TYRA

Investor Call and Webcast

March 1, 2023

Disclaimers

Forward-looking statements and market data

We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, research and development plans, the anticipated timing and phase of development, costs, design and conduct of our ongoing and planned preclinical studies and clinical trials for our product candidates, the timing and likelihood of regulatory filings and approvals for our product candidates, the potential to develop product candidates and the safety and therapeutic benefits of our product candidates, our ability to commercialize our product candidates, if approved, the impact of the COVID-19 pandemic and other epidemic diseases on our business, the pricing and reimbursement of our product candidates, if approved, the timing and likelihood of success, plans and objectives of management for future operations, and future results of anticipated product development efforts, are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negative of these terms or other similar expressions. The inclusion of forward-looking statements should not be regarded as a representation by us that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: we are early in our development efforts, have only recently begun testing our lead product candidate in clinical trials and the approach we are taking to discover and develop drugs based on our SNÅP platform is novel and unproven and it may never lead to product candidates that are successful in clinical development or approved products of commercial value; potential delays in the commencement, enrollment, and completion of preclinical studies and clinical trials; results from preclinical studies or early clinical trials not necessarily being predictive of future results; our dependence on third parties in connection with manufacturing, research and preclinical testing; acceptance by the FDA of INDs or of similar regulatory submissions by comparable foreign regulatory authorities for the conduct of clinical trials of TYRA-300 in pediatric achondroplasia; an accelerated development or approval pathway may not be available for TYRA-300 or other product candidates and any such pathway may not lead to a faster development process; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval, and/or commercialization; the potential for our programs and prospects to be negatively impacted by developments relating to our competitors, including the results of studies or regulatory determinations relating to our competitors; unfavorable results from preclinical studies; our ability to maintain undisrupted business operations due to the COVID-19 pandemic or other epidemic diseases, including delaying or disrupting our preclinical studies and clinical trials, manufacturing, and supply chain; regulatory developments in the United States and foreign countries; our ability to obtain and maintain intellectual property protection for our product candidates and proprietary technologies; we may use our capital resources sooner than we expect; and other risks described in our prior filings with the Securities and Exchange Commission (SEC), including under the heading “Risk Factors” in our annual report on Form 10-K and any subsequent filings with the SEC. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.

TYRA Corporate Overview

Differentiated assets

Precision small molecules targeting large opportunities in FGFR biology

Lead asset: TYRA-300, the first oral FGFR3-selective inhibitor in the clinic

Accelerated design

SNAP CHEMISTRY DESIGN

NASDAQ: TYRA

CASH:* $263.2M

*Sept. 30, 2022

TYRA

Introduction to Skeletal Dysplasias

Opportunity for next-gen FGFR3 inhibitors

Our Skeletal Dysplasias Program

Dr Michael Bober

Medical Director of the Skeletal Dysplasia Program
Nemours Children’s Hospital, Delaware

Achondroplasia

- Most common skeletal dysplasia
- ~1 in 26,000 live births
- Short-limbed dwarfism
  - Rhizomelic
  - Average intelligence
- Autosomal dominant
  - 100% penetrant
  - 80% de novo
  - 3 - 5% recurrence risk for an AS couple with a previous child born with achondroplasia
- Caused by mutation in FGFR-3

