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Infigratinib Clinical Program in Achondroplasia Investor Webinar

Jan 9th, 2026

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Agenda

1	Overview of Achondroplasia		Janet Legare, MD University of Wisconsin School of Medicine and Public Health
2	Infigratinib Clinical Development Program		Daniela Rogoff, MD, PhD Chief Medical Officer Skeletal Dysplasias, BridgeBio
3	The Opportunity for a Daily Oral in ACH		Justin To CEO Skeletal Dysplasias, BridgeBio
4	Q&A		

Overview of Achondroplasia

Janet Legare, MD

Director of Midwest Regional Bone Dysplasia Clinic

Professor of Pediatrics

University of Wisconsin School of Medicine and Public Health

Achondroplasia has health consequences beyond stature

- **Most common form of disproportionate short stature** – more than 90% of disproportionate short stature
- Incidence 1: 25,000-30,000
- **About 300,000 affected persons worldwide; 15,000 in North America**
- **Many health consequences besides living with dwarfism**
- Autosomal dominant inheritance – 50% risk in every pregnancy if parent is affected
- Majority are new mutations with a 80% *de novo* mutation rate – means **80% of patients with achondroplasia have average stature parents**

Achondroplasia presents in all ethnicities equally

[1] Pauli et al., 1995; [2]Pauli 2019

When does diagnosis usually occur?

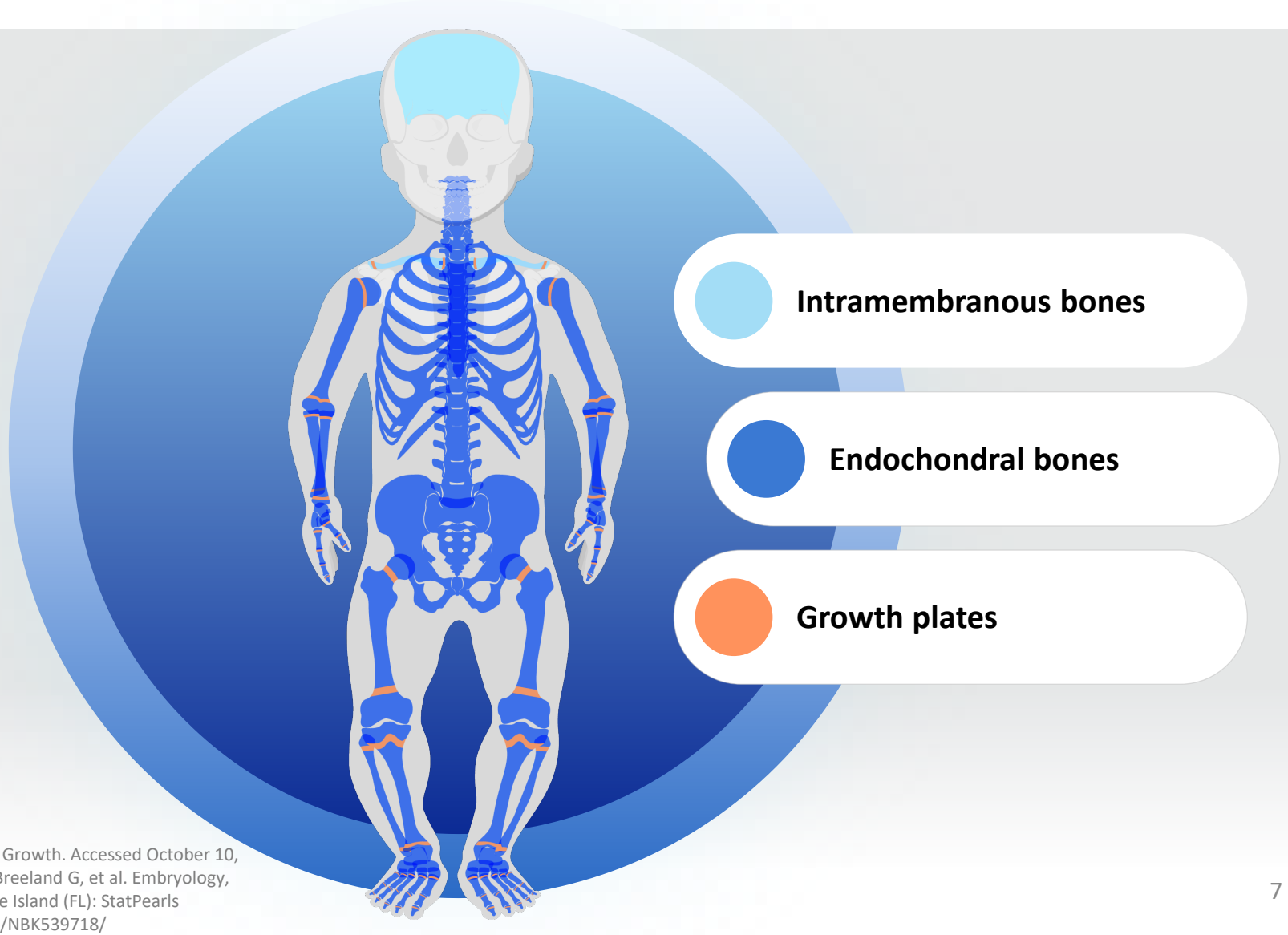
- Detectable during the third trimester – usually after 26 weeks – only diagnosed prenatally if mother is having growth ultrasounds during this time
  - Most cases are diagnosed by the 1st month of life
- About 33% diagnosed prenatally, 33-40% in first 48 hours, another 15% by the first month of life. About 10% still diagnosed after 4 months of age
- May be diagnosed earlier if a parent has achondroplasia and looking for it– only 20% of cases are inherited

FGFR3 variant can be confirmed in virtually all cases of clinically diagnosed achondroplasia

[1]ISUOG. Achondroplasia – Patient Information. [2]Pauli RM. Achondroplasia: A Comprehensive Clinical Review. Orphanet J Rare Dis, 2019. [3]Stoll C et al. Prenatal Diagnosis of Achondroplasia. Bone Abstracts, 2018. [4]Horton WA et al. Achondroplasia. Lancet, 2007. [5]Data from CLARITY database for 2010 decade

There are two types of bone growth or ossification: endochondral- and intramembranous- ossification

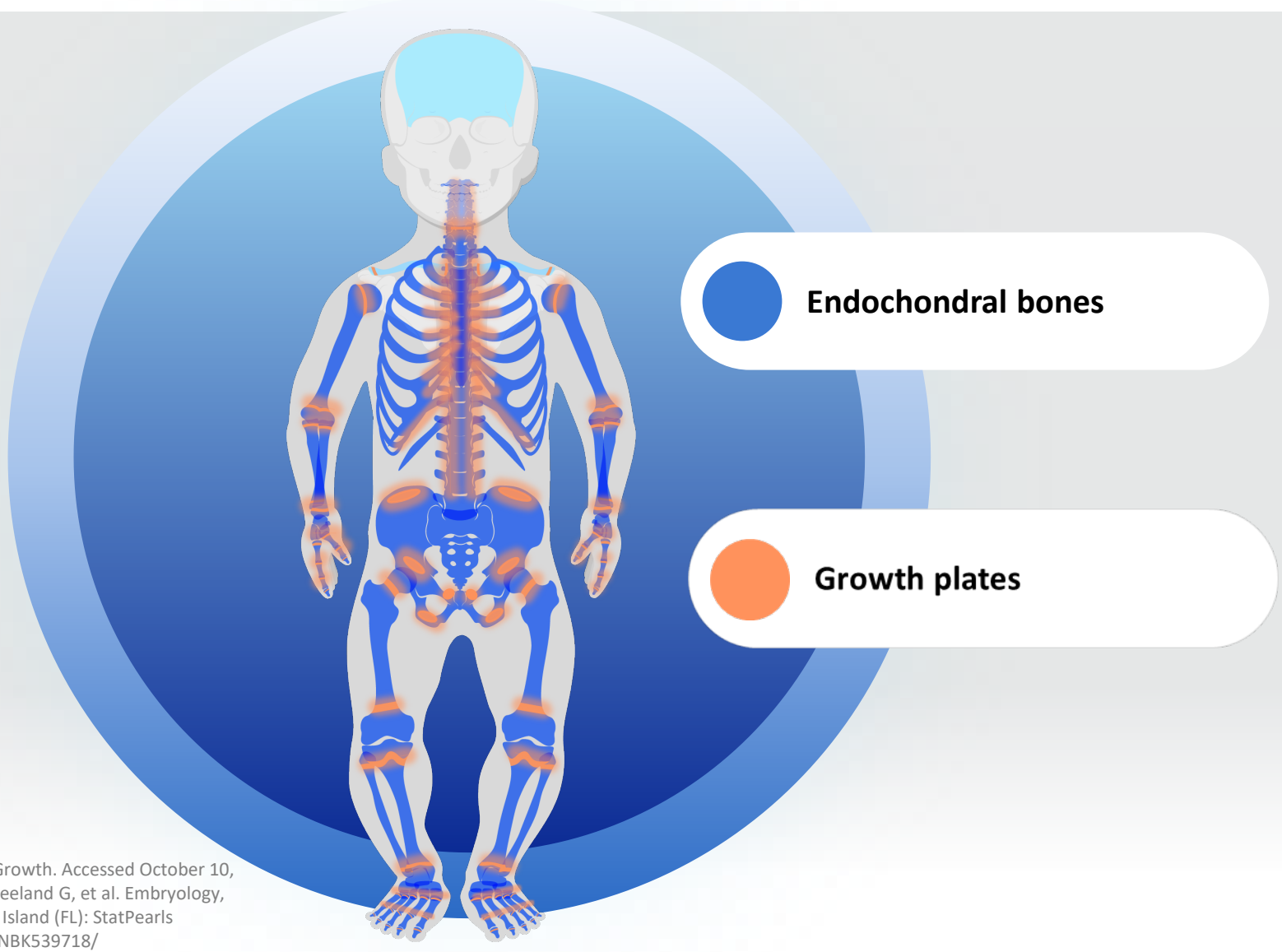
- **There are two types of ossification:** endochondral and intramembranous[1,2]



1. National Cancer Institute. SEER Training Modules. Bone Development & Growth. Accessed October 10, 2025. https://training.seer.cancer.gov/anatomy/skeletal/growth.html; 2. Breeland G, et al. Embryology, Bone Ossification. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539718/

