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COMPANY PRESENTATION

December 2019

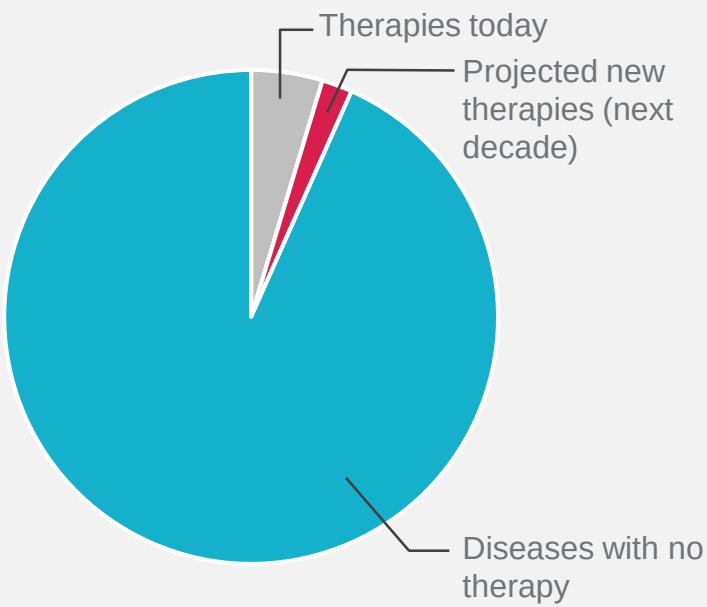
Forward-Looking Statements

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.

Certain information contained in this Presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company's own internal estimates and research. While the Company believes these third-party sources to be reliable as of the date of this Presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. In addition, all of the market data included in this Presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while the Company believes its own internal research is reliable, such research has not been verified by any independent source.

We are at Day 1 in the era of genetic medicine

Vast opportunity to help patients



Advancing science and clinical genetics

- 100’s of disease-causing genes ID’ed per year. 10’s of high fidelity, causal, RGD-polygenic disease links
- Dramatic reduction in cost of exome and genome sequencing
- Increased availability of patient registries and longitudinal databases
- Better molecular biology tools to interrogate and manipulate gene products. Better disease models

Genetic disease drug discovery is lower risk, faster, and has higher returns than traditional drug discovery

Relative to all-comer diseases, genetic disease R&D offers…

>3x **Cumulative probability of success**

36% **Faster development time**

1.7x **Total return on investment**

Reduced **Discovery risk**

- Dramatic reduction in target risk
- Either replacing or directly modulating driver of disease

Reduced **Development risk**

- Reduction in toxicity risk, especially associated with protein replacement
- Faster and smaller trials

Reduced **Regulatory risk**

- For these high unmet need diseases, the FDA actively works with companies

Reduced **Commercial risk**

- Faster and more predictable uptake, lower marketing costs
- Favorable reimbursement, more competitive protection

Sources: Meekings et.al, Drug Discovery Today, Hay et al., Nature Biotechnology, “Clinical Development Success Rates for Investigational Drugs”, Jan. 2014, Shareholder Representative Services, Life Sciences M&A – New Findings on Deal Mechanics, Oct. 2012, DiMasi et al., Nature, “Trends in Risks Associated with New Drug Development”, Mar. 2010, Delloitte, Tufts , EvaluatePharma with team analysis

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Sources of sustainable competitive advantage

- **Distinctive early stage asset selection,** based on a deep understanding of clinical unmet need, genetics, and underlying molecular pathophysiology
- **Experienced, product-focused R&D leadership,** that can define go / no-go's, required product attributes, and can drive programs through the clinic efficiently
- **Efficient corporate structure** that cuts no corners on science and medicine, but limits G&A, infrastructure, needless management, and other waste
- **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

BridgeBio is led by an experienced team

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

Charles Homcy, MD Chairman of Pharmaceuticals	PORTOLA PHARMACEUTICALS, MyoKardia, globalblood THERAPEUTICS, Takeda MILLENNIUM THE TAKEDA ONCOLOGY COMPANY	VELCADE (bortezomib) FOR INJECTION, Andexxa (Coagulation Factor Xa (Recombinant), inactivated - zhzo), Oxbryta (voxelotor)
Frank McCormick, PhD, FRS Chairman of Oncology	Cetus, ONYX PHARMACEUTICALS, UCSF	Nexavar (sorafenib) tablets, Kyprolis (carfilzomib) for injection
Richard Scheller, PhD 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 Chief Scientific Officer	globalblood THERAPEUTICS, PORTOLA PHARMACEUTICALS, Takeda MILLENNIUM THE TAKEDA ONCOLOGY COMPANY	(eptifibatide) Injection INTEGRILIN, Andexxa (Coagulation Factor Xa (Recombinant), inactivated - zhzo), Oxbryta (voxelotor)
Eli Wallace, PhD Chief Scientific Officer in Residence, Oncology	ARRAY BIOPHARMA, Peloton Therapeutics	MEKTOVI (binimetinib) 15 mg tablets, Tucatinib (HER2i, Ph3), PT2997 (HIF2αi, Ph3)
Robert Zamboni, PhD 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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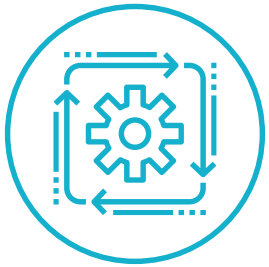
BridgeBio: A product platform and current pipeline



Product Platform

Systematic disease mapping

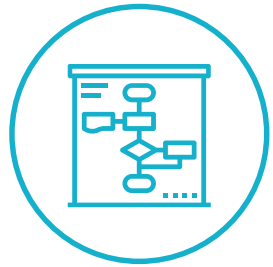
World-class R&D minds and capabilities

Partnered with leading institutions

Focus on capital efficiency

Best owner mentality

Continued growth and terminal value



Current Pipeline

15 programs targeting diseases at their source

Four Phase 2 or Phase 3 clinical programs

Focus on Mendelian diseases and targeted oncology

Small molecules, biologics, gene therapies

5+ therapeutic areas

Present value and near-term catalysts

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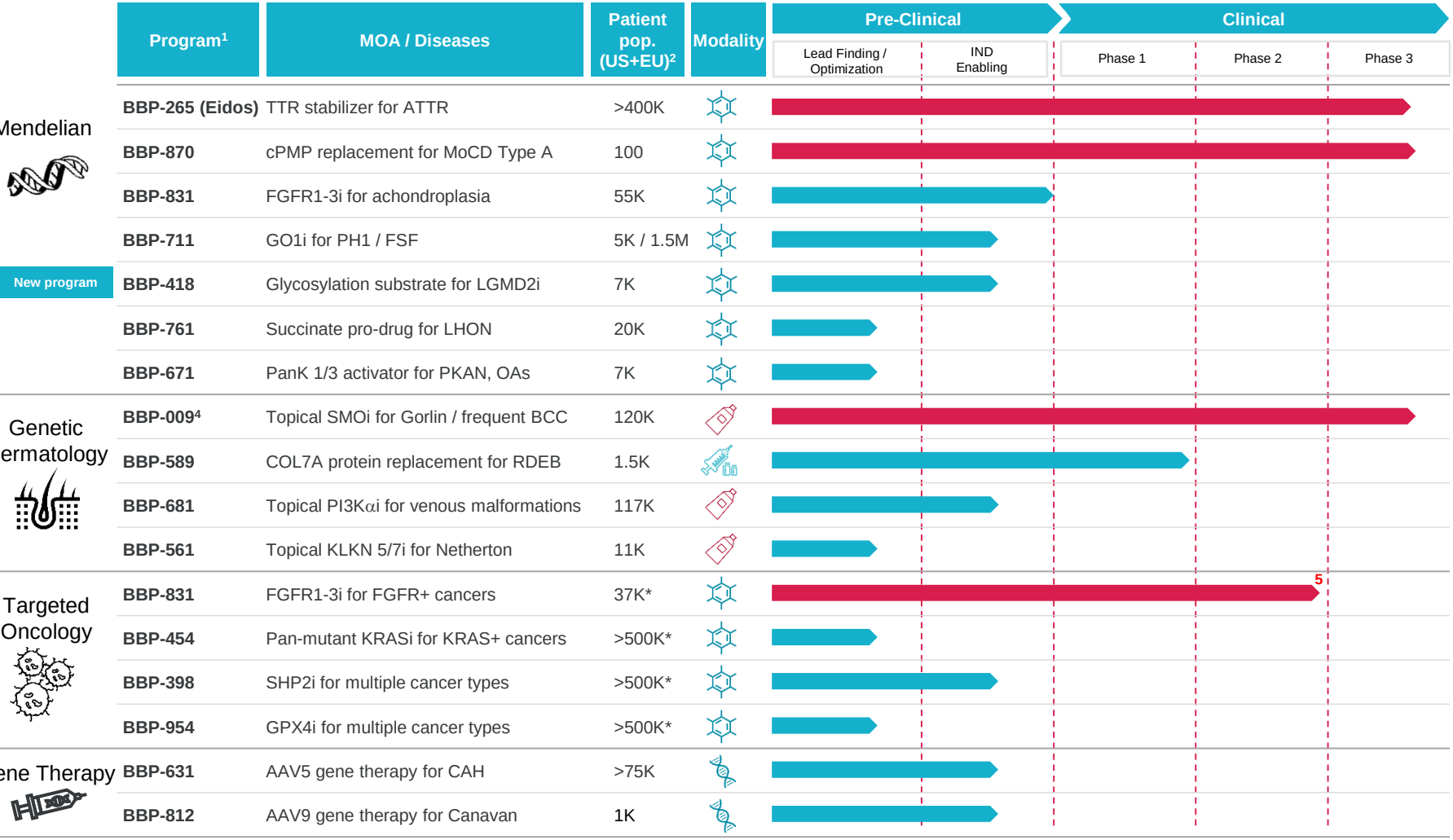
We have built a sizeable pipeline over the past ~4 years