c 1643 - Diego de Velazquez

Fibroblast Growth Factor Receptor -3

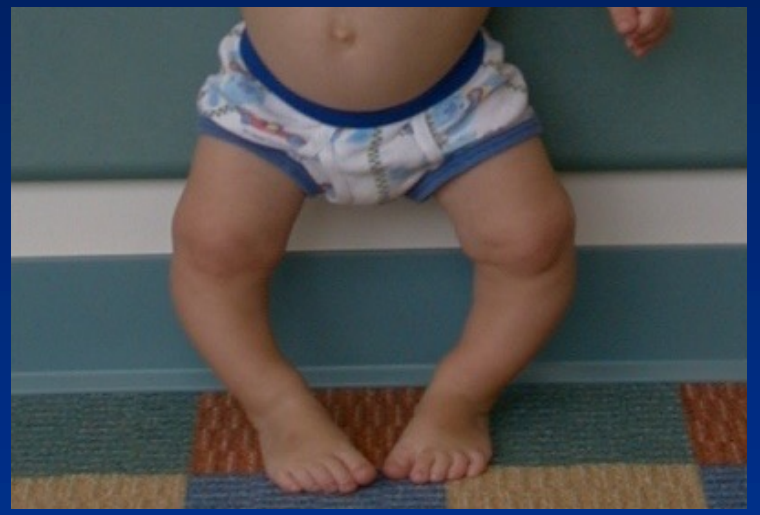
A transmembrane receptor in the fibroblast growth factor signaling pathway

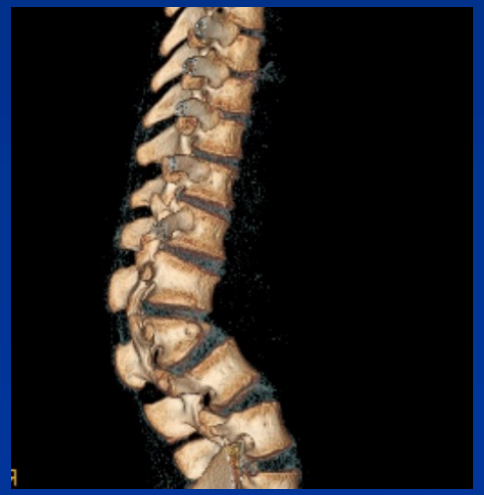
The FGFR-3 pathway functions as a negative regulator of chondrocyte proliferation and differentiation within the growth plate

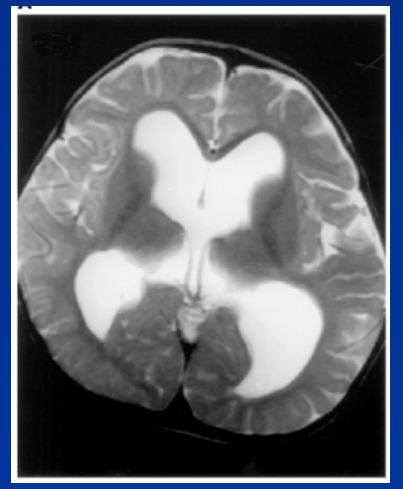
Gene responsible for a family of skeletal dysplasias including achondroplasia

Major Medical Problems

- Otitis Media
  - Serous
  - Infectious
- Obstructive Apnea
- Foramen Magnum Stenosis
- Hydrocephalus
- Spinal Stenosis
  - Thoracolumbar kyphosis
- Genu Vara







TYRA

Introduction to Skeletal Dysplasias

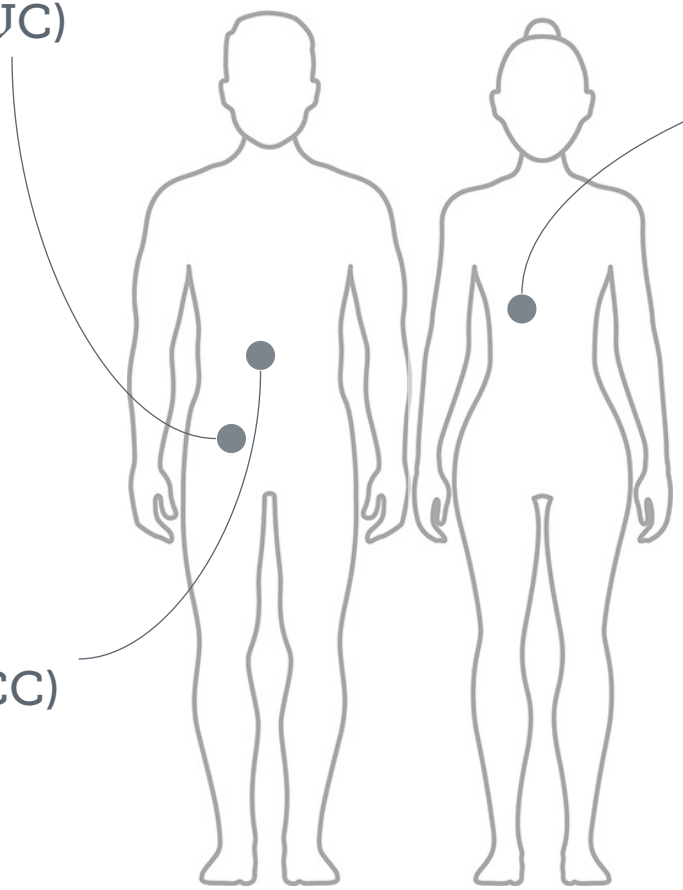
Opportunity for next-gen FGFR3 inhibitors