Achondroplasia affects bones that grow through cartilage growth plates, specifically endochondral bone

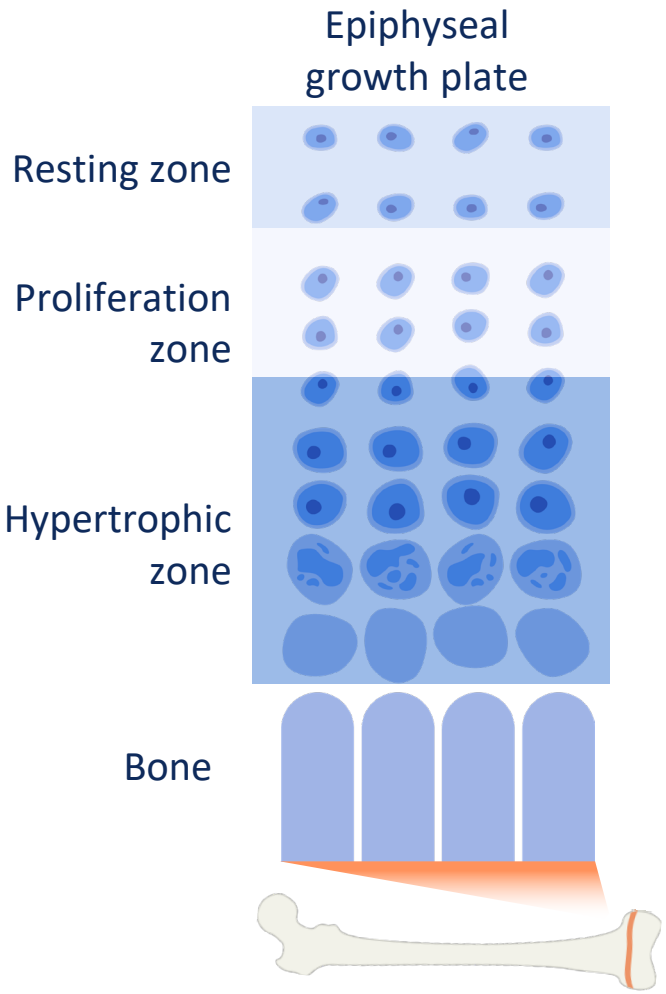
- **Endochondral ossification** occurs in all bones except for top of skull, lower jaw, and collar bones, **and contributes to length of bones**[1,2]



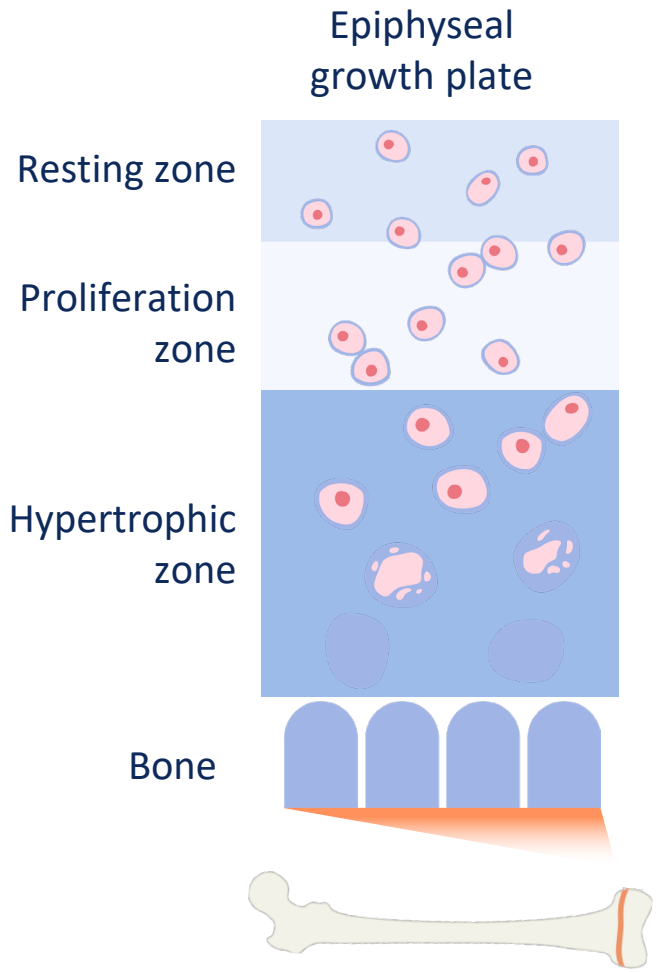
1. National Cancer Institute. SEER Training Modules. Bone Development & Growth. Accessed October 10, 2025. https://training.seer.cancer.gov/anatomy/skeletal/growth.html; 2. Breeland G, et al. Embryology, Bone Ossification. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK539718/

