BRIDGEBIO

PROPEL 3 Phase 3 Topline Results of Infigratinib in Achondroplasia

February 12, 2026

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Agenda

1	**Introduction**	Neil Kumar, Ph.D. Chief Executive Officer, BridgeBio
2	**PROPEL 3 Summary**	Justin To Chief Executive Officer, Skeletal Dysplasias
3	**PROPEL 3 Phase 3 Topline Results**	Daniela Rogoff, M.D., PhD. Chief Medical Officer, Skeletal Dysplasias
4	**Program Next Steps**	Justin To Chief Executive Officer, Skeletal Dysplasias
5	**Launch Readiness**	Matt Outten, M.B.A. Chief Commercial Officer, BridgeBio

Introduction

Neil Kumar, Ph.D.
Chief Executive Officer,
BridgeBio

PROPEL3

Infigratinib for Achondroplasia

A sincere THANK YOU to families, advocates, investigators, clinical research staff, and collaborating research partners

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

> 8,500
individuals impacted by our therapies

Obtained Approval for 3 Medicines

Attruby™ (acoramidis) 356 mg tablet

Nulibry® (fosdenopterin) for injection

TRUSELTIQ™ (infigratinib) capsules

3 Positive Phase 3 Results

fortify ✓

CALIBRATE ✓

PROPEL3 ✓

CANaspire

+ 1 Ongoing Pivotal Trials

15 active trials in ecosystem	**> 70** papers published	**> 35** academic partnerships	**19** INDs created	**< $40M** spend to proof-of-concept data

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

Source: internal estimates. IND: Investigational New Drug application; FTE: Full Time Employee.

We target well-described diseases at their source, underpinning our industry-leading probability of technical success

Probability of Technical Success (POTS)

Clinical Dev. Stage	Industry Benchmark[1]	BBIO Historical[2]
Phase 1	52%	86%
Phase 2	29%	71%
Phase 3	58%	86%
Cumulative	9%	52%

Approaching engineering-like success

- **Certain Rare/Genetic Disease sub-categories are particularly likely to succeed**
- **For example, protein replacements for rare monogenic diseases have an exceptional track record**

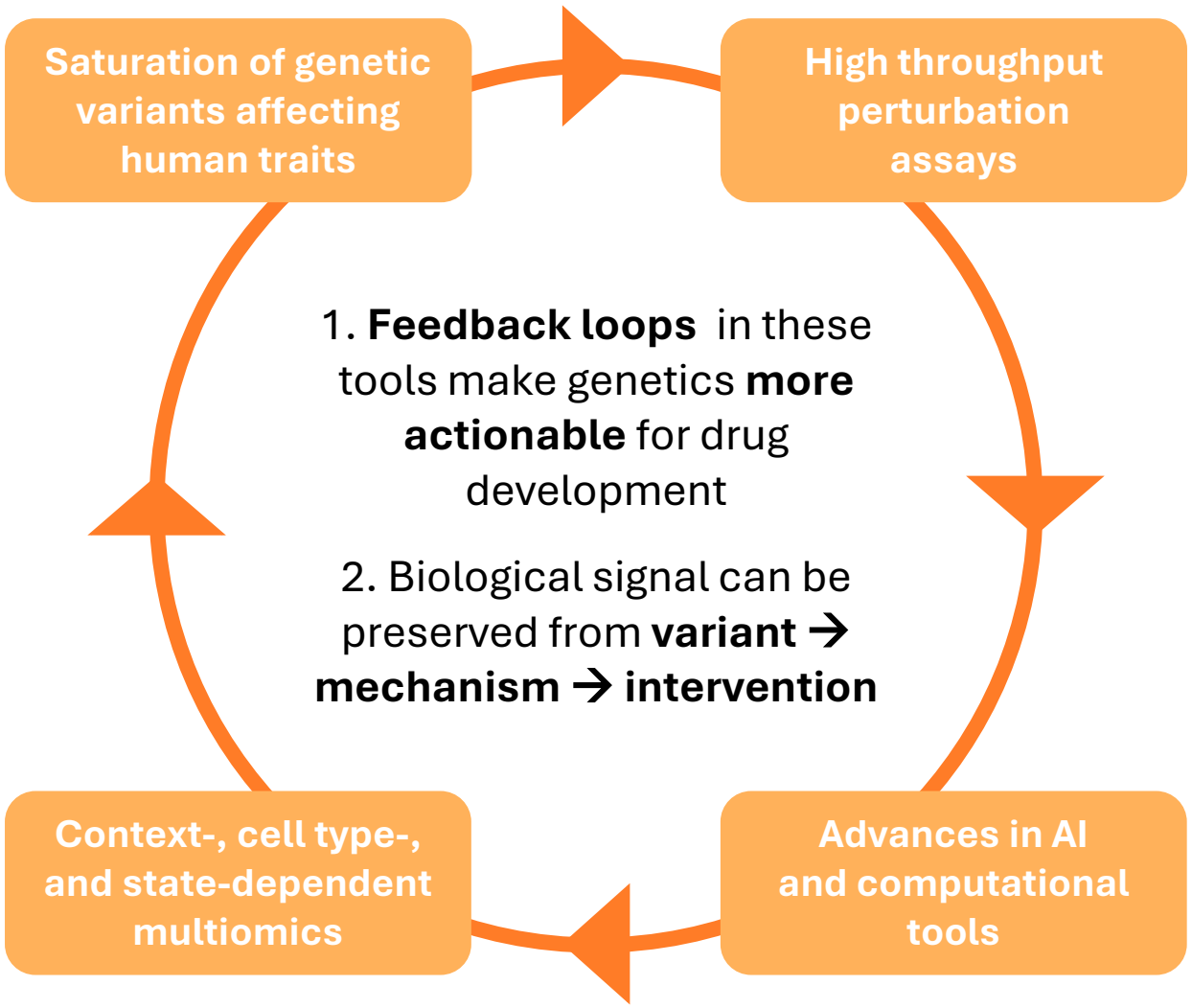
POTS for monogenic protein replacement therapies } **88%**