	Program[1]	MOA / Diseases	Patient pop. (US+EU)[2]	Modality
Mendelian	**BBP-265 (Eidos)**	TTR stabilizer for ATTR	>400K	
	BBP-870	cPMP replacement for MoCD Type A	100	
	BBP-831	FGFR1-3i for achondroplasia	55K	
	BBP-711	GO1i for PH1 / FSF	5K / 1.5M	
New program	**BBP-418**	Glycosylation substrate for LGMD2i	7K	
	BBP-761	Succinate pro-drug for LHON	20K	
	BBP-671	PanK 1/3 activator for PKAN, OAs	7K	
Genetic Dermatology	**BBP-009[4]**	Topical SMOi for Gorlin / frequent BCC	120K	
	BBP-589	COL7A protein replacement for RDEB	1.5K	
	BBP-681	Topical PI3Kαi for venous malformations	117K	
	BBP-561	Topical KLKN 5/7i for Netherton	11K	
Targeted Oncology	**BBP-831**	FGFR1-3i for FGFR+ cancers	37K*	
	BBP-454	Pan-mutant KRASi for KRAS+ cancers	>500K*	
	BBP-398	SHP2i for multiple cancer types	>500K*	
	BBP-954	GPX4i for multiple cancer types	>500K*	
Gene Therapy	**BBP-631**	AAV5 gene therapy for CAH	>75K	
	BBP-812	AAV9 gene therapy for Canavan	1K	



Phase 2/3 clinical development[3] Small molecule Topical small molecule Biologics Gene therapy

[1] Each of our programs is housed in a separate subsidiary; [2] Patient population: Prevalence except for asterisked figures which represent incidence; [3]A clinical trial we believe could support filing an application for marketing authorization, although the FDA and other regulatory authorities have not indicated their agreement or that additional trials will not be required; [4] 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. See "Business — Our Material Agreements—BBP-009 (Patidegib): Option Agreement with LEO Pharma A/S." [5]Planned New Drug Application (NDA) submission for the treatment of cholangiocarcinoma (CCA) as a second-line or later therapy (2L)

How to judge our performance – the ABC's

A ***high POTS***

Targeting genetic disease @ its source

×

B ***low $ spend / program***

Target less than $15 M to IND, where dollars are riskiest

×

C ***large # of programs***

Mirroring the early stage opportunity -- always discovering the next PellePharm or Eidos – 16 growing to 25

A B ***High ROI***

A C ***High probability of real revenue***

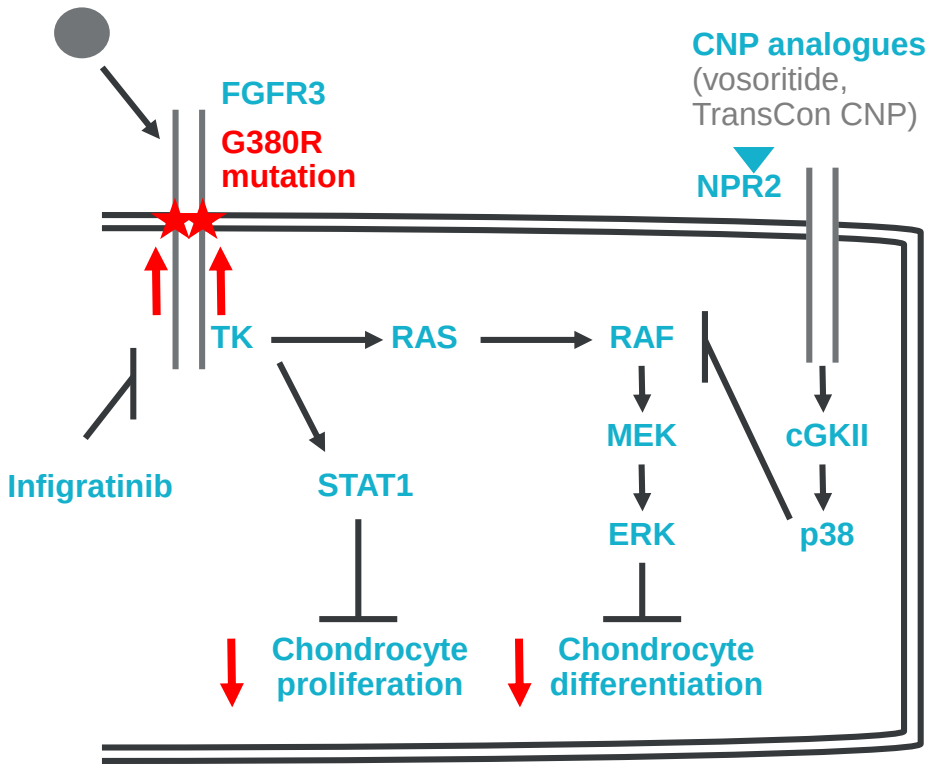
A B C ***High probability of real revenue, coming from many small plus a few large markets – 'synthetic' plus real blockbusters***

4 areas of near-term value in the BridgeBio portfolio

	Portfolio area	Next update
1	Low-dose infigratinib (FGFR1-3i) for achondroplasia	Begin dosing children in the Ph2 study 2020, data 2021
2	Genetic dermatology	Ph1/2 POC data in RDEB (rCOL7A) 2020; Ph3 data in Gorlin syndrome (topical SMOi) 2021
3	Targeted oncology	Infigratinib Ph2 update in CCA and NDA submission 2020; SHP2i IND 2020
4	Neuromuscular diseases	Begin rolling NDA submission in MoCD type A (cPMP replacement) 2019; Gene therapy IND 2020 (Canavan)

1 Achondroplasia – pathway and clinical features

Slight **FGFR3 gain-of-function** mutation leads to **dysregulated growth plate development**



Clinically, this leads to the following symptoms:

1. **Disproportionate short stature**, often leading to functional impairment
2. **Narrowed foramen magnum**, which can result in the need for urgent surgery
3. **Spinal stenosis** and compression, kyphosis, and back pain

By inhibiting FGFR3 directly, infigratinib targets both chondrocyte proliferation and differentiation defects that cause ACH

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

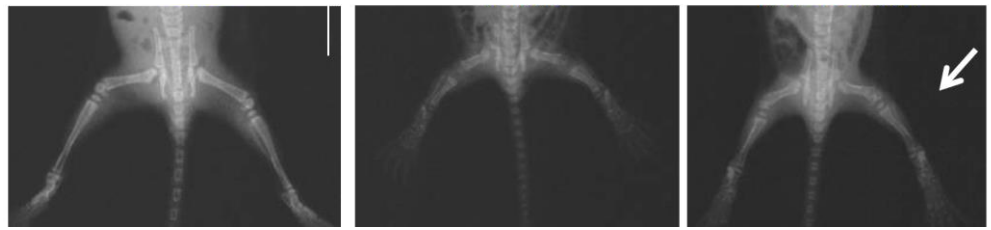
Infigratinib improves all the key drivers of clinical symptomology in the ACH mouse model