Typical vs. Irregular Growth at Growth Plate

Typical Growth Plate

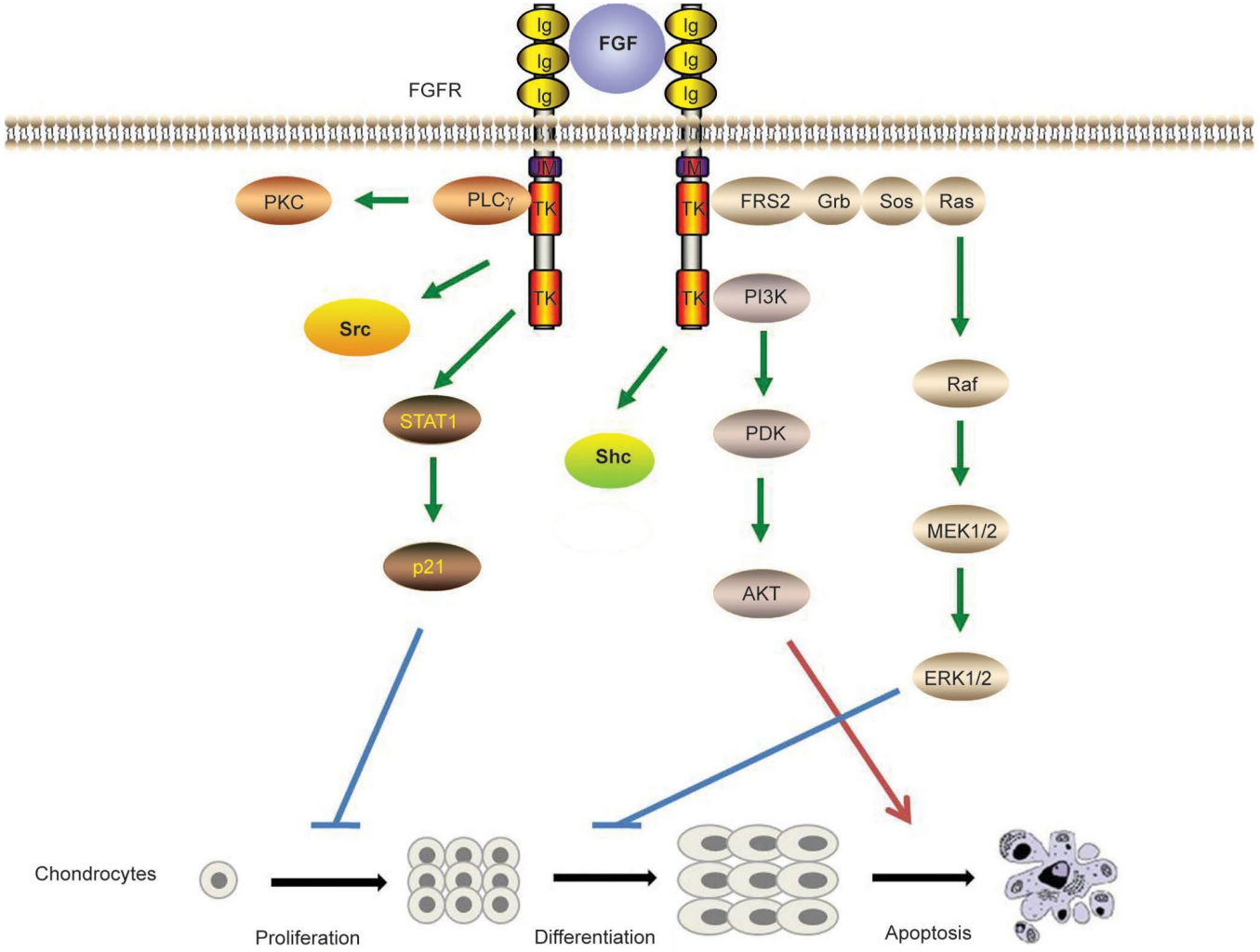


Achondroplasia Growth Plate



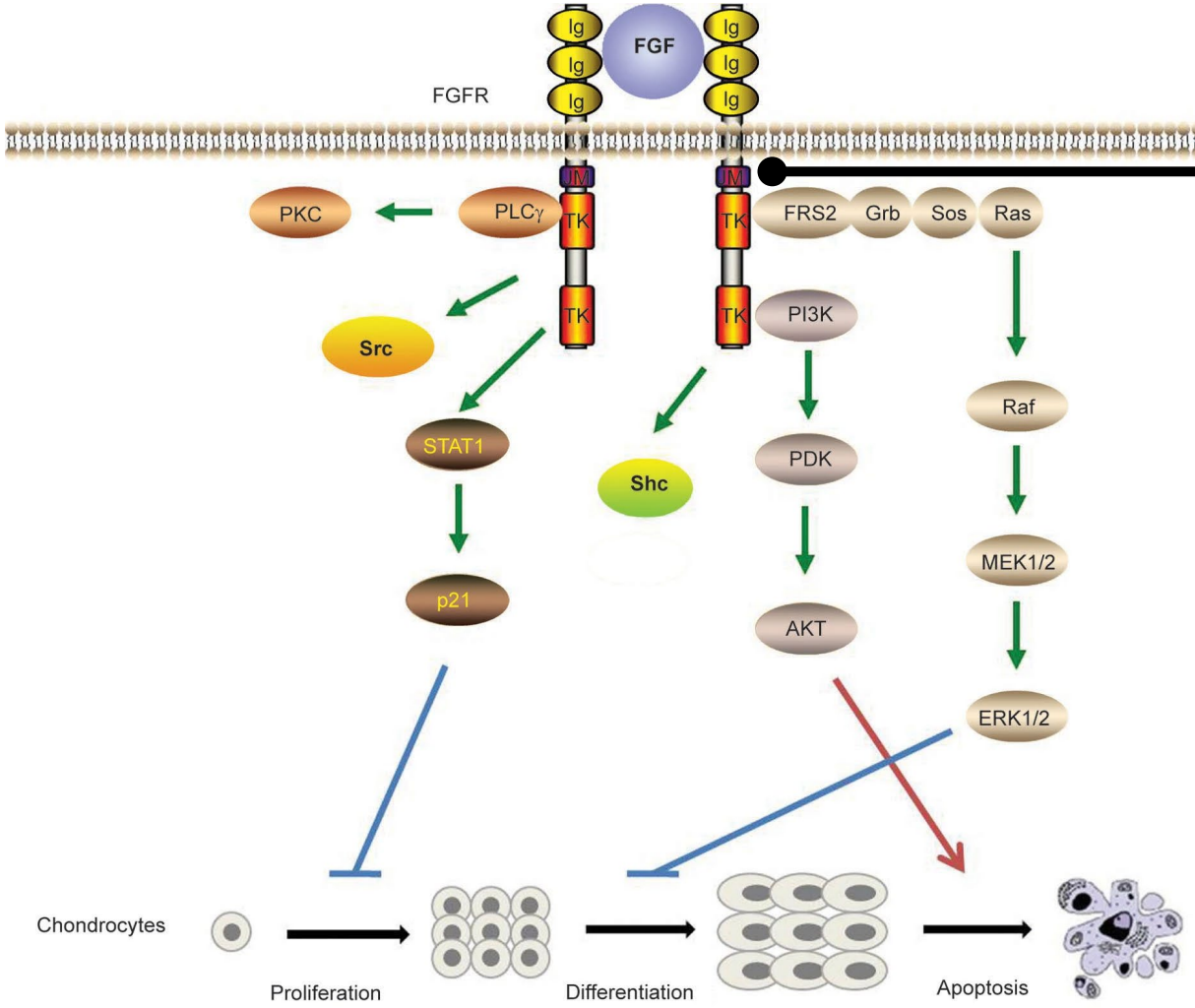
- Chondrocyte hypertrophy accounts for ~60% of longitudinal bone growth

In achondroplasia, FGFR3 gain-of-function variants disrupt endochondral ossification and limit bone growth



[1] Figure from Su, N., Jin, M. & Chen, L. Role of FGF/FGFR signaling in skeletal development and homeostasis: learning from mouse models. *Bone Res* **2**, 14003 (2014). https://doi.org/10.1038/boneres.2014.3

In achondroplasia, FGFR3 gain-of-function variants disrupt endochondral ossification and limit bone growth

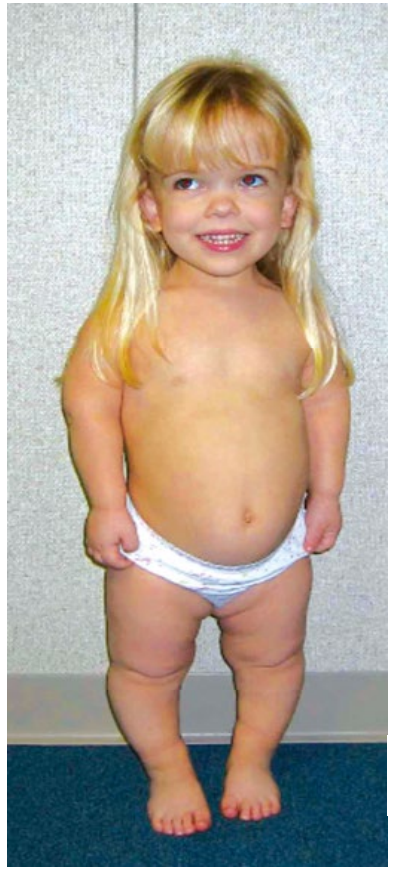


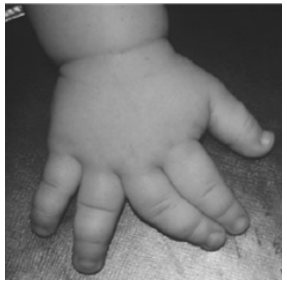
FGFR3 tyrosine kinase inhibitors **directly target** overactive FGFR3 signaling, potentially addressing the mechanistic driver of skeletal dysplasia[2-4]

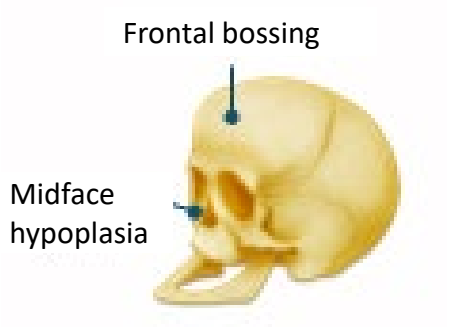
[1]Figure from Su, N., Jin, M. & Chen, L. Role of FGF/FGFR signaling in skeletal development and homeostasis: learning from mouse models. *Bone Res* 2, 14003 (2014). https://doi.org/10.1038/boneres.2014.3; 2. Savarirayan R, et al. N Engl J Med. 2025;392(9):865–874. 3. Komla-Ebri D, et al. J Clin Invest. 2016;126(5):1871–1884. 4. Demuynck B, et al. J Bone Miner Res. 2024;39(6):765–774.

Clinical Picture

Examples of Features





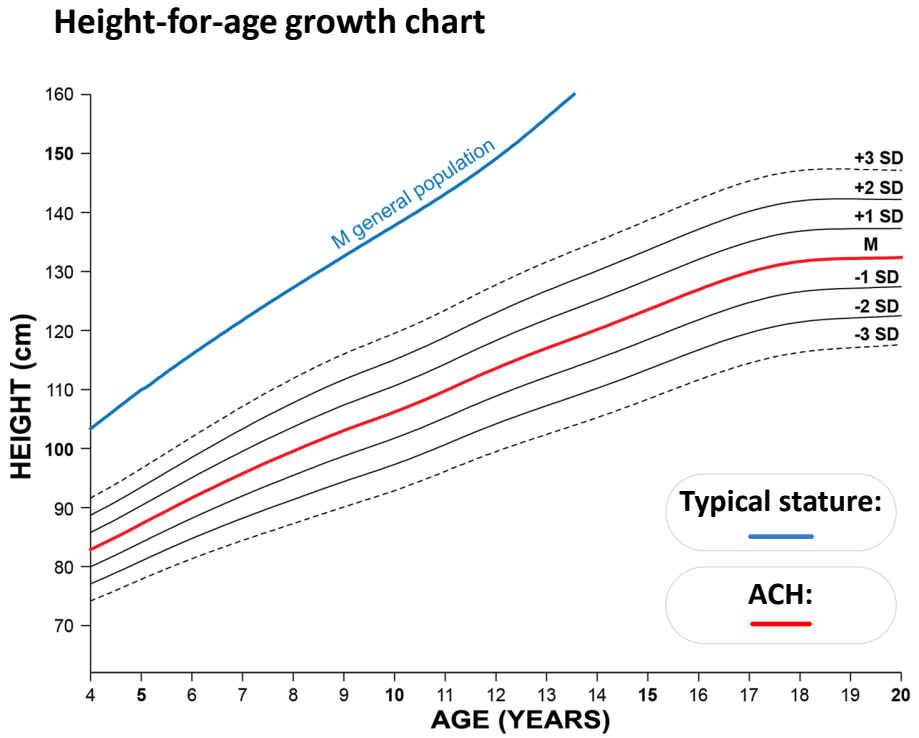


Clinical Presentation

- Short stature: average height of 4’4” in males and 4’1” in females
- Rhizomesomelia
- Short fingers/trident configuration
- Macrocephaly with frontal and biparietal bossing
- Midface hypoplasia
- Small chest
- Tibial varus
- Clinical presentation and medical sequalae occur because of which bones are affected
- Achondroplasia affects bone growth, not the growth of the underlying structures

Osteochondrodysplasias. OBGYNKey. https://obgynkey.com/osteochondrodysplasias/

Children living with achondroplasia have a very different growth trajectory from those of the general population



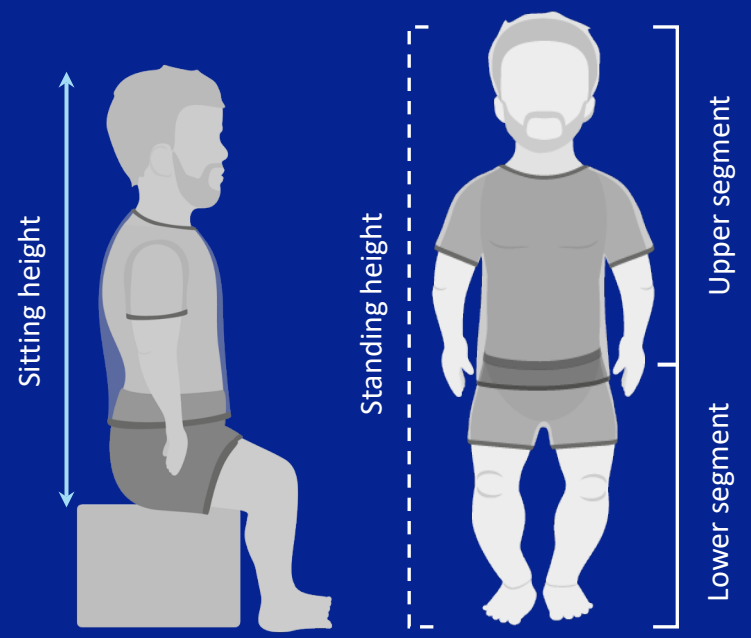
- Height z-scores (or height SDS) is the difference of an individual's height from the average height expected for their age and sex, expressed in standard deviations
- For children living with achondroplasia (ACH), the height z-score must be calculated against ACH-specific growth charts
- Height z-score adjusts for age and sex

An improvement in ACH-specific z-score shows growth beyond what's typical for ACH

Growth charts adapted from Neumeyer et al., Am J Med Genet A 2021

Proportionality links changes in linear growth to potential benefits beyond height