- **Genzyme and Shire achieved high rates of POTS across disease states as a result**

Source: [1] Biotechnology Innovation Organization (BIO), QLS Advisors, and Informa Pharma Intelligence, *Clinical Development Success Rates and Contributing Factors 2011–2020* (2021). [2] Internal data on file (2015 – 2025). Includes programs initiated at BridgeBio that continue to be advanced at sister companies BridgeBio Oncology Therapeutics and GondolaBio. [3] Gorzelany, J.A. and Souza, M.P., Science Translational Medicine, "Protein Replacement Therapies for Rare Diseases: A Breeze for Regulatory Approval?", Mar. 2013

We are at day 1 of genetic medicine: Our engine sits on top of an explosion of new science and promising substrate

- Larger Biobanks: UKB, AOU, ADG, etc.
- WGS for non-coding rare variants
- Long-read sequencing for complex SVs



- Perturb-seq to perturb every single gene across the genome
- MAVE to perturb every possible variant within a gene

- Cell atlases: Human Cell Atlas, Tabula Sapiens
- Tissue mapping: HuBMAP
- Large-scale compendia: CellxGENE, TenK10K

- Variant effect prediction: Alphamissense
- Pangenome graphs
- Disease prediction: MILTON

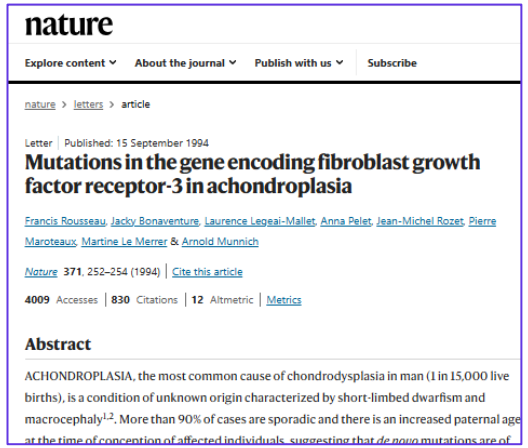
PROPEL 3
Phase 3 Topline Overview

Justin To
Chief Executive Officer
Skeletal Dysplasias

Infigratinib: Connecting the dots between genetics, nonclinical, and clinical data to do more for families and individuals living with achondroplasia

We are developing the first therapeutic option that directly targets FGFR3 gain-of-function because we believe that we can and should do more and better for families and individuals living with achondroplasia.

1994


nature

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nature > letters > article

Letter | Published: 15 September 1994

Mutations in the gene encoding fibroblast growth factor receptor-3 in achondroplasia

Francis Rousseau, Jacky Bonaventure, Laurence Legeai-Mallet, Anna Pelet, Jean-Michel Rozet, Pierre Maroteaux, Martine Le Merrer & Arnold Munnich

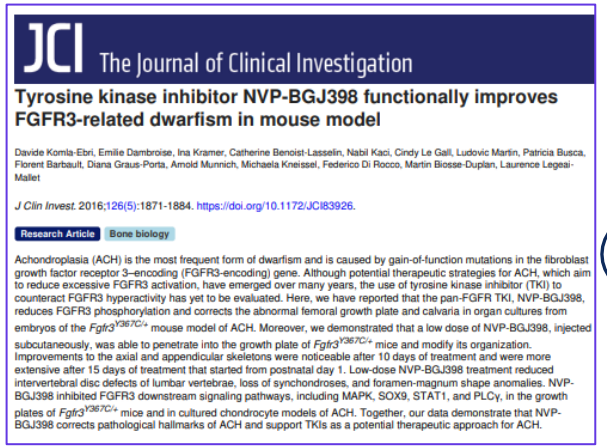
Nature 371, 252–254 (1994) | Cite this article

4009 Accesses | 830 Citations | 12 Altmetric | Metrics

Abstract

ACHONDROPLASIA, the most common cause of chondrodysplasia in man (1 in 15,000 live births), is a condition of unknown origin characterized by short-limbed dwarfism and macrocephaly[1,2]. More than 90% of cases are sporadic and there is an increased paternal age at the time of conception of affected individuals, suggesting that *de novo* mutations are of

2016


JCI The Journal of Clinical Investigation

Tyrosine kinase inhibitor NVP-BGJ398 functionally improves FGFR3-related dwarfism in mouse model

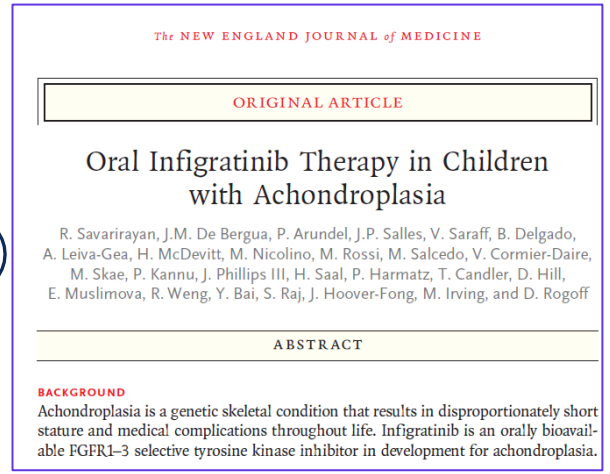
Davide Komla-Ebri, Emilie Dambroise, Ina Kramer, Catherine Benoist-Lasselin, Nabil Kaci, Cindy Le Gall, Ludovic Martin, Patricia Busca, Florent Barbault, Diana Graus-Porta, Arnold Munnich, Michaela Kneissel, Federico Di Rocco, Martin Biosse-Duplan, Laurence Legeai-Mallet

J Clin Invest. 2016;126(5):1871-1884. https://doi.org/10.1172/JCI83926.