1 Disproportionate short stature

21% increase in femur length

33% increase in tibia length

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

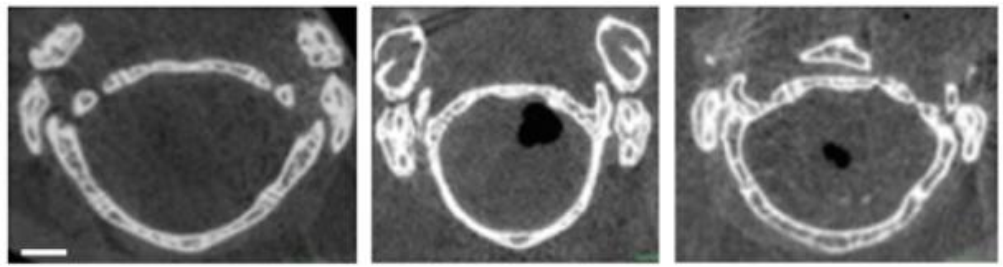


2 Narrowed foramen magnum

17% increase in FM area

6% increase in AP skull length

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

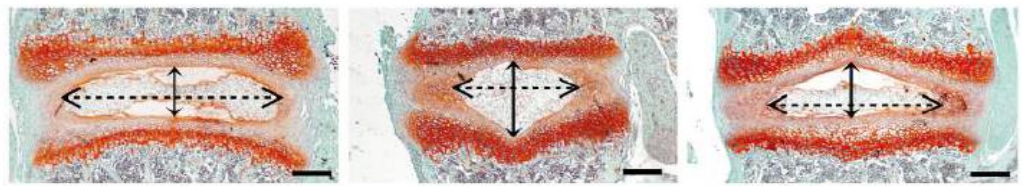


3 Spinal stenosis

12% increase in L4-L6 length

73% increase in disc width

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



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

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Infigratinib showed significant improvements in key disease features in the achondroplasia mouse model

Preclinical data from infigratinib

Ph1/2 clinical study to initiate by 2020 for infigratinib, the only oral asset in development

Percent increase compared to non-treated mouse

Asset	Company	Status	Mouse model	Tibia length	Femur length	Foramen magnum area	L4-L6 height
Infigratinib	bridgebio	Pre-Ph1/2	FGFR3$^{Y367C/+}$	33%	21%	16%	12%

Source: Komla-Ebri et al. J Clin Inv 2016
Note: Infigratinib dose of 2mg/kg

Preclinical data from other investigational achondroplasia therapies

Percent increase compared to non-treated mouse

Asset	Company	Status	Mouse model	Tibia length	Femur length	Foramen magnum area	L4-L6 height
Vosoritide (BMN111)	BIOMARIN	Pivotal	FGFR3$^{Y367C/+}$	7%	5%	No known publically available data	3%
TransCon CNP[1]	ascendis pharma	Ph1/2	FGFR3$^{Y367C/+}$	12%*			
TA-46	Therachon achieving potential	Ph1/2	FGFR3$^{ach/+}$	9%*	7%*		

Source: 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 FGFR3Y367C/+, FGFR3ACH/+ mouse unless otherwise noted
1 Based on vosoritide continuous infusion; *Value estimated using DigitizeIt

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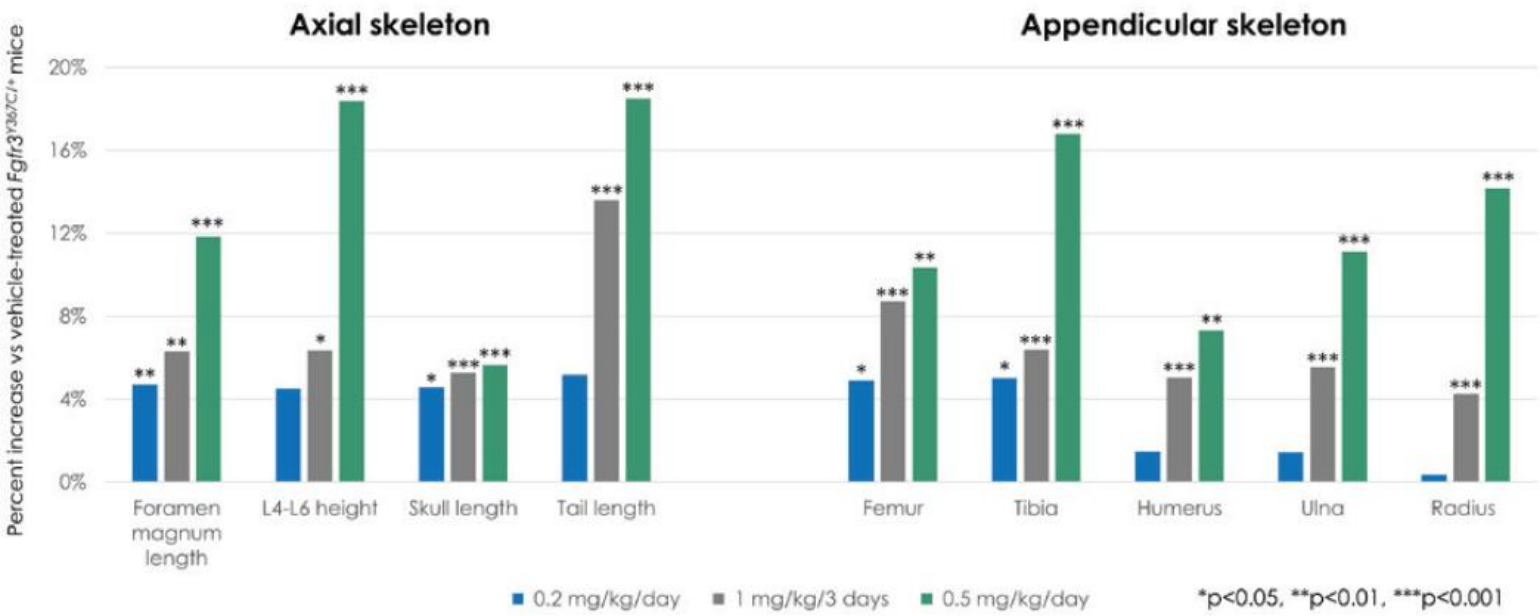
We showed significant and dose-dependent effects on key features of the achondroplasia model at ASHG 2019

These data continue to support our therapeutic hypothesis for this program

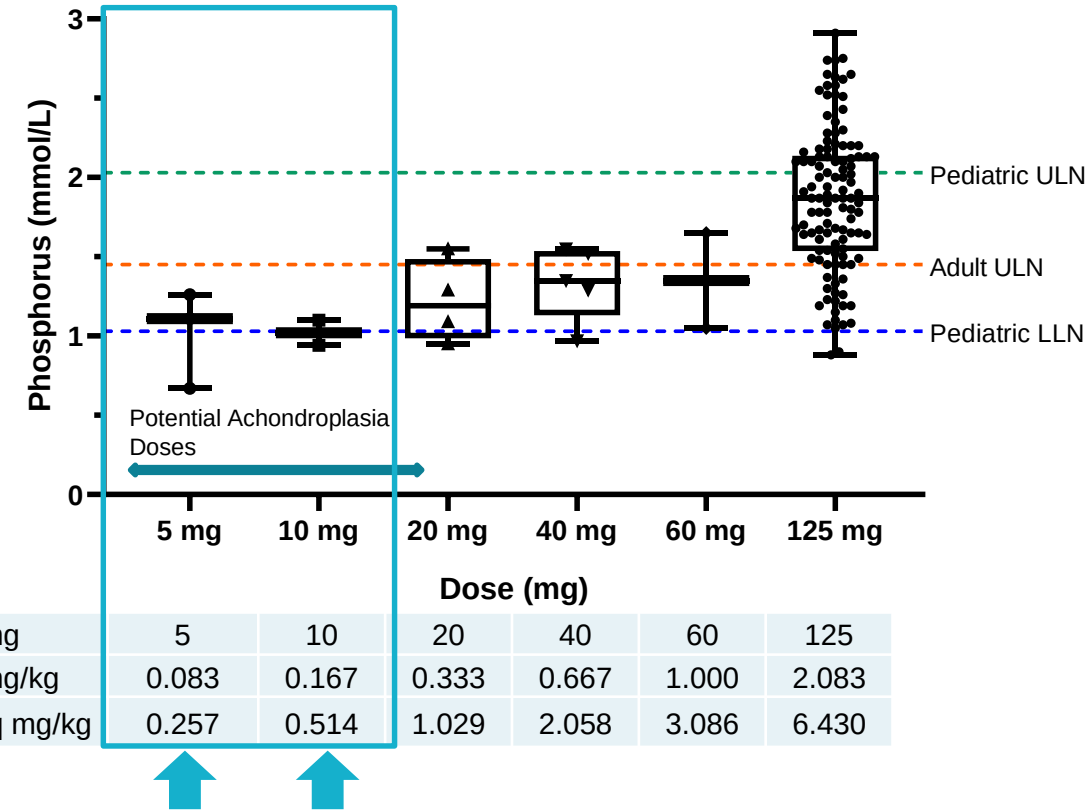


All doses had highly significant effects on survival relative to untreated animals

