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# COMPANY PRESENTATION

**June 2020**

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Statements in this Presentation that are not statements of historical fact are forward-looking statements. Such forward-looking statements include, without limitation, statements regarding our research and clinical development plans, expected manufacturing capabilities, strategy, regulatory matters, market size and opportunity, future financial position, future revenue, projected costs, prospects, plans, objectives of management, and the Company's ability to complete certain milestones. Words such as "believe," "anticipate," "plan," "expect," "intend," "will," "may," "goal," "potential" and similar expressions are intended to identify forward-looking statements, though not all forward-looking statements necessarily contain these identifying words. These forward-looking statements are based on the beliefs of the Company's management as well as assumptions made by and information currently available to the Company. Such statements reflect the current views of the Company with respect to future events and are subject to known and unknown risks, including business, regulatory, economic and competitive risks, uncertainties, contingencies and assumptions about the Company, including, without limitation, risks inherent in developing therapeutic products, future results from the Company's ongoing and planned preclinical studies and clinical trials, the Company's ability to obtain adequate financing to fund its preclinical studies and planned clinical trials and other expenses, trends in the industry, the legal and regulatory framework for the industry and future expenditures. In light of these risks and uncertainties, the events or circumstances referred to in the forward-looking statements may not occur. The actual results may vary from the anticipated results and the variations may be material. These forward-looking statements should not be taken as forecasts or promises nor should they be taken as implying any indication, assurance or guarantee that the assumptions on which such forward-looking statements have been made are correct or exhaustive or, in the case of the assumptions, fully stated in the Presentation. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date this Presentation is given. This Presentation discusses product candidates that are under preclinical study or clinical trial and which have not yet been approved for marketing by the U.S. Food and Drug Administration (the "FDA"). No representation is made as to the safety or effectiveness of these product candidates for the therapeutic use for which such product candidates are being studied.

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# We are building a leading genetic disease company

## Core attributes…

1. **Distinctive early stage asset selection**
2. **Experienced, product-focused R&D team**
3. **Efficient corporate structure**
4. **The willingness and scale to fail**
5. **Focus at the level of individual diseases and assets**

## …applied many times…

eidos a bridgebio company

pellepharm a bridgebio company

MYOKARDIA

GBT

***+ 18 BridgeBio programs***

## …a pipeline of potential blockbusters and synthetic blockbusters*

- **Two potential $1B+ franchises in Phase 2 or later**
- **Two planned NDA submissions this year**
- **Several early-stage potentially large franchises**
  - KRAS
  - GPX4
  - Congenital adrenal hyperplasia
  - Leber's hereditary optic neuropathy
- **Multiple IND submissions planned in 2020**
- **Four new programs announced in January 2020**

*Blockbuster defined as program with $1bn+ opportunity



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# BridgeBio is led by a world-class team of experienced drug developers

## We rely on some of the top R&D minds in this industry to select assets…

| | | |
|---|---|---|
| **Charles Homcy, MD**<br>Chairman of Pharmaceuticals | PORTOLA PHARMACEUTICALS, MyoKardia, GBT, Takeda MILLENNIUM THE TAKEDA ONCOLOGY COMPANY | VELCADE (bortezomib) FOR INJECTION, Andexxa (Coagulation Factor Xa (Recombinant), inactivated - zhzo), Oxbryta (voxelotor) |
| **Frank McCormick, PhD, FRS**<br>Chairman of Oncology | Cetus, ONYX PHARMACEUTICALS, UCSF | Nexavar (sorafenib) tablets, Kyprolis (carfilzomib) for injection |
| **Richard Scheller, PhD**<br>Chairman of R&D | Genentech, 23andMe | TECENTRIQ atezolizumab, PERJETA pertuzumab for injection, Kadcyla trastuzumab emtansine, OCREVUS ocrelizumab, VENCLEXTA venetoclax tablets |

## …and put them in the hands of one of the most productive groups of R&D operators in the industry

| | | |
|---|---|---|
| **Uma Sinha, PhD**<br>Chief Scientific Officer | GBT, PORTOLA PHARMACEUTICALS, Takeda MILLENNIUM THE TAKEDA ONCOLOGY COMPANY | (eptifibatide) Injection INTEGRILIN, Andexxa (Coagulation Factor Xa (Recombinant), inactivated - zhzo), Oxbryta (voxelotor) |
| **Eli Wallace, PhD**<br>Chief Scientific Officer in Residence, Oncology | ARRAY BIOPHARMA, Peloton Therapeutics | MEKTOVI (binimetinib) 15 mg tablets, TUKYSA tucatinib 50 mg \| 150 mg tablets, Koselugo (selumetinib) 10 mg & 25 mg capsules<br>PT2997 (HIF2αi, Ph3) |
| **Robert Zamboni, PhD**<br>Chemistry | MERCK FROSST | SINGULAIR (montelukast, MSD), ARCOXIA (etoricoxib), VIOXX (rofecoxib, MSD) |

**Together, our R&D team is responsible for 100+ INDs and 20+ approved products**



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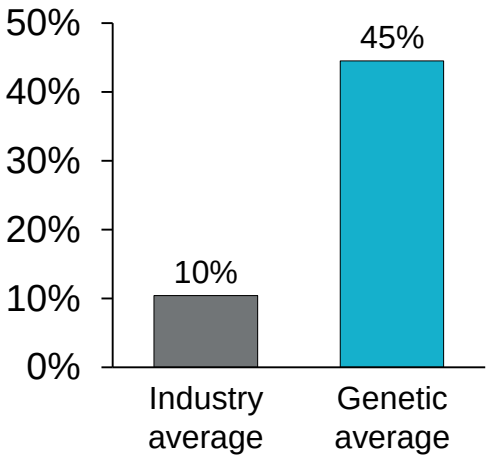
# We believe genetic disease drug discovery is lower risk, faster, with potentially higher returns than traditional drug discovery

**>4x** Higher cumulative probability of success

**>65%** Faster time to Phase 3*

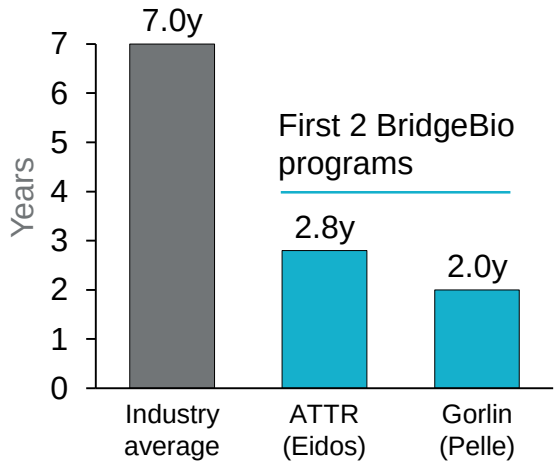
**>8x** Better return on investment*

**Probability of success from Ph1 to launch**



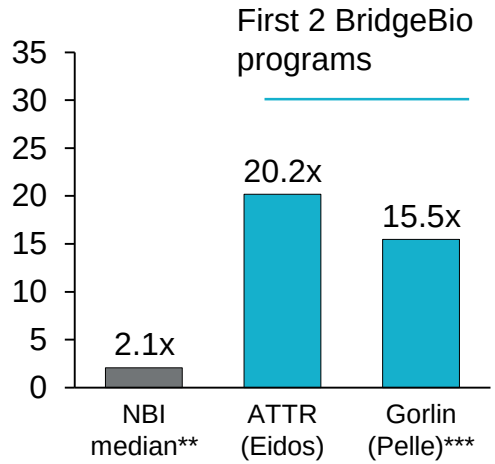

Sources: Hay et al., Nature Biotechnology, "Clinical Development Success Rates for Investigational Drugs", 2014

**Time from lead optimization to Ph3**




Sources: Paul et al., Nat Rev Drug Disc, "How to improve R&D productivity: the pharmaceutical industry's grand challenge.", 2010

**Total return on investment**
[Enerprise value]/[APIC - cash on hand]*




*As of 2/28/2020 close
**Includes all NBI constituents with market value <$20bn
***Calculated as total consideration from LEO Pharma transaction divided by total burn to date
Sources: FactSet

**Targeting genetic disease has higher average probability of success and BridgeBio has demonstrated higher ROI and shorter development time in its first 2 programs**