Research Article | Bone biology

Achondroplasia (ACH) is the most frequent form of dwarfism and is caused by gain-of-function mutations in the fibroblast growth factor receptor 3–encoding (FGFR3-encoding) gene. Although potential therapeutic strategies for ACH, which aim to reduce excessive FGFR3 activation, have emerged over many years, the use of tyrosine kinase inhibitor (TKI) to counteract FGFR3 hyperactivity has yet to be evaluated. Here, we have reported that the pan-FGFR TKI, NVP-BGJ398, reduces FGFR3 phosphorylation and corrects the abnormal femoral growth plate and calvaria in organ cultures from embryos of the $Fgfr3^{Y367C/+}$ mouse model of ACH. Moreover, we demonstrated that a low dose of NVP-BGJ398, injected subcutaneously, was able to penetrate into the growth plate of $Fgfr3^{Y367C/+}$ mice and modify its organization. Improvements to the axial and appendicular skeletons were noticeable after 10 days of treatment and were more extensive after 15 days of treatment that started from postnatal day 1. Low-dose NVP-BGJ398 treatment reduced intervertebral disc defects of lumbar vertebrae, loss of synchondroses, and foramen-magnum shape anomalies. NVP-BGJ398 inhibited FGFR3 downstream signaling pathways, including MAPK, SOX9, STAT1, and PLCγ, in the growth plates of $Fgfr3^{Y367C/+}$ mice and in cultured chondrocyte models of ACH. Together, our data demonstrate that NVP-BGJ398 corrects pathological hallmarks of ACH and support TKIs as a potential therapeutic approach for ACH.

2024


The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Oral Infigratinib Therapy in Children with Achondroplasia

R. Savarirayan, J.M. De Bergua, P. Arundel, J.P. Salles, V. Saraff, B. Delgado, A. Leiva-Gea, H. McDevitt, M. Nicolino, M. Rossi, M. Salcedo, V. Cormier-Daire, M. Skae, P. Kannu, J. Phillips III, H. Saal, P. Harmatz, T. Candler, D. Hill, E. Muslimova, R. Weng, Y. Bai, S. Raj, J. Hoover-Fong, M. Irving, and D. Rogoff

ABSTRACT

BACKGROUND

Achondroplasia is a genetic skeletal condition that results in disproportionately short stature and medical complications throughout life. Infigratinib is an orally bioavailable FGFR1–3 selective tyrosine kinase inhibitor in development for achondroplasia.

Today

PROPEL3

Sources: Rousseau et al., 1994, Komla-Ebri et al., 2016, Savarirayan et al., 2025, **Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.*

Infigratinib exceeded the bar for a potential best-in-class therapeutic option, in the broadest age range studied across any achondroplasia trial

Target Clinical Profile for Commercial Success	Outcomes
CFBL in AHV: More than +1.5 cm/yr against placebo at Week 52	**Met the primary endpoint** with mean difference against placebo of **+2.10 cm/yr** (p<0.0001), and a LS mean difference against placebo of **+1.74 cm/yr** (p<0.0001), the largest change observed in a randomized trial for ACH.
CFBL in height Z-score (ACH charts): More than +0.3 SD on treatment arm at Week 52	The LS mean improvement on the tx arm was +0.41 SD, the largest improvement observed in a RCT in ACH. LS mean difference against placebo was +0.32 SD (p<0.0001), the largest difference observed in a RCT in ACH.
Proportionality: More than 0.05 decrease in upper to lower body ratio on treatment arm	In a pre-specified exploratory analysis of children <8yrs of age (>50% of trial), there was a statistically significant LS mean change from baseline against placebo of -0.05 (p<0.05). In the overall population, infigratinib achieved a LS mean decrease of -0.05 on the tx arm, with a favorable LS treatment difference of -0.02 versus placebo at Week 52 (p=0.1849)
Safety: No symptomatic hypotension. Less than 10% low-grade hyperphosphatemia rate.	Well-tolerated safety profile, **consistent with no inhibition of FGFR1 and FGFR2**. ▪ No discontinuations related to study drug ▪ No serious adverse events related to study drug ▪ 3 cases of hyperphosphatemia (4%), all mild, asymptomatic, transient and did not require dose reduction or discontinuation ▪ No FGFR1 or FGFR2 associated AEs (E.g., retinal or corneal) ▪ No AEs associated with CNP analogues: symptomatic hypotension, ISRs, or hypertrichosis

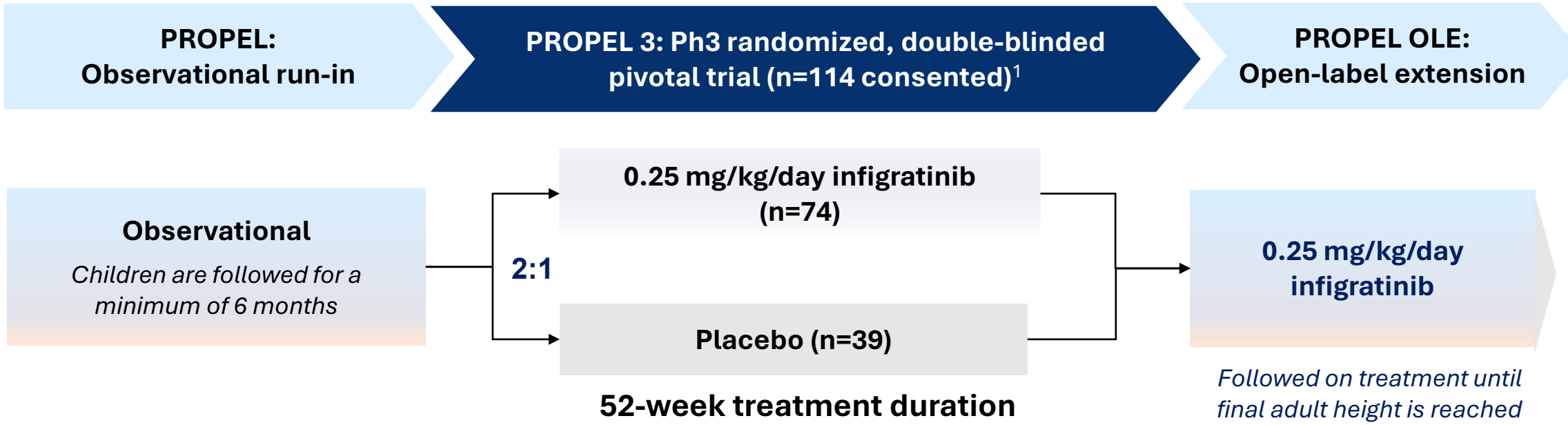
BBIO data on file; Tx : treatment; pbo : placebo; ISR : injection site reactions; AEs : adverse events; RCT : randomized controlled trial