Source: ASHG 2019

Human clinical data suggests potential activity in achondroplasia below the threshold for on-target adverse events

We expect our active achondroplasia dose could be >50x lower than our oncology dose, well below known toxicity thresholds in humans



Human mg	5	10	20	40	60	125
Human mg/kg	0.083	0.167	0.333	0.667	1.000	2.083
Mouse sq mg/kg	0.257	0.514	1.029	2.058	3.086	6.430

5 mg, n=3
10 mg, n=2
20 mg, n=4
40 mg, n=5
60 mg, n=2
125 mg, n= 114

Pediatric
ULN = 2.03 mmol/L
LLN = 1.03 mmol/L
Adult
ULN =1.45 mmol/L
LLN = 0.8 mmol/L

Estimated mouse equivalent doses are similar to prior slide

Pediatric normal ranges: source https://www.healthcare.uiowa.edu/path_handbook/appendix/heme/pediatric_normals.html
Age range 1 – 15 years of age

Source: ASHG 2019

We initiated the PROPEL observational study in 3Q19 and are actively enrolling at multiple sites

PROPEL

PROPEL: observational study in children with ACH

- **Objectives:** anthropometric characteristics, medical events, treatments, etc.
- **Timing:** enrolling children in Australia and the UK now; expanding to other countries throughout 2019 and 2020

PROPEL2: Phase 2 dose finding study

- Participants in the observational study may have the opportunity to move onto PROPEL2
- **Objectives:** safe and tolerable dose; preliminary efficacy
- **Timing:** starting in 2020

PROPEL3: Phase 3 registrational trial

- Participants in the observational study may have the opportunity to move onto PROPEL3
- **Objective:** efficacy
- **Timing:** TBD, depending on PROPEL2 results

Visit www.clinicaltrial.gov for more information (NCT04035811)

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2 Genetic dermatology

Program	MOA / Diseases	Pre-clinical: Dis-covery	Pre-clinical: Pre-IND	Clinical: Phase 1	Clinical: Phase 2	Clinical: Phase 3	Next update
BBP-009	SMOi for Gorlin syndrome						Ph3 topline data in 2021
BBP-589	Recombinant COL7A for dystrophic epidermolysis bullosa						Ph1/2 topline data in 2020
BBP-681	Topical PI3Ka inhibitor for venous and lymphatic malformations						IND filing in 2020
BBP-561	Topical KLKN 5/7i for Netherton						Clinical candidate nomination

Other players in this space

Krystal

Abeona THERAPEUTICS

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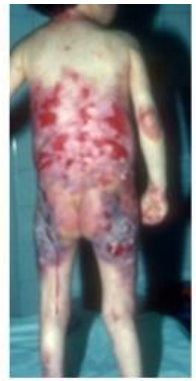
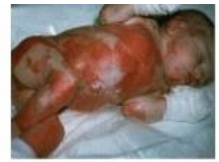
RDEB is a devastating rare genetic disease with no approved drugs

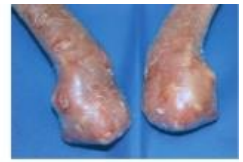
RDEB Impacts Multiple Tissues and Organs and is Associated with Early Death

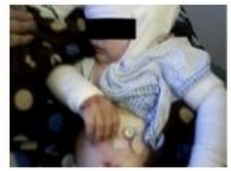
- Skin: Extensive scarring
- Severe impact to the GI track
- Corneal erosions
- Dystrophy and loss of nails
- Joint contractures
- Pseudosyndactyly (mitten deformity of the hand and feet)
- Patients often develop aggressive squamous-cell carcinoma, a major contributor to mortality; Cumulative risk of mortality at ~22% by age 55

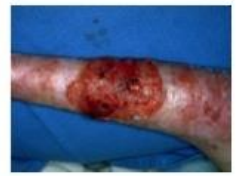

Painful erosions and blisters

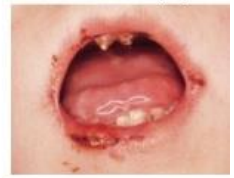

Extensive costly bandaging


Early morbidity with scarring


Mitten deformity requiring reconstructive surgery


Poor nutrition (e.g., from esophageal strictures) often requires a G-tube


High risk of mortality due to squamous cell carcinoma


Oral and dental problems

RDEB prevalence is estimated at ~1.5 per million on average; at least ~1,500 – 2,000 RDEB patients in the US/EU

Current standard of care for RDEB is largely palliative and results in profound impact on the lives of patients and families

"I was born with the worst disease you've never heard of…"

--- RDEB patient

Symptoms	Treatment
Open Wounds	Bathing, Wound Dressings
Infection	Topical Antiseptics, Antibiotics
Pain Management	Analgesics, NSAIDs, opioids, anti-anxiety drugs
Nutritional Compromise	Nutrient Supplements, G-tube Feeding
Pseudosyndactyly; esophageal strictures	Surgeries

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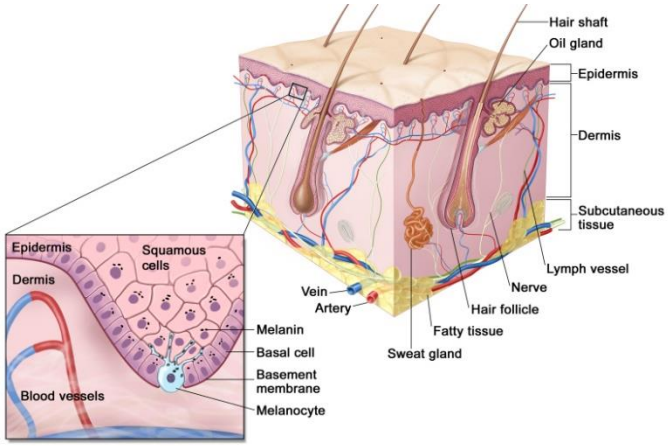
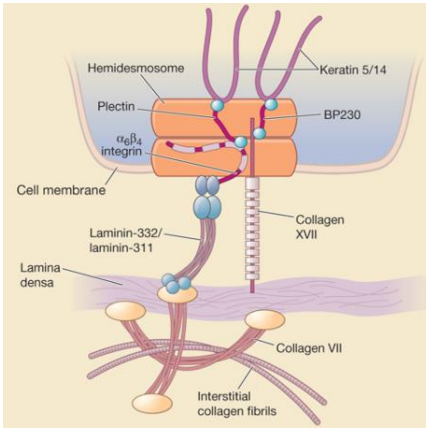
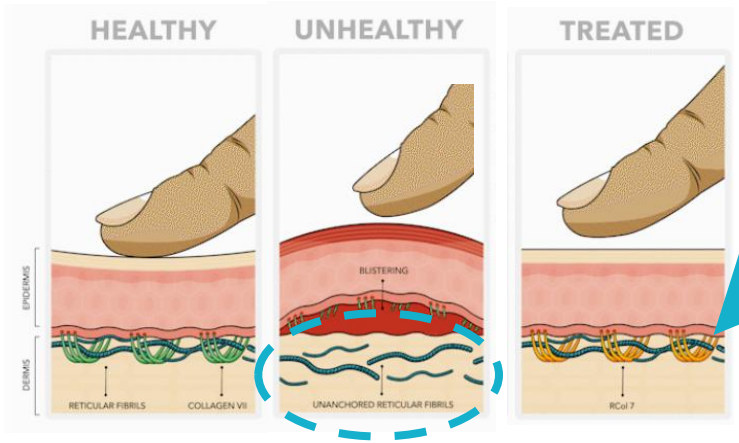
Disease mechanism and our treatment strategy