*For first two BridgeBio programs



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# Assessing BridgeBio

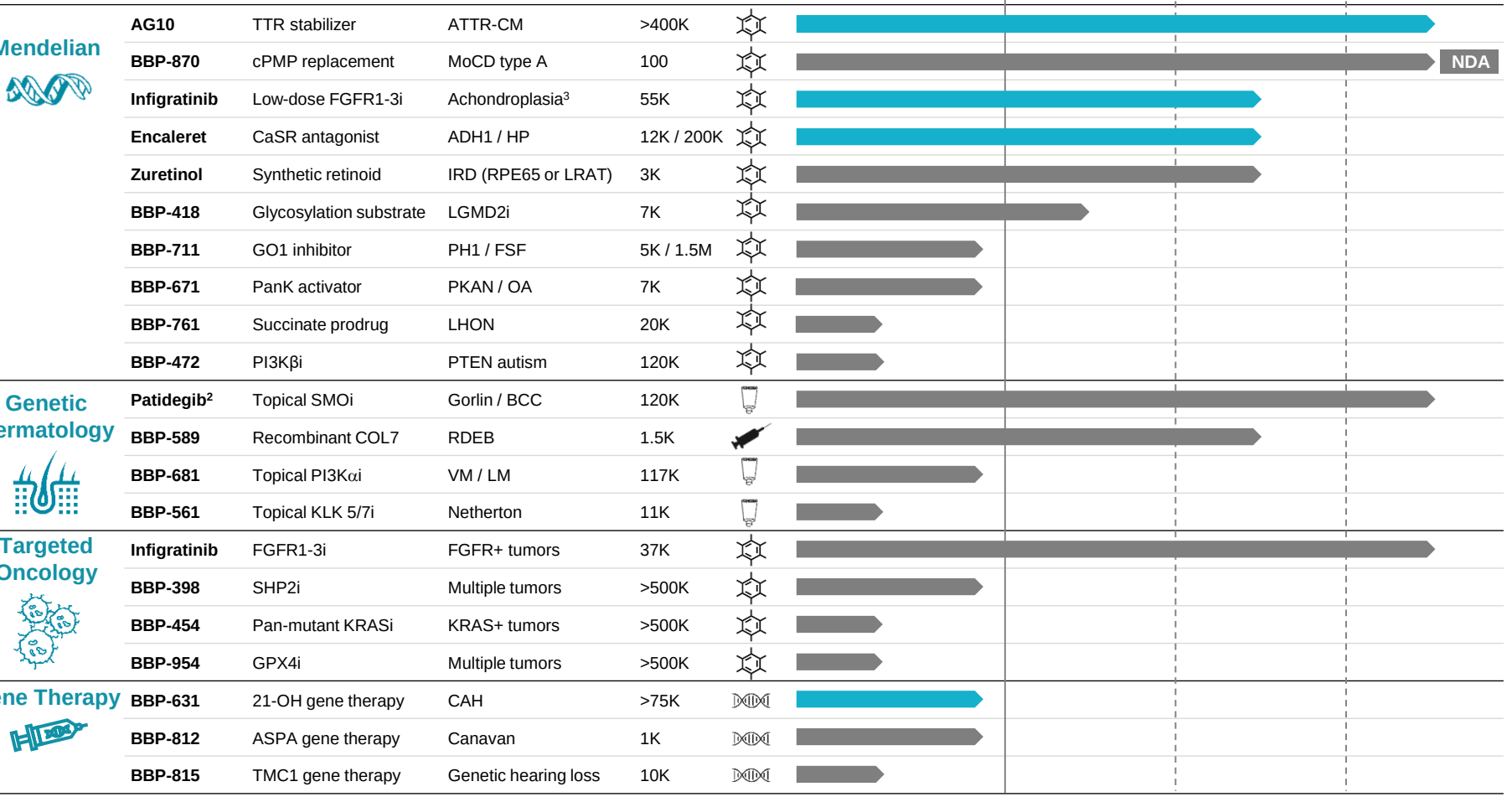
| | Criteria | Relevance |
|---|---|---|
| 1 | High probability of success | • Historically higher probability of success for genetic disease drugs<br>• BridgeBio's early programs have outperformed historical probabilities |
| 2 | Number of programs | • We find great science and unlock its potential for patients<br>• Always searching for the next PellePharm or Eidos<br>• Scale allows for objective assessment and failure |
| 3 | Capital efficiency | • Generate value by making each program ROI-positive<br>• Driven by judicious use of capital at the high-risk preclinical stages |

# Our pipeline of 20+ development programs spans multiple therapeutic areas and drug modalities

Small molecule | Topical small molecule | Biologics | Gene therapy

= Key value drivers over the next 18 months

| Portfolio segment | Program[1] | Drug mechanism | Diseases | Patient pop. (US+EU) | Modality |
|---|---|---|---|---|---|
| Mendelian | **AG10** | TTR stabilizer | ATTR-CM | >400K | |
| | **BBP-870** | cPMP replacement | MoCD type A | 100 | |
| | **Infigratinib** | Low-dose FGFR1-3i | Achondroplasia[3] | 55K | |
| | **Encaleret** | CaSR antagonist | ADH1 / HP | 12K / 200K | |
| | **Zuretinol** | Synthetic retinoid | IRD (RPE65 or LRAT) | 3K | |
| | **BBP-418** | Glycosylation substrate | LGMD2i | 7K | |
| | **BBP-711** | GO1 inhibitor | PH1 / FSF | 5K / 1.5M | |
| | **BBP-671** | PanK activator | PKAN / OA | 7K | |
| | **BBP-761** | Succinate prodrug | LHON | 20K | |
| | **BBP-472** | PI3Kβi | PTEN autism | 120K | |
| Genetic Dermatology | **Patidegib[2]** | Topical SMOi | Gorlin / BCC | 120K | |
| | **BBP-589** | Recombinant COL7 | RDEB | 1.5K | |
| | **BBP-681** | Topical PI3Kαi | VM / LM | 117K | |
| | **BBP-561** | Topical KLK 5/7i | Netherton | 11K | |
| Targeted Oncology | **Infigratinib** | FGFR1-3i | FGFR+ tumors | 37K | |
| | **BBP-398** | SHP2i | Multiple tumors | >500K | |
| | **BBP-454** | Pan-mutant KRASi | KRAS+ tumors | >500K | |
| | **BBP-954** | GPX4i | Multiple tumors | >500K | |
| Gene Therapy | **BBP-631** | 21-OH gene therapy | CAH | >75K | |
| | **BBP-812** | ASPA gene therapy | Canavan | 1K | |
| | **BBP-815** | TMC1 gene therapy | Genetic hearing loss | 10K | |




[1] Each of our programs is housed in a separate subsidiary; [2]We are party to an option agreement pursuant to which LEO Pharma A/S has been granted an exclusive, irrevocable option to acquire PellePharm, including the BBP-009 program. If the option is exercised by LEO Pharma A/S, we will no longer have rights to develop and commercialize BBP-009. [3]Protocol accepted by Australian local ethics committed, IND submission to FDA expected 2020.



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# Four key value drivers over the next 18 months

| Program | Population (US+EU) | Status | Upcoming event(s) |
|---|---|---|---|
| **AG10: TTR stabilizer for ATTR**<br>▪ Most potent TTR stabilizer; MOA mimics protective genetic variant<br>▪ Data to date suggest potential best-in-class clinical profile | **>400K** | Enrolling ATTR-CM Ph3 | ▪ Complete enrollment 1H21<br>▪ Data 1H22 |
| **Low-dose infigratinib (FGFRi) for achondroplasia**<br>▪ Only agent designed to directly target genetic cause of ACH<br>▪ Differentiated pre-clinical data on cranial and spinal defects | **55K** | Enrolling Ph2 study | ▪ Dose first child 2020<br>▪ Data 2021 |
| **Gene therapy for congenital adrenal hyperplasia (BBP-631)**<br>▪ One of largest potential gene therapy markets<br>▪ Only approach capable driving endogenous cortisol production | **>75K** | GLP tox ongoing | ▪ File IND 2020<br>▪ Data 2021 |
| **Encaleret: CaSR antagonist for autosomal dominant hypocalcemia type 1 (ADH1)**<br>▪ Directly targets ADH1 genetic driver; potentially first-in-class | **12K** | Ph2-ready | ▪ Dose first patient 2020<br>▪ Data 2021 |