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

PROPEL 3
Phase 3 Topline Results

Daniela Rogoff, M.D., Ph.D.
Chief Medical Officer
Skeletal Dysplasias

PROPEL 3 - Phase 3 Registrational Study Design



Key inclusion criteria

- Children 3 – <18 years old with open epiphyses

Primary endpoint:

- Change from baseline in annualized height velocity (AHV) at week 52 compared to placebo

Key secondary endpoints:

- Change from baseline in height z-score (in relation to ACH tables)
- Change from baseline in upper body : lower body segment ratio

Other secondary endpoints:

- Change in physical functioning; HRQoL, cognitive function, participant and caregiver evaluation of treatment benefit (qualitative interview)

1-One participant withdrew consent before dosing, Clinicaltrials.gov ID: NCT06164951, HRQoL: health-related quality of life, ACH: achondroplasia

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Demographic and baseline characteristics

Baseline demographics	Infigratinib (n=74)	Placebo (n=39)	All Participants (n=113)
Age (yrs), mean (SD) (min, max)	7.92 (2.7) (3.7 – 14.4)	7.74 (2.5) (3.9 – 14.9)	7.86 (2.7) (3.7 – 14.9)
Age group, n (%)			
3 – <5	10 (13.5)	4 (10.3)	14 (12.4)
5 – <11	50 (67.6)	31 (79.5)	81 (71.7)
11 – <18	14 (18.9)	4 (10.3)	18 (15.9)
Sex			
Male, n (%)	42 (56.8)	23 (59.0)	65 (57.5)
Female, n (%)	32 (43.2)	16 (41.0)	48 (42.5)
Baseline AHV (cm/year), mean (SD)	4.28 (1.39)	4.57 (1.45)	4.38 (1.41)
Height z-score (ACH reference), mean (SD)	0.09 (0.87)	-0.00 (1.00)	0.05 (0.91)

The study population was adequate to evaluate the study objectives, with well balanced treatment arms

BBIO data on file; SD : standard deviation; AHV : annualized height velocity

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Primary endpoint: Change from baseline to Week 52 in AHV

	Infigratinib (n=74)	Placebo (n=39)
Baseline AHV (cm/yr), Mean (SE)	4.28 (0.16)	4.57 (0.23)
Week 52 AHV (cm/yr), Mean (SE)	5.75 (0.15)	3.95 (0.14)
CFBL AHV at Week 52, Mean (SE)	1.48 (0.22)	-0.62 (0.29)
Infigratinib vs. placebo difference, Mean (95% CI) **P-value[1]**	**2.10 (1.38, 2.81)** **p < 0.0001**	
CFBL AHV at Week 52, LS Mean (SE)	1.58 (0.19)	-0.16 (0.23)
Primary Endpoint: Infigratinib vs. placebo difference, LS Mean (95% CI) **P-value[2]**	**1.74 (1.31, 2.17)** **p < 0.0001**	

Infigratinib has demonstrated the largest CFBL in AHV at Week 52 in a randomized trial

BBIO data on file; 1- Nominal; 2- ANCOVA model; CFBL : change from baseline; AHV : annualized height velocity

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Subgroup analyses: Change from baseline to Week 52 in AHV by age group

Age subgroups	3 to < 5 years		5 to < 11 years		11 to < 18 years	
	Infigratinib (n=10)	Placebo (n=4)	Infigratinib (n=50)	Placebo (n=31)	Infigratinib (n=14)	Placebo (n=4)
Infigratinib vs. placebo difference, Mean	2.30		1.93		2.78	
Infigratinib vs. placebo difference, LS Mean	1.47		1.84		1.40	

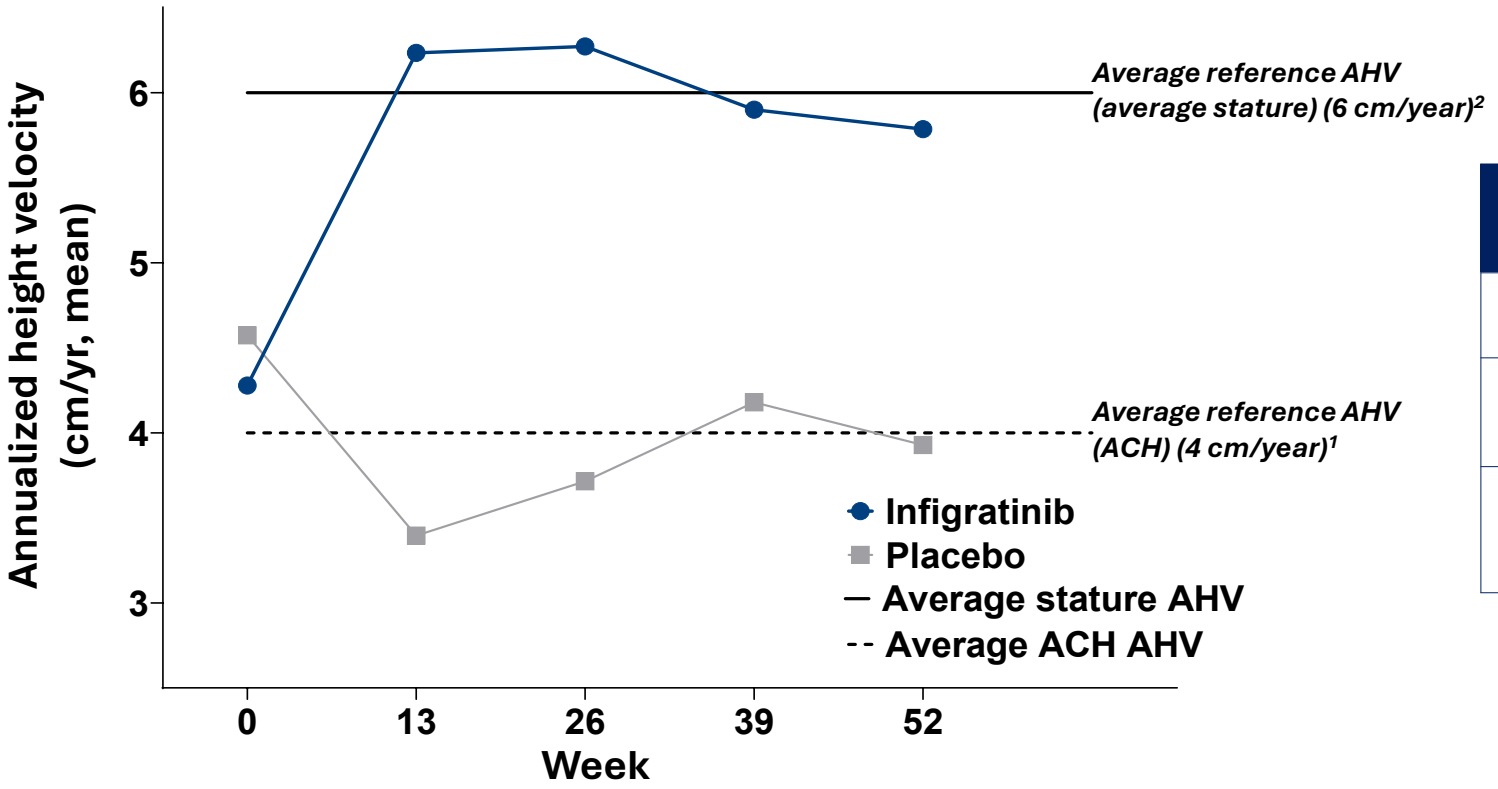
Across all of the above age subgroups, infigratinib demonstrated the largest CFBL in AHV at Week 52 in a randomized trial for ACH