- Collagen type VII protein (C7) forms the anchoring fibril (AF) which secures the association of the epidermal basement membrane with the underlying dermis layer
- Anchoring fibril is identified in many tissues/organs, including: ***skin***, ***chorioamnion, cornea***, ***oral mucosa***, ***cervix***, ***esophagus***, and ***anal canal***
- Mutations within the *COL7A1* gene lead to absence or deficient level of functional collagen type VII protein (C7); thus in RDEB patients, the integral stability of the affected tissue/organ is compromised
- We are developing **PTR-01/BBP-589**, intravenously delivered recombinant C7 to treat the systemic manifestations of RDEB



Anchoring fibril (AF) made of C7 protein

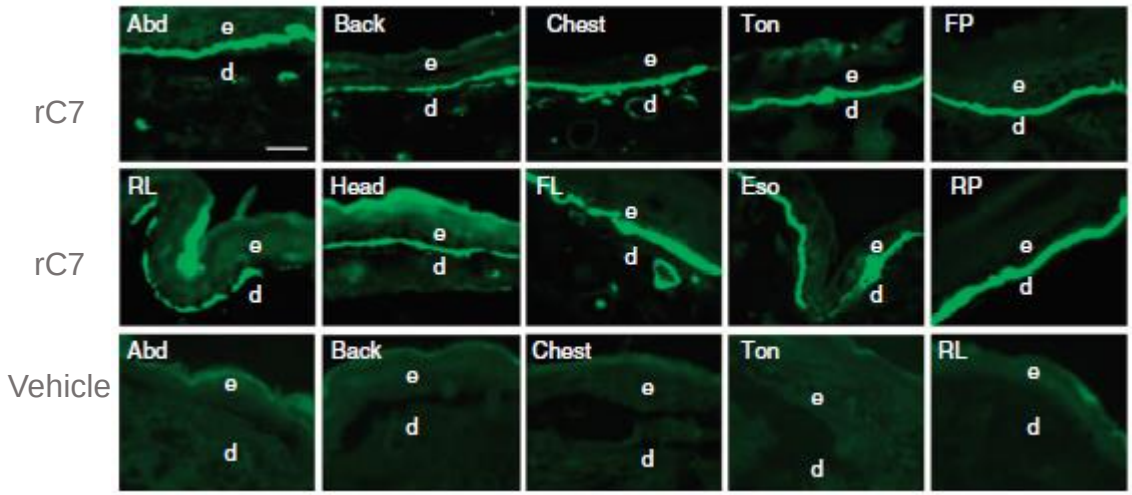


IV delivered recombinant C7 locates to the basement membrane, forms anchoring fibrils and re-tethers the epithelium

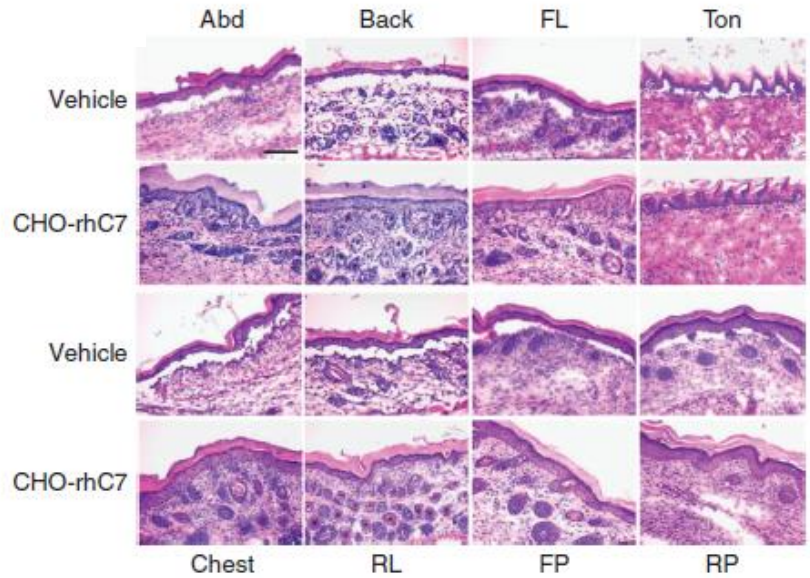
Clear preclinical proof-of-concept: rC7 distributes to the epidermis and forms anchoring fibrils

Single intravenous injection of rC7 distributed to the multiple skin sites of RDEB mice

e, *Epidermis****; d,*** *dermis;* ***Abd****, abdomen;* ***Eso****, esophagus;* ***FL****, front leg;* ***FP****, front paw;* ***RL****, rear leg;* ***RP****,rear paw;* ***Ton****, tongue*

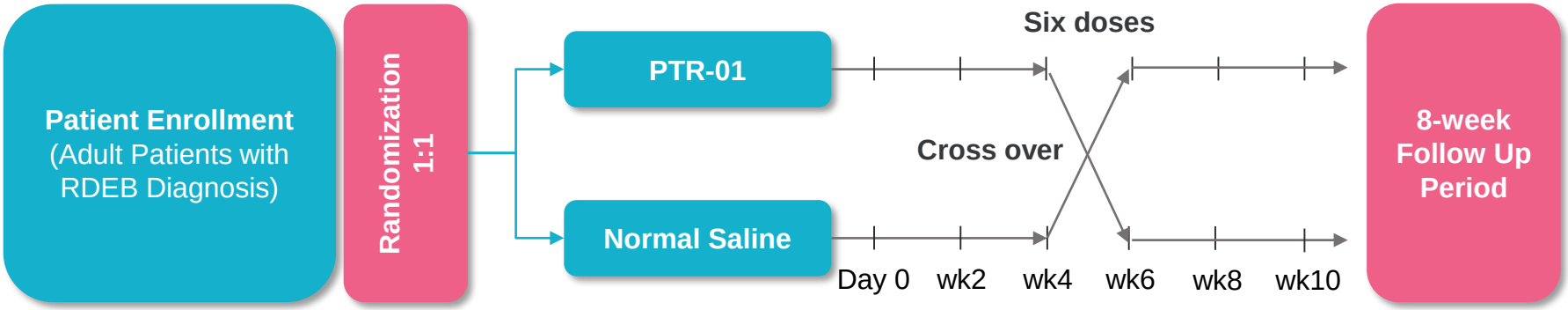


Following rC7 injection, RDEB mice skin exhibited good dermal–epidermal adherence compared to vehicle injected mice



Ongoing randomized, dose-escalation Phase 1/2 proof-of-concept clinical study in adults with RDEB

- First patient dosed in 1Q19
- **Anticipate clinical POC in 2020** (safety, tolerability, skin biopsy)



Cohort 1: 0.1 mg/Kg N=2 → **Cohort 2: 0.3 mg/Kg N=4** → **Cohort 3: 1 mg/Kg N=8**

- **Primary endpoints**
  - Safety and tolerability
- **Key secondary endpoints**
  - Pharmacokinetics
  - Change in skin biopsy (rC7 and anchoring fibrils)
  - Change in healing of up to 5 chronic wounds
  - Patient reported outcomes, including itch and quality-of-life assessments

Key secondary endpoints designed to show POC efficacy including key biomarkers, wound, blister formation and overall quality of life assessment

3 Targeted oncology

Program	MOA / Diseases	Pre-clinical: Dis-covery	Pre-clinical: Pre-IND	Clinical: Phase 1	Clinical: Phase 2	Clinical: Phase 3	Next update
Infigratinib (BBP-831)	FGFR1-3 inhibitor for FGFR+ tumors			▶	▶	▶	Ph2 pivotal CCA data 2020, NDA 2020
BBP-398	SHP2 inhibitor for multiple solid tumor types			▶			IND submission in 2020
BBP-454	Pan-mutant KRAS inhibitor for KRAS+ cancer			▶			Clinical candidate nomination
BBP-954	GPX4 inhibitor for multiple solid tumor types			▶			Clinical candidate nomination

Other players in the targeted oncology space

MIRATI THERAPEUTICS

TP Therapeutics® Turning Point Medicines for Life!