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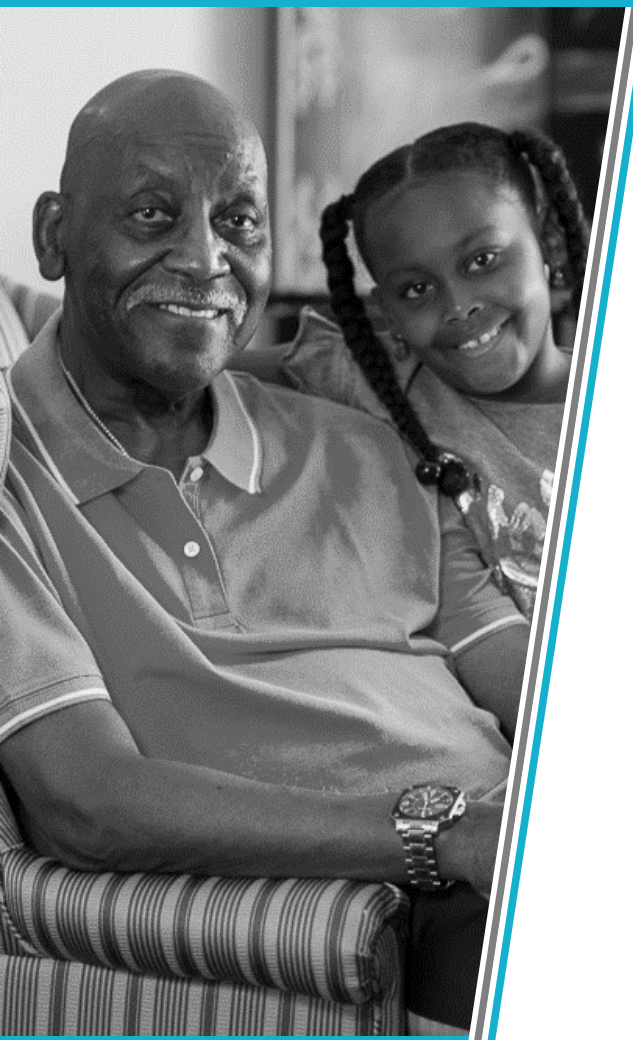
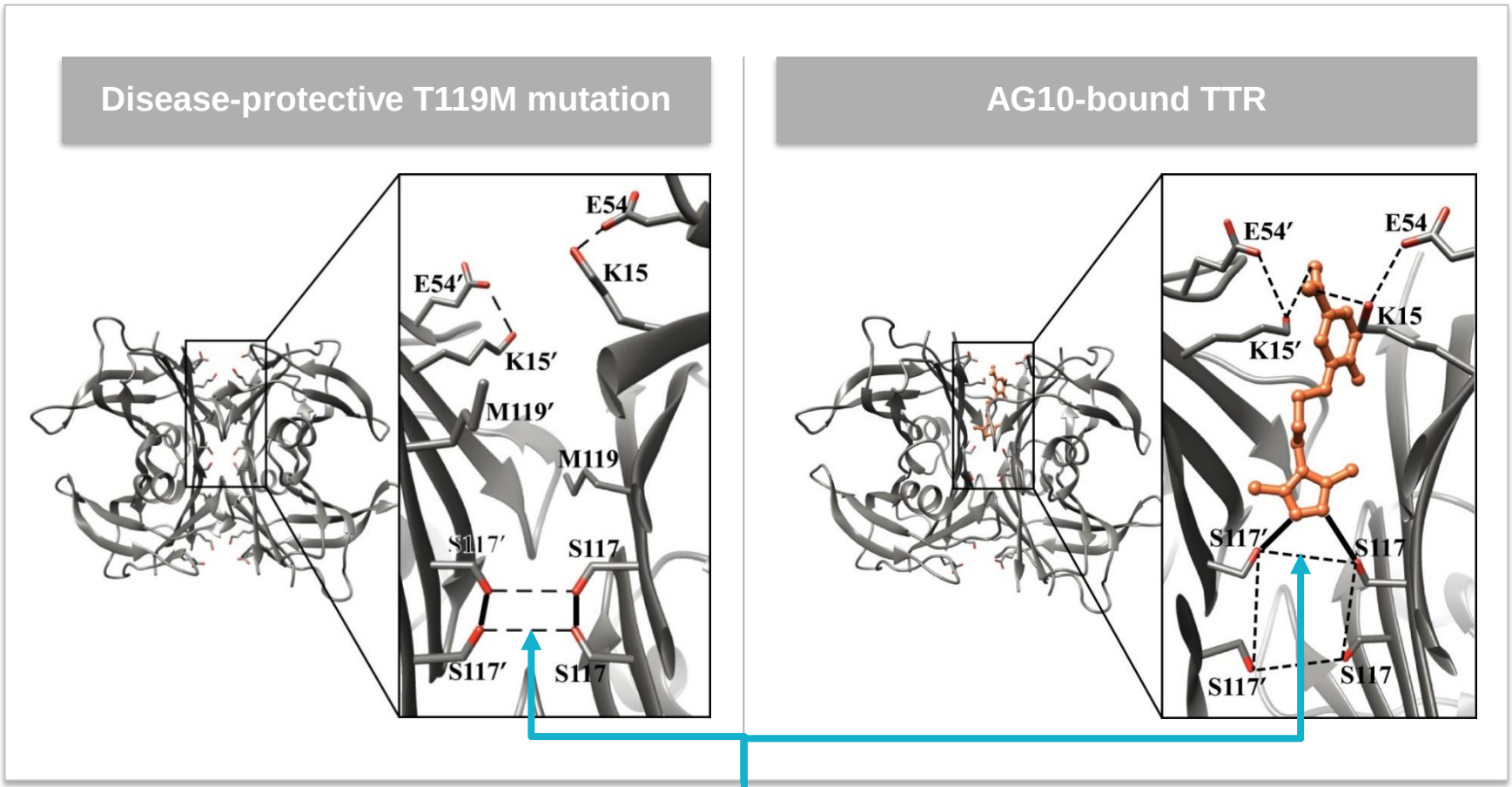
# AG10 for TTR amyloidosis (Eidos)



Art, ATTR-CM patient

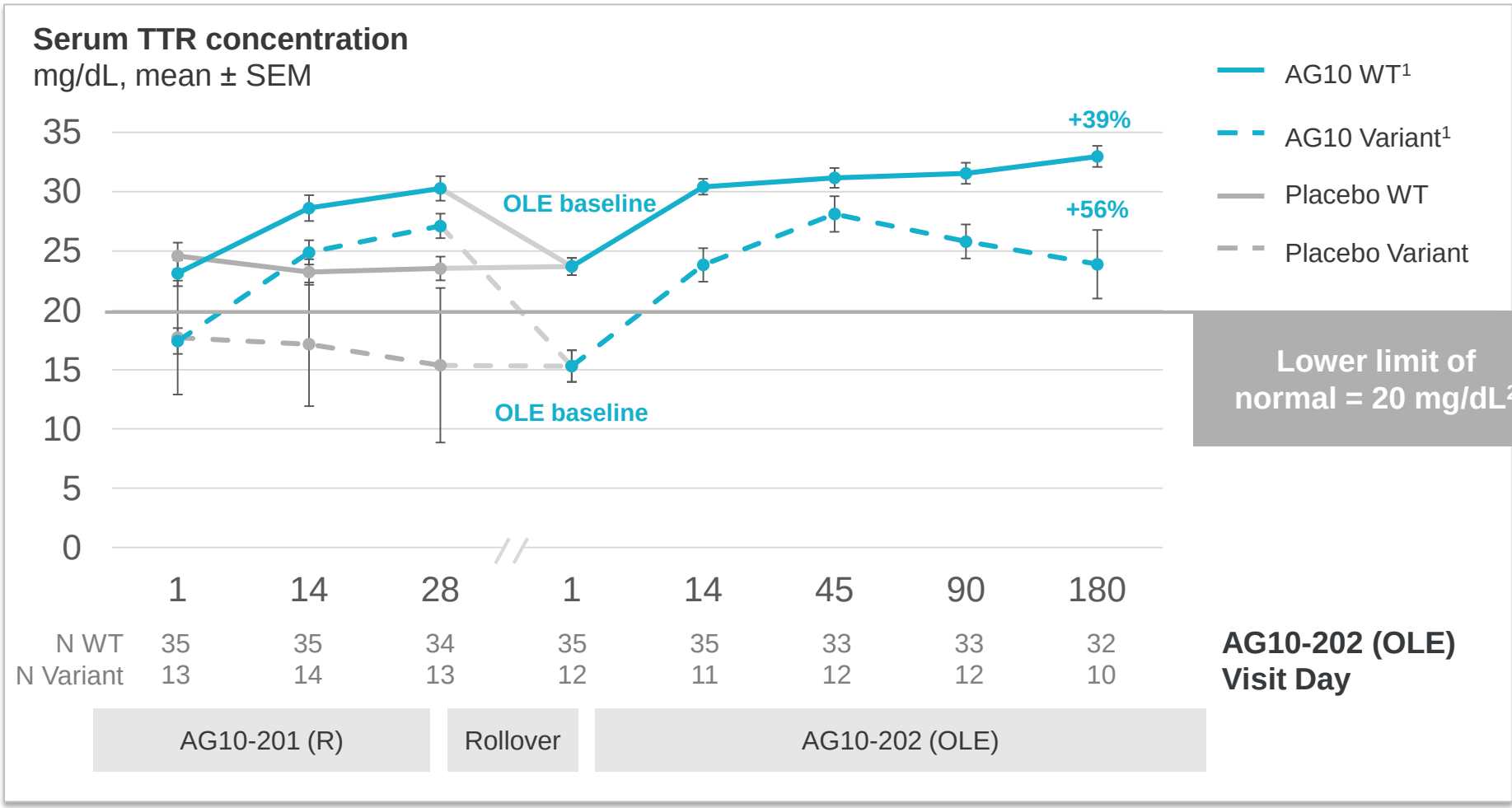
- **Addressing large and growing need in ATTR**, a fatal disease affecting >400K patients
- **Targeting the disease at its source** by stabilizing TTR, a genetic and clinically validated mechanism
- **Advancing AG10, a potential best-in-class drug** that mimics naturally occurring rescue mutation
- Phase 2 open label extension study suggests potential to **reduce mortality and cardiovascular hospitalizations** at 15 months
- **Executing Phase 3 study in ATTR-CM** with top-line data expected in 2022