BBIO data on file; AHV : annualized height velocity; CFBL : Change from Baseline

**Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.*

Secondary endpoint: Absolute AHV at Week 52

Absolute Annualized Height Velocity (AHV)



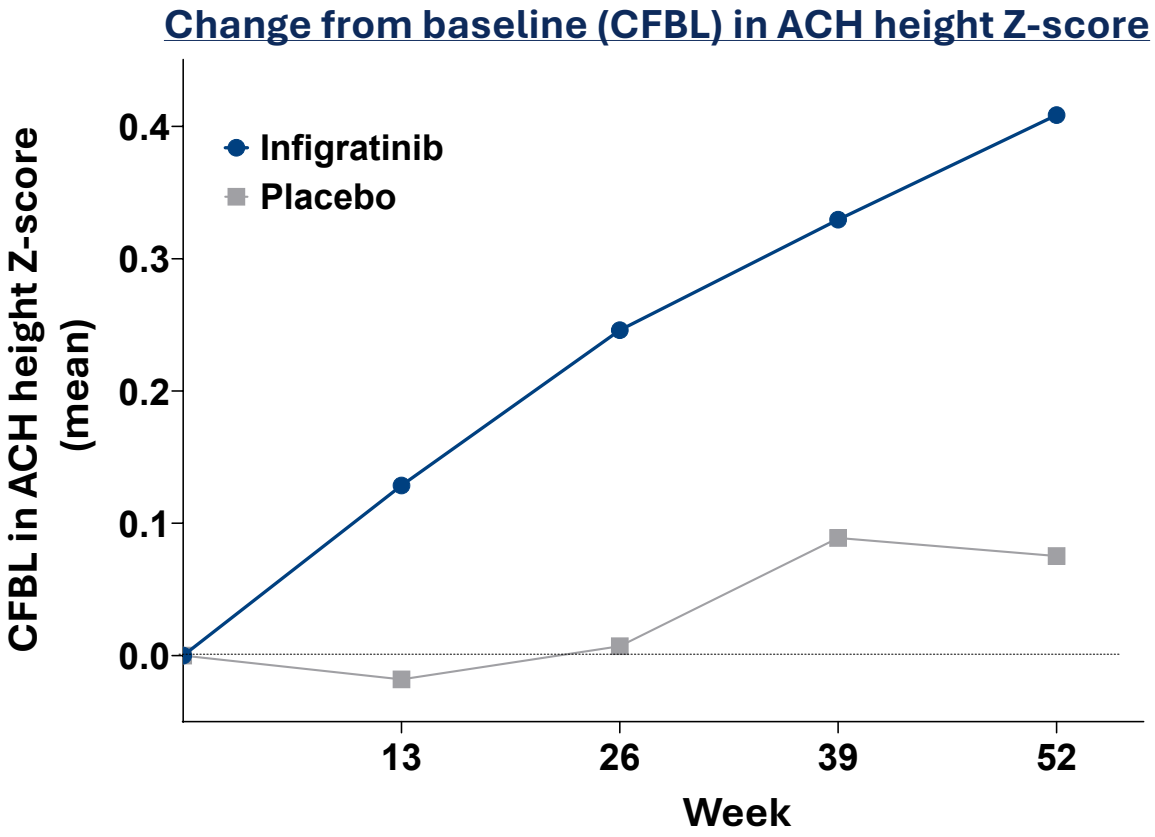
Absolute AHV at Week 52	Infigratinib (n=74)	Placebo (n=39)
LS Mean	**5.96**	**4.22**
Infigratinib vs. placebo difference, LS mean	1.74	
P-value	p<0.0001	

Infigratinib has demonstrated the largest absolute AHV (LS mean) at Week 52 in any randomized trial

BBIO data on file; ACH: achondroplasia; 1-Hoover-Fong et al., 2021; 2-Sperling, 2020

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Key secondary endpoint: Change from baseline in height Z-score in relation to an achondroplasia population over 52 weeks



ACH Height Z-score at Week 52	Infigratinib (n=74)	Placebo (n=39)
LS Mean	0.41	0.09
Infigratinib vs. placebo difference, LS mean	0.32	
P-value	p<0.0001	