agios

LOXO ONCOLOGY

blueprint™ MEDICINES

REVOLUTION MEDICINES

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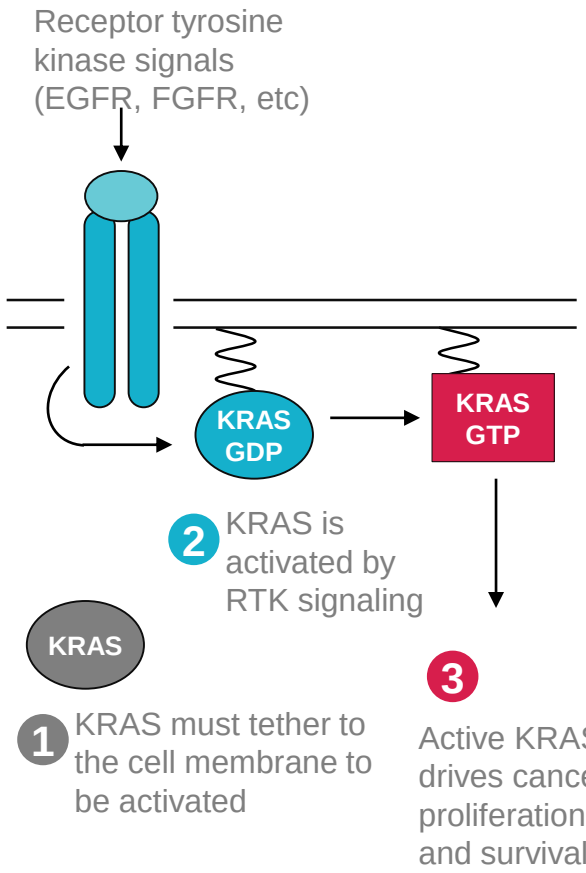
We have two shots on goal with our pan-mutant KRAS inhibitor programs – each with a unique and novel MOA

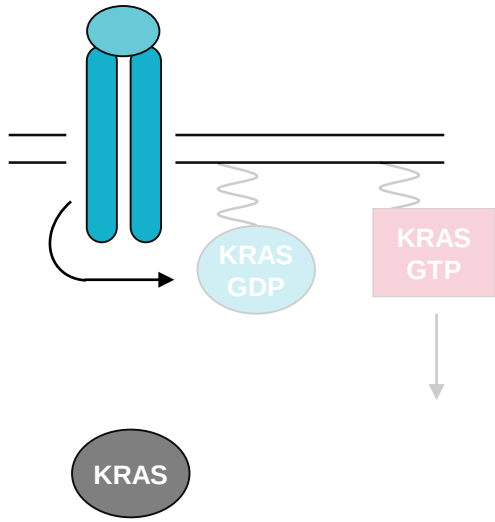
KRAS activation in cancer is a multi-step process



Our two programs target different steps of the KRAS activation process

1st approach: C185 targeting

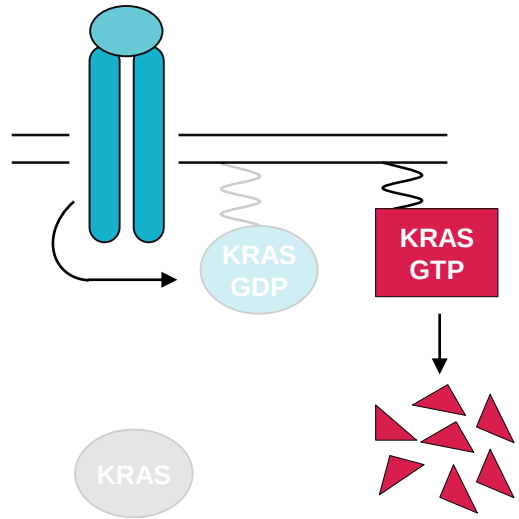
- Blocks KRAS from tethering
- Blocks activated KRAS GTP from signaling



KRAS tethering is blocked – cancer growth is inhibited

2nd approach: H95 targeting

- Directly binds activated KRAS
- Triggers KRAS degradation, blocking the signal



Activated KRAS is degraded – cancer growth is inhibited

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4 Neuromuscular diseases

Program	MOA / Diseases	Pre-clinical: Dis-covery	Pre-clinical: Pre-IND	Clinical: Phase 1	Clinical: Phase 2	Clinical: Phase 3	Next update
BBP-870	cPMP replacement for MoCD type A						Begin rolling NDA submission by YE19
BBP-418	Glycosylation substrate for LGMD2i						IND submission and Ph1 initiation
BBP-812	Gene therapy for Canavan disease						IND submission in 2020
BBP-671	PanK activator for PKAN and organic acidemias						Clinical candidate nomination

Other players in the neuromuscular space

SAREPTA THERAPEUTICS

avexis

IONIS

PTC THERAPEUTICS

AUDENTES THERAPEUTICS

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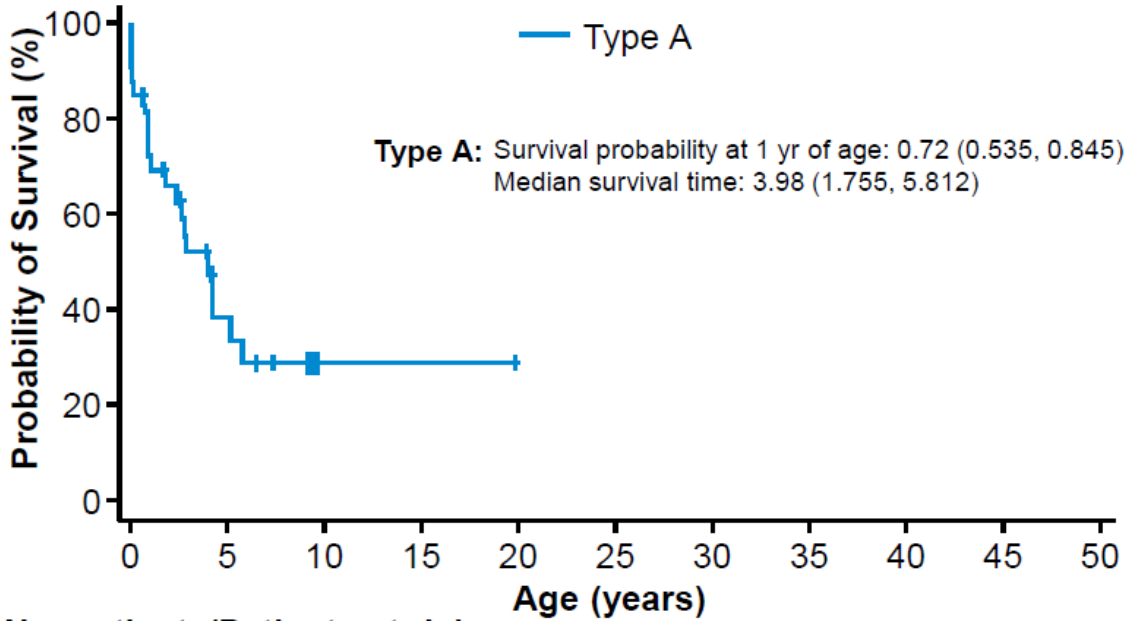
We presented data from our natural history study in MoCD type A at SSIEM 2019

- Median survival time of <4y highlights urgent need for a new medicine
- Data will play an important role in our NDA data package
- **Our rolling NDA submission has initiated!**

Kaplan-Meier Estimates of Survival Probability (FAS)



Type A: Survival probability at 1 yr of age: 0.72 (0.535, 0.845)
Median survival time: 3.98 (1.755, 5.812)

No. patients/Patients at risk

Type A:	0/33	18/8	20/1	20/1	20/0	20/0	20/0	20/0	20/0	20/0	20/0

Note: Kaplan-Meier curves step down at time points at which a death has been observed; slashes represent patients whose observation time was censored as of the last contact date.

