 patients are symptomatic including one third with severe symptoms

Meta-analysis of published ADH1 case reports

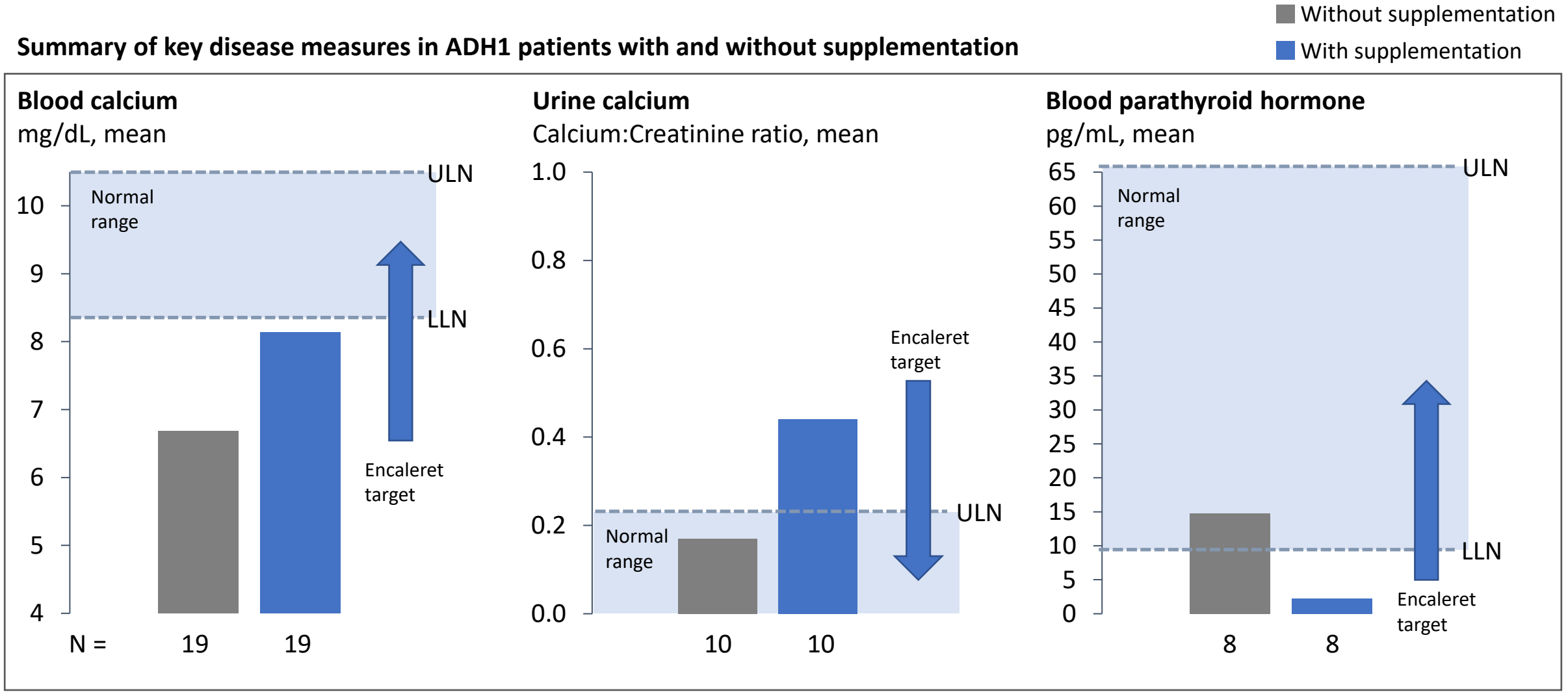


In aggregate, ~60% of familial ADH1 cases experienced hypocalcemia-related symptoms with one third experiencing severe symptoms

Source: 31 published reports, cumulatively 252 confirmed ADH1 cases over 24 years

Current therapy for ADH1 (oral calcium, activated Vitamin D) raises blood Ca but does not address disease mechanism; increases UCa, suppresses PTH

Summary of key disease measures in ADH1 patients with and without supplementation

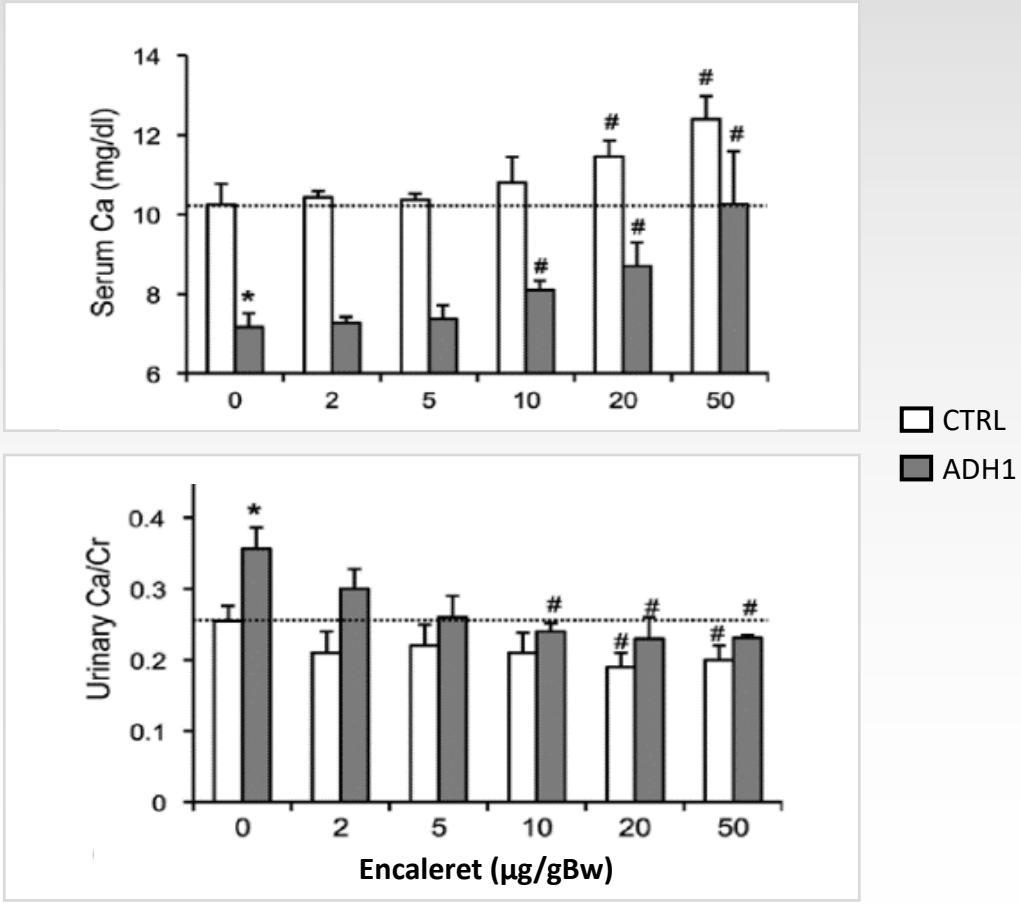


ULN = upper limit of normal, LLN = lower limit of normal

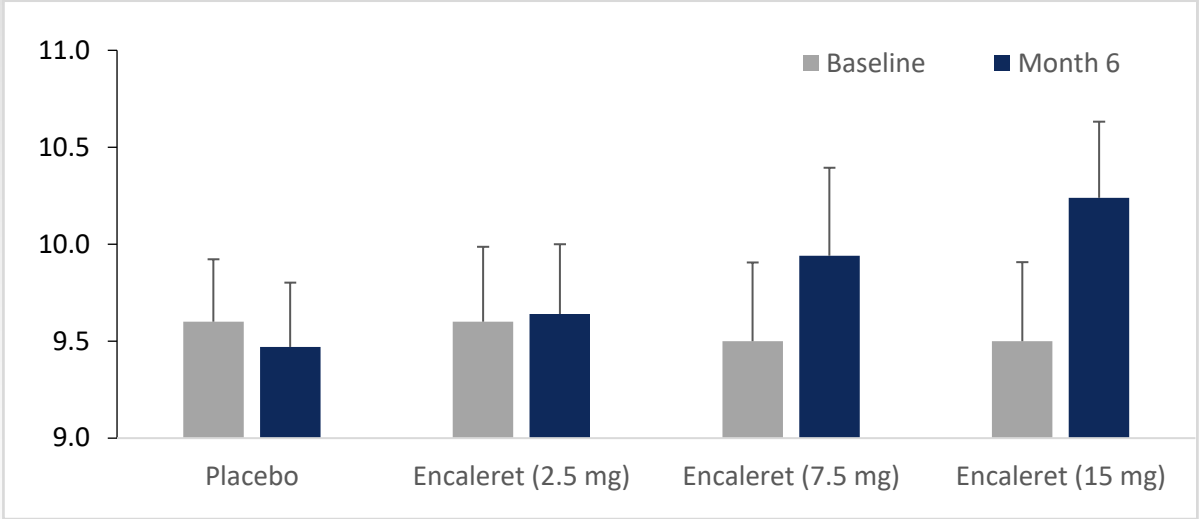
Source: Pearce et al. Clin Endocrinol (Oxf).1996. PTH values reported as below detection limit or undetectable were recorded as "0"

Encaleret proof of mechanism in a mouse model of ADH1 and in humans with wild-type CaSR

Encaleret normalized serum and urine calcium in a mouse model of ADH1[1]



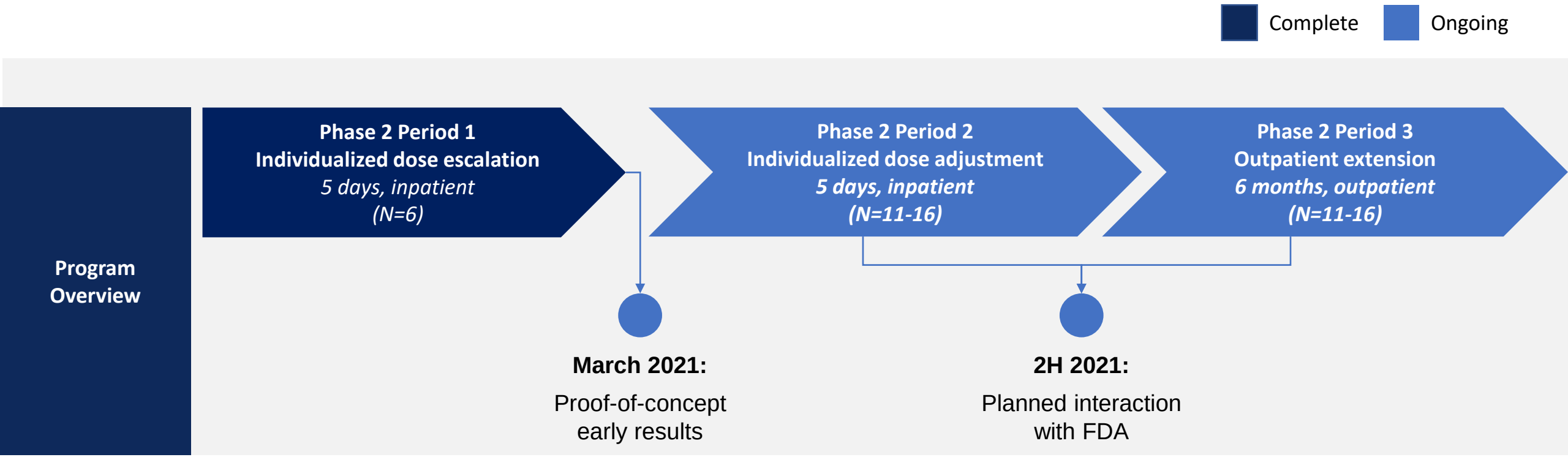
Encaleret was well-tolerated and increased serum calcium in clinical trials in patients with osteoporosis[2, 3]