# AG10 structurally mimics disease-protective mutation by hyper-stabilizing TTR




**Strong inter-monomer H-bonds observed via X-ray crystallography**
**Unique binding mode vs other stabilizers**

# Serum TTR levels, a prognostic indicator of survival, increased upon AG10 treatment and were maintained throughout Ph 2 study

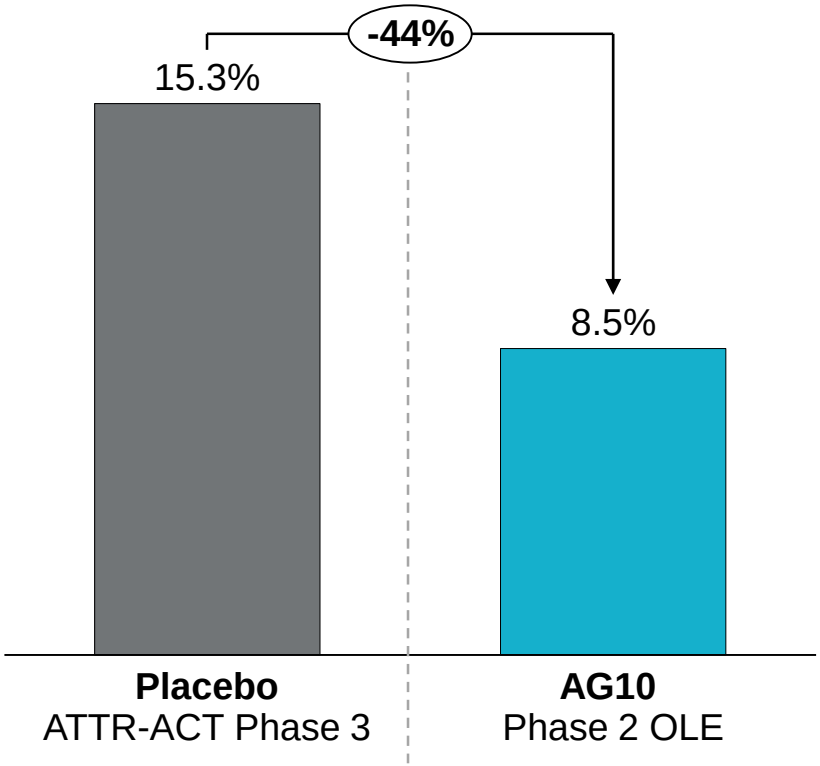



1 400mg and 800mg BID AG10 groups pooled during randomized portion
2 Defined as the lower limit of the reference interval for the serum prealbumin (TTR) clinical laboratory assay

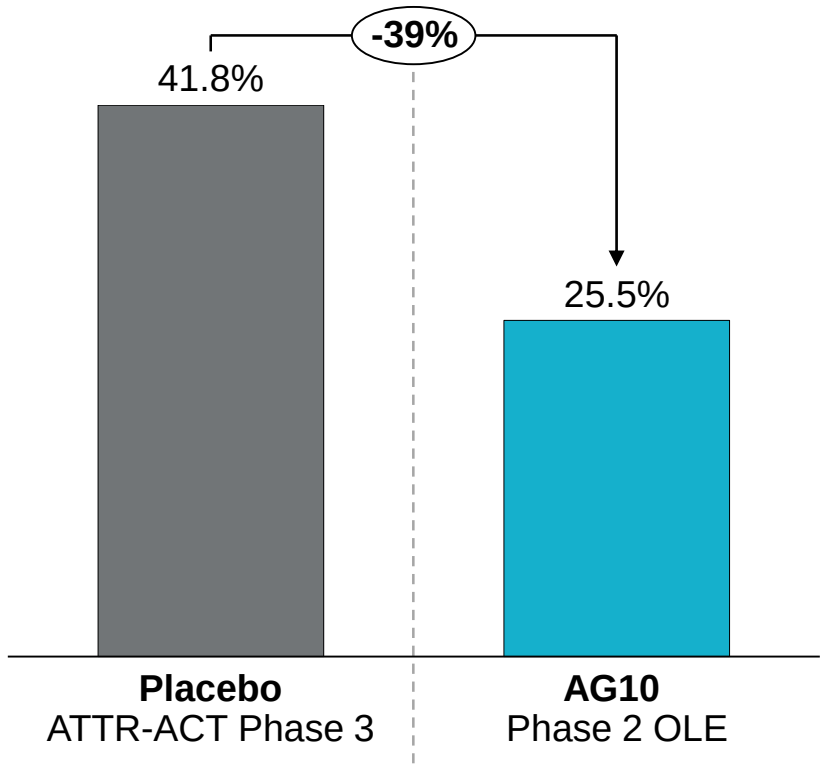
Source: Judge, DP et al. American Heart Association Scientific Sessions 2019

# Deaths and CV hospitalizations reported in AG10 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 (%)




**Phase 3 ATTRibute study expected to complete enrollment in 1H21**

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

# Low-dose FGFR inhibitor (infigratinib) for achondroplasia

Claudia, child with 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 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

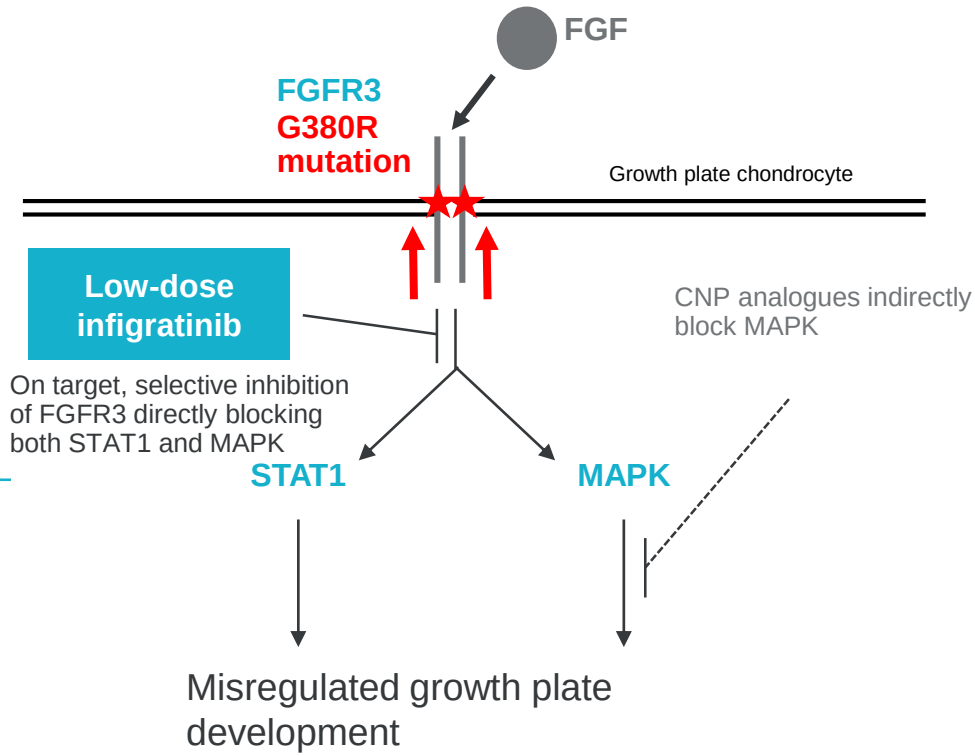
# Potential best-in-class approach to treating achondroplasia directly at its genetic source

