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rigorous science

Investor webcast: PROPEL 2 data update and ACCEL program initiation

June 4th 2024



Forward-looking statements

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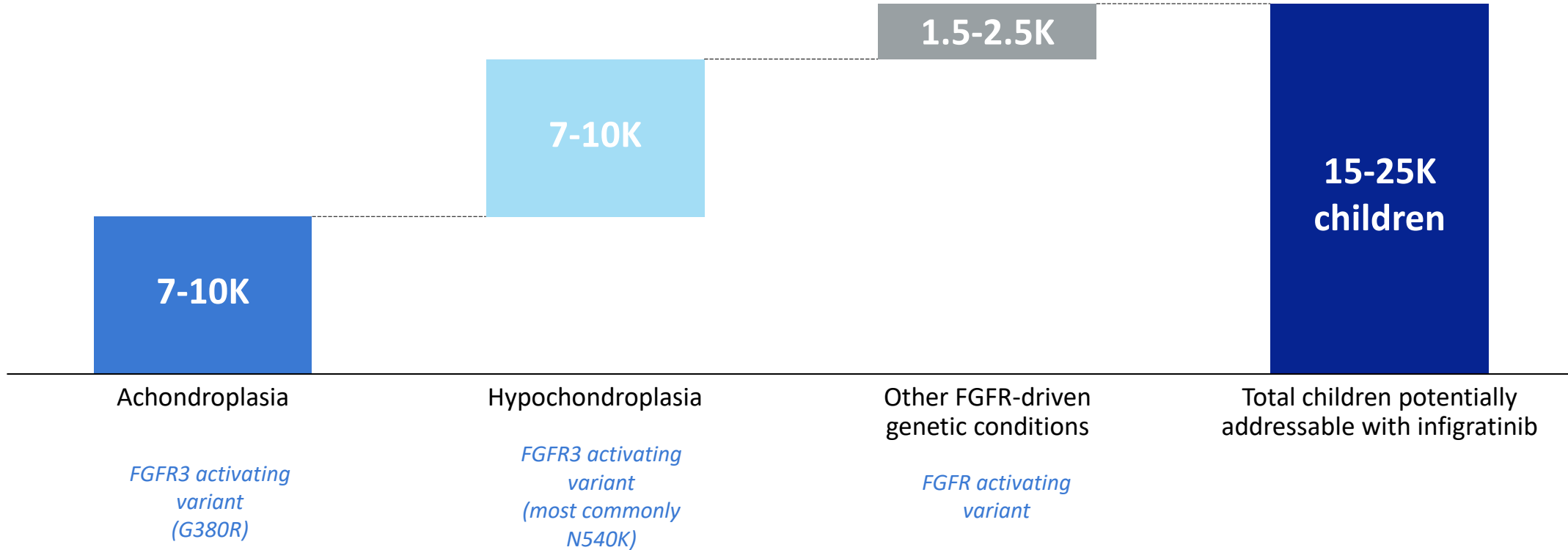
**To the children, families,
advocates, and physicians
who have been a part of
this program:
Thank you**

Developing new treatment options relies
entirely on your guidance, dedication, and effort



Achondroplasia, hypochondroplasia, and related FGFR-driven genetic conditions represent a large unmet medical need

Children eligible for FGFR inhibitor treatment in the US and Europe



BridgeBio is committed to developing a treatment option for children with FGFR-driven conditions

We are developing infigratinib as a treatment option based on three key principles

Objectives

- ✓ **Maximize efficacy**
For all the manifestations of ACH—not just height— which matter for families and physicians
- ✓ **Demonstrate safety**
Avoiding hypotension & injection site reactions with no hyperphosphatemia, ocular effects or VEGFR3 or JAK2 off-target effects
- ✓ **Avoid injections**
For children and families, to reduce burden and pain of treatment