Upper to Lower (U:L) body segment ratio is a measurable marker of disproportion in ACH[2]



$$\text{U:L ratio} = \frac{\text{Sitting height*}}{\text{Standing height - Sitting height}}$$

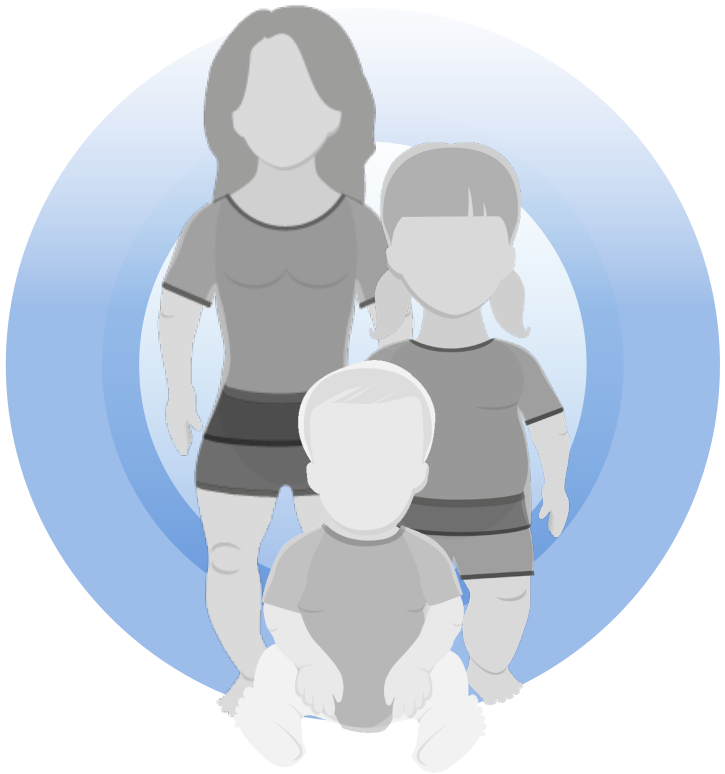
- U:L body segment ratio never reaches 1 in achondroplasia because trunk growth continues while lower extremity growth is limited[1]
- Disproportion persists across childhood compared with peers of typical stature[1]
- Disproportionality is associated with delayed gross motor milestones, balance and challenges in daily functioning e.g., driving, independent ambulation[3]

Proportionality is an important endpoint in achondroplasia

*Crown-rump length is used instead of sitting height in infants.

[1]Hoover-Fong JE, et al. *Am J Clin Nutri.* 2008;88(2):364–371. [2]Hoover-Fong JE, et al. *Adv Ther*. 2025;42(3):1291–1311., [3]Ireland PJ, Johnson S, Donaghey S, et al. Developmental milestones in infants and young Australasian children with achondroplasia. J Dev Behav Pediatr 2010; 31: 41-7.

Achondroplasia is associated with multisystem medical challenges across the lifespan



Neurologic[1–3]

- Foramen magnum stenosis (FMS)
- Hydrocephalus

Respiratory and Sleep[1–3]

- Obstructive sleep apnea (OSA)
- Central sleep apnea (CSA)

Gastrointestinal/Feeding[1,3]

- Feeding and swallowing difficulties

Metabolic/Weight[1]

- Increased risk of obesity

Musculoskeletal/Orthopedic[1–5]

- Hypotonia
- Ligament laxity
- Thoracolumbar kyphosis (TLK)
- Lumbar lordosis
- Lumbar spinal stenosis (LSS)
- Genu varum
- Joint contractures (elbows, hips)

Craniofacial (ENT/Dental)[1–6]

- Chronic or recurrent otitis media or otitis media with effusion
- Dental crowding, malocclusion, open bite

AAP published new Clinical Guidelines in 2020 (Hoover-Fong et al., 2020)

ENT, ear, nose, and throat.

[1]Pauli RM. *Orphanet J Rare Dis*. 2019;14(1):1. [2]Savarirayan R, et al. *Nat Rev Endocrinol*. 2022;18(3):173–189. [3]Sforza E, et al. *Genes (Basel)*. 2023;14(1):199. [4]Ireland PJ, et al. *Appl Clin Genet*. 2014;7:117–125. [5]Hoover-Fong JE, et al. *Pediatrics*. 2020;145(6):e20201010. [6]Wright M, et al. *Arch Dis Child*. 2012;97(2):129–134.

Goals of Therapeutic Intervention

- Improve health
- Decrease medical sequelae
- FDA requires a measurement – height/annual growth velocity
- Emerging data have associated increased height with greater mobility, function, and QoL[1,2]

Height is the metric, but improved health is the goal

QoL, quality of life.

[1]Backeljauw P, et al. Growth Horm IGF Res. 2021;57–58:101392. [2]Lorne H, et al. Eur J Med Genet. 2020;63(4):103816.

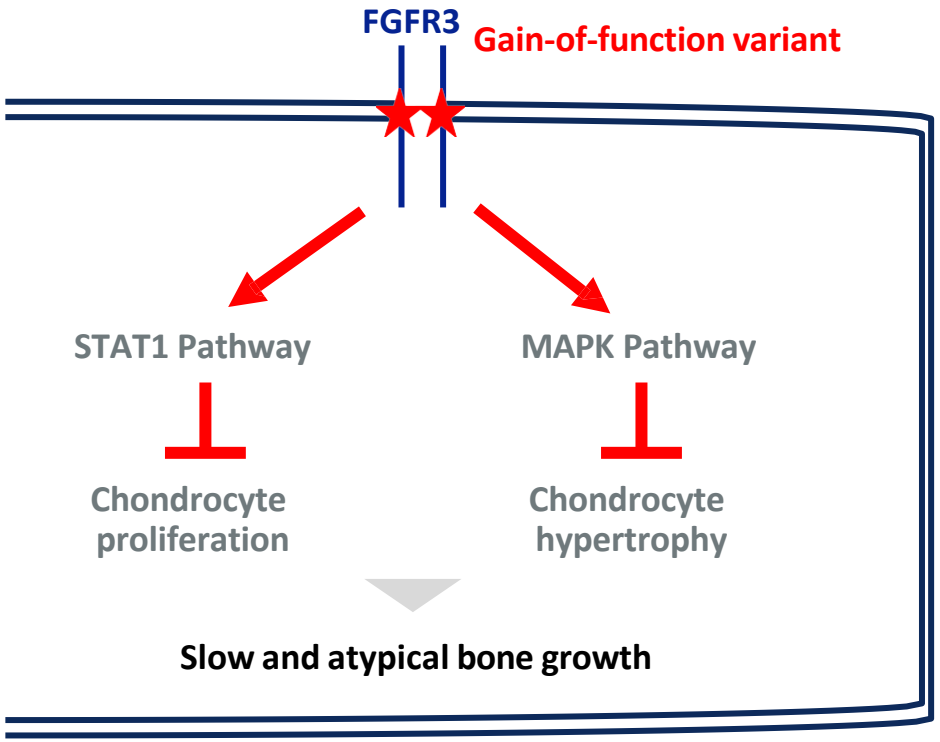
Infigratinib Clinical Development Program

Daniela Rogoff, MD, PhD

Chief Medical Officer,
Skeletal Dysplasias

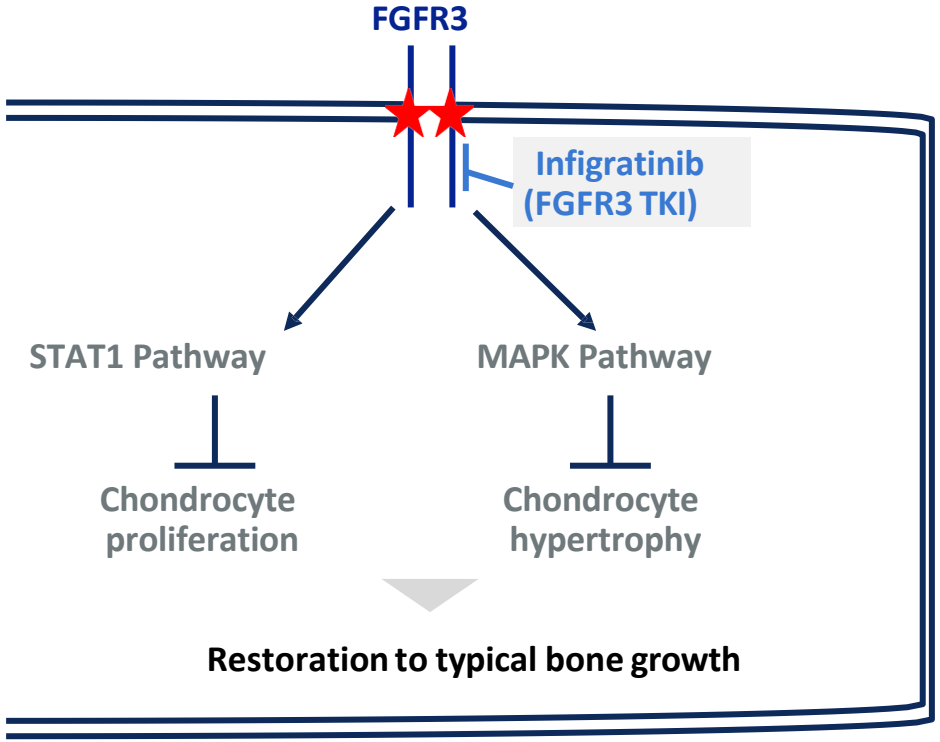
Infigratinib is an oral, potentially first-in-class FGFR1-3 inhibitor in development as a therapeutic option for achondroplasia

Mechanism of the condition



FGFR3 acts as a "molecular brake" on chondrocyte proliferation and hypertrophy; **in ACH, this brake is stuck** due to gain-of-function mutations resulting in the receptor being always "on" and consequently, the **bone growth significantly slowed down**

Infigratinib Mechanism of Action

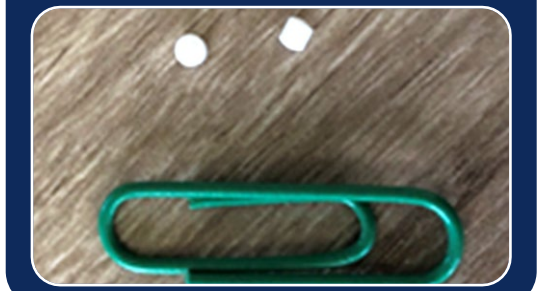


Infigratinib "releases" the brake, potentially resuming normal chondrocyte function, allowing for **restoration of bone growth**