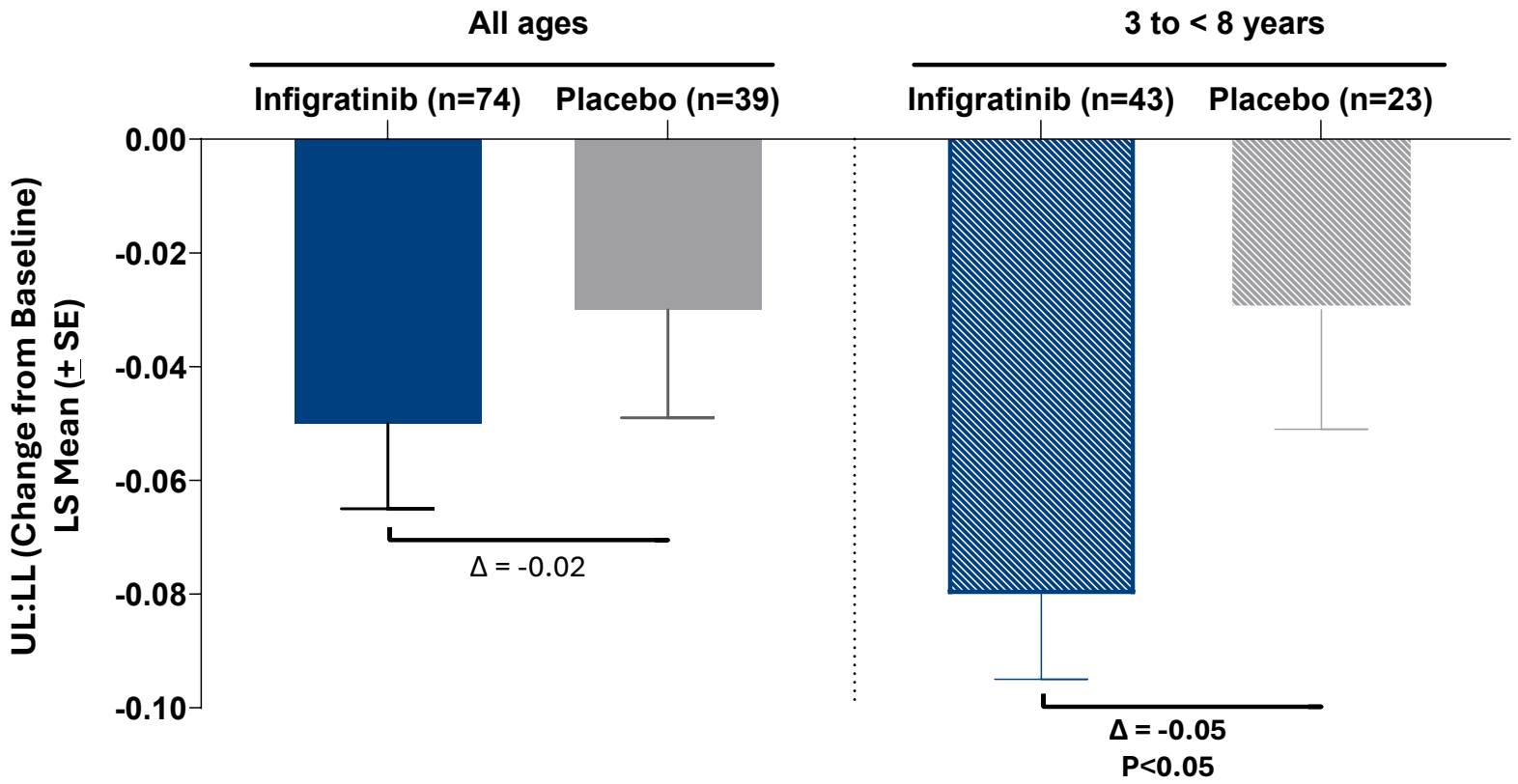
Infigratinib significantly increased the height Z-score in relation to an ACH population over 52 weeks by 0.32SDs (p<0.0001)

BBIO data on file

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Key secondary endpoint: Change from baseline to Week 52 in upper-to-lower body segment ratio

Change from baseline in upper to lower body segment ratio (UL:LL) at Week 52



Infigratinib is the first therapeutic option to demonstrate a statistically significantly result on proportionality against placebo in a RCT for ACH

BBIO data on file; Exploratory analysis pre-specified in the SAP; RCT : randomized controlled trial

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Infigratinib at 0.25 mg/kg was well-tolerated, with no safety signal indicating inhibition of FGFR1 or 2

- The majority of treatment-emergent adverse events (TEAEs) were grade 1 or 2 in severity and typical for children of these ages
- TEAEs were balanced between the treatment and placebo arms
- No serious adverse events related to study drug
- No discontinuations due to AEs related to study drug