Design principles

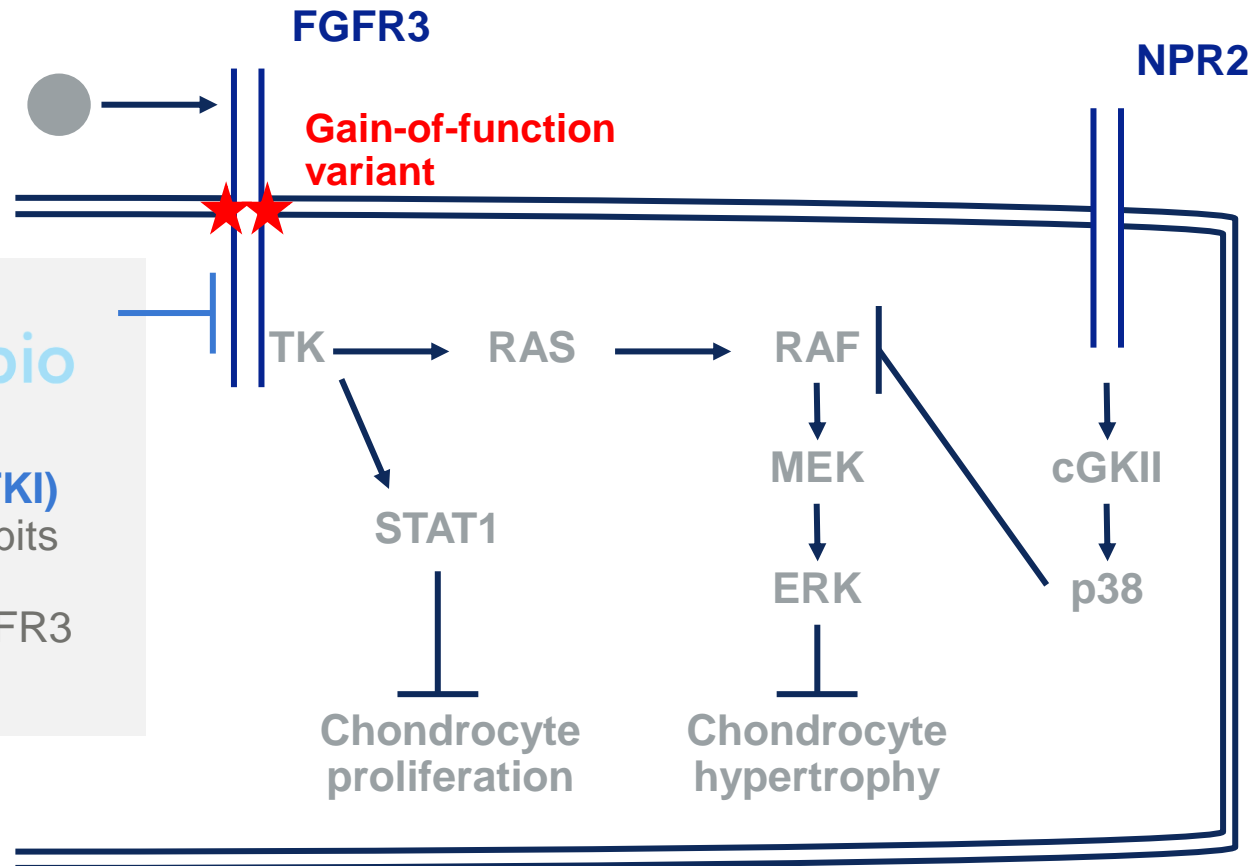
Target the condition directly at the source (FGFR3)

Use low doses to ensure safety

Provide an oral treatment option

Infigratinib is the only treatment option in the clinic that could incorporate all of these features

Infigratinib addresses the first design principle by directly targeting the underlying cause of achondroplasia and hypochondroplasia: FGFR3 overactivity



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Infigratinib
(FGFR1-3 TKI)
Directly inhibits the gain-of-function FGFR3 receptor

- Infigratinib targets achondroplasia and hypochondroplasia **directly at the source**: FGFR3 overactivity