- In prior osteoporosis development program (>1,200 participants), dose-dependent increases in mean serum calcium were observed
- Encaleret was well-tolerated; hypercalcemia events were more frequent among participants receiving higher doses
- Increasing serum calcium levels is target effect in ADH1

Sources: 1 Dong B., et al. J Bone & Min 2015; #p<0.05 compared to baseline data; *p<0.05 compared to control animals; 2 Halse J., et al. J Clin Endocrinol Metab 2014; 3 Data on File; 4 Cosman F., et al. Osteoporos Int 2015

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)

Encaleret Ph baseline characteristics

Characteristic	Encaleret N = 6	*Normal Range*
Age, mean (range)	40 (22-60)	
Female, n (%)	3 (50%)	
Nephrocalcinosis, n (%)	4 (67%)	
ECG QT_cB (msec)	452 ± 9	*< 440*
Corrected Calcium (mg/dL)*	7.6 ± 0.6	*8.4 –10.2*
Intact PTH (pg/mL)*	3.4 ± 4.5	*15 – 65*
Phosphate (mg/dL)*	4.5 ± 0.7	*2.5 – 4.5*
Magnesium (mg/dL)*	1.6 ± 0.4	*1.6 – 2.6*
24h Urine Calcium (mg/24h)	436 ± 255	*< 250-300*
Supplements		
Elemental Calcium (mg/day) [mean (range)]	2317 (800-4000)	
Calcitriol (µg/day) [mean (range)]	0.9 (0.5-2.0)	
***CASR* Variants**	C131Y (2), P221L (2), E604K (1), A840V (1)	

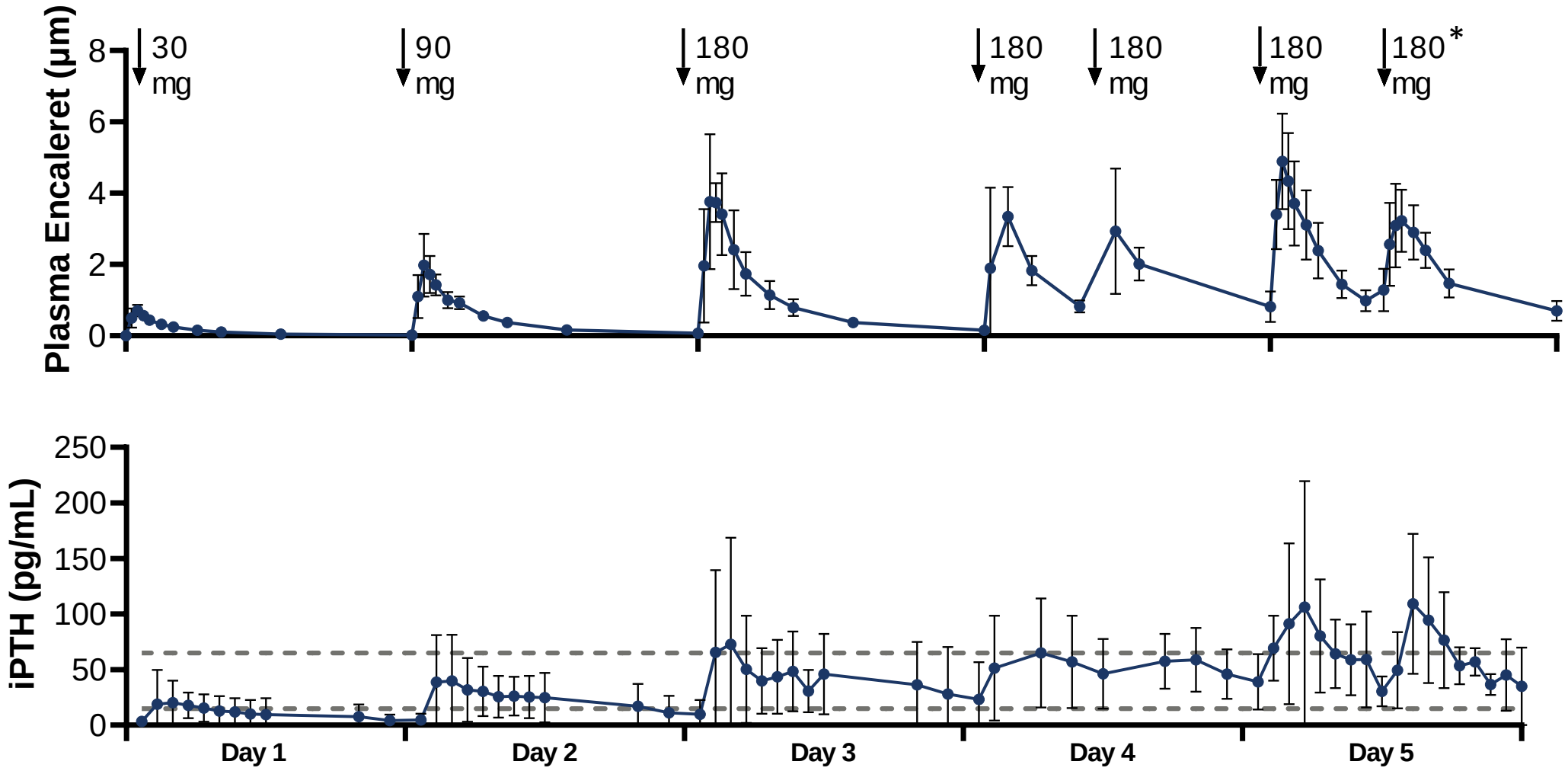
ECG QT_cB = electrocardiogram Bazett-corrected Q-T interval
*Measurements taken pre-dose Day 1 (mean ± SD).

Encaleret was generally well-tolerated with no serious adverse events reported after 5 days

	N = 6
Number of subjects experiencing any Serious Adverse Event	**0 (0%)**
Number of subjects experiencing any Adverse Event	**5 (83%)**
Mild	5 (83%)
Moderate	0 (0%)
Severe	0 (0%)
Number of Adverse Events Reported	**9**
Mild	9 (100%)
Moderate	0 (0%)
Severe	0 (0%)

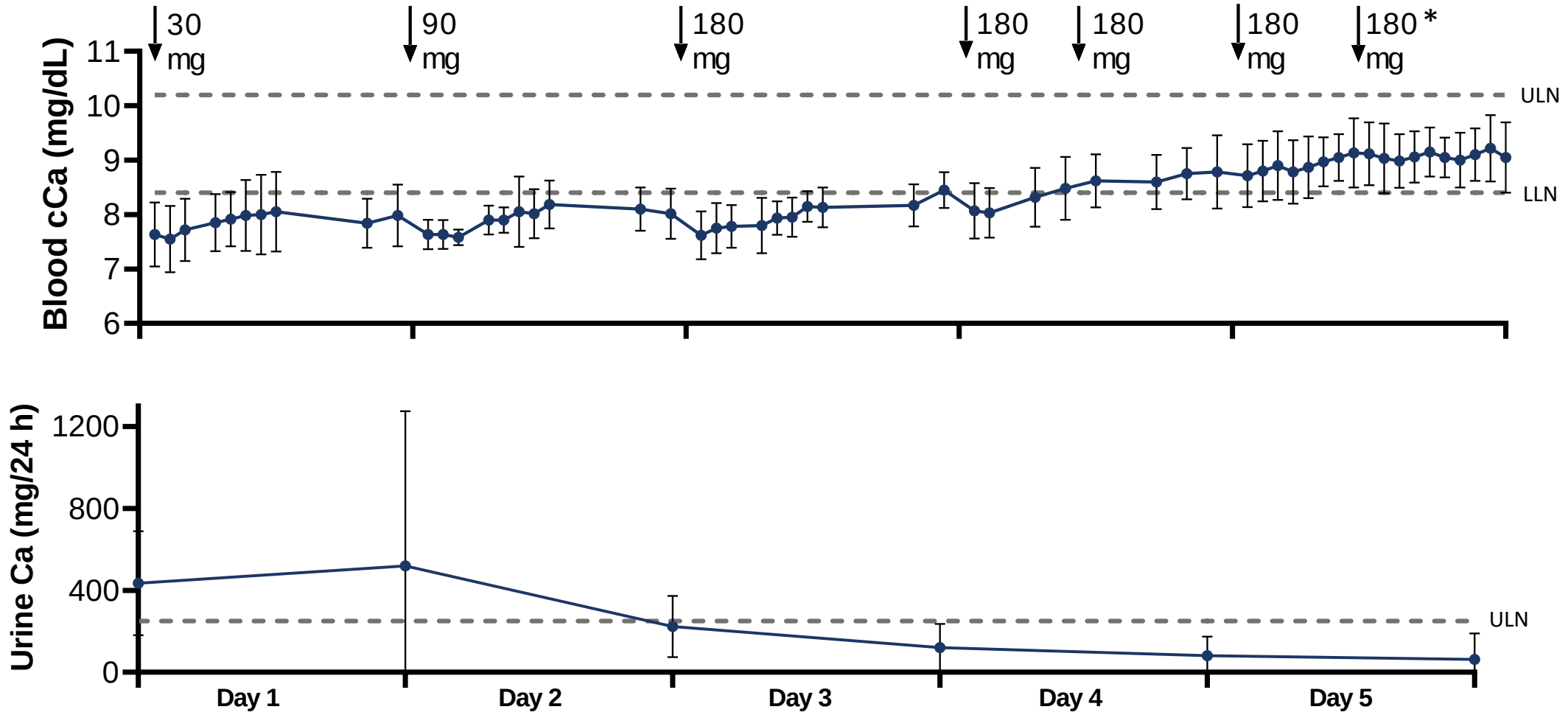
Only treatment-related AE was mild, transient, asymptomatic hypophosphatemia (<2 mg/dL) in 2 subjects