AHA UPDATE: AG10 (BBP-265) our transthyretin stabilizer for the treatment of transthyretin amyloidosis

Mechanism of Disease

Native transthyretin circulates in blood as a tetramer

Dissociation into toxic monomers initiates pathogenesis

Monomers aggregate and deposit as amyloid, causing disease
ATTR-CM, ATTR-PN

Impact on patients:

- Progressive, fatal cardiomyopathy (median life expectancy 3-5 yrs) and polyneuropathy
- Both manifestations includes significant disability
- We believe the diagnosed population of ATTR-CM is growing rapidly due to awareness and accurate, non-invasive, diagnostic methods

Mechanism of Drug

AG10 stabilizes TTR tetramer, potentially preventing disease

Dissociation into toxic monomers initiates pathogenesis

Monomers aggregate and deposit as amyloid, causing disease
ATTR-CM, ATTR-PN

AG10 is designed to bind TTR in a way that mimics a naturally-occurring protective mutation

Program Highlights

400k+
ATTR-CM patients worldwide

10k+
ATTR-PN patients worldwide

Clinical status:

Pre-IND	Phase 1	Phase 2	Phase 3

ATTR-CM Ph3 study ongoing (FPI 1Q19)

ATTR-PN Ph3 study expected 1Q20

Catalysts:

- Complete enrolment in Ph3 ATTR-CM 2H20
- Ph3 ATTR-CM 12-month data in 2021
- Potential ATTR-CM NDA submission in 2021

Key data:

- Ph2 ATTR-CM data presented in at AHA 2019
  - Normalized serum TTR in all actively treated patients at d28 and through 65 weeks on therapy
  - Durable stabilization of cardiac biomarkers over 6m
  - 8.5% all cause mortality and 25% CV hospitalization at 15m
  - TTR stabilization of ≥90% in all actively treated pts at d28
- Ph3 ATTRibute study initiated in 1Q19
  - Potential registration on 12m 6MWD endpoint, followed by 30m CV outcome/hospitalization endpoint

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Preclinical data: We showed comparative stabilization potency between AG10 and tafamidis

Near complete tetramer stabilization by AG10 across a range of mutations

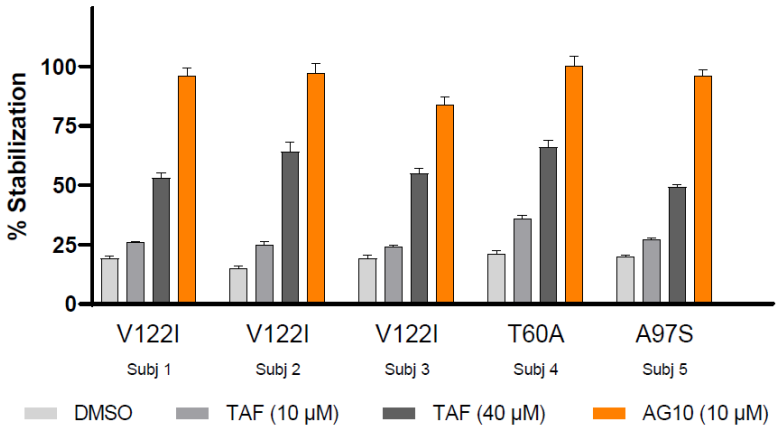


Figure 2. *Western Blot quantitation of tetrameric TTR in plasma samples subjected to low pH conditions.* ***A)*** *Representative Western Blot image with tetrameric TTR stabilization at 72 hr. WT depicts pooled normal human plasma.* ***B)*** *Percent stabilization of tetrameric TTR after 72 hr acidification. Results from five individuals with destabilizing TTR mutations are shown. Mean and standard error shown.*

Near complete target occupancy by AG10

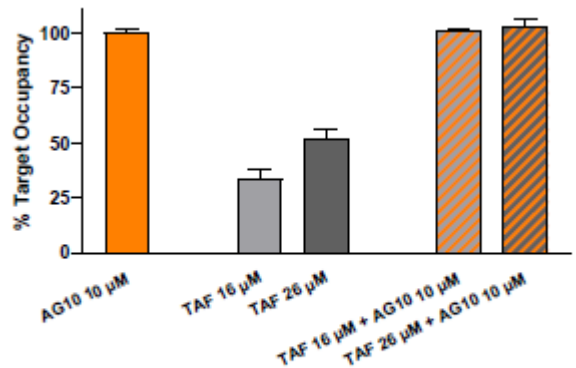
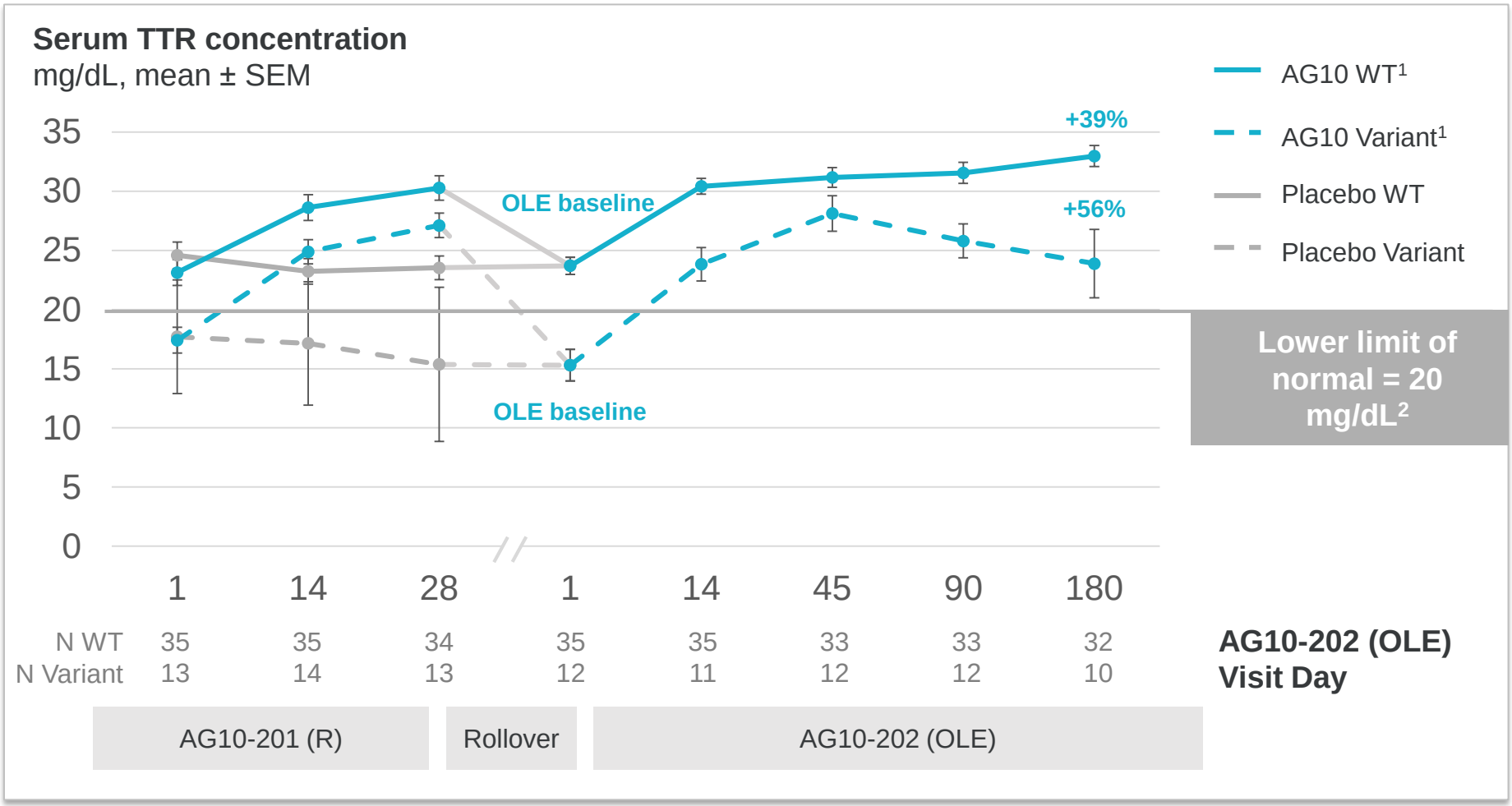


Figure 4. *FPE characterization of TTR binding site occupancy in serum incubated with stabilizer, n = 12 for each condition tested. Target occupancy with mean and standard deviation shown.*

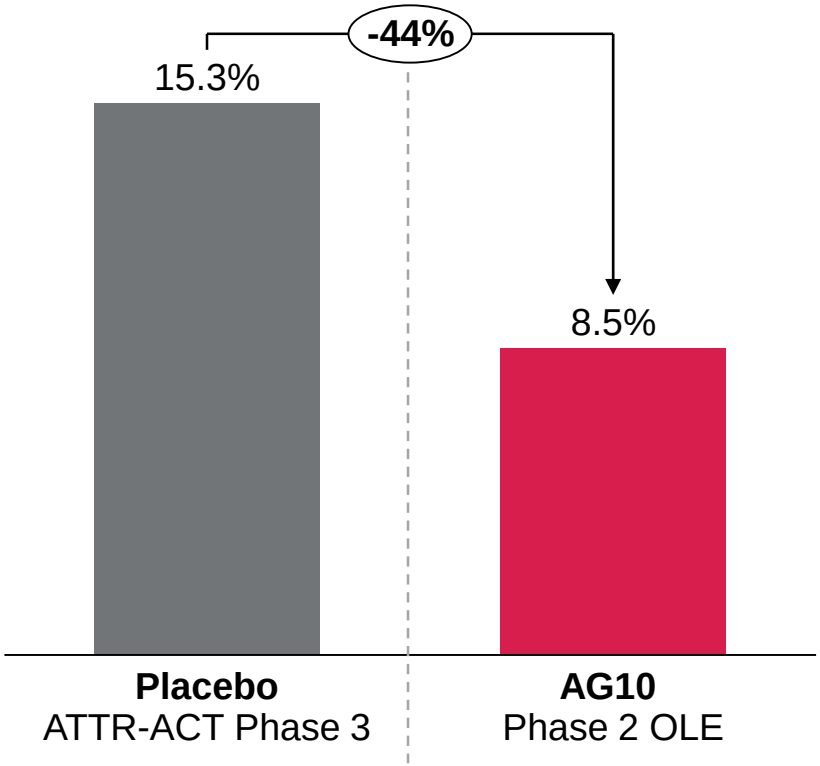
Ph2 OLE data: Serum TTR levels increased upon AG10 treatment and were maintained throughout study duration