- Infigratinib **inhibits both downstream pathways** responsible for the clinical phenotype associated with achondroplasia

- Thus infigratinib's mechanism of action provides an **advantageous approach to treatment of achondroplasia**

Agenda

Cohort 5 in ACH: Data Update

Cohort 5 in ACH: Context (Dr. Savarirayan)

PROPEL 3 for ACH

Hypochondroplasia: Overview (Dr. Savarirayan)

ACCEL program for Hypochondroplasia: Overview

Closing

Cohort 5 (0.25 mg/kg) of the PROPEL2 Trial: Infigratinib continues to be well-tolerated, with no related AEs

- In Cohort 5 (the highest dose escalation level of 0.25 mg/kg):
 - No serious adverse events (SAEs), no AEs that required treatment discontinuation
 - Most 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 ocular adverse events**
 - **0 hyperphosphatemia events**
 - No accelerated progression of the bone age
- Safety profile for cohorts 1-4 has continued with no new hyperphosphatemia events, ocular events, or SAEs

Infigratinib in PROPEL 2 cohort 5 is well-tolerated with no safety signals identified through month 18

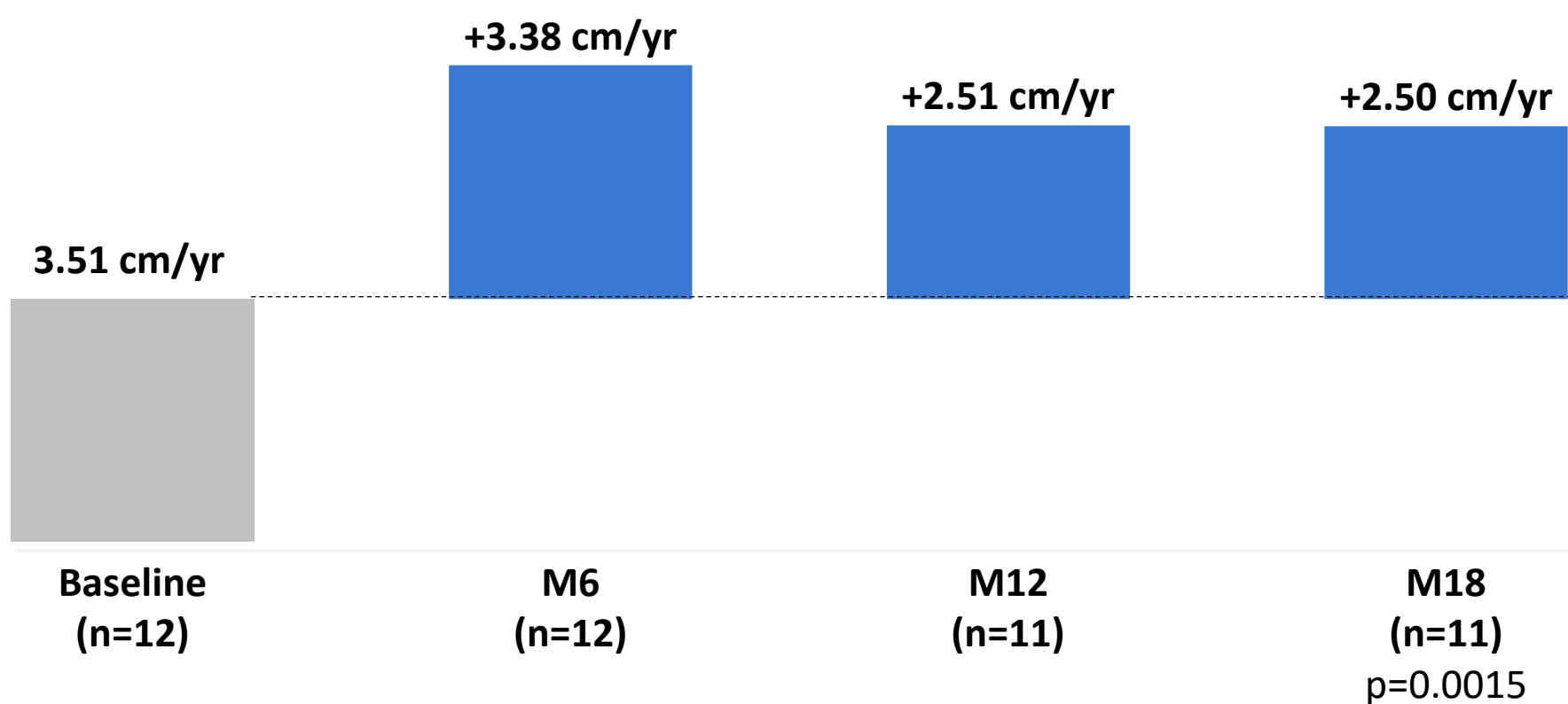
Reported AEs across all cohorts are common in the pediatric population, especially in children with achondroplasia