## 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 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
- Demonstrate clear macro and microscopic improvements on foramen magnum, intervertebral discs, and long bones in validated preclinical model




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

## Preclinical data from infigratinib and other investigational achondroplasia therapies

Percent increase compared to non-treated mouse

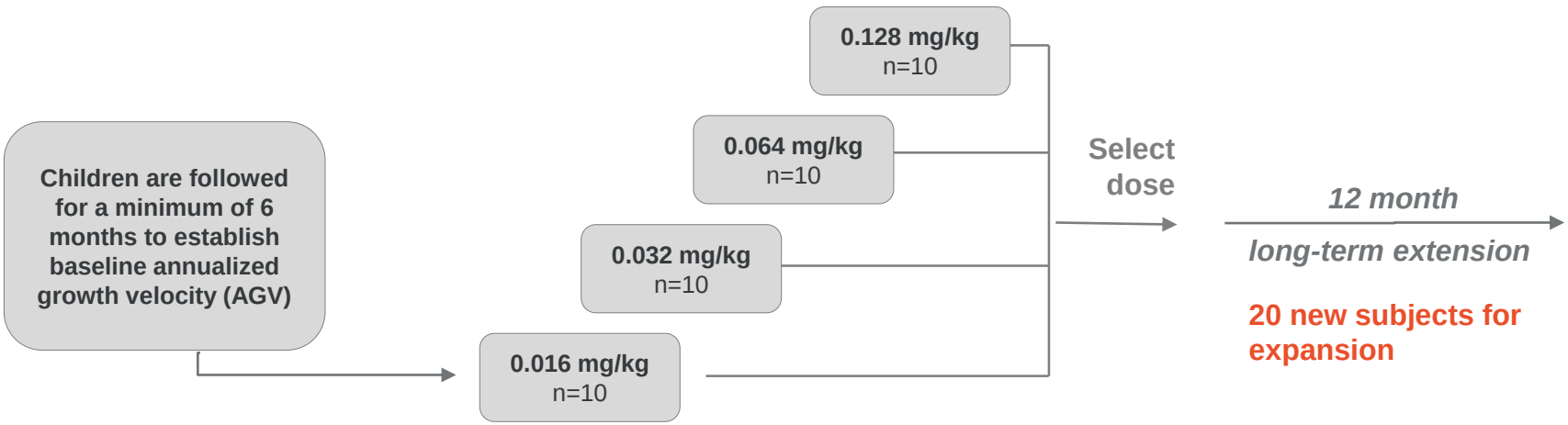
| Company/ Asset | MOA | Route | Status | Mouse model | Tibia length | Femur length | Foramen magnum area | L4-L6 height |
|---|---|---|---|---|---|---|---|---|
| bridgebio Infigratinib | Selective FGFR1-3i | Oral | Ph2 lead-in ongoing[2] | FGFR3$^{Y367C/+}$ | 33% | 21% | 17% | 12% |
| BIOMARIN Vosoritide (BMN111) | CNP analogue | Daily SQ | Pivotal | FGFR3$^{Y367C/+}$ | 7% | 5% | | 3% |
| ascendis pharma TransCon CNP[1] | CNP analogue | Weekly SQ | Ph2 lead-in ongoing | FGFR3$^{Y367C/+}$ | 12%* | | | |
| Pfizer Reifercept (TA-46) | FGFR3 decoy | Weekly SQ | Ph1 | FGFR3$^{ACH/+}$ | 9%* | 7%* | | |

No known publicly available data

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. [2]Protocol submitted to Australian local ethics committed, IND submission to FDA expected 2020.

# The PROPEL clinical program is enrolling with data expected in 2021

**Observational run-in** | **Ph2 Dose-finding (n=40)** | **Expansion (n=20)**




**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

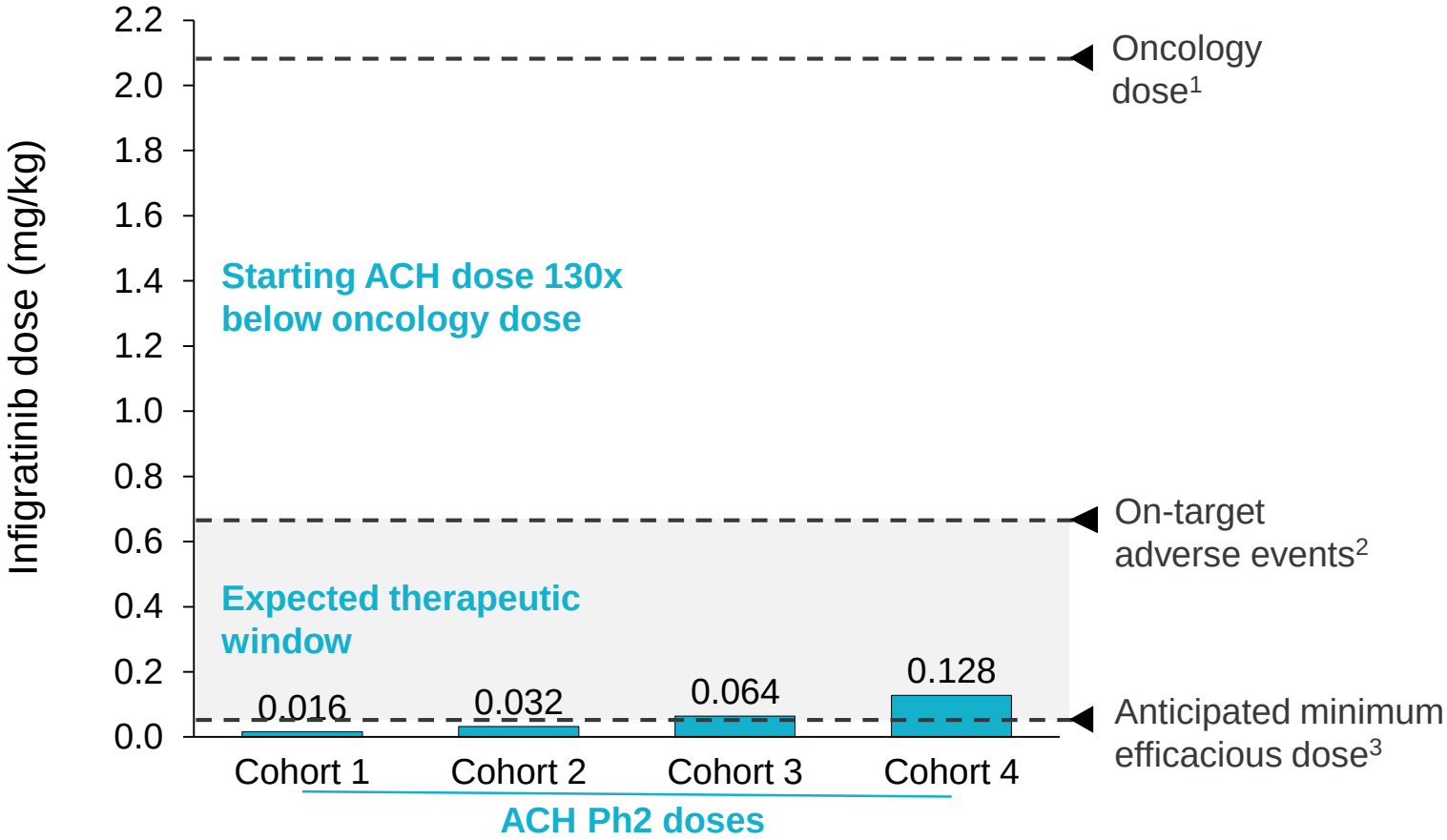
**Primary objectives**

- Long-term safety and efficacy

# 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

# Gene therapy for congenital adrenal hyperplasia (CAH)

Maris, child with CAH

## Program overview:

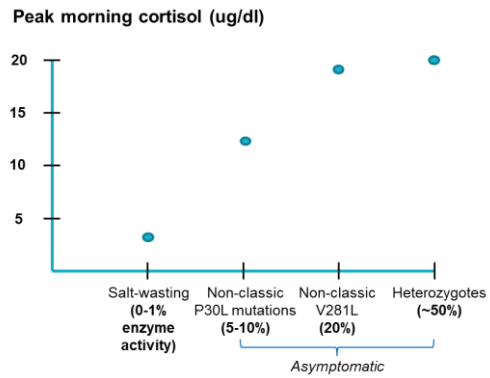
- **Prevalence**: 75,000 (US+EU) – One of the largest known AAV gene therapy markets
- **Genetic driver**: 21-hydroxylase inactivation
- **Pathophysiology**: Inability to produce cortisol causes need for supraphysiologic doses of synthetic steroids, 3x increase mortality risk, hirsutism, Cushingoid symptoms