Our Skeletal Dysplasias Program

Alterations in the FGFR family: a major unmet need

UROTHELIAL CARCINOMA (UC)
~50% FGFR3
~40,000/yr (US)

INTRAHEPATIC CHOLANGIOCARCINOMA (ICC)
10–20% FGFR2
~1,700/yr (US)



HEPATOCELLULAR CARCINOMA (HCC)
~30% FGF19 (FGFR4/3 ligand)
~9,000/yr (US)

7%
Cancer patients with FGFR mutations

Note: oncology figures represent 2022 US incidence across all stages of the disease

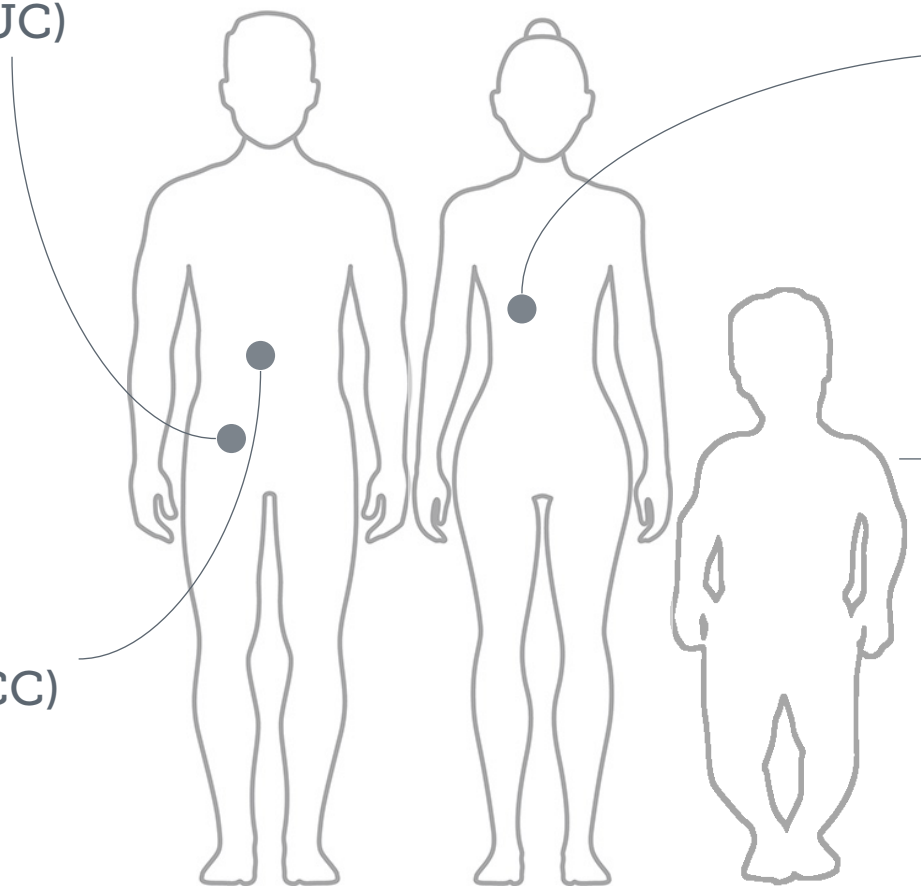
Alterations in the FGFR family: a major unmet need