AEs occurring in $\geq 10\%$ of study participants	Total (%) N = 72
Nasopharyngitis	29 (40.3%)
COVID-19	24 (33.3%)
Headache	24 (33.3%)
Vomiting	22 (30.6%)
Pain in extremity	20 (27.8%)
Ear infection	19 (26.4%)
Pyrexia	18 (25.0%)
Abdominal pain	11 (15.3%)
Cough	11 (15.3%)
Diarrhea	11 (15.3%)
Rhinitis	11 (15.3%)
Viral infection	11 (15.3%)
Upper respiratory tract infection	10 (13.9%)
Abdominal pain upper	8 (11.1%)
Ear pain	8 (11.1%)
Nausea	8 (11.1%)
Oropharyngeal pain	8 (11.1%)
Otitis media	8 (11.1%)

Infigratinib continues to demonstrate a well-tolerated safety profile

Cohort 5: The best-in-class change from baseline in AHV at M6 is sustained at M12 and M18

Mean change from baseline in annualized height velocity (AHV)

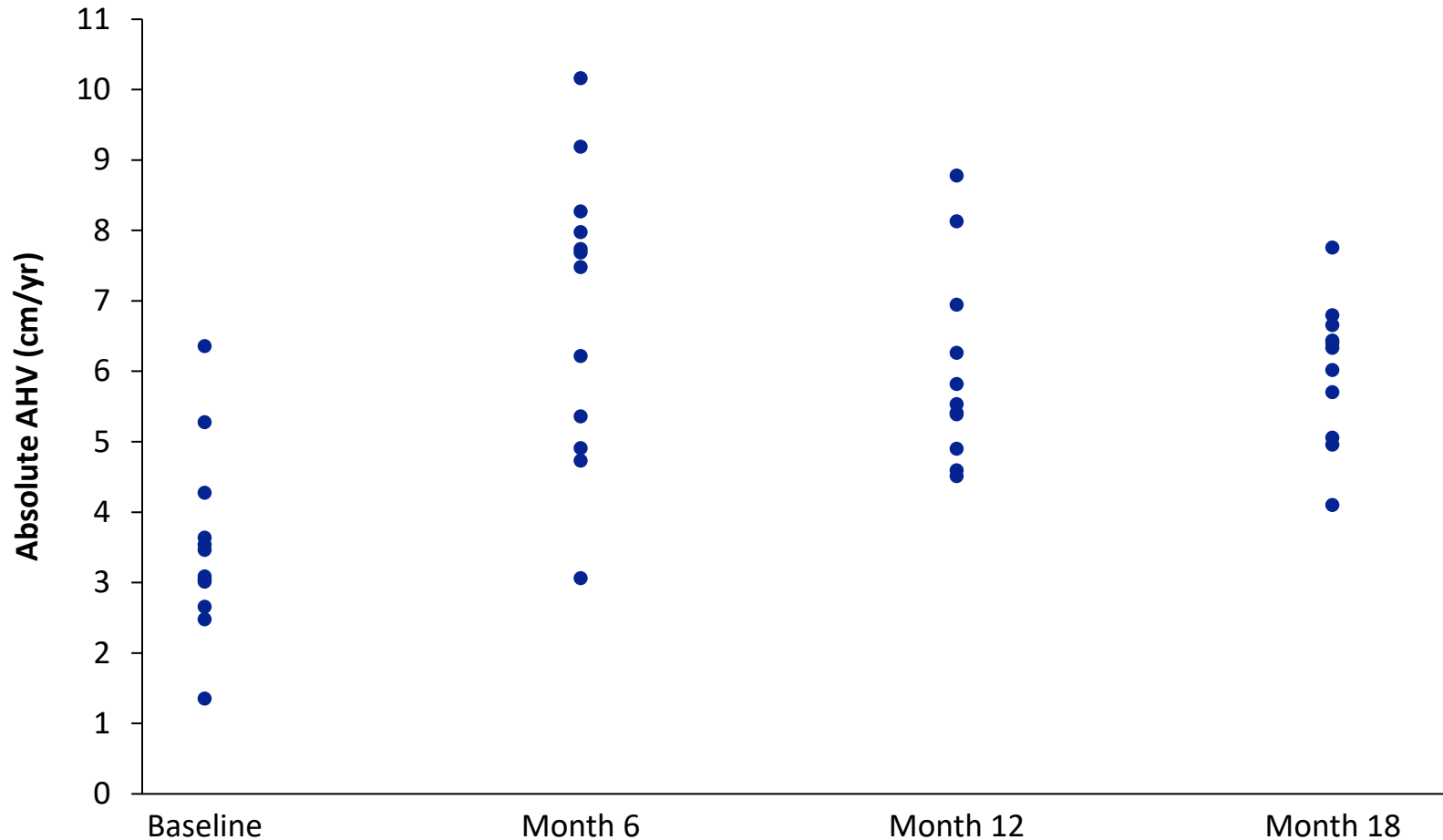


Baseline demographics	
Female:Male ratio	7:5
Mean age at screening (yr)	7.24
<5	8%
5 - <8	58%
8 - <11	25%
>=11	8%
Baseline AHV (cm/yr) Mean (SD)	3.51 (1.3)

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

The response to infigratinib was broad, with 10 out of 11 kids having an increase from baseline in AHV at M18 compared to baseline