Dose dependent-increases in PTH mirrored encaleret levels



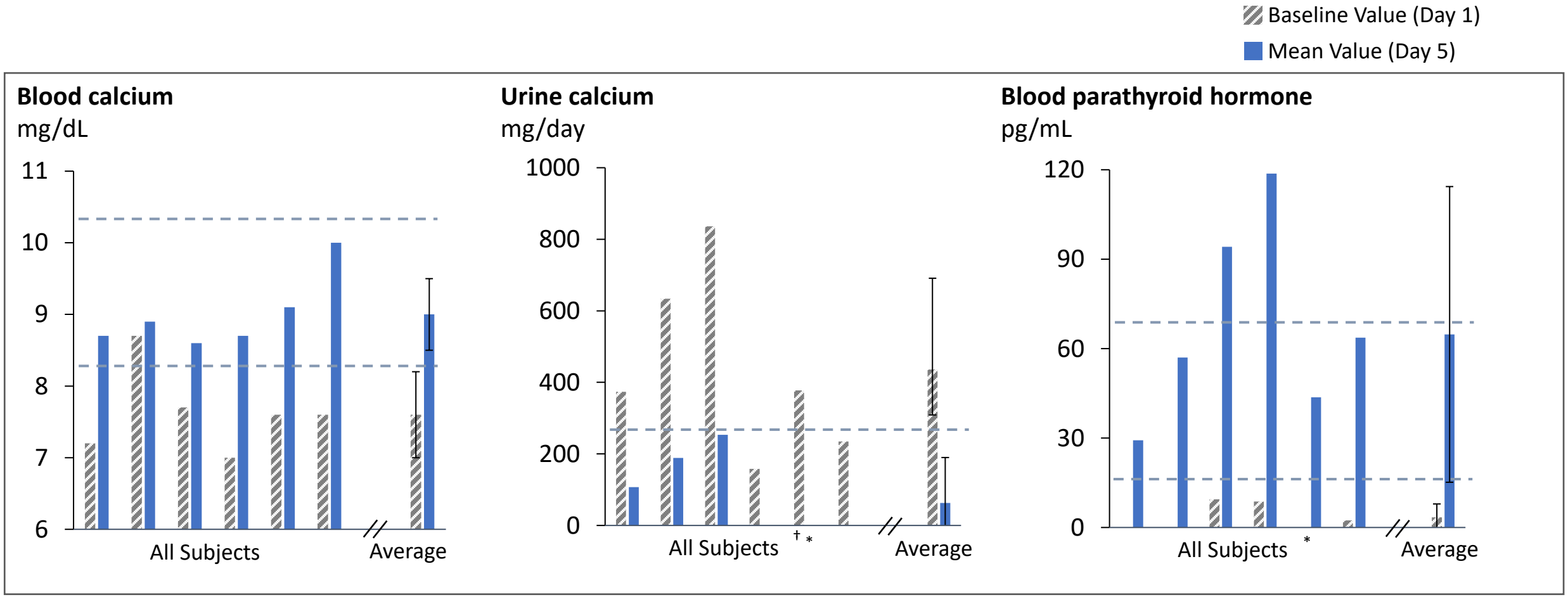
Data shown as mean ± SD. *One subject reduced second dose on Day 5 to 120 mg. Dashed line reflects normal range of PTH 15-65 pg/mL.

Encaleret normalized blood and urine calcium



Data shown as mean ± SD. Values below limit of assay quantitation were marked as "0". *One subject reduced second dose on Day 5 to 120 mg. Dashed line reflects normal ranges: calcium, 8.4-10.2 mg/dL; 24-hr urine calcium, < 250-300 mg/day.

All trial participants had normal blood and urine calcium by Day 5



*Values below limit of assay quantitation recorded as "0". † Day 4 values used in two subjects given Day 5 values unavailable.
Dashed lines reflect normal ranges.

Conclusions

- Encaleret was well-tolerated when administered in escalating oral doses once or twice daily over 5 days, with no serious adverse events reported and no adverse events of moderate or severe intensity
- Blood calcium, PTH, and phosphate were normalized and maintained within the normal range on average by day 5
- Urinary calcium excretion was reduced to below the upper limit of normal or undetectable in all participants while on encaleret and eucalcemic
- Consistent changes from baseline in blood and urine mineral measurements provide proof-of-concept data that encaleret may be an effective treatment for ADH1
- Data support further development of encaleret in ADH1

Next steps for encaleret include generating further evidence in ongoing Phase 2 study

2020

- ✓ Initiate Phase 2 study in ADH1
- ✓ Receive ODD from FDA for ADH

2021

- ✓ Report Phase 2 proof-of-concept results
- ❑ Complete enrollment of Cohort 2 in Phase 2 study
- ❑ Interaction with FDA

Planned activities

- Phase 3 registrational study in ADH1
- Pediatric development program in ADH1
- Evaluation of encaleret in non-genetic hypoparathyroidism

Art
ATTR-CM patient

Acoramidis (AG10) for transthyretin (TTR) amyloidosis (ATTR)

ATTR overview

Prevalence

400,000+ worldwide, largely undiagnosed today

Genetic driver

Destabilizing TTR variants or factors of aging, leading to amyloid accumulation

Pathophysiology

Systemic disease most commonly presenting as cardiomyopathy or peripheral neuropathy

Features of a potential best-in-class medicine for ATTR

Near-complete stabilization of TTR, preventing the formation of amyloid deposits

Preserve TTR tetramer, which has known beneficial roles and is highly evolutionarily conserved

Oral dosing, a convenient and flexible solution for ATTR patients and their families

Acoramidis was designed to treat ATTR at its source

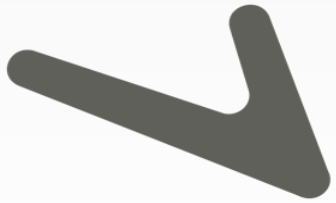
Disease mechanism

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

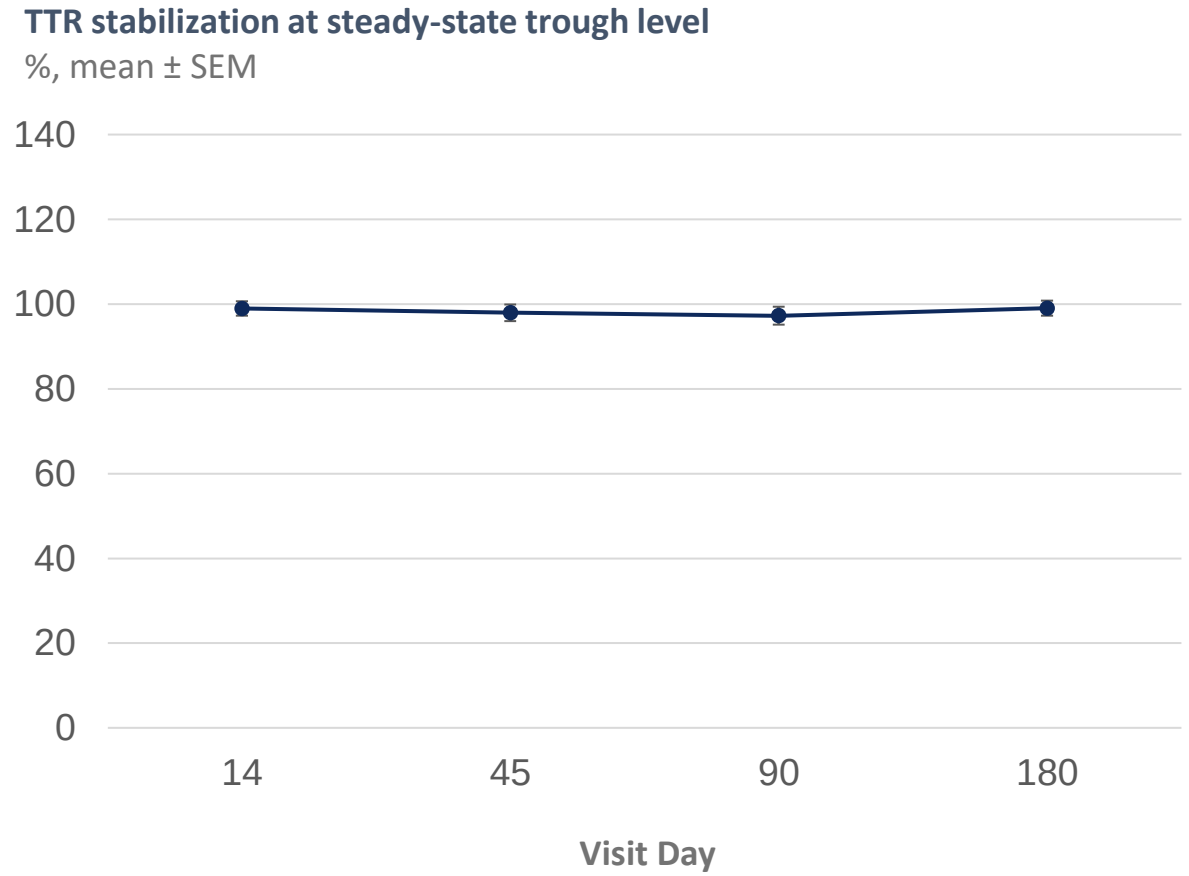
Acoramidis was designed to mimic protective T119M mutation by stabilizing TTR tetramers to slow or halt disease progression

Acoramidis has been well-tolerated and demonstrated near-complete TTR stabilization in pre-clinical, Ph1, and Ph2 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%)

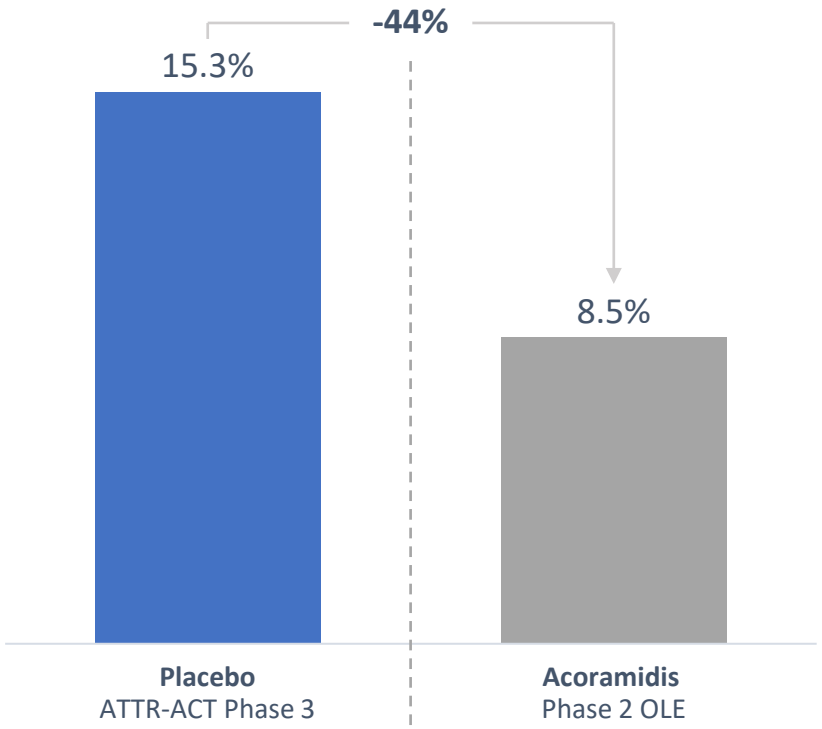
Phase 2 TTR stabilization[2]



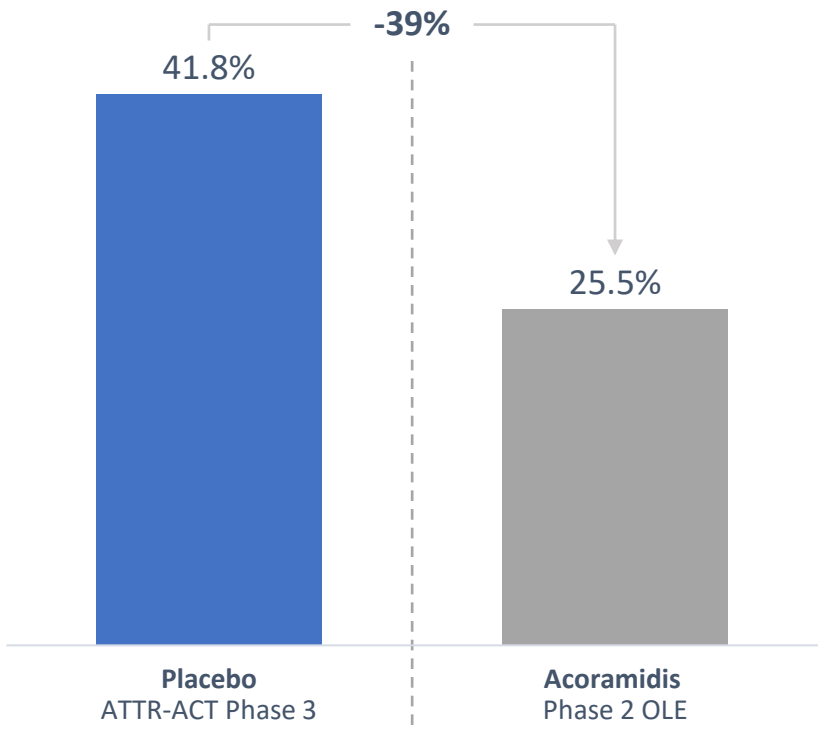
1 Judge, D.P. et al. JACC Vol. 74, No. 3, 2019:285 – 95
2 Judge, D.P. et al. American Heart Association 2019