UROTHELIAL CARCINOMA (UC)

~50% FGFR3
~40,000/yr (US)

INTRAHEPATIC CHOLANGIOCARCINOMA (ICC)

10–20% FGFR2
~1,700/yr (US)



HEPATOCELLULAR CARCINOMA (HCC)

~30% FGF19 (FGFR4/3 ligand)
~9,000/yr (US)

ACHONDROPLASIA (ACH)

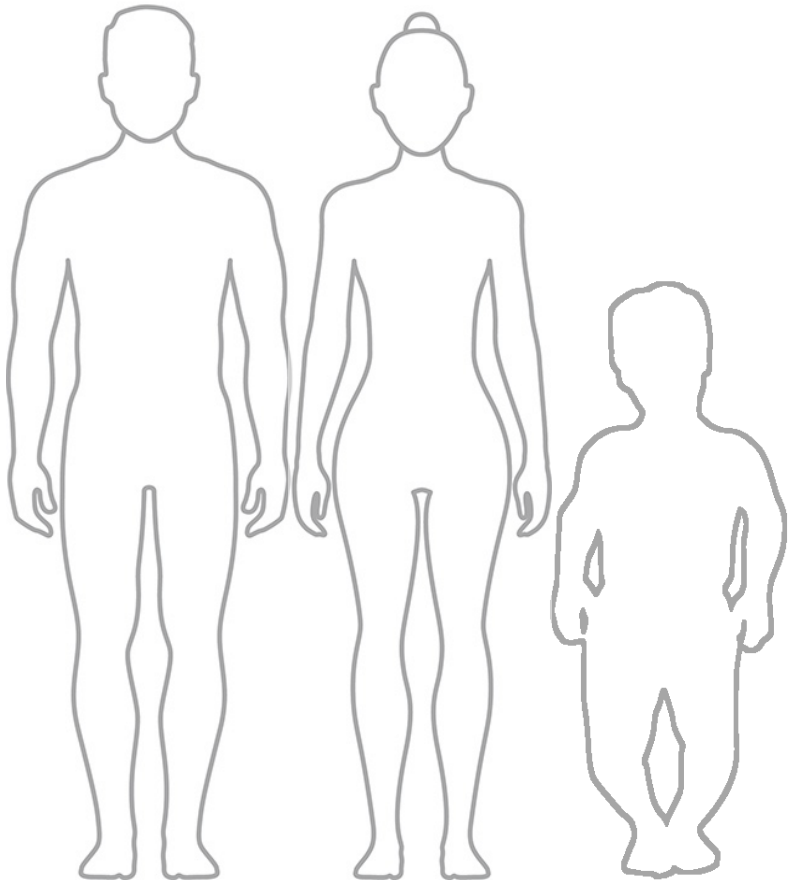
~97% FGFR3
~3,000/yr (US)

OTHER FGFR3-RELATED SKELETAL DYSPLASIAS

~40,000/yr (US)

Note: oncology figures represent 2022 US incidence across all stages of the disease; skeletal dysplasias represent 2022 US pediatric prevalence