Absolute AHV individual values, cm/yr

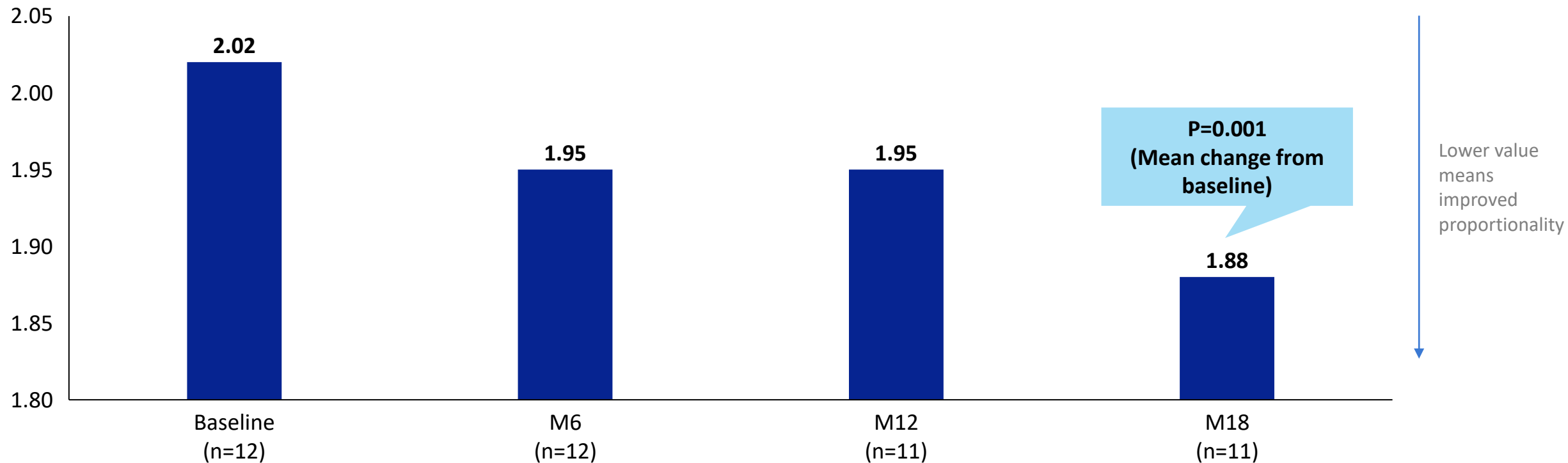


91% of participants had an increase in AHV from Baseline to M18

73% of participants had an increase of greater than 25% in AHV from Baseline to M18

Infigratinib shows a persistent decrease in upper/lower body segment ratio at the cohort 5 dose

Upper body to lower body segment ratio