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 (%)

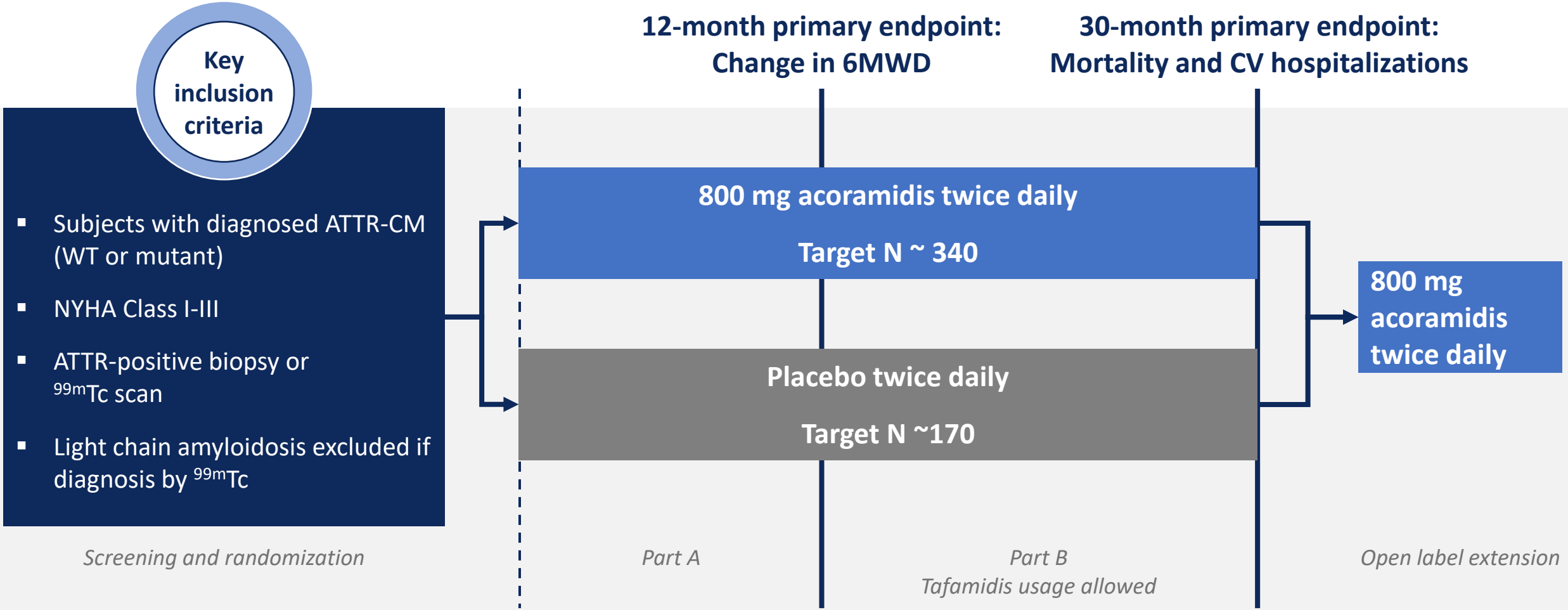


Cardiovascular hospitalizations at 15 months
Participants with ≥1 CV hospitalization (%)



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

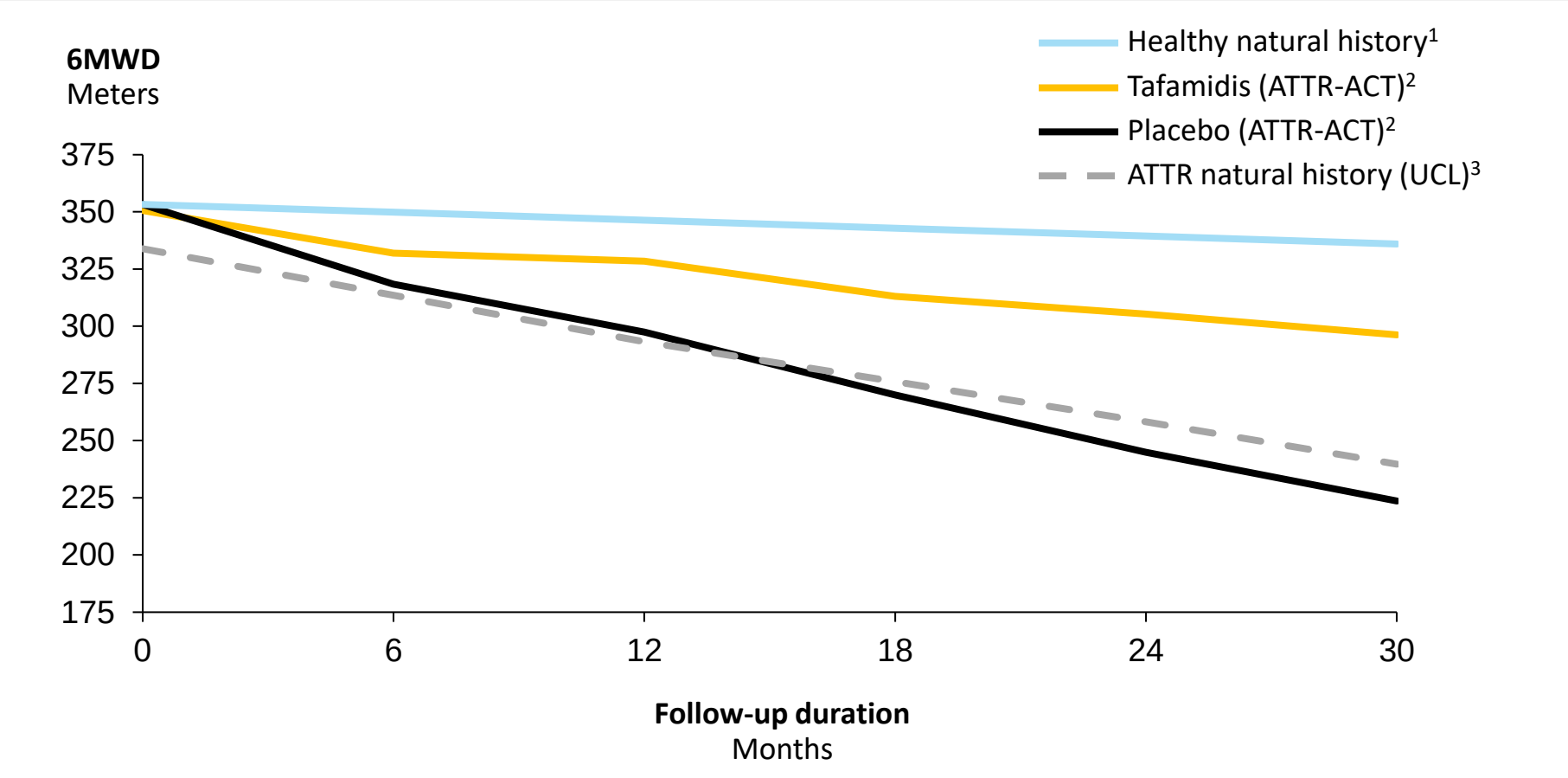
ATTRibute-CM will provide 12-month functional outcome data and 30-month mortality and CV hospitalization data



6MWD = Six-minute walk distance; 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 6MWD data in ATTR-CM and healthy cohorts



Optimal profile for acoramidis would markedly slow or halt decline in 6MWD in trial participants

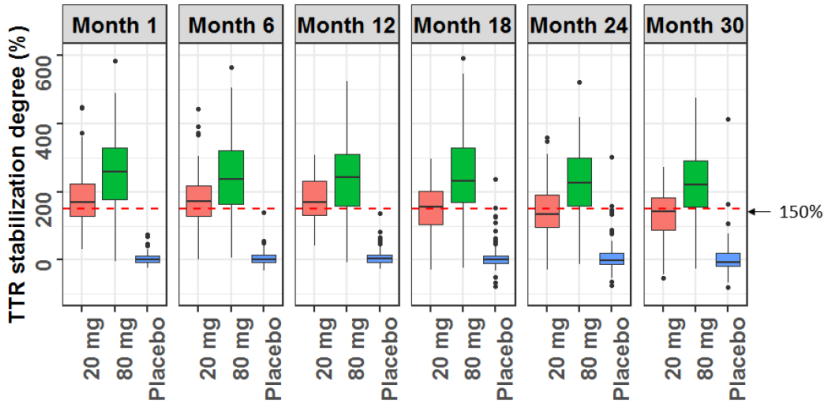
1. Enright, P.L. et al. Chest 2003. N = 3333 healthy elderly adults, baseline set to match ATTR-ACT placebo group
2. Maurer, M.S. et al. NEJM 2018. N = 264 (tafamidis), N = 177 (placebo) ATTR-CM trial participants
3. Lane, T.L. et al. Circulation 2019. N = 1034 ATTR-CM patients

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

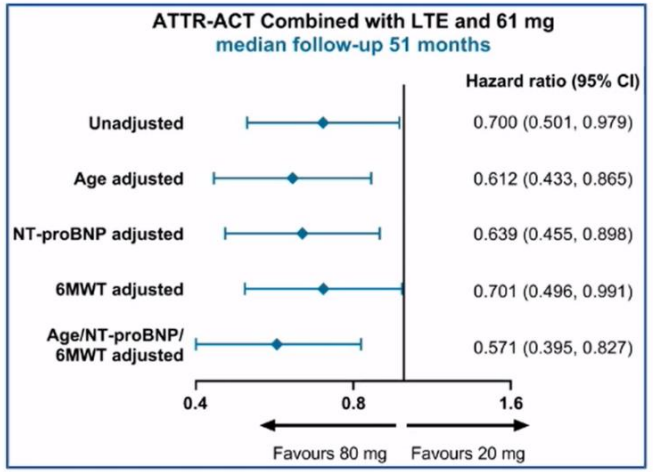
Phase 3 ATTR-ACT study tested two doses of tafamidis (20 mg & 80 mg) vs. placebo

- 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

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 late 2021 or early 2022

2021

ATTRibute CM

ATTR-CM
WT and hereditary
Functional outcomes

2023

ATTRibute CM

ATTR-CM
WT and hereditary
Functional outcomes
+
Composite mortality and morbidity

2024

ATTRibute PN

ATTR-PN
Hereditary
Functional outcomes

ATTRibute CM

ATTR-CM
WT and hereditary
Functional outcomes
+
Composite mortality and morbidity

2025+

Prevention in high risk populations

Head-to-head comparisons

ATTRibute PN

ATTR-PN
Hereditary
Functional outcomes

ATTRibute CM

ATTR-CM
WT and hereditary
Functional outcomes
+
Composite mortality and morbidity