Areas of interest

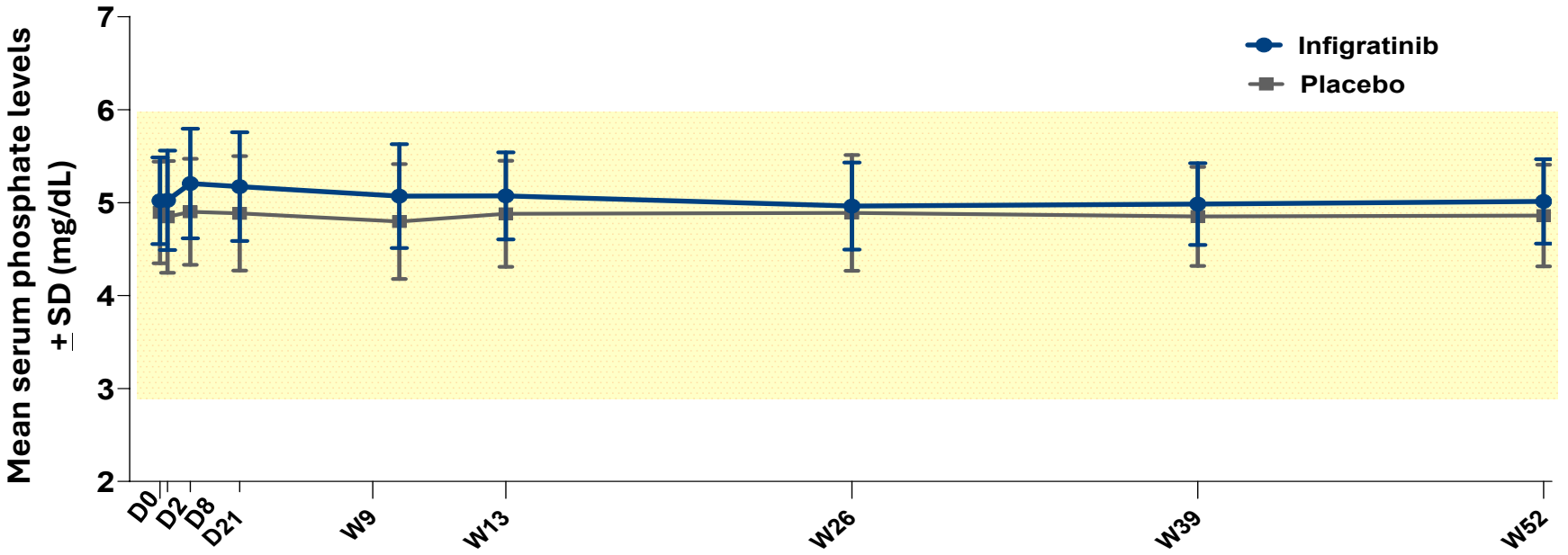
- 3 cases of hyperphosphatemia, all mild, asymptomatic, and transient; no cases required a dose reduction or discontinuation
- No AEs associated with the inhibition of FGFR1 or FGFR2 (e.g. retinal or corneal)

BBIO data on file

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

Mean serum phosphate levels were comparable across infigratinib and placebo at all measured time points

Mean Serum Phosphate Levels



For all participants, the maximal change in phosphate levels was within the typical intra-subject variability in the pediatric population[1]

Mean phosphate levels (mg/dL)	Baseline	Day 2	Day 8	Day 21	Week 9	Week 13	Week 26	Week 39	Week 52
Infigratinib	*5.0*	*5.0*	*5.2*	*5.2*	*5.1*	*5.1*	*5.0*	*5.0*	*5.0*
Placebo	*4.9*	*4.9*	*4.9*	*4.9*	*4.8*	*4.9*	*4.9*	*4.9*	*4.9*

Mean phosphate levels at all measured time points were similar and within normal ranges

Shaded area show reference range of serum phosphorus for study central lab reference ranges for ages 3 to 13 years old;
BBIO data on file; D:day; W : week; 1 – Hanudel (2020)

*Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.

NDA and MAA submissions expected in 2H 2026

Advance infigratinib towards registration in achondroplasia

Submit New Drug Application to FDA
2H 2026

Submit Marketing Authorization Application to EMA
2H 2026

Detailed PROPEL 3 results planned for presentation at scientific and advocacy conferences
2026

**Infigratinib is an investigational agent that is not approved for use in skeletal dysplasia by any regulatory authority.*

PROPEL3

Infigratinib for Achondroplasia

THANK YOU to the study participants, their families, investigators, clinical research staff, advocates and collaborating research partners

"Taken together, these best-in-class results highlight the transformative potential for infigratinib to address aspects of achondroplasia beyond linear height, and with a product administered orally."

Ravi Savarirayan, M.D., Ph.D. of Murdoch Children's Research Institute in Melbourne, Australia

Program Next Steps

Justin To
Chief Executive Officer
Skeletal Dysplasias

We should continue to do more and do better for families and individuals with ACH; Today is just the beginning

PROPEL OLE will follow children on infigratinib (0.25mg/kg) until they reach final or near-final adult height

Our commitment to the community continues

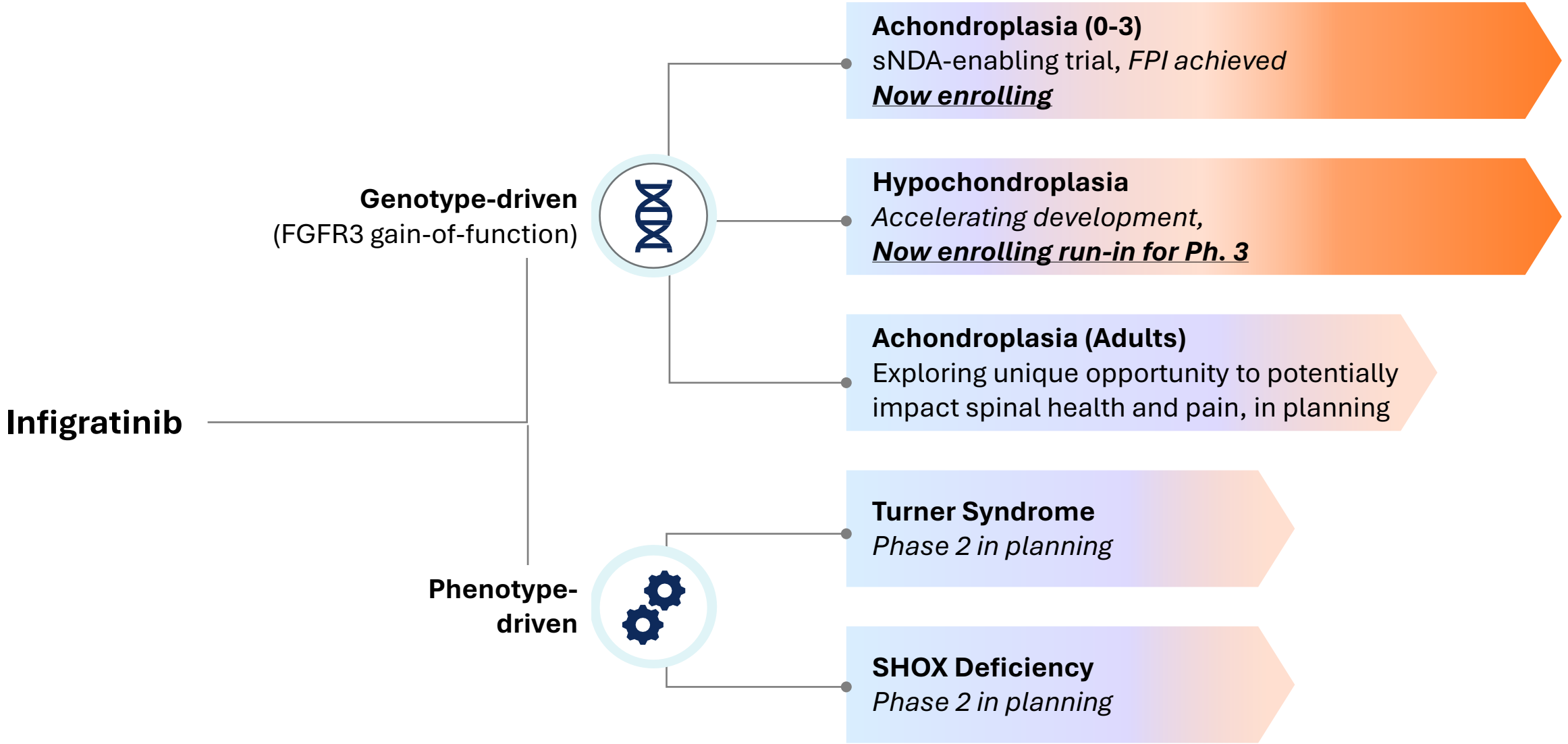
Longer Term Objectives:
Changes over time impacting overall health in ACH on treatment