Approved FGFR inhibitors have significant liabilities



Approved drugs are pan-FGFR inhibitors

pemigatinib	erdafitinib
futibatinib	infigratinib

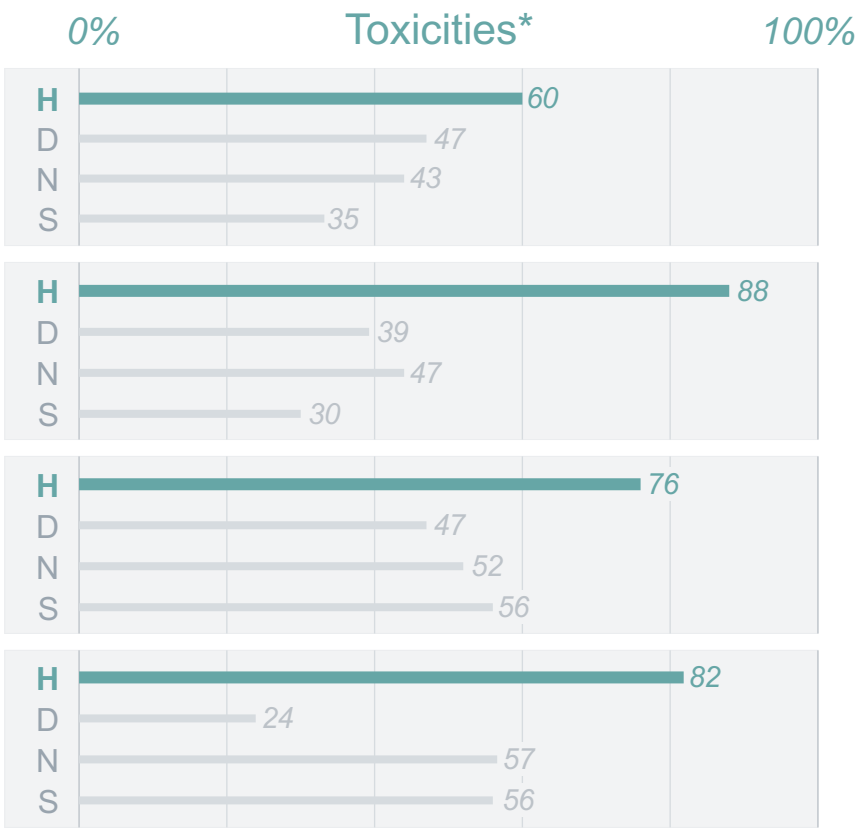
Liabilities

Tolerability	Off-target toxicities drive frequent dose interruptions and reductions
Acquired resistance	Resistance mutations limit the durability of current drugs

FGFR1 drives hyperphosphatemia

	SELECTIVITY	
	FGFR	Design
pemigatinib	1–3	Reversible
futibatinib	1–4	Covalent
erdafitinib	1–4	Reversible
infigratinib	1–3	Reversible