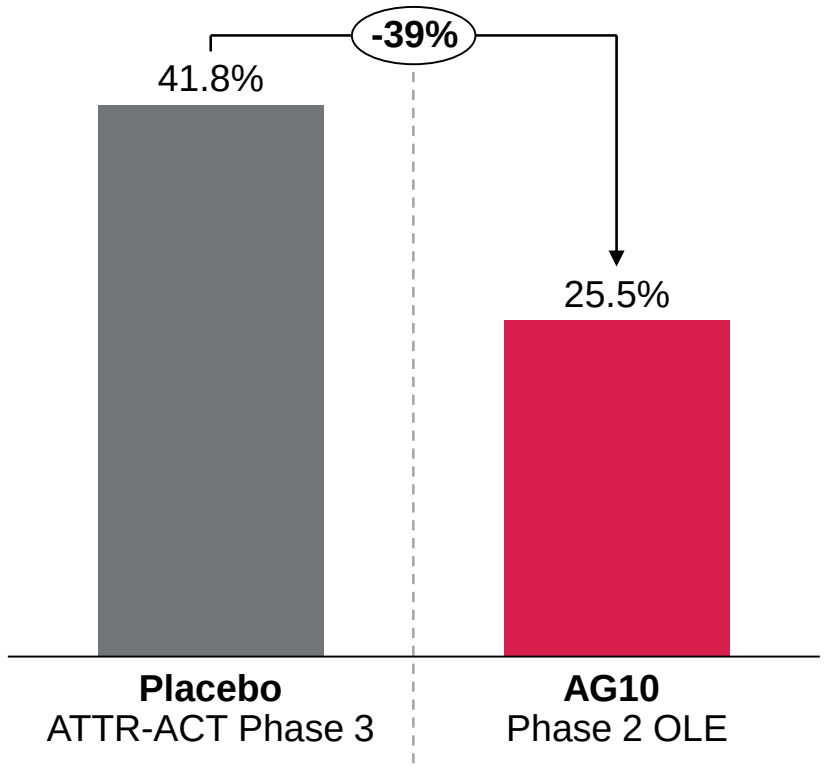
1 400mg and 800mg BID AG10 groups pooled during randomized portion
2 Defined as the lower limit of the reference interval for the serum prealbumin (TTR) clinical laboratory assay

Ph2 OLE data: 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 2H20

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

Where we are headed: Profile by year-end 2021

“Shots on goal”

Based on our projected 2019-2021 catalyst map

Pipeline Advancement + **Add New Programs**

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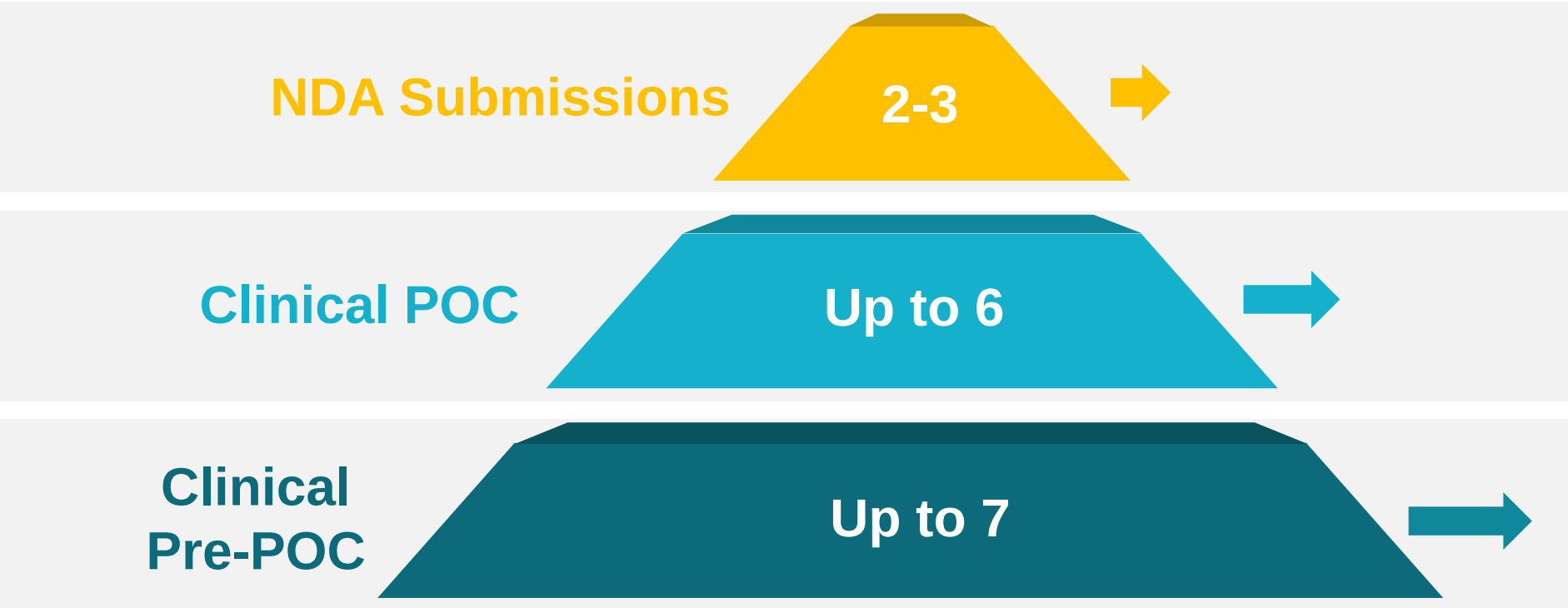
2-3 new programs/year



NDAs: BBP-831 (2L CCA), BBP-870, BBP-265
Clinical Proof-of-Concept (POC): BBP-831 (achon), BBP-631, BBP-009, BBP-681, BBP-589, 0-1 additional
Clinical: BBP-812, BBP-398, BBP-711, BBP-561, BBP-671, 1-2 additional
Note: The above represents potential non-risk adjusted outcomes

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Our cash balance provides runway through key value inflections across the portfolio

ESTIMATED

2019		2020	2021
1H	**2H**	**FY**	**FY**
✓ Initiate BBP-265 (AG10) Ph3 in ATTR-CM	✓ Initiate achondroplasia observational run-in study (infigratinib)	❑ Topline Ph1/2 data from BBP-589 in RDEB	❑ Topline AG10 Ph3 data in ATTR-CM (12m 6MWD)
✓ Initiate BBP-009 Ph3 in Gorlin	✓ Announce new program (BBP-418 for LGMD2i)	❑ Updated infigratinb Ph2 CCA data	❑ Topline BBP-009 Ph3 data in Gorlin
✓ Open infigratinib Ph3 in 1L CCA for enrollment	✓ CAH gene therapy (BBP-631) NHP data at ESGCT	❑ Initiate infigratinib adjuvant urothelial carcinoma Ph3 study	❑ Infigratinib Ph2 PoC data in achondroplasia
✓ Initiate BBP-589 Ph1/2 in RDEB	✓ AG10 Ph2 ATTR-CM OLE data at AHA (November 16)	❑ Initiate dosing in infigratinib Ph2 in achondroplasia	❑ CAH gene therapy (BBP-631) clinical PoC data
	✓ Initiate BBP-870 rolling NDA in MoCD Type A	❑ Submit infigratinib NDA for 2L CCA	❑ Infigratinib approval and launch in 2L CCA
		❑ Multiple new INDs	❑ BBP-870 approval and launch in MoCD Type A
		❑ Multiple new program announcements	

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