Infigratinib sprinkle capsules are being developed for oral administration[1,2]

Capsules
(17 mm long*)

Granules
(2 mm long)



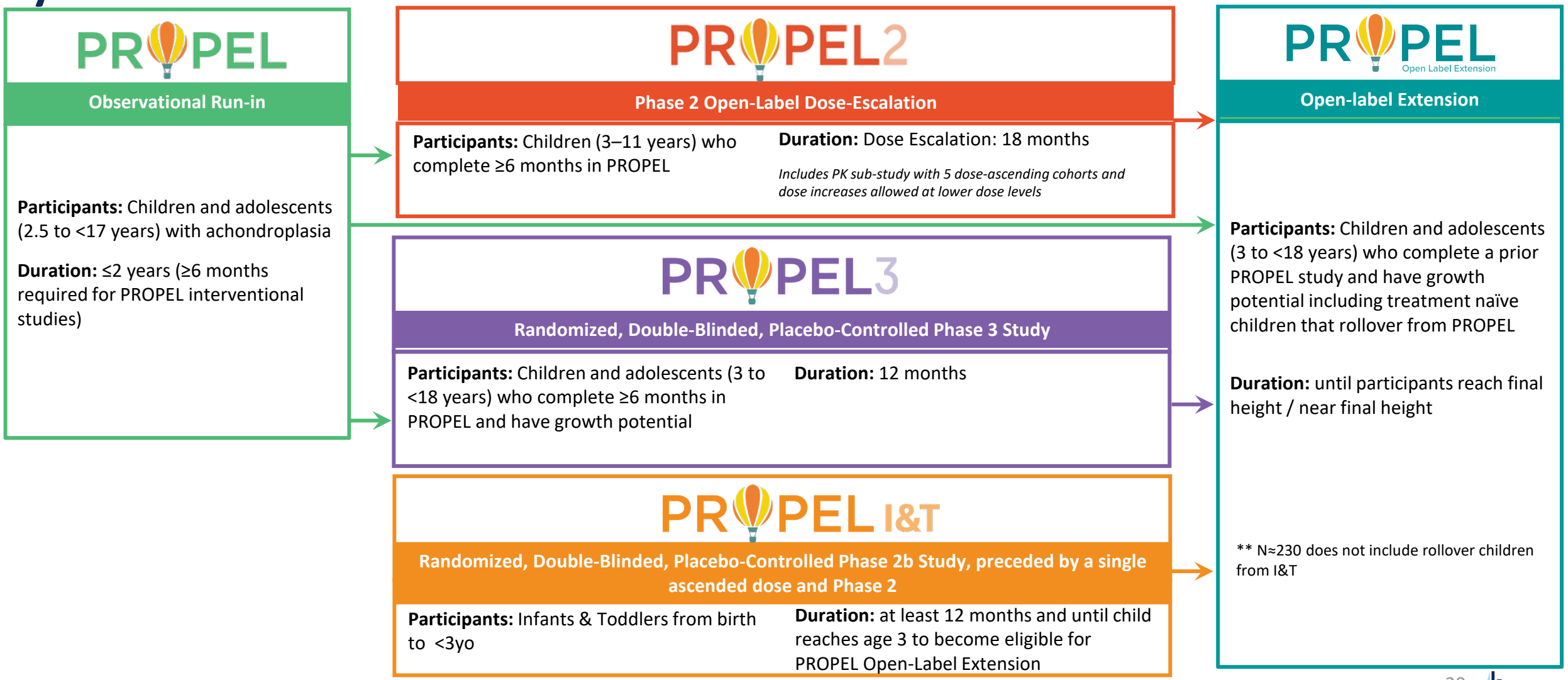
- Infigratinib is being studied in children over 3 years of age with achondroplasia (0.25 kg/mg/day) as a sprinkle capsule
- Capsules can be swallowed whole or content (granules) sprinkled on soft food
- The dosage strength of each capsule depends on how many granules are inside
- Each child's dose is based on their weight

*Size 2 capsules are shown in photo.

[1]Savarirayan R, et al. *N Engl J Med*. 2024;392(9):865–874. [2]BridgeBio data on file.

Infigratinib is an investigational agent that is not approved for use in the treatment of achondroplasia by any regulatory authority

The infigratinib clinical program for achondroplasia encompasses 5 clinical studies, evaluating safety and efficacy in ~300 children ages 0 to <18 years*



ACH, achondroplasia; AHV, annual height velocity; CFB, change from baseline; HRQoL, health-related quality of life; PK, pharmacokinetics; QoL, quality of life; TEAE, treatment-emergent adverse event
*with growth potential; **For these children, infigratinib given until final or near-final height reached. - PROPEL: NCT04035811; PROPEL2: NCT04265651; PROPEL3: NCT06164951; PROPEL Open-label Extension: NCT05145010

PROPEL 2: Infigratinib was well-tolerated, with no safety signals identified

In Cohort 5 (highest dose escalation level of 0.25 mg/kg/day):

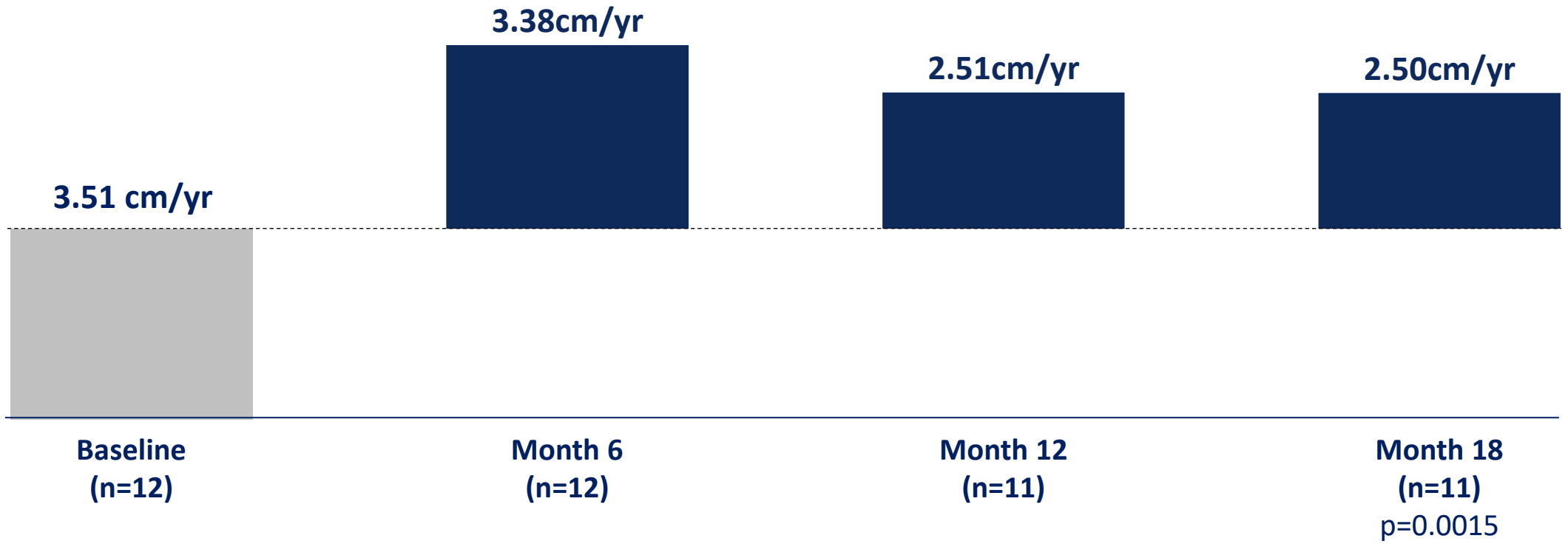
- No serious adverse events (SAEs)
- No adverse events (AEs) that required treatment discontinuation
- Most treatment-emergent adverse events (TEAEs) were grade 1 in severity and **none of the TEAEs** were assessed as related to study drug

- **0 subjects with grade 3 TEAEs**
- **0 retinal adverse events**
- **0 hyperphosphatemia events**
- No accelerated progression of bone age

BridgeBio data on file

PROPEL 2 – Cohort 5: Change from baseline in AHV over time demonstrated durability of treatment effect

Mean change from baseline in annualized height velocity (AHV)

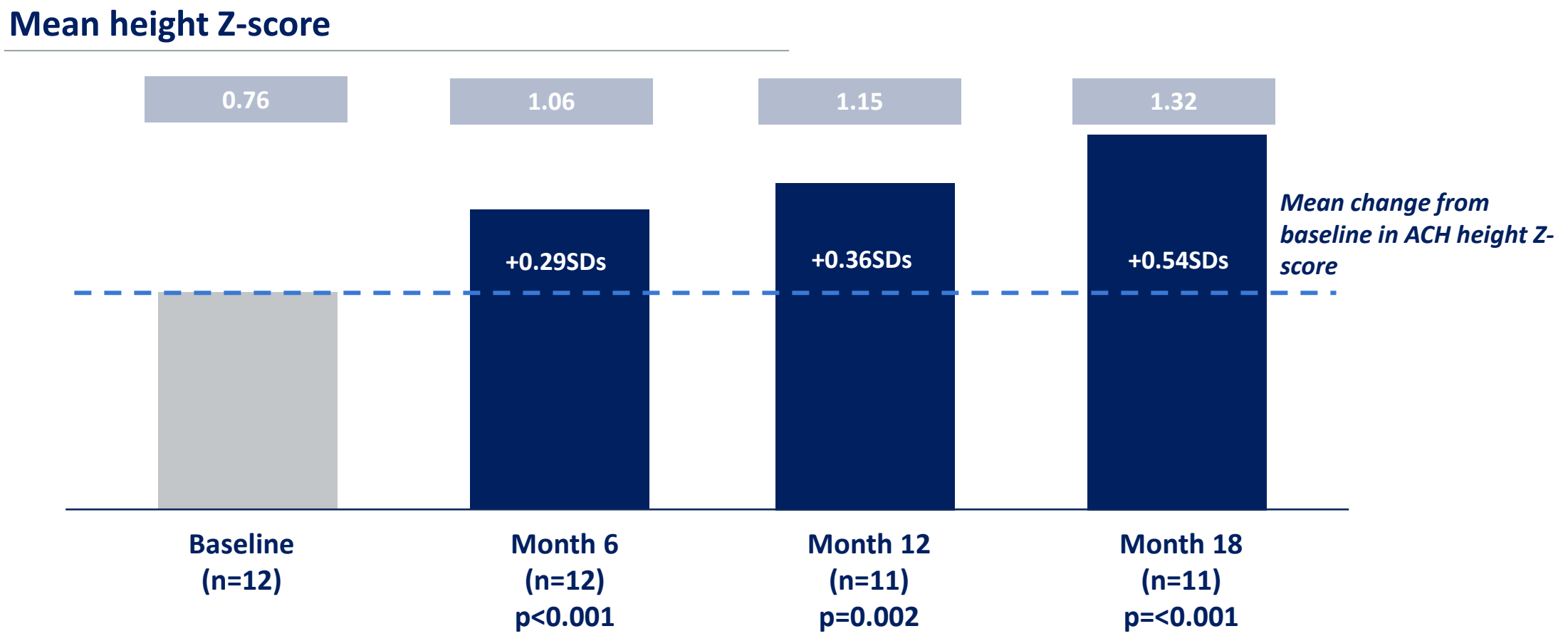


At each timepoint, change from baseline AHV is higher than that reported by any other treatment option