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

- **Low threshold to correct phenotype**, validated by human clinical genetics
- **Only approach designed to induce endogenous cortisol production**, potentially allowing steroid withdrawal
- **Durable transgene delivery to the adrenal gland of NHP** with IV dosing of our construct
- **Preliminary Ph1/2 data anticipated in 2021**

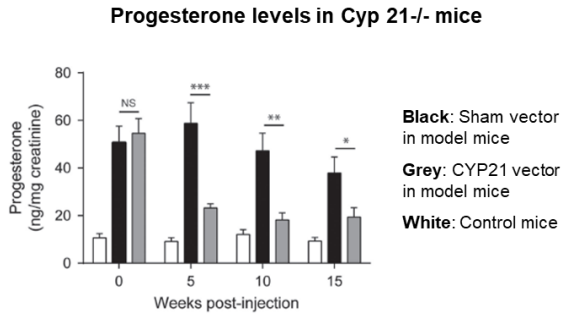
# CAH: NHP study showed durable transgene expression; 5-10% of WT enzyme may be sufficient for clinical impact

## 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**

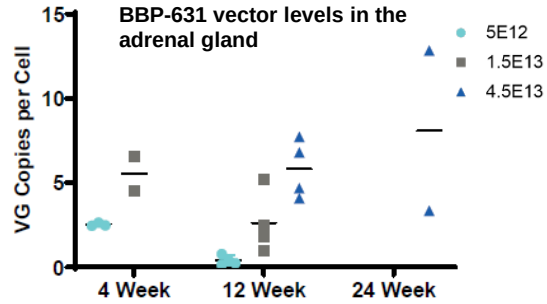
## Mouse studies show a VGC of only 0.13 at 18 wks was sufficient for phenotypic correction



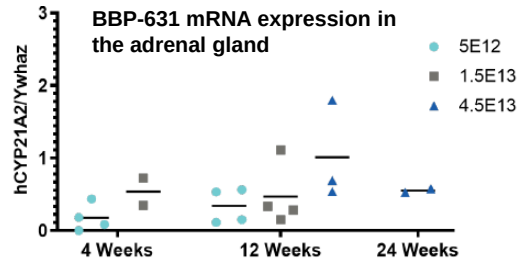

VGC from the whole adrenal gland was 0.13 at wk 18

- 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




- Mean vector genome copies per cell appear stable at 24 wks




- Transgene expression is dose-dependent and stable out at 24 wks

Source Perdomini, *Gene Therapy* 2017; ESGCT 2019

# Encaleret for disorders of calcium homeostasis, including autosomal dominant hypocalcemia type 1 (ADH1)

## Targets hypocalcemia/hypercalciuria by selectively antagonizing the calcium-sensing receptor (CaSR)

- Opportunity identified in collaboration with global experts at the NIH

## Potential 1st in class CaSR antagonist with differentiated profile for ADH1 and hypoparathyroidism

- Initial development in genetically-defined population of ADH1, driven by CaSR activating mutations (~12K carriers in US)
- Potential for expansion into post-surgical chronic hypoparathyroidism (~200K patients in US & EU)

## Prior clinical experience enables accelerated development

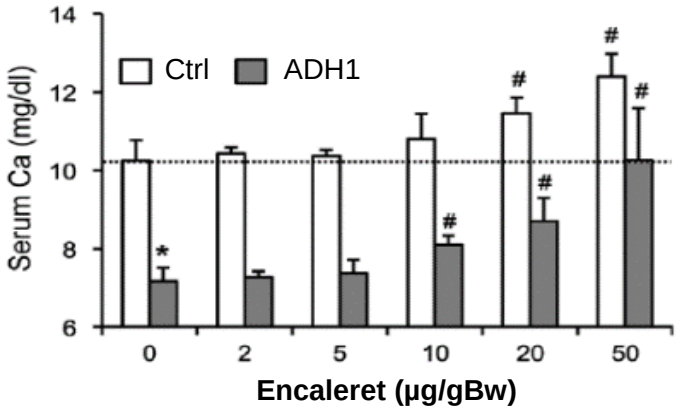
- Well tolerated in >1,200 human subjects and increased serum calcium in a dose-dependent manner
- Phase 2 study in ADH1 planned to initiate in 2020 with proof-of-concept data anticipated in 2021

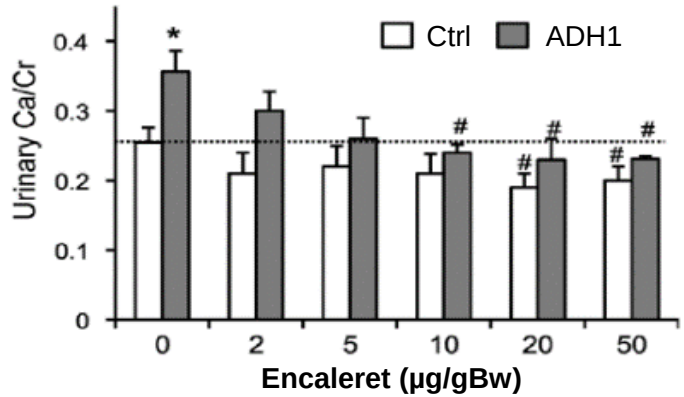
# Encaleret targets ADH1 at its source by normalizing hyper-active calcium sensing receptor

## Rationale for calcilytic use in ADH1

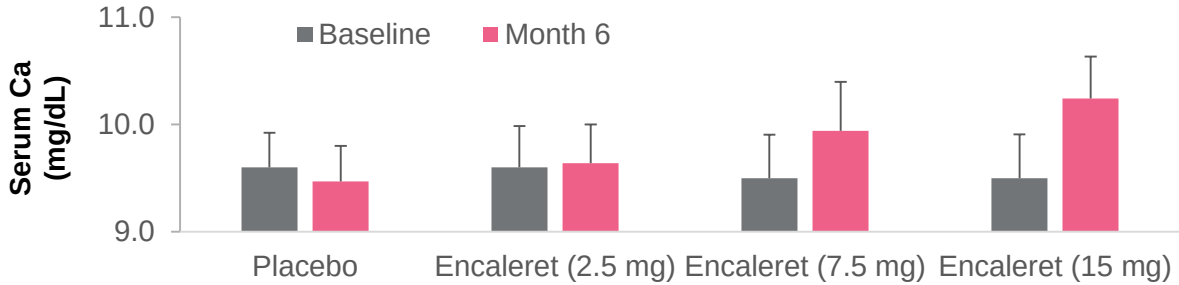
- ADH1 is caused by activating mutations in the CaSR leading to hypocalcemia and hypercalciuria
- Prior generation calcilytic partially addressed ADH1 phenotype despite limited exposure[1]