Examples of endpoints:

- Physical Functioning (energy, daily activities, reach, mobility)
- Aspects of daily living (WeeFIM, PedsQL, QoLISSY)
- Skeletal and spine (bowing, bone angles, vertebrae distances)
- Overall pain (pain-NRS)
- Episodes and severity of otitis media (ear infection)
- Episodes and severity of sleep apnea
- Body proportionality
- ...and more

Source: Clinicaltrials.gov (NCT05145010)

We are on Day 1 of the opportunity for infigratinib



Launch Readiness

Matt Outten, M.B.A.
Chief Commercial Officer
BridgeBio

Leveraging our proven commercial structure to successfully launch infigratinib

Built on the foundation of Attruby's commercial success and tailored to the achondroplasia market opportunity

Established commercial infrastructure and team with track record of multiple blockbuster launches

Proven competitive launch playbook
Enabling faster and more efficient mobilization against multi-billion-dollar incumbents

Ensuring broad access
Leverage best-in-class data to ensure broad access and coverage across all books of business

A world-class commercial team

Matt Outten
Chief Commercial Officer, *BridgeBio*

- Broad commercial leadership expertise with significant success across multiple highly competitive markets
- Led $5B+ portfolio, 12 FDA approvals spanning 6 disease states and 7 indications (IMBRUVICA, Pharmacyclics)
- Commercial lead on $21B pharma M&A deal, securing purchase at $261 per share

Scott Collins
SVP, Market Access

Extensive market access experience with consistent coverage across rare disease and oncology leading large field-based access teams

Hudson Boyer
SVP, Commercial Analytics & Ops

Launches in rare disease, hematology, and immunology; strategy consulting and equity analyst background

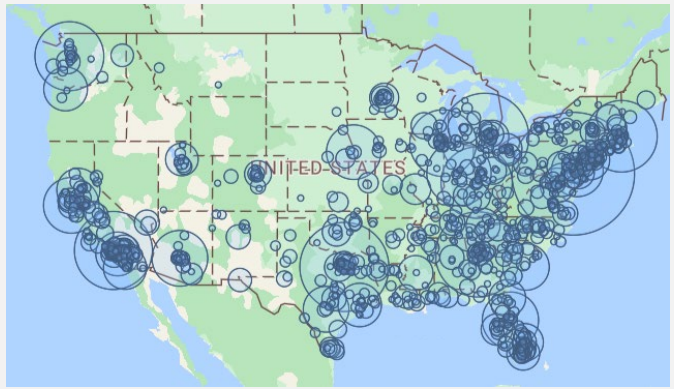
Aaron McIllwain
SVP, Sales and Marketing

Global competitive launch experience with expertise in across rare disease in competitive markets

Large concentrated market with global $5Bn opportunity

State of the market

Addressable market in the U.S.

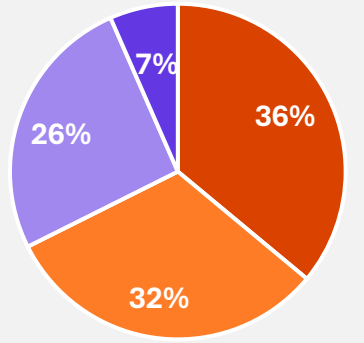


~2.9k

addressable individuals at launch in the U.S.

U.S. Prescriber base

People living with achondroplasia predominantly managed by geneticists & pediatric endos



- Geneticists
- Pediatric Endocrinologist
- Other
- Pediatric Orthopedics

We are preparing for a global launch

Infrastructure and capabilities in place to support a global launch

~55k

addressable individuals globally

Source: BBIO data on file

Combining compelling data with a proven commercial platform

Key highlights

Best-in-class efficacy with largest AHV, height-z score and **only statistically significant improvement in proportionality** to date across any ACH trial

Well-tolerated option, with no SAEs related to study drug or discontinuations due to AEs related to study drug

First- and only oral therapeutic option providing freedom from burden of daily or weekly injections, symptomatic hypotension or ISRs

World-class commercial team with proven launch and rare disease expertise

Strategic platform

Commercial objectives

Strategic area	Objective
Reinforce best in class treatment	Establish **infigratinib** as the **first and only precision oral standard of care**
Anchor FGFR3 as the core driver	FGFR3 inhibition is the validated biological foundation of ACH
Accelerate treatment switching	Rapid conversion from injectables to a more convenient oral alternative
Enable broad and global access	Secure payer support and remove barriers to care globally

Q&A Session