BridgeBio data on file. NOTE: 1 child dropped out before month 12 assessments as study site closed due to PI departure. This child was a responder at month 6.

Infigratinib is an investigational agent that is not approved for use in the treatment of achondroplasia by any regulatory authority

PROPEL 2 – Cohort 5: Cumulative increase in AHV corresponds to an increase in the height z score referenced to ACH-specific growth charts



BridgeBio data on file. NOTE: 1 child dropped out before month 12 assessments as study site closed due to PI departure. This child was a responder at month 6.

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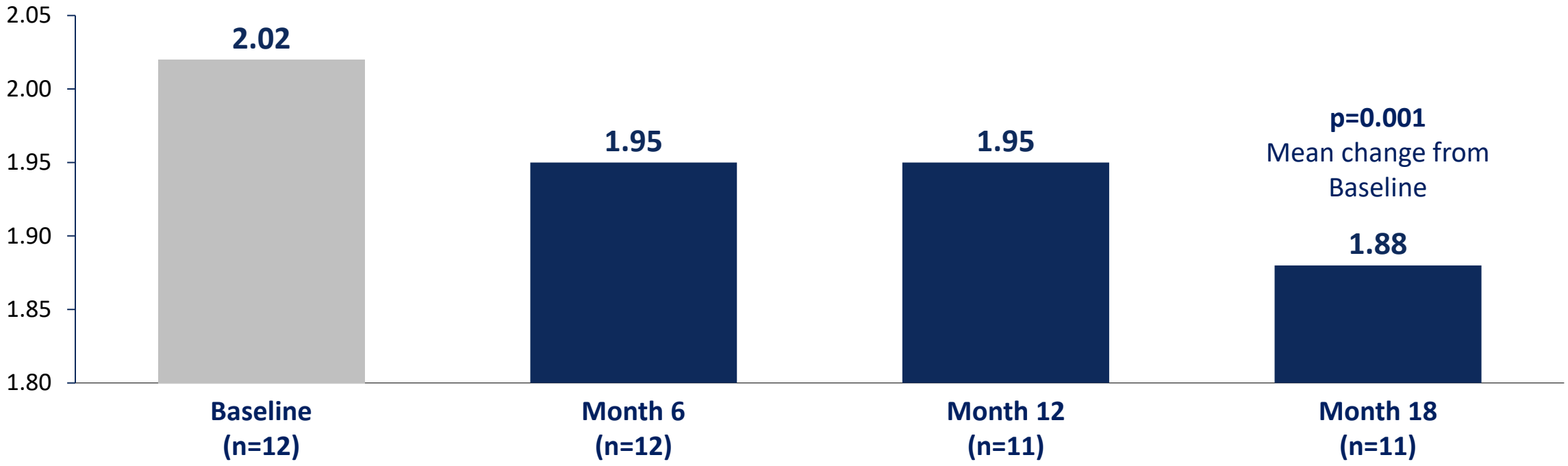
PROPEL 2 – Cohort 5: Infigratinib showed a persistent decrease in upper/lower body segment ratio

Upper to lower body segment ratio

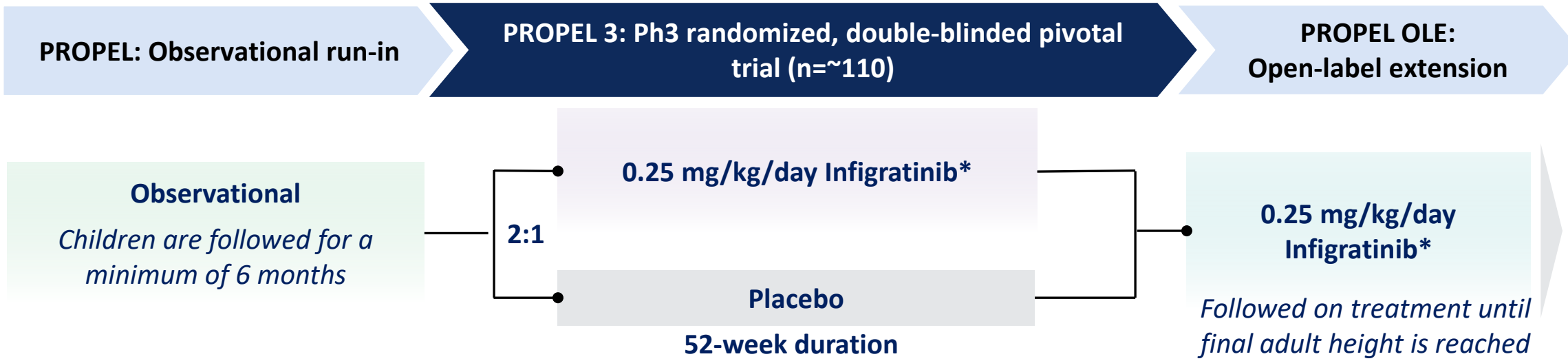


Infigratinib showed statistically significant improvement in upper to lower body segment ratio at 18 months, demonstrating potential for a meaningful effect on body proportionality and functionality if maintained

BridgeBio data on file. NOTE: 1 child dropped out before month 12 assessments as study site closed due to PI departure. This child was a responder at month 6.

Infigratinib is an investigational agent that is not approved for use in the treatment of achondroplasia by any regulatory authority

PROPEL 3 study design and key endpoints[1]



Key inclusion criteria

- Children **3 – <18 years old** with open growth plates

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
- Change from baseline in upper body : lower body segment ratio

Other secondary endpoints:

- Safety, change in physical functioning PedsQL, participant and caregiver evaluation of treatment benefit (qualitative interview)

[1]Clinicaltrials.gov ID: NCT06164951

Infigratinib is an investigational agent that is not approved for use in the treatment of achondroplasia by any regulatory authority

The Opportunity for a Daily Oral in Achondroplasia

Justin To

CEO, Skeletal Dysplasias

Infigratinib: Defining characteristics of a potentially best-in-class program in the ACH landscape

Designed to target achondroplasia at its genetic source: FGFR3 overactivation
Addresses not just overactivation of the MAPK pathway (chondrocyte hypertrophy), but also STAT1 (chondrocyte proliferation) and all other downstream pathways

Achieved profound efficacy in animal models, beyond just long bone growth
In mouse models of achondroplasia, treatment with infigratinib showed an increase in proximal and distal long bone length (femur +21%, humerus +12%, tibia +33%, ulna 22%, and radius +24%) and foramen magnum area (+17%)[5]

Demonstrated the largest degree of efficacy (across multiple dimensions[1]) across any clinical trial for ACH[2]

1. Mean change from baseline in AHV: +2.51 cm/yr at M12
2. Mean absolute AHV: >6 cm/yr at M12
3. Mean change in height Z-score compared to ACH growth charts: +0.36 SD at M12
4. Mean improvement in upper-to-lower body segment ratio (proportionality): Decrease of 0.12 at M18 (P=0.001)

Received the only Breakthrough Designation from the FDA for ACH
Met the regulatory requirement of showing preliminary evidence of substantial improvement over SoC

Designed to be taken as a daily oral, avoiding side effects associated with CNPs and repeated injections
Avoids symptomatic hypotension[1], injection site reactions[1], and the psychosocial burden of receiving/administering repeated injections[3,4]

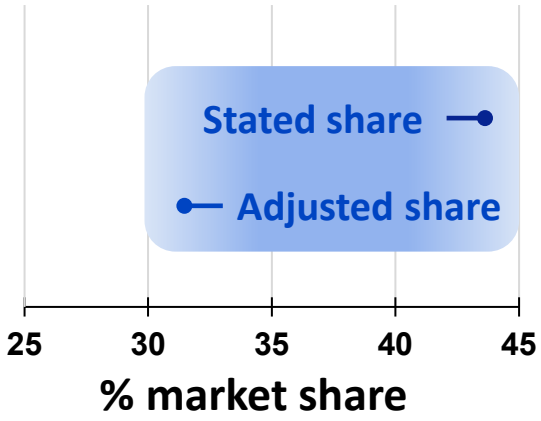
*[1]Savarirayan et al, NEJM, 2024; [2]For monotherapy trials in ACH; [3]Antal et al, Journal of Pediatric Psychology, 2011; [4]Jacobse et al, European Journal of Pediatrics, 2019; [5]Komla-Ebri et al, J Clin Invest. 2016.