Statistically significant proportionality improvements after only 18 months demonstrates strong potential for a meaningful effect on body proportionality

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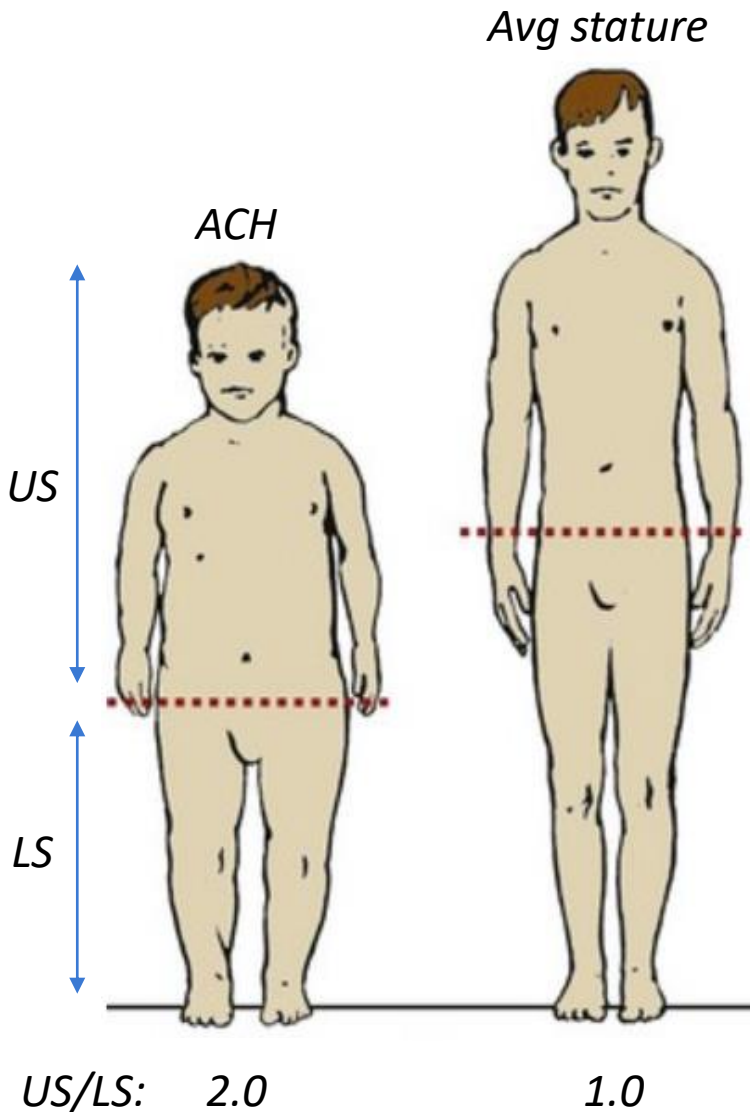
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Proportionality links changes in linear growth to potential benefits beyond height