Claudia,
child with achondroplasia

Low-dose FGFR inhibitor (infigratinib) for achondroplasia

Achondroplasia overview

Prevalence

55,000 (US+EU) –
one of the most common genetic conditions

Genetic driver

FGFR3 activation

Pathophysiology

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

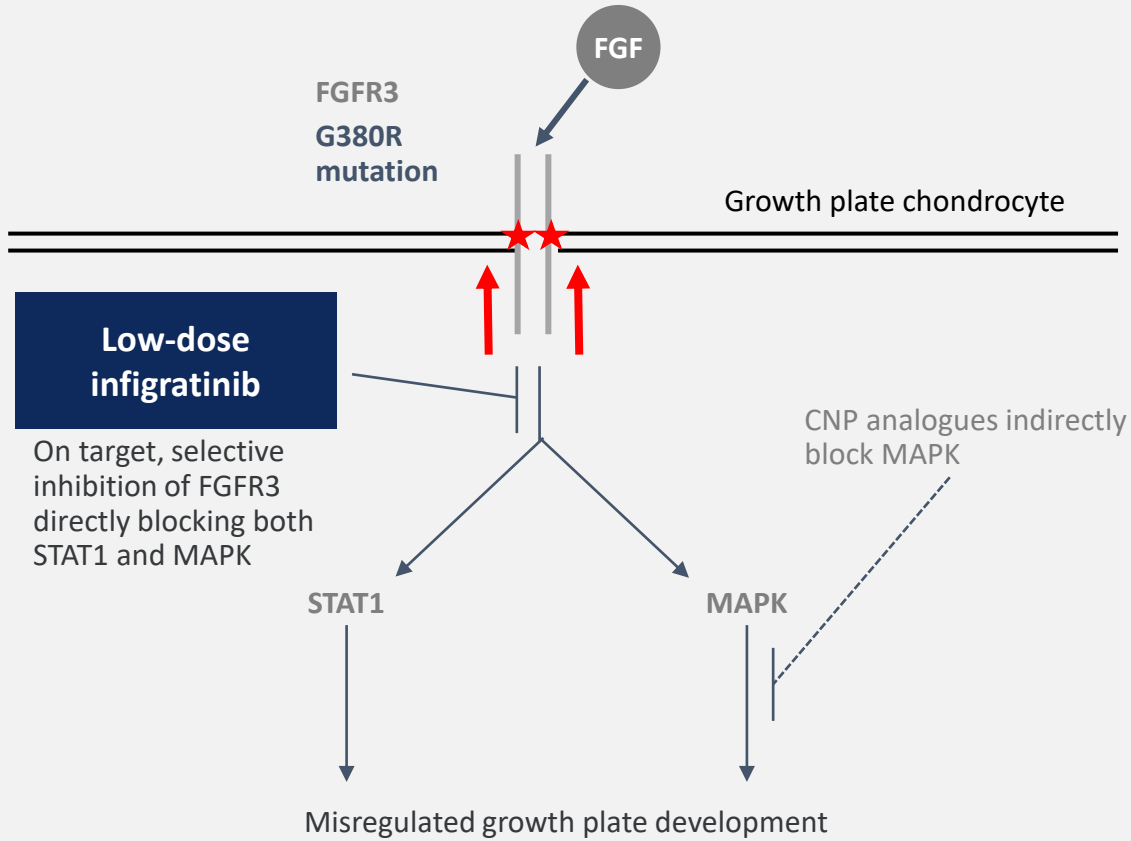
Features of a potential best-in-class medicine for achondroplasia

Direct targeting of FGFR3 and normalization of both STAT1 and MAPK signaling pathways

Potential to address all drivers of symptoms, including cranial, spinal and stature issues

Oral dosing, the most convenient solution for children with achondroplasia and their families

Potential best-in-class approach targeting achondroplasia directly at its genetic source



ACH FGFR3 gain-of-function mutation causes:

- 2-3x over-activation of the receptor
- Up-regulation of downstream pathways STAT1 and MAPK
- Aberrant growth plate development, which causes cranial, spinal, and stature symptoms

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

Low-dose infigratinib improves all the key drivers of clinical symptomology in validated ACH mouse model

FGFR3 WT
No treatment

FGFR3$^{Y367C/+}$
No treatment

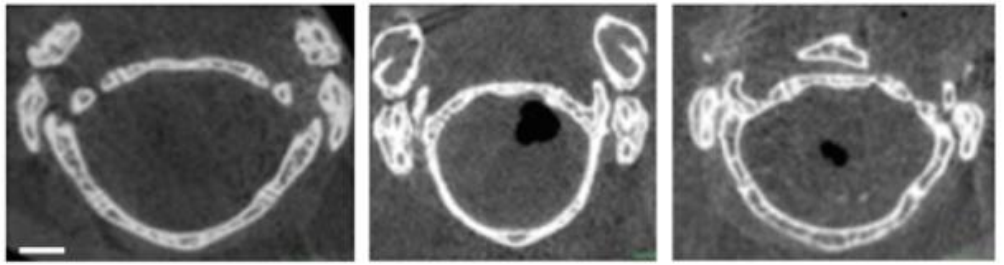
FGFR3$^{Y367C/+}$
Infigratinib tx

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

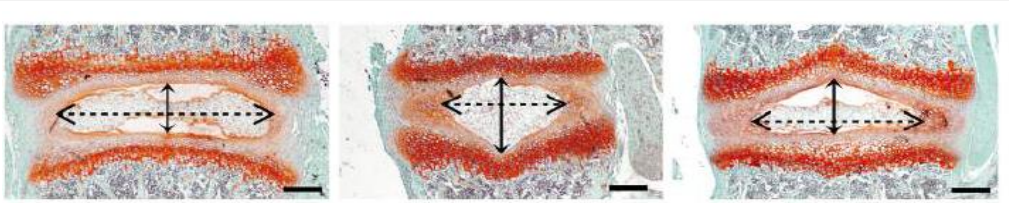


2 Disorders of the spine

12% increase in L4-L6 length

73% increase in disc width

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

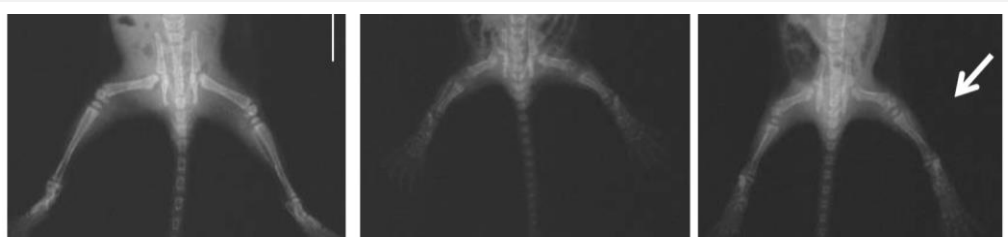


3 Disproportionate short stature

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

Low-dose infigratinib showed potential best in-class preclinical profile in validated achondroplasia mouse model

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%		

Preclinical data from infigratinib and other investigational achondroplasia therapies

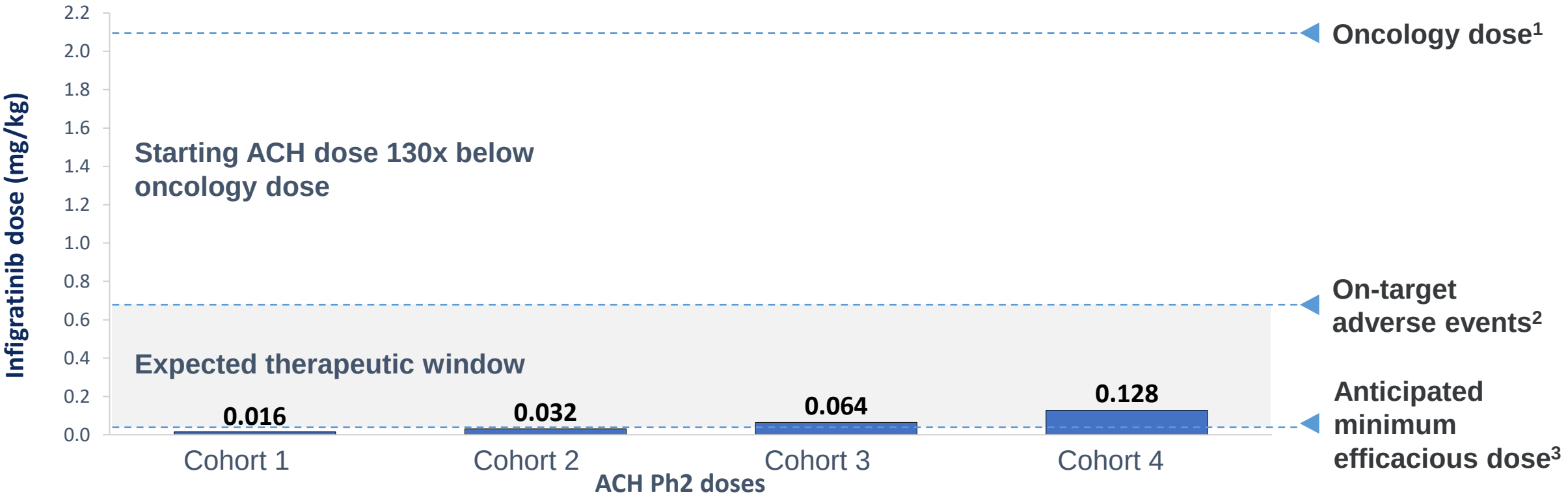
Percent increase compared to non-treated mouse

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.

We have a wide anticipated therapeutic index in achondroplasia

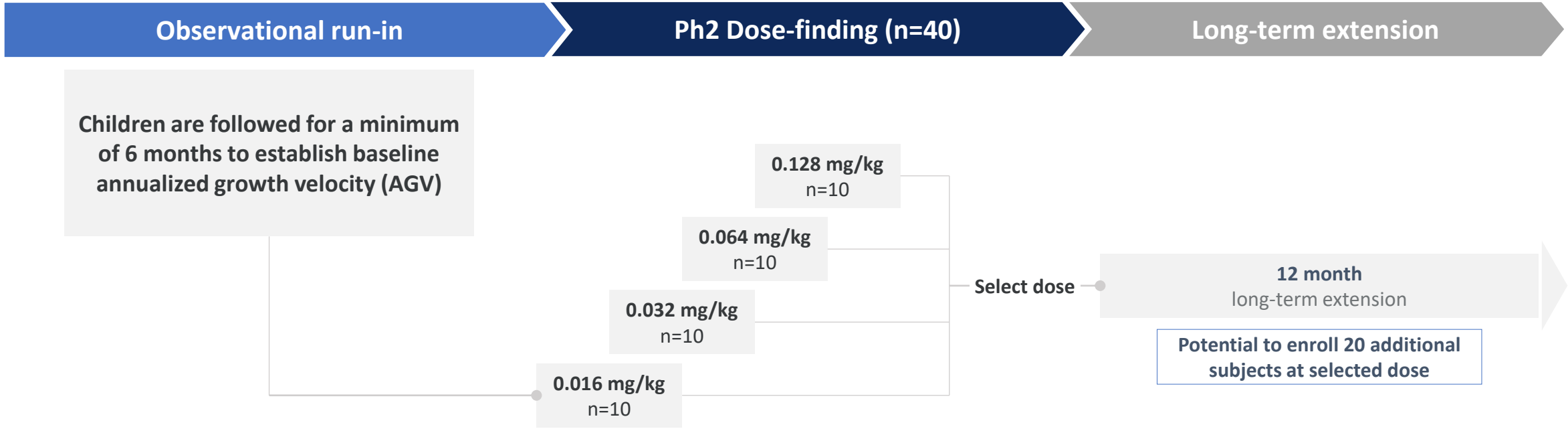
Infigratinib has been tested in >700 humans in our oncology program, providing significant data on PK, tolerability and safety