BBIO has developed a validated evidence-based perspective to forecasting market share performance

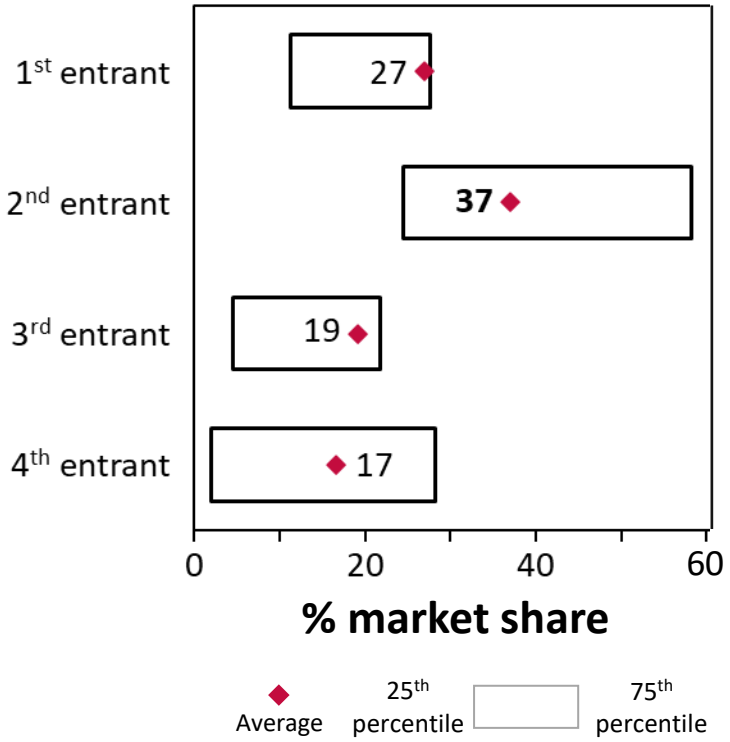
Comprehensive market surveys indicated 30-40%+ Attruby share

Attruby future competitive market share

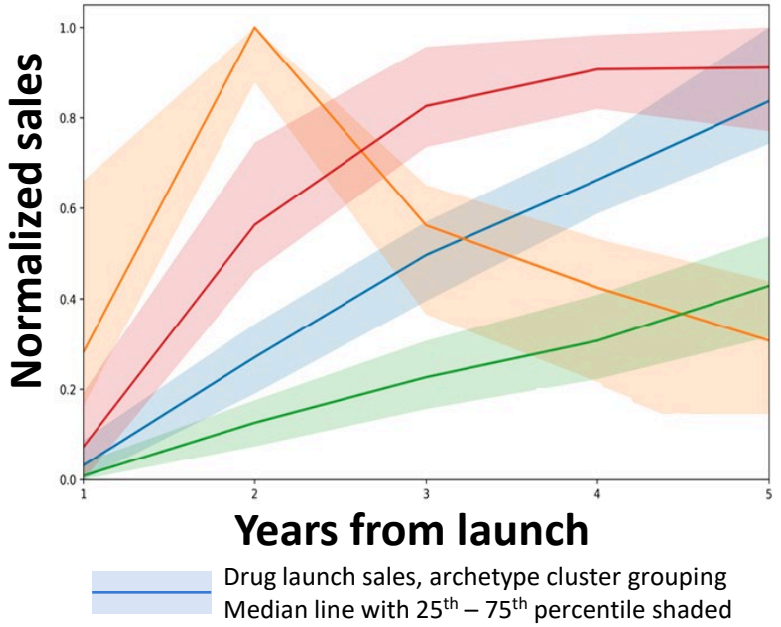
ATTR-CM experienced prescribers
N = 200



Analog research shows 2nd entrants achieve average ~37% share



ML-based empirical analysis enables high fidelity drug launch modeling



*We have analyzed **>900 drug launches** and built a **proprietary algorithm** capable of categorizing launch profiles into **archetypes** and **predicting future revenue***

Note: Stated share adjusted for historical performance of demand study market research on new product launches.
Source: BridgeBio market research HCP surveys (n = 184 & n=200), Evaluate Pharma, PhAST Rx, Advisory board, IQVIA LAAD data set

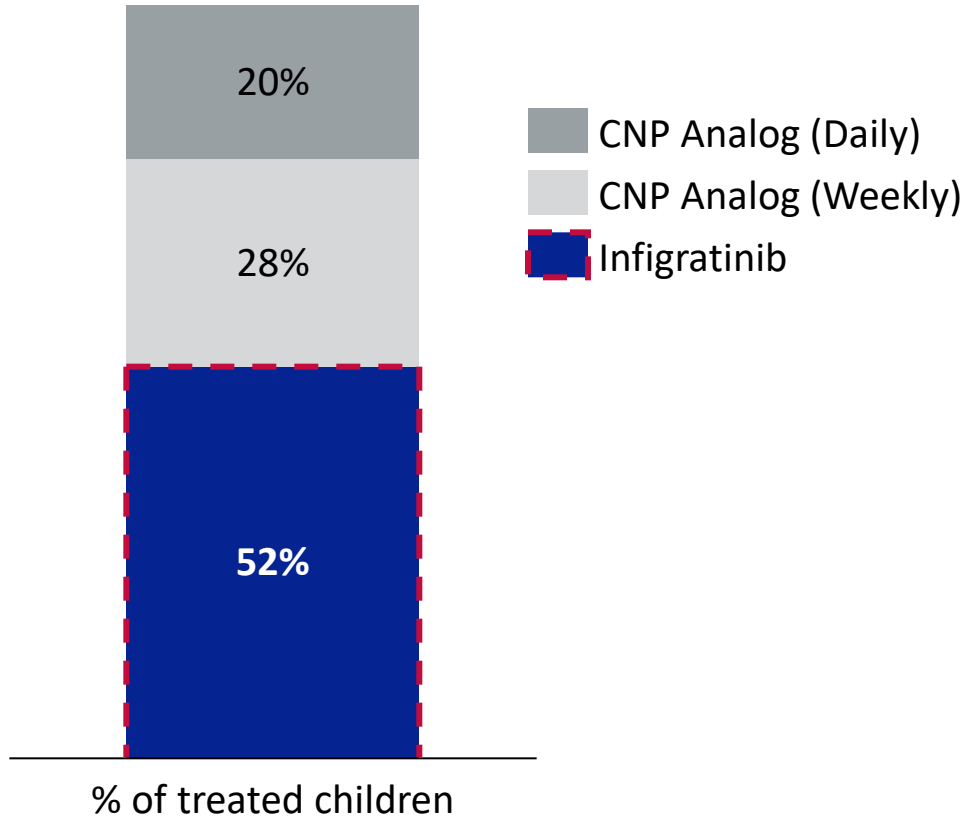
Using these techniques, our market research indicates that infigratinib could capture >50% of treated market share, primarily driven by the favorable oral administration and MOA

Attribute	TPP for testing market share
Indication	• Children **(3 – 18 years)** with achondroplasia and open epiphyses
MOA	• **Selective FGFR1-3 tyrosine kinase inhibitor**
Dosing and Administration	• **Once daily capsules** (containing minitablets swallowed whole/chewed/sprinkled on soft foods)
Primary Endpoint	• Statistically significant improvement in change from baseline in annualized height velocity (AHV): **+1.5 cm/year** vs. placebo
Safety & Tolerability	• Well-tolerated AE profile: No injection site reactions or symptomatic hypotension. Less than 10% rate of hyperphosphatemia.

Potential share for ACH children

% of treated children

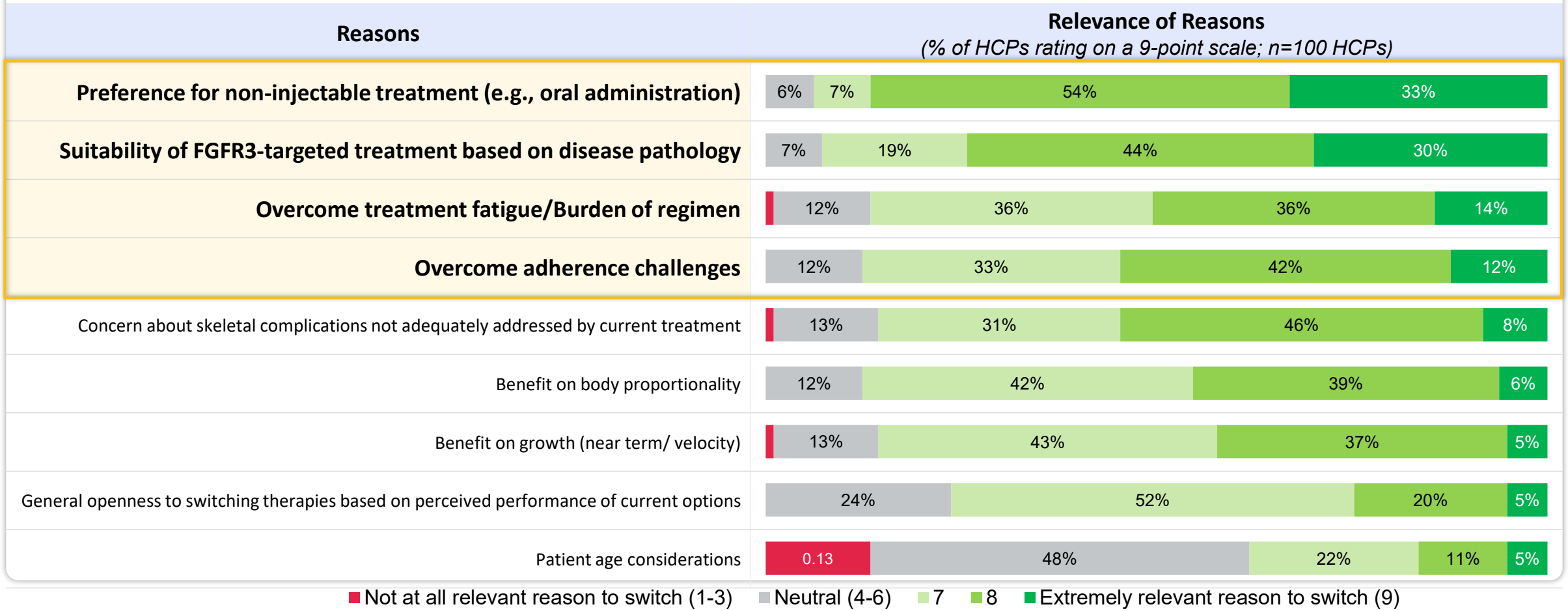
(N = 95 HCPs; represents ~37% of current market)



[1]BridgeBio market research (Analyses from ACH demand forecast Aug – Oct 2025); % of children; patient-weighted responses from n=95 HCPs; Q: Based on the information you just reviewed, please think about how you might decide to prescribe pharmacological therapy to the next 10 children you see who fall into each of the following clinical scenarios. For each scenario, how many of these children would you expect to prescribe each of the following treatment options?