## Encaleret normalized serum and urine calcium in a mouse model of ADH1[2]



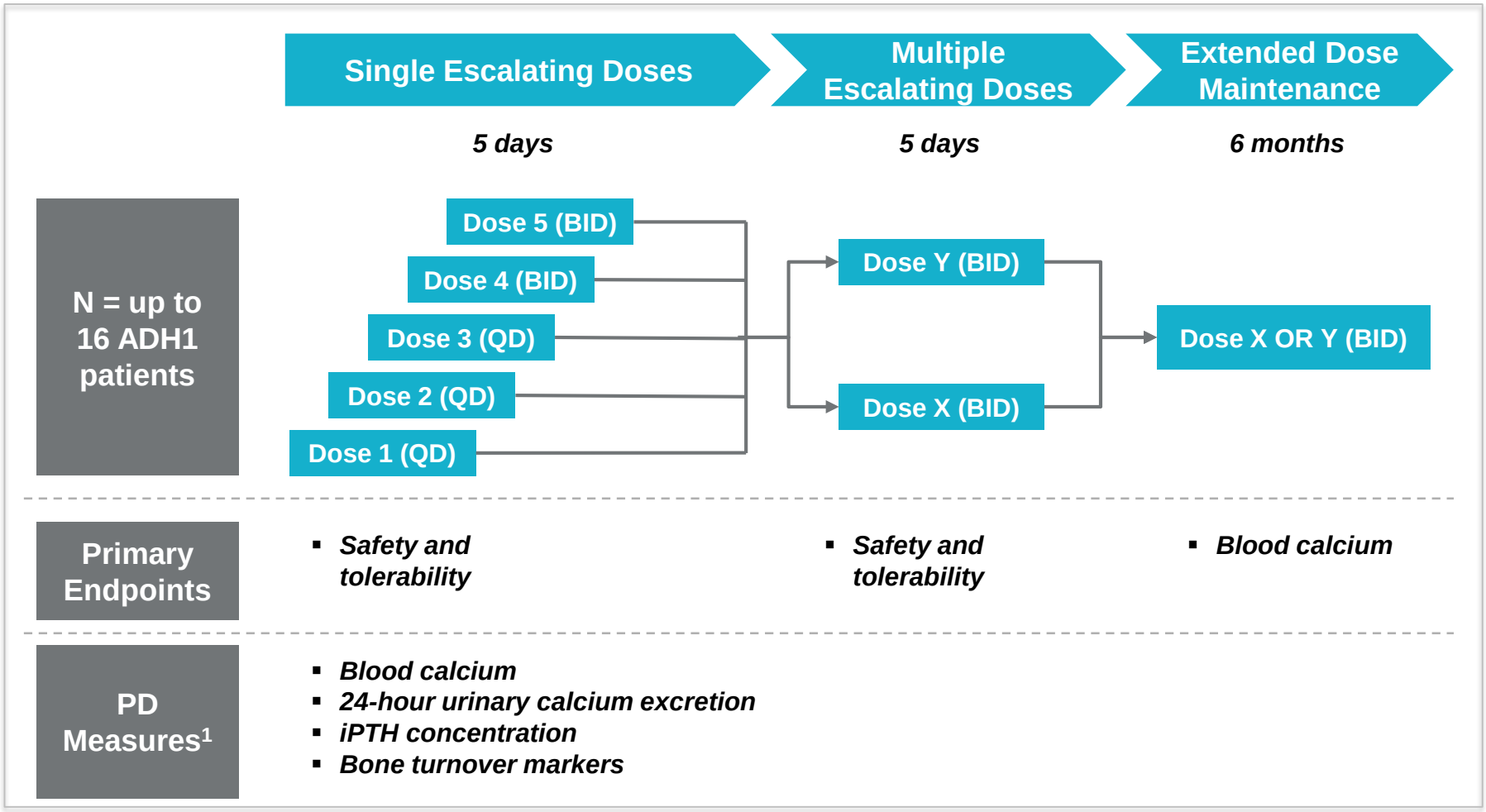




## Encaleret increased serum calcium in clinical trials in patients with osteoporosis[3]




Source: 1 Roberts, M.S., et al. J Bone & Min 2019; 2 Dong B., et al. J Bone & Min 2015; 3 Data on file

# Phase 2b, open-label dose-ranging study will evaluate safety, tolerability, and efficacy of encaleret in ADH1




**Top-line, proof-of-concept results of encaleret in ADH1 are expected in 2021**

[1] Pharmacodynamic measurements to be collected through duration of study.

# Early oncology portfolio

Basia, pancreatic cancer patient

## World-class oncology team drives our discovery and development

- **Eli Wallace,** CSO Oncology
- **Frank McCormick**, Chairman of Oncology
- **Richard Scheller**, Chairman of R&D

## Our KRAS platform has produced 3 pan-mutant programs:

- 1 – H95 approach, designed to block effector signaling
- 2 – KRAS:PI3K blocker approach, designed to block PI3K effector signaling
- 3 – C185 approach, designed to block KRAS prenylation and activation

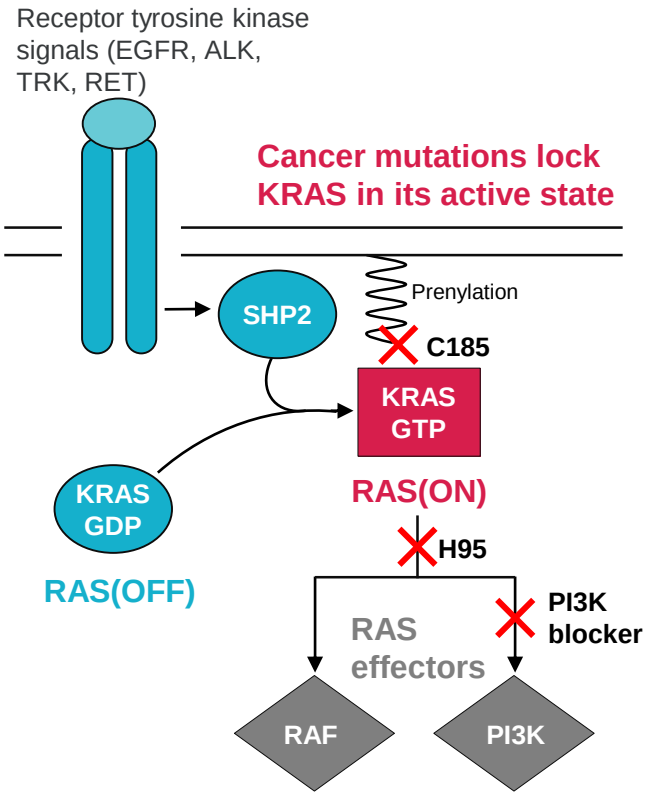
## We are also prosecuting novel targets with extensive academic validation

- **SHP2i** for multiple tumors (10+ recent papers in *Nature, Science, Nature Medicine*)
- **GPX4i** for multiple tumors (10+ recent papers in *Nature, Cell, Science, Cancer Cell*)

| Program | MOA | Disease | Stage | Next anticipated update |
|---|---|---|---|---|
| Pan-mutant KRAS inhibitors | (1) H95 approach<br>(2) PI3K blocker<br>(3) C185 approach | KRAS+ cancer | Discovery | Clinical candidate nomination |
| SHP2 inhibitor | Allosteric inhibitor | Multiple tumors | Pre-IND | IND submission in 2020 |
| GPX4 inhibitor | Covalent inhibitor | Multiple tumors | Discovery | Clinical candidate nomination |

# 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 |
|---|---|---|---|---|
| **Program 1: H95 targeting** | ▪ Directly binds activated KRAS through H95<br>▪ Inhibits KRAS from signaling through effectors | ✓ | ✓ | ✓ |
| **Program 2: PI3K effector blocking** | ▪ Blocks specific interaction between KRAS and PI3Ka<br>▪ Blocks PI3K / AKT effector signaling | ✓ | ✓ | ✓ |
| **Program 3: C185 targeting** | ▪ Blocks KRAS from tethering<br>▪ 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**



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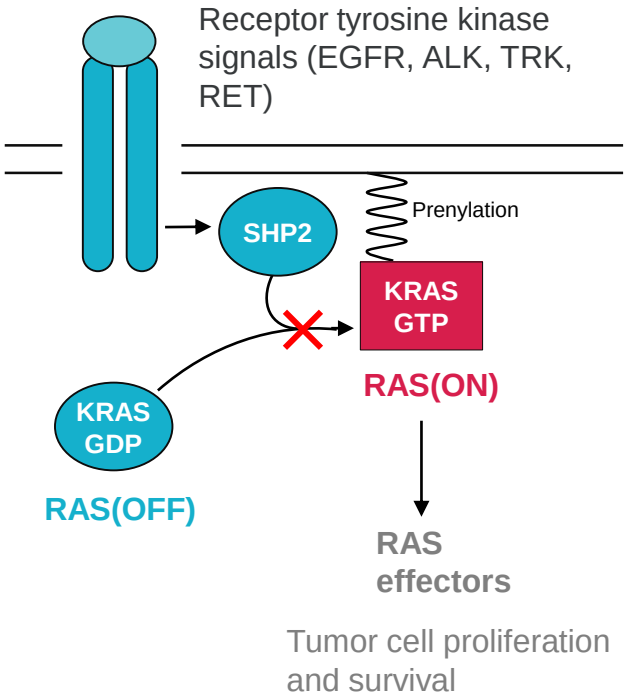
# SHP2: Our compound is expected to enter the clinic in 2020

- Our compound traps SHP2 in the inactive state, thereby blocking downstream MAPK signaling
- In collaboration with MD Anderson, optimized our SHP2i for use in combination (no QTC prolongation or hypertension)
- Well tolerated in 28d GLP-tox studies – MTD not reached in dogs (25mpk) or rats (100mpk)
- First SHP2 inhibitor clinical data, (RVMD Q1 2020) demonstrates monotherapy antitumor activity*