Most common and dose-limiting side effect is phosphorus elevation (on-target through FGFR1 inhibition), which occurs significantly above our planned achondroplasia doses



[1]Based on 125mg dose and 60kg adult; [2]Based on estimated TD_{50} at 40mg and 60kg adult; [3]Based on PK modeling and allometric scaling from animal models

The PROPEL clinical program is enrolling with data expected in 2H 2021



Key inclusion criteria

- Children 2.5 – 10 years old
- Clinical and molecular ACH diagnosis

Primary objectives

- Baseline annualized growth velocity (AGV)

Primary objectives

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

Primary objectives

- Long-term safety and efficacy

Maris,
child with CAH

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

Program overview

Prevalence

75,000 (US+EU) – One of the largest known AAV gene therapy markets

Genetic driver

21-hydroxylase inactivation

Pathophysiology

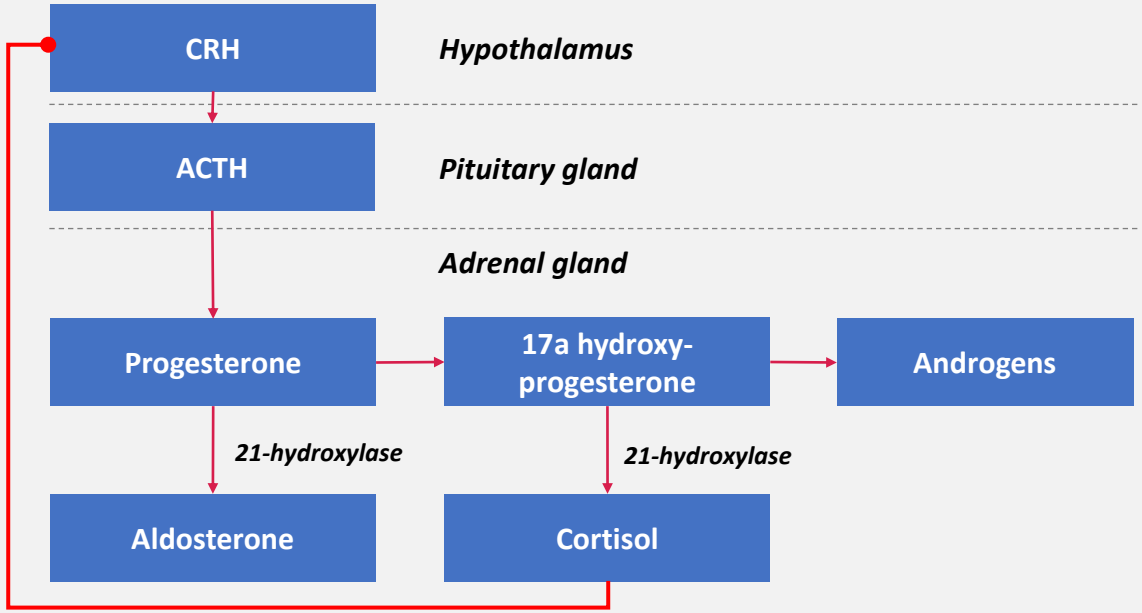
Inability to produce cortisol causes need for supraphysiologic doses of synthetic steroids, 3x increase in mortality risk, hirsutism, Cushingoid symptoms

We believe CAH is an ideal indication for AAV gene therapy:

- **Low threshold to correct phenotype**, validated by human clinical genetics (~5-10% of WT enzyme activity)
- **Only approach designed to induce endogenous cortisol and mineralocorticoid production**, potentially allowing steroid withdrawal
- **Durable transgene delivery to the adrenal gland of NHPs** with IV dosing of our construct
- **Next catalyst:** initial data from first-in-human study

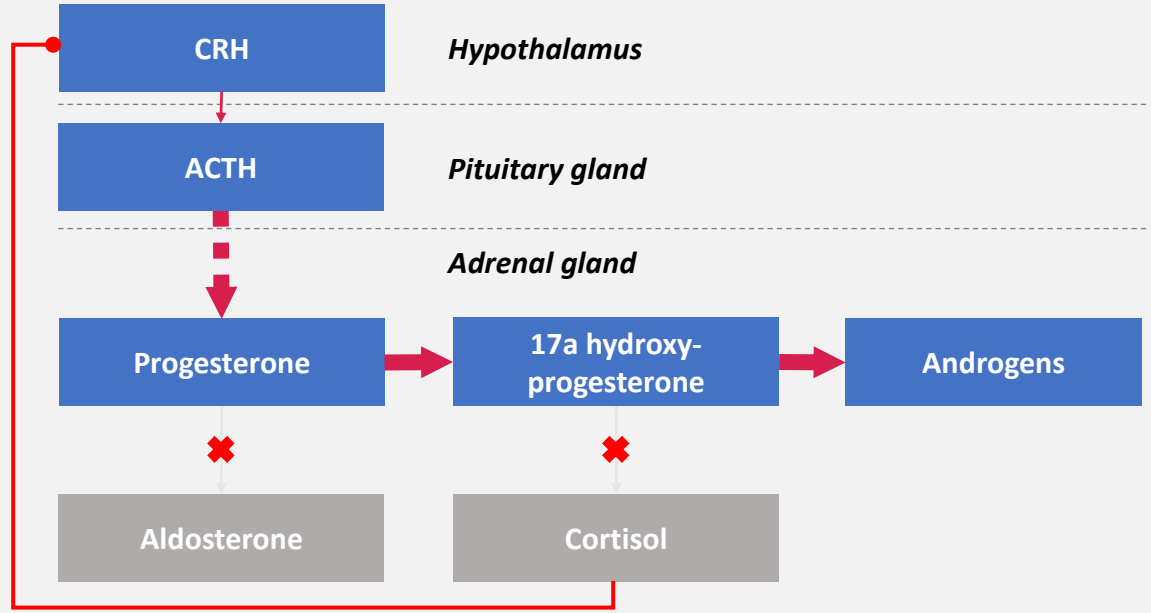
Gene therapy is the only modality designed to treat CAH at its source and allow for production of endogenous cortisol

Healthy Hypothalamic-Pituitary-Adrenal Axis



In a functional HPA system, cortisol and aldosterone are produced as needed by the body. Cortisol serves as a “brake” on the CRF/ACTH system

Hormonal dysregulation with 21OHD; no cortisol “brake” on ACTH, shunting of 17OHP to androgens

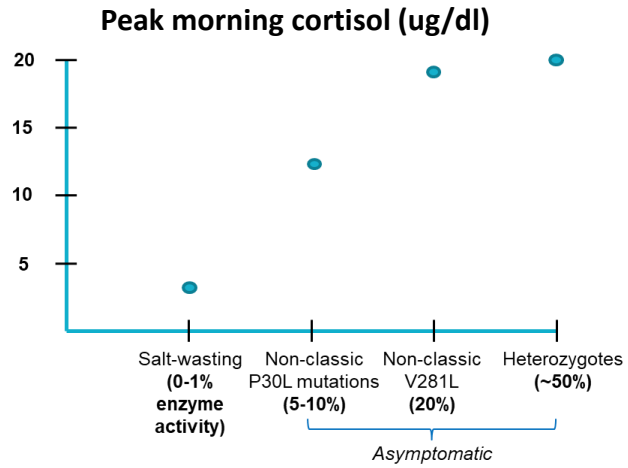


In CAH, cortisol and aldosterone are not able to be produced. The lack of a “cortisol brake” results in buildup of progesterone and 17OHP, leading to an excess of androgen production

CAH patients have 3-4X higher mortality than the general population, and suffer significant morbidity ranging across cardiovascular and metabolic disease, bone disease, infertility, chronic fatigue, and other disorders.

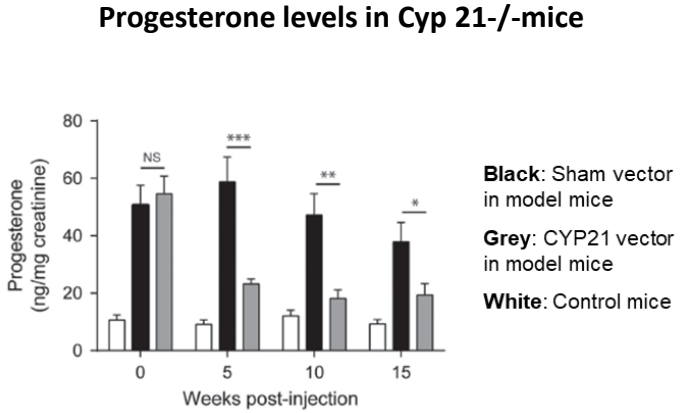
CAH: NHP study showed durable transgene expression; 5-10% of WT enzyme may be sufficient for clinical impact

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



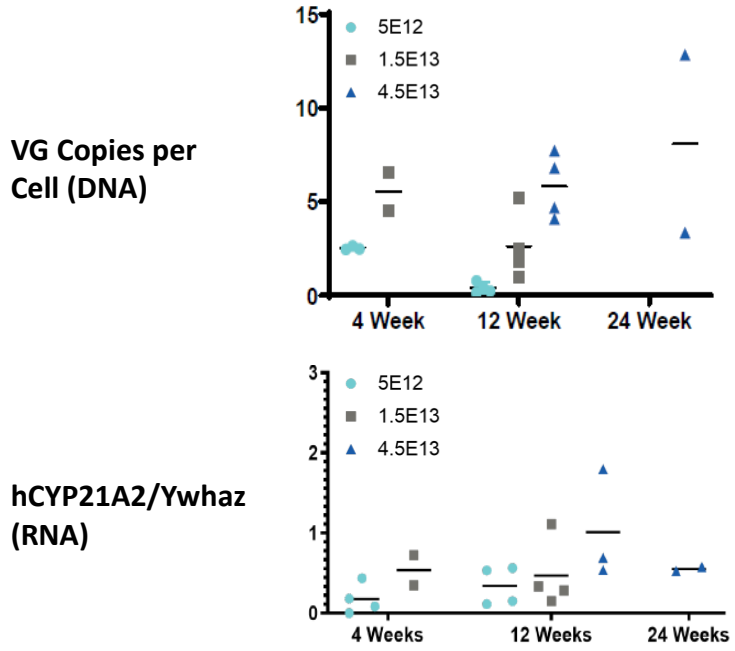
- Due to the high enzymatic efficiency/selectivity of 21-OHase, **only a small amount of enzyme is required to rescue the phenotype**

Mouse studies show a VGC of only 0.13 at 18 wks was sufficient for phenotypic correction



- At 15 weeks in treated mice, **progesterone** (the key substrate of 21OHase in mice) was **significantly reduced vs untreated mice**