A) Insights on market share: The leading prescription drivers for clinicians are having a child-friendly oral option and a mechanism targeting FGFR3

Relevance of Reasons to Switch TO Infigratinib[1]
(% of HCPs; n=100 HCPs)



[1]BridgeBio market research (Analyses from ACH demand forecast Aug – Oct 2025); Q. Based on the information you just reviewed and thinking about a hypothetical future scenario where Product Y is available for the treatment of pediatric achondroplasia (ACH), please rate the level of relevance of each potential reason to switch children living with achondroplasia TO Product Y, on a 9-point scale, where 1= not at all relevant reason to switch and 9= extremely relevant reason to switch.; n=100 HCPs

B) Insights on market size: There is a $5B market opportunity globally, with the vast majority of individuals not on a treatment option

Addressable achondroplasia population[1-4]

55,000 globally

Represents diagnosed and addressable ACH population with open growth plates

Estimated number of ACH children currently treated[1-4]

~55K addressable globally

~4,500 individuals on a treatment option

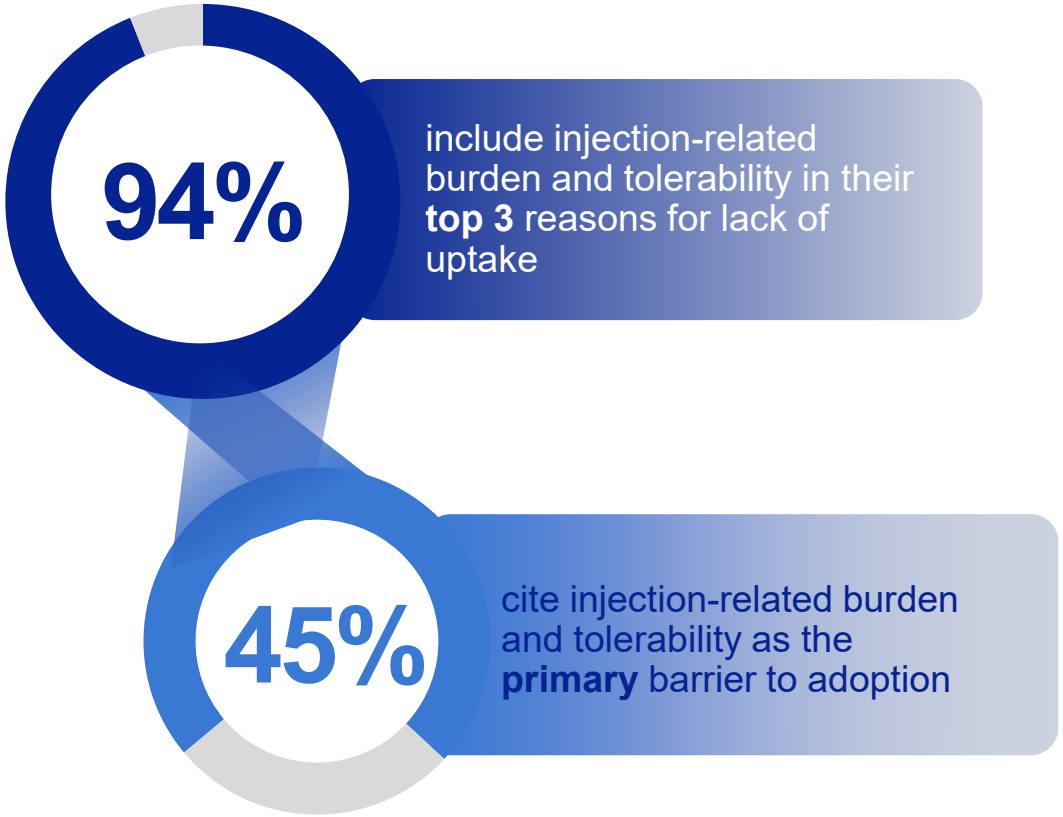
Up to 50K addressable individuals not on a treatment option

Availability of a safe oral treatment option could significantly expand the market potential for achondroplasia medicines

[1]CDC birth estimates; [2]EU Eurostats birth estimates; [3]Foreman, et al. Am J Med Genet. 2020; [4]Analyst estimates

C) Insights on market growth: Injection-related burden is frequently cited as the leading barrier against adoption of approved options today

Barriers to treatment uptake despite approved option among U.S. clinicians experienced with therapy[1]



Top reasons for families not being treated today[2]

For those interested in treatment, injections and dosing burden are consistently cited as the leading reason for children NOT being on therapy today[2]

> *"Most children are scared of injections, so parents usually hesitate to provide this treatment."*
> *- Ped Endocrinologist, CoE*

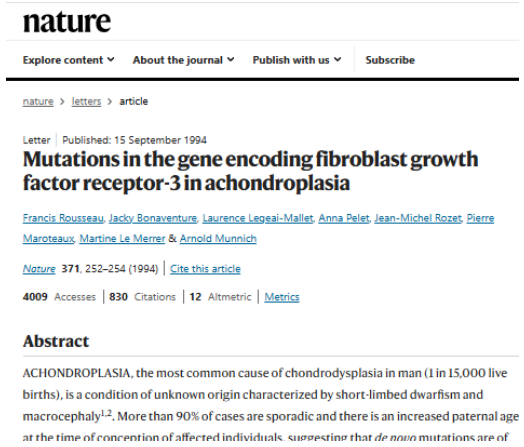
> *"The psychological stress associated with daily injections does bother most of the parents."*
> *- Geneticist, non-CoE*

> *"He doesn't want to get shots every day. That was a big turn-off. He said if it was just a medicine that he took every day, he could do that; but the shot, he's like, 'no, thank you.' "*
> *- Caregiver of ACH child*

[1]BridgeBio market research (Analyses from ACH demand forecast Aug – Oct 2025); [2] BridgeBio market research (Qualitative Market Research Interviews, Conducted Aug - Oct 2025)

Infigratinib: Connecting the dots between genetics, nonclinical data, and clinical data to do more and to do better for families 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

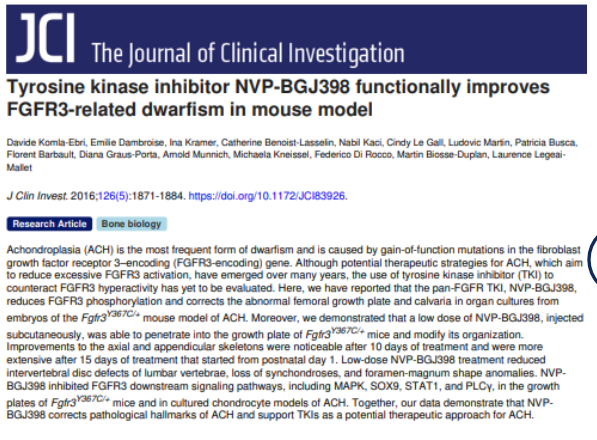
Nature 371, 252–254 (1994) | Cite this article

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

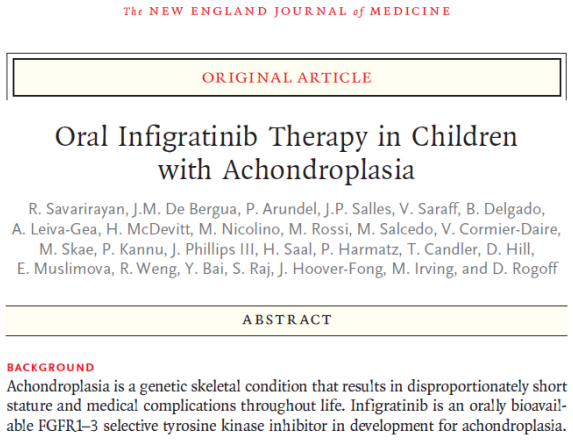
Davide Komla-Ebri, Emilie Dambroise, Ina Kramer, Catherine Benoist-Lasselin, Nabil Kaci, Cindy Le Gall, Ludovic Martin, Patricia Busca, Florent Barbault, Diana Graus-Porta, Arnold Munnich, Michaela Kneissel, Federico Di Rocco, Martin Biosse-Duplan, Laurence Legeai-Mallet

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.

Topline Q1 2026

PROPEL3

(Last participant last visit completed)

What does being able to "do more" and "do better" look like in PROPEL 3?

- **Safety:** No injection site reactions or symptomatic hypotension. Less than 10% low-grade hyperphosphatemia rate on treatment arm.
- **Linear growth:**
  - Change from baseline in AHV: More than +1.5cm/yr vs placebo
  - Change from baseline in height Z-score (ACH): More than +0.3SD from baseline on treatment
- **Proportionality:**
  - Decrease of 0.05 or more from baseline on treatment
  - **Home run scenario:** The first statistically significant result in a pivotal trial
- **Broadest age range (3-18) in a Ph3 trial:** Larger age range on potential initial label, even if negative impact to overall point estimate based on other trials (+0.77 cm/yr[1] in children >11 yrs, and +1.02 cm/yr[2] in <5 years)
- **A commitment to looking at more than just height:** PROPEL OLE will continue to study longer term impact on skeletal changes, quality of life, and continued impact on proportionality

[1]Savarirayan et al, Lancet, 2020; [2]Ascendis ApproaCH topline results, September 2024

Infigratinib is an investigational agent that is not approved for use in the treatment of achondroplasia by any regulatory authority

Infigratinib: PROPEL 3 topline in Q1 2026 and expansion opportunities

PROPEL 3 topline results expected in Q1 2026

- Last participant last visit achieved
- Ph. 3 topline results to be shared in future webinar, with full results presented at a medical congress

Advancing toward regulatory milestones and launch readiness

- Planning for key 2026 regulatory interactions, supported by Breakthrough Therapy Designation
- Building the capabilities required to support a successful global launch for infigratinib

Pursuing the full potential of infigratinib in FGFR3-related conditions

- Completed enrollment of Phase 2 portion of ACCEL 2/3 study in hypochondroplasia; proof-of-concept results expected in 2H 2026
- Initiated PROPEL I&T, the Infant and Toddler study in achondroplasia (0-<3 years)
- Expansion potential in FGFR3-implicated growth disorders (Turner Syndrome, SHOX Deficiency)

Q&A

bridgebio

hope through
rigorous science

Thank you