- Achondroplasia is a **disproportionate** skeletal dysplasia (upper body segment to lower body segment ratio of ~ 2.0 versus ~ 1.0 in average statured children)
- Disproportionality is associated with delayed motor milestones, balance and impaired functionality in children with achondroplasia
- The cumulative improvements in proportionality seen with infigratinib are encouraging with the **potential to enhance functionality, activities of daily living and quality of life**

Proportionality is a critical endpoint in achondroplasia

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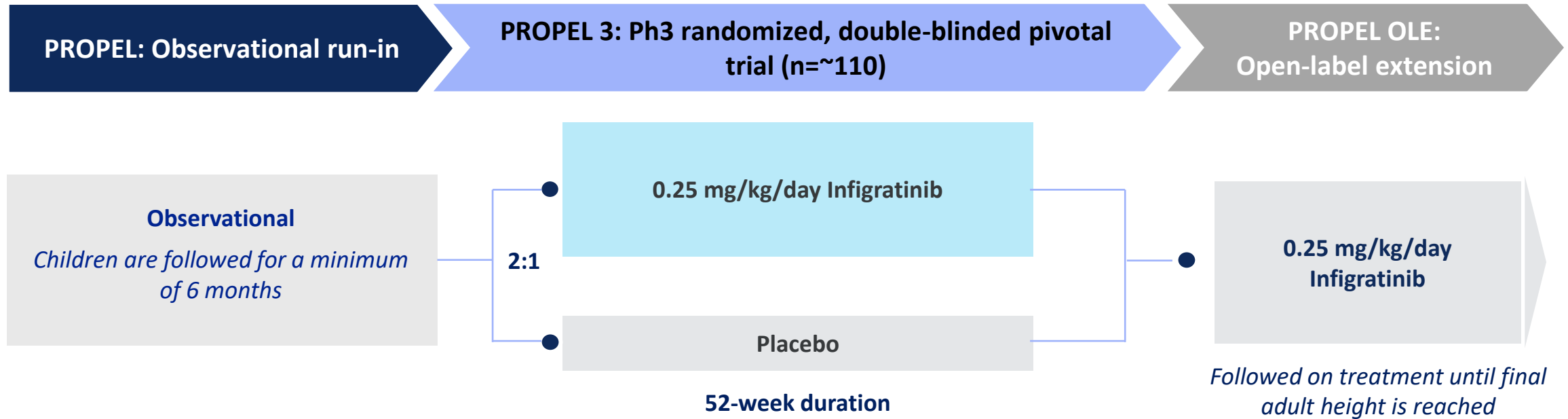
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PROPEL 3, the phase 3 trial of infigratinib for achondroplasia, is underway with last participant in expected by end of 2024



Key inclusion criteria

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

Primary endpoint:

Change from baseline in annualized height velocity at week 52

Key secondary endpoints:

Change from baseline in height z-score
Change from baseline in upper body:lower body segment ratio