NHP studies show sustained VGC and RNA out to 6 months

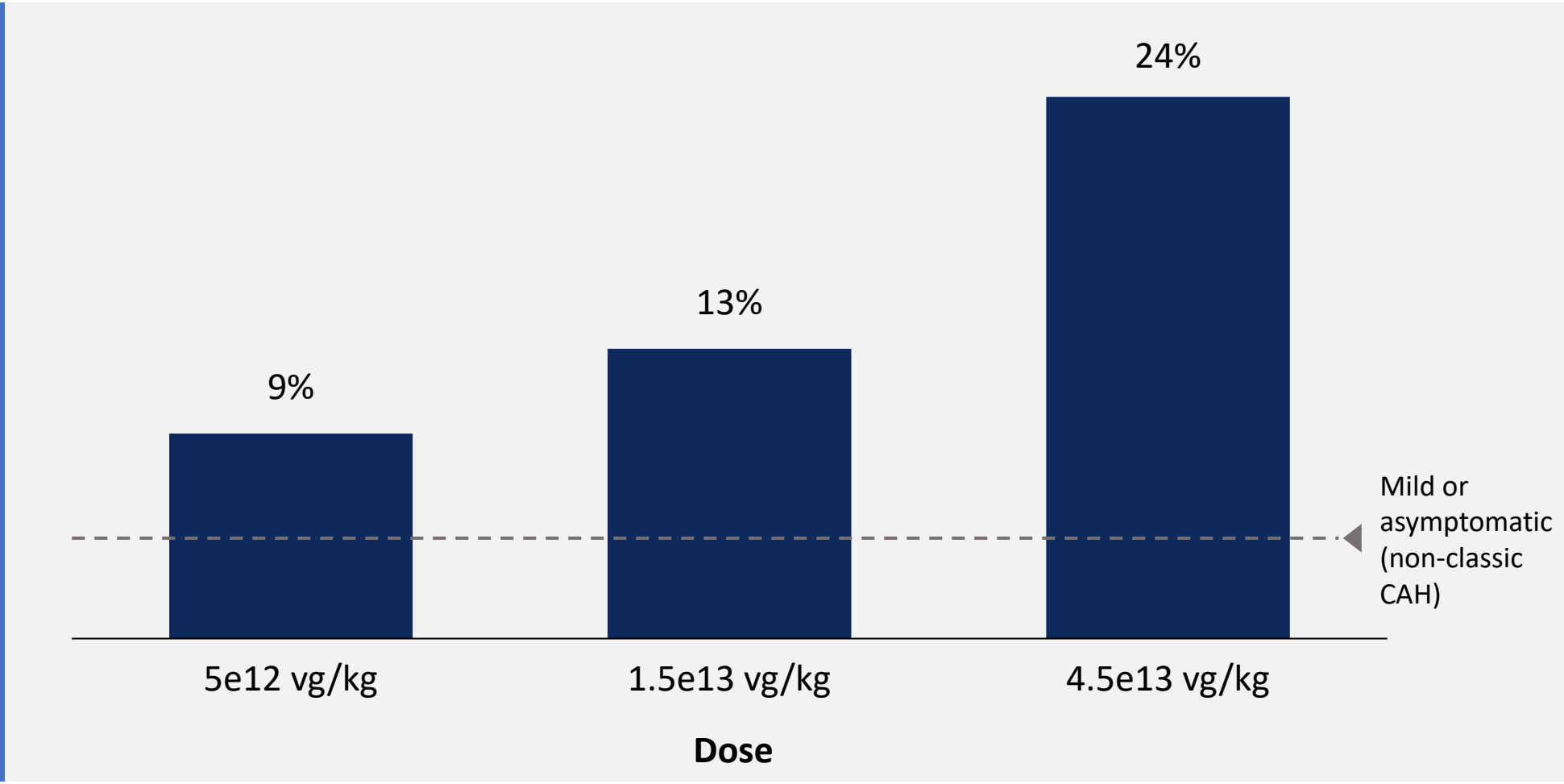


- Transgene expression is dose-dependent and stable out at 24 wks
- **We can durably transduce the NHP adrenal gland with our construct at >20x the vector required to correct the CAH phenotype in mice**

Source: Perdomini, Gene Therapy 2017; ESGCT 2019

NHP protein data using mass spec methods suggests potentially therapeutic levels of 21-hydroxylase enzyme

- We have developed mass-spec methods to quantify protein expression by identifying differential peptides between human and NHP 21-OH
- These data suggest dose-dependent enzyme expression in the adrenal cortex from 9%-24% of WT levels
- Genotype-phenotype relationship suggests as little as 5% of WT enzyme activity is associated with the mild/asymptomatic non-classic form of CAH



Source: Data on file

Basia, pancreatic cancer patient (>90% KRAS-driven)

BridgeBio oncology research

World-class oncology team drives our discovery and development

Eli Wallace
CSO Oncology Research

ARRAY BIOPHARMA | Peloton Therapeutics

Pedro Beltran
SVP Oncology

AMGEN | UNITY BIOTECHNOLOGY

Frank McCormick
Chairman of Oncology

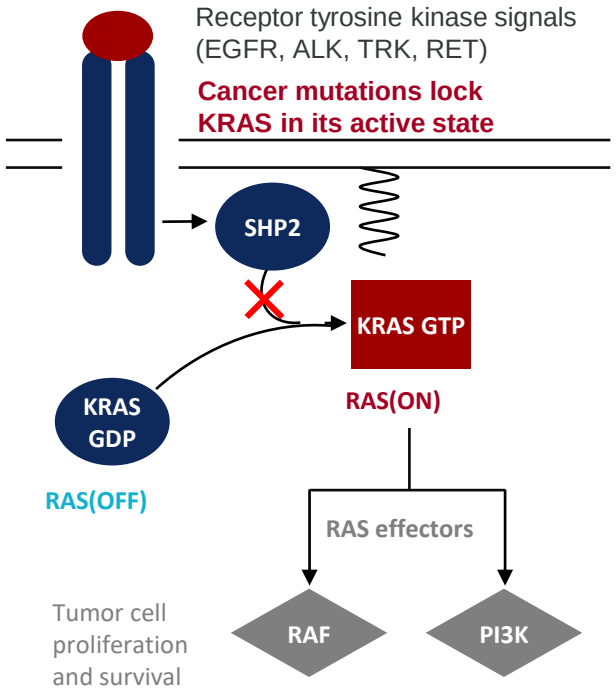
ONYX PHARMACEUTICALS | UCSF

Richard Scheller
Chairman of R&D

Genentech | 23andMe

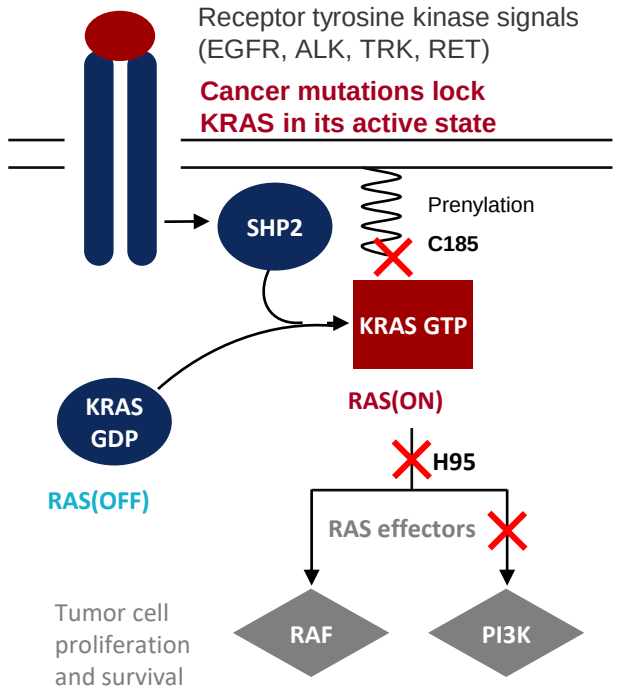
Three disclosed oncology research targets

SHP2 (BBP-398)



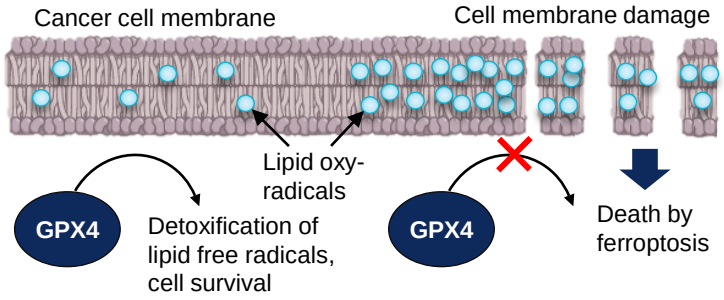
- Potential best in class oral compound
  - Optimized safety, PK and PD profile
  - Maximizes combination therapy potential
- First-in-human study initiated 4Q20

KRAS



- Multiple unexploited sites
- Comprehensive pan-mutant targeting approaches

GPX4



- Potential first in class compound for novel cancer target
- In vivo monotherapy activity and combo potential

Partnerships afford us exceptional collaborators and resources

NIH NATIONAL CANCER INSTITUTE

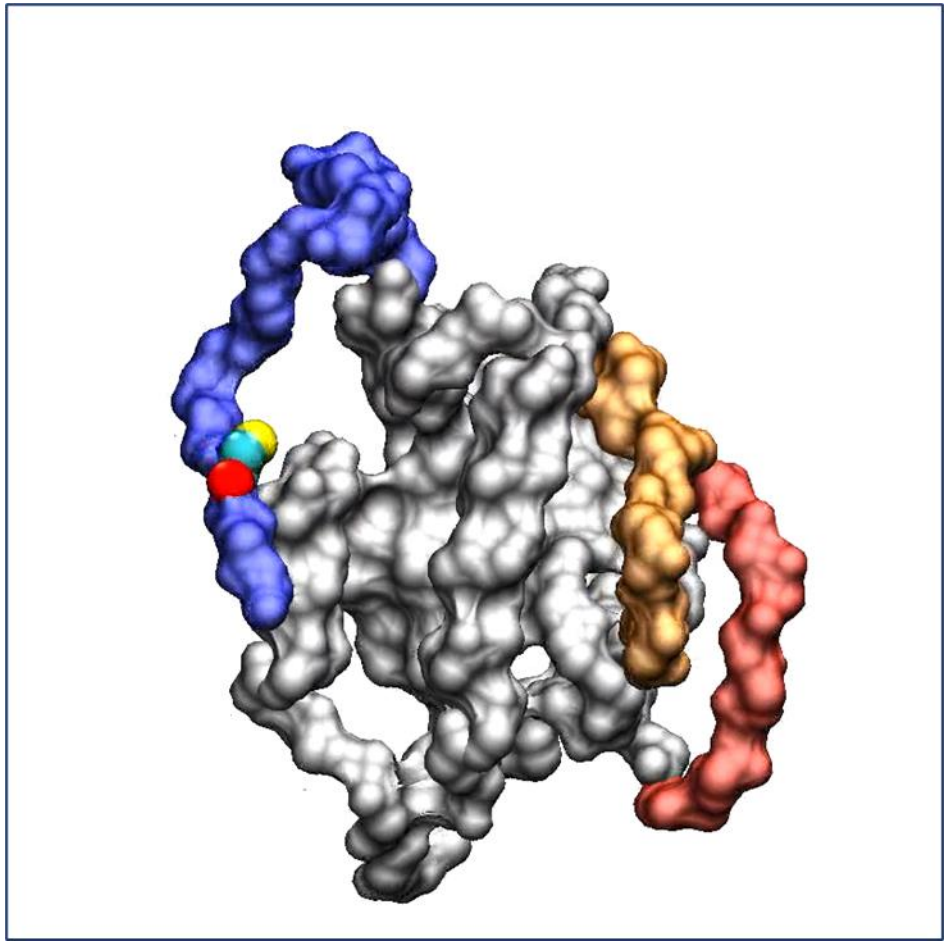
- Partnership with the National RAS Initiative, including 60 of the world's **foremost academic RAS researchers**
- Cutting edge RAS **structural biology expertise**
- Utilization of **cutting-edge instrumentation and techniques**, as well as the **expertise** to lead experiments