## Preclinical SHP2i data

| *In vitro* properties | BBIO | RVMD* |
|---|---|---|
| pERK $IC_{50}$ (nM) cellular assay | <40 | <40 |
| hERG Patch clamp $IC_{50}$ (µM) | >100 | ? |
| **Monotherapy anti-tumor activity** | | |
| KRASG12C xenograft | ✓ | ✓ |
| EGFR mutant xenograft | ✓ | ✓ |
| **Combination enhanced anti-tumor activity** | | |
| G12Ci | ✓(AMG 510) | ✓(MRTX 849) |
| MEKi | ✓(trametinib) | ✓(cobimetinib) |
| EGFRi osimertinib | ✓ | ✓ |

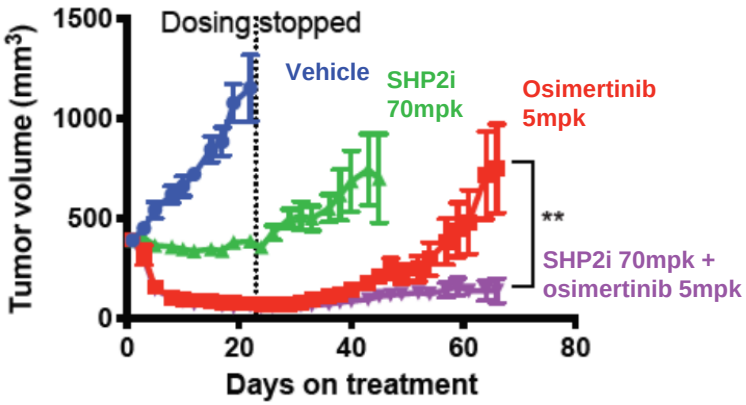
**Preclinical profile demonstrates activity in-line with SHP2i class and potential for better tolerability**

Source: Data on file, *Revolution Medicines S-1

# SHP2: Preclinical rationale for development in NSCLC

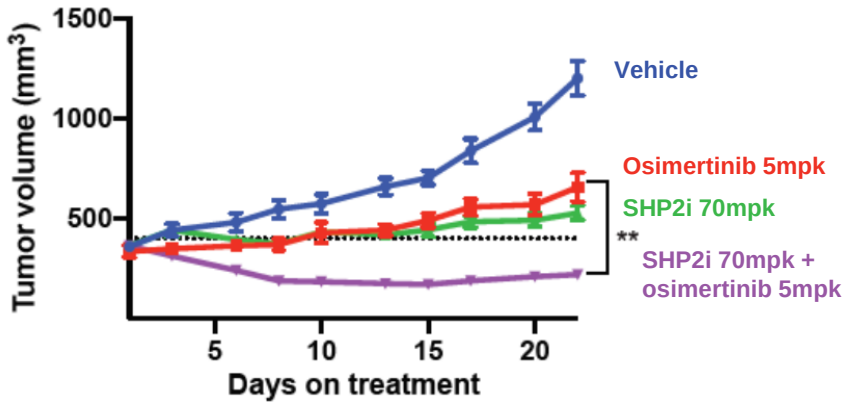
**NSCLC: Deep and durable tumor regressions in combination with market-leading EGFRi osimertinib**

## EGFR+ NSLC model (HCC827)




- Our compound + osimertinib deep tumor regression
- Effect was durable at least 40d after dosing stopped

## EGFR+, osimertinib-resistant NSLC model (HCC827-ER, METamp)




- Our compound + osimertinib produced regression in an osimertinib resistant model
- Provides rationale for testing in osimertinib failures

Source: NSCLC - 2019 AACR-NCI-EORTC meeting, CRC - data on file



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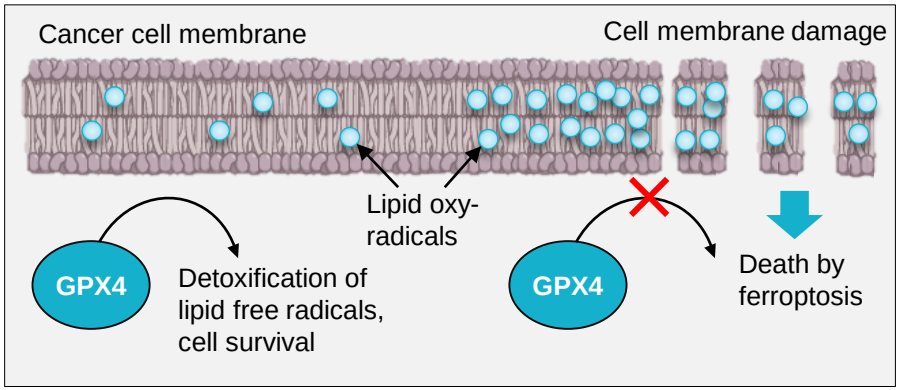
# GPX4: Potential first-in-class therapy for a novel cancer target

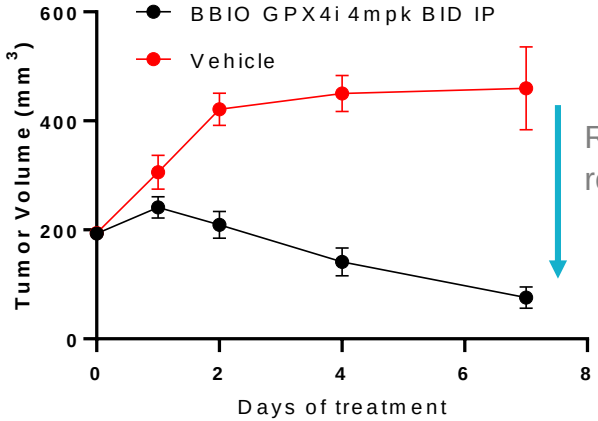
## GPX4 is the key negative regulator of ferroptosis

- GPX4 neutralizes toxic free radicals at the lipid membrane, protecting cells from death (ferroptosis)
- We are developing covalent inhibitors of GPX4 designed to induce ferroptosis in cancer cells
- Recent high profile publications provide preclinical in vivo rationale for monotherapy and combinations with IO, kinase inhibitors and chemotherapy




## In vivo monotherapy activity in RCC xeno model

Model: 786-O RCC xenograft (VHL LOF, p53 LOF)




**Synergy with targeted therapies and immunotherapy using in vitro models**

**Optimization of oral lead compounds ongoing**

Source: Data on file

# Three late-stage programs continue to progress toward the market

| Program | 2019 | 2020 | 2021 |
|---|---|---|---|
| **Fosdenopterin:** cPMP replacement for MoCD type A | ✓ Initiate rolling NDA submission | ❑ Complete rolling NDA submission | ❑ FDA approval / launch<br>❑ Potential PRV sale |
| **Infigratinib:** FGFRi for 2L cholangiocarcinoma with FGFR2 fusion | ✓ Complete enrollment in Ph2 pivotal study | ❑ Complete NDA submission | ❑ FDA approval / launch |
| **Topical patidegib:** SMOi for Gorlin syndrome | ✓ Complete enrollment in Ph3 study | ❑ Last patient last visit | ❑ Topline data<br>❑ Potential payment from Leo |

**We are building a track record of late-stage clinical and regulatory execution**



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# Multiple catalysts anticipated in 2020-2021

ESTIMATED

## 2020

### 1H

- ✓ New program announcements
- ✓ FGFRi for cancer: FPI Ph3 adjuvant urothelial carcinoma study
- ✓ FGFRi for cancer: FPI Ph2 FGFR fusion tumor agnostic Ph2 study

### 2H

- ❑ Low-dose FGFRi for achondroplasia: Begin dosing Ph2
- ❑ cPMP for MoCD type A: Complete NDA submission
- ❑ CaSR antagonist for ADH1: Dose first patient in Ph2 study
- ❑ FGFRi for cancer: Pivotal 2L CCA data
- ❑ FGFRi for cancer: Submit NDA for 2L CCA
- ❑ New IND filings

## 2021

### FY

- ❑ TTR stabilizer for ATTR: Complete enrollment of ATTR-CM Ph3
- ❑ Topical SMOi for Gorlin: Topline Ph3 data
- ❑ Low-dose FGFRi for achondroplasia: Ph2 PoC data
- ❑ CAH gene therapy: Ph1/2 PoC data
- ❑ CaSR antagonist for ADH1: Ph2 POC data
- ❑ FGFRi for cancer: 2L CCA approval and launch
- ❑ cPMP for MoCD type A: Approval and launch
- ❑ Recombinant COL7 for RDEB: Topline Ph1/2 data

**$928mn in cash and equivalents as of 1Q20 expected to provide runway into 2022**



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