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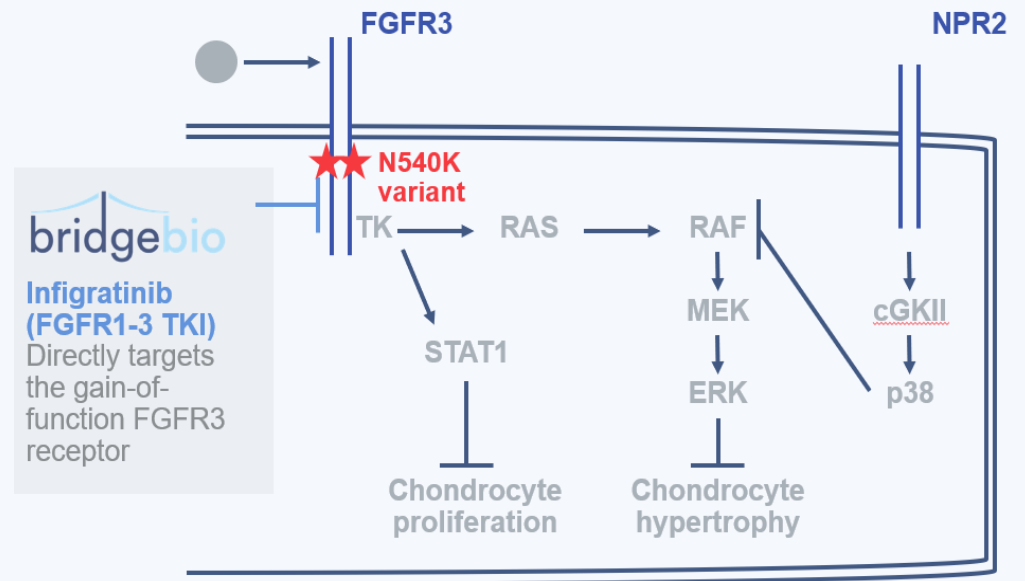
Closing

Hypochondroplasia is a FGFR3-related skeletal dysplasia with a need for treatment options



- Autosomal dominant condition
- Similar incidence to achondroplasia
- Greater genetic heterogeneity in *FGFR3* pathogenic variants (e.g. N540K in addition to others)
- Clinical features:
 - Moderate disproportionate short stature
 - Head circumference larger than average
 - Tibia bowing
- Medical complications are milder and less frequent than in achondroplasia
 - Motor milestones less delayed
 - Reports of epilepsy, temporal lobe abnormalities & other cognitive functions^{2,3}

Infigratinib directly targets FGFR3 signaling



Infigratinib directly targets the underlying cause of hypochondroplasia, FGFR3 overactivity

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Similar to the PROPEL program in ACH, the ACCEL clinical program consists of an observational run-in and an interventional study with long-term follow-up

ACCEL

Observational Run-in

Children and adolescents (2.5 to <17 years) with hypochondroplasia (HCH)

Primary objective: Establish baseline height velocity

Primary endpoints: Annualized height velocity (AHV)

ACCEL 2/3

Phase 2/3 Open-Label Phase followed by a Double-Blinded, Randomized, Placebo-Controlled Study

Ph2: Open-Label Phase

Children (5 – 11 years with growth potential) with HCH who completed ≥6 months in ACCEL

Primary objectives: Obtain preliminary efficacy, safety and tolerability of infigratinib in HCH

Primary endpoints: Change from baseline in HV and safety endpoints

Pivotal Ph3: Double-Blind, Randomized, Placebo-Controlled Phase

Children and adolescents (3 – <18 years with growth potential) with HCH who completed ≥6 months in ACCEL

Primary objectives: Evaluate safety and efficacy of infigratinib in HCH

Primary endpoints: Change from baseline in AHV vs. placebo at 52 wks

ACCEL OLE

Open-Label Extension (planned)

Eligible children and adolescents who completed either portion will be offered treatment with infigratinib until final height/near final height

Primary objectives: Evaluate long term safety, tolerability and efficacy of infigratinib in HCH

The ACCEL trials will also evaluate changes in growth, body proportions and HCH-related complications

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Conclusions



Best-in-class efficacy and well-tolerated safety profile of infigratinib in achondroplasia are sustained through 18 months in the current data, now with a **statistically-significant improvement in proportionality**



PROPEL 3 pivotal study of infigratinib in achondroplasia is enrolling quickly, on track for **last participant in by end of 2024**



Expansion of **infigratinib in hypochondroplasia is now initiated**, with the ACCEL clinical trial open and **first participant consented**



BridgeBio is committed to **exploring infigratinib's scientific potential** and is evaluating **additional FGFR-driven genetic conditions** including Crouzon, Apert, & Muenke syndromes

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Thank you