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**

Crystal structure enables a static understanding of the target …

KRAS4b model based on crystal



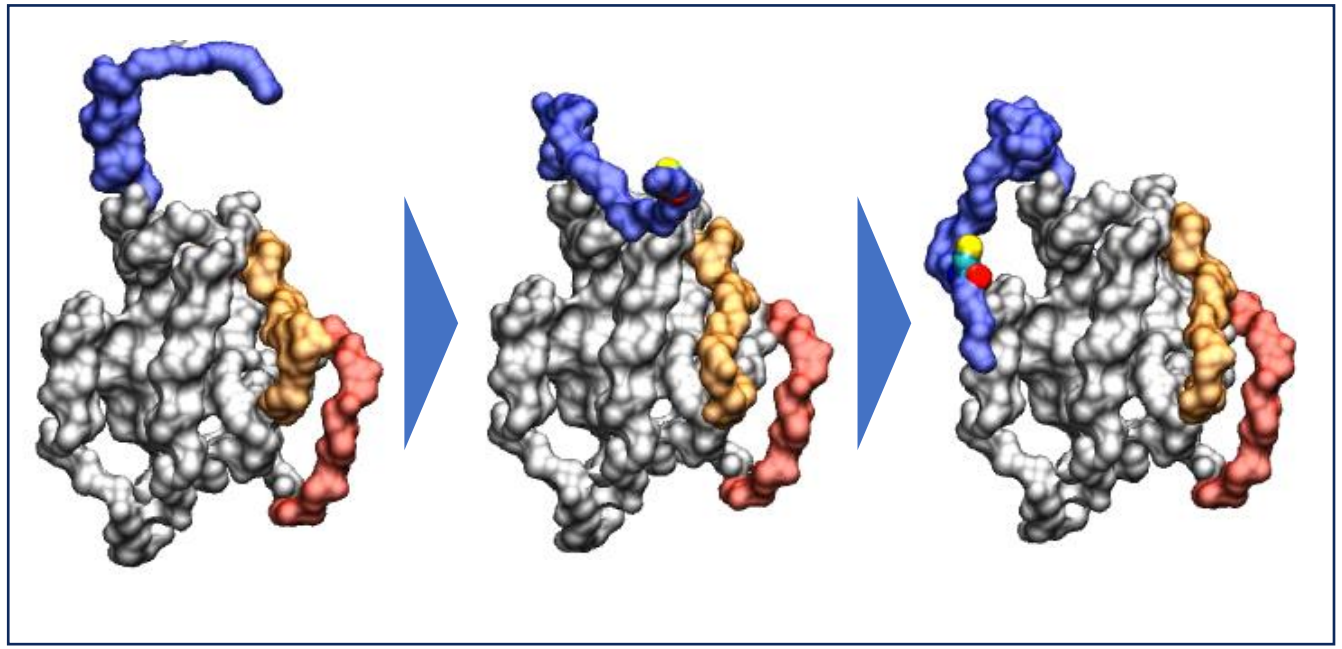
G-domain
G-domain switch I
G-domain switch II
Hypervariable region

One therapeutic approach is to inhibit KRAS4b **membrane localization** by targeting **hypervariable region**

Static model reveals only a **subset of potential binding sites** for pharmacological compounds

… whereas molecular dynamics simulation reveals transient conformations and interactions

KRAS4b simulation



G-domain
G-domain switch I
G-domain switch II
Hypervariable region

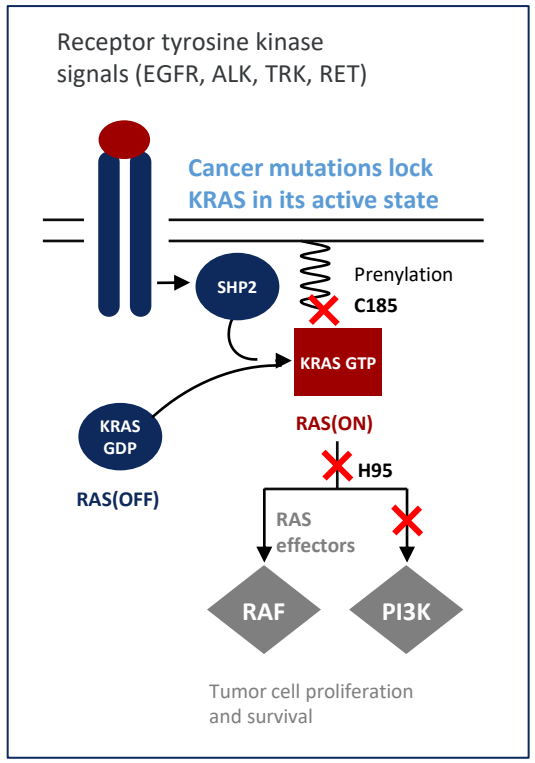
Reveals possible KRAS4b **HVR transient localization to G-domain**

Elucidates potential transient druggable pocket where **compounds could react covalently with C185**

Enables *in silico* SAR to **inhibit KRAS4b membrane localization**

KRAS: multiple shots on goal with our pan-mutant inhibitor programs – each with a unique MOA targeting a novel pocket

KRAS pathway in cancer

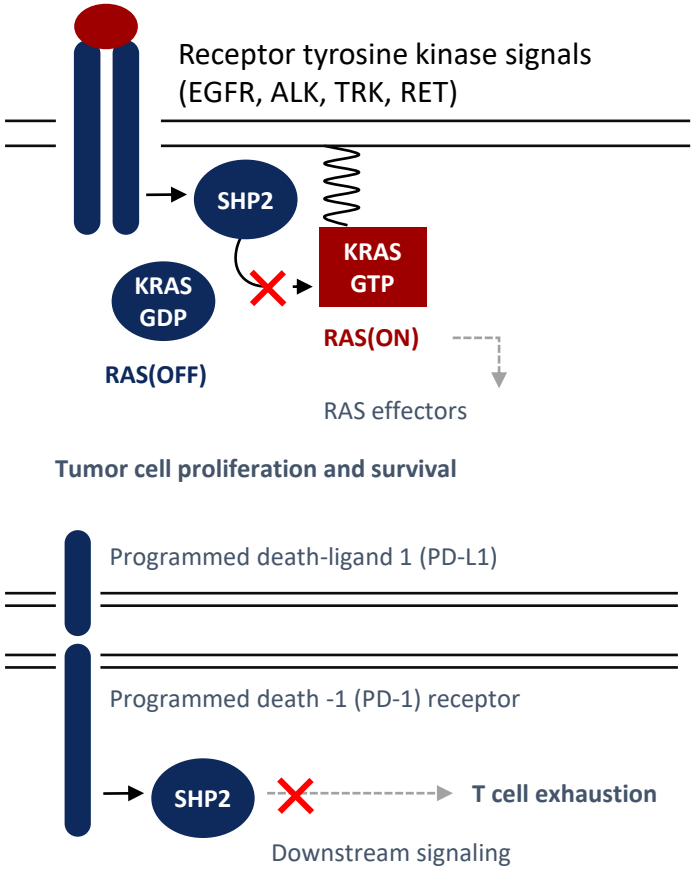


Program	MOA	Targets KRAS GTP	Pan-mutant	Crystal structure	Molecular Dynamics
Program 1: H95 targeting	▪ Directly binds activated KRAS through H95 ▪ Inhibits KRAS from signaling through effectors	✓	✓	✓	✓
Program 2: PI3K effector blocking	▪ Blocks specific interaction between KRAS and PI3Ka ▪ Blocks PI3K / AKT effector signaling	✓	✓	✓	✓
Program 3: C185 targeting	▪ Blocks KRAS from tethering ▪ Blocks conversion of inactive KRAS GDP to active KRAS GTP	✓	✓		✓

Our programs are designed to address all KRAS driver mutations, which occur in >30% of all cancers

SHP2: Our compound shows best-in-class potential

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: ~15 hours[1]

- Allows for recovery above EC50 and reduced MAPK-driven tox

Potentially differentiated safety profile for combination therapy

- hERG IC50 (µM)*: >100: No QT prolongation

SHP2i combination potential

US + EU incidence, '000s

Supporting evidence[2]

Peclin data:

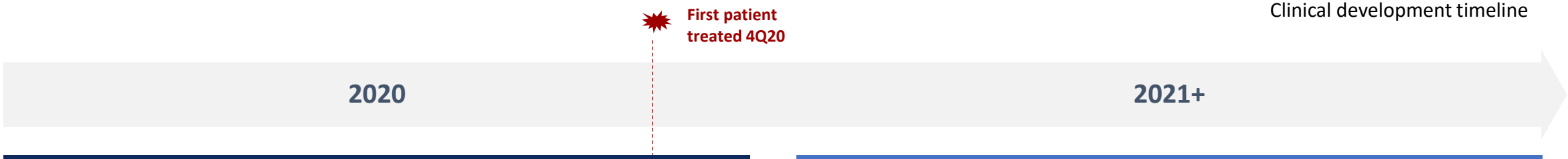
~1 million patients annually

1 Predicted human PK based on preclinical in vivo data 2 Preclinical data of combination efficacy with SHP2i

SOURCE: US incidence estimated from SEER, TCGA and Kiuru & Busam "The NF1 gene in tumor syndromes and melanoma"; all scaled for WW incidence

SHP2: BBP-398 monotherapy study initiated in 2020; combo trials to follow

Clinical development timeline



Mono therapy

- **Monotherapy study initiated 2H 2020**
- Dose escalation followed by dose expansion
- Starting dose 80mg

Combo Therapy

- **Partnered with Perceptive-backed LianBio in China for initial combo therapy trials**
- Dose escalation followed by dose expansion
- Priority combinations include osimertinib, and G12Ci in NSCLC

Initial clinical combinations of focus based on SHP2i preclinical data

	SHP2i Combination Partner	Tumor growth inhibition
KRAS G12Ci	AMG 510	**~130%**
EGFRi	Osimertinib	**~125%**
PD-1	Anti-mouse PD-1	**~90%**
MEK	Trametinib	**~80%**
CDK4/6 and MEK	Trametinib + palbociclib	**~110%**

2021 is a pivotal year with major catalysts across the pipeline

ANTICIPATED

2021		2022
1H	2H	FY
☑ **BBIO / EIDX merger closure:** Shareholder meeting January 19	☐ **Acoramidis (ATTR stabilizer) for ATTR-CM:** Ph3 ATTRibute topline data	☐ **Acoramidis (ATTR stabilizer) for ATTR-CM:** NDA submission
☑ **NULIBRY (fosdenopterin) for MoCD type A:** FDA approval	☐ **Low-dose infigratinib (FGFRi) for achondroplasia:** Ph2 proof-of-concept data	☐ **KRAS inhibitor program:** Clinical candidate selection
☑ **Encaleret (CaSRi) for ADH1:** Ph2 proof-of-concept data	☐ **AAV5 gene therapy for CAH:** Initial data from first-in-human study (late ’21 / early ‘22)	☐ **SHP2 inhibitor for RAS and RTK driven cancer:** Monotherapy Phase 2 dose selection
☐ **High-dose infigratinib (FGFRi) for second-line cholangiocarcinoma:** FDA approval	☐ **COL7 replacement for RDEB:** Data from Ph2 study (late ’21 / early ’22)	☐ **Ribitol for LGMD2i:** Ph2 proof-of-concept data

$1bn+ in cash and equivalents as of March 2021 anticipated to provide runway into 2023