SAFETY



*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis

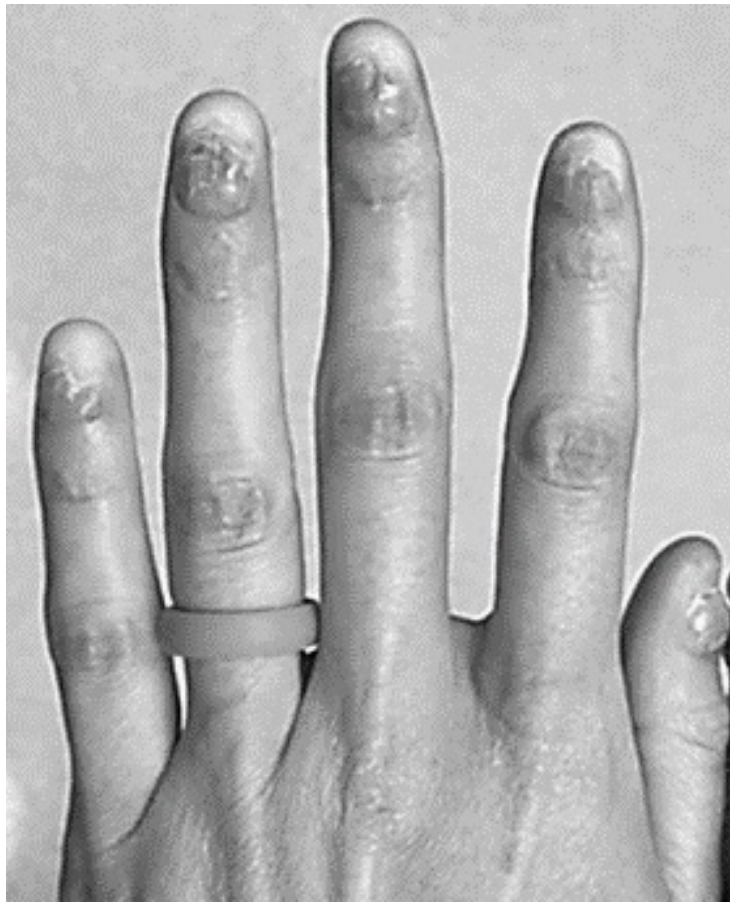
Source: product labels and websites

FGFR2 drives stomatitis and nail toxicity

	SELECTIVITY		SAFETY
	FGFR	Design	0% Toxicities* 100%
pemigatinib	1–3	Reversible	H 60, D 47, N 43, S 35
futibatinib	1–4	Covalent	H 88, D 39, N 47, S 30
erdafitinib	1–4	Reversible	H 76, D 47, N 52, S 56
infigratinib	1–3	Reversible	H 82, D 24, N 57, S 56

Source: product labels and websites

*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis

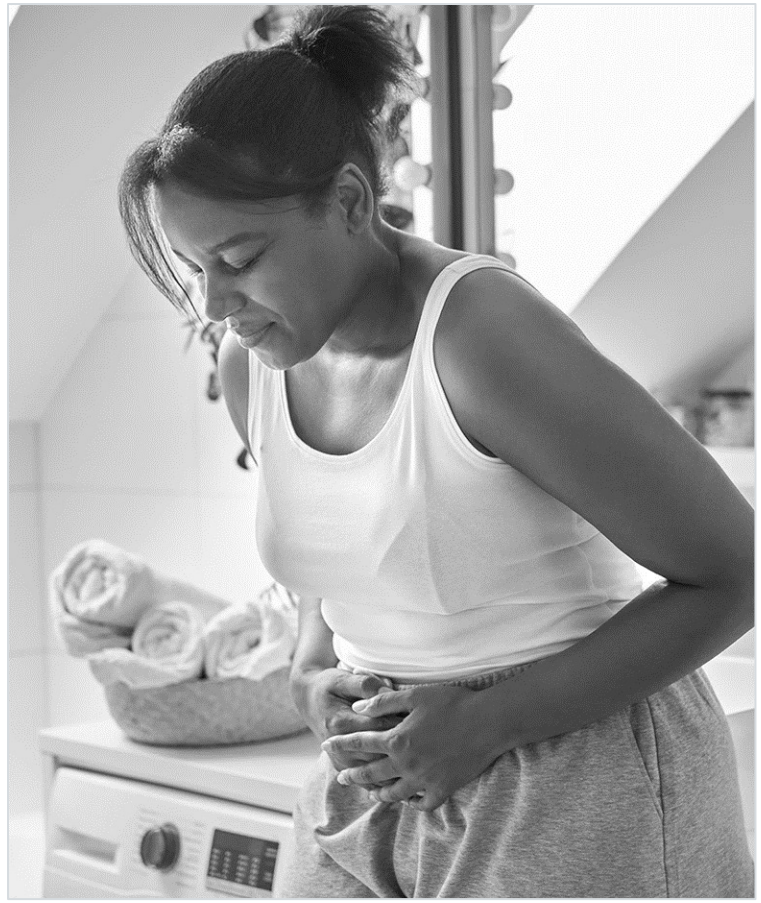


FGFR4 drives gastrointestinal and liver toxicity

	SELECTIVITY		SAFETY
	FGFR	Design	0% Toxicities* 100%
pemigatinib	1–3	Reversible	H 60, **D** 47, N 43, S 35
futibatinib	1–4	Covalent	H 88, **D** 39, N 47, S 30
erdafitinib	1–4	Reversible	H 76, **D** 47, N 52, S 56
infigratinib	1–3	Reversible	H 82, **D** 24, N 57, S 56

Source: product labels and websites

*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis



FGFR1/2/4 toxicities limit dosing

	SELECTIVITY		SAFETY		
	FGFR	Design	0% Toxicities* 100%	Discontinuation/ Reduction	DLT Driver
pemigatinib	1–3	Reversible	H 60 D 47 N 43 S 35	23%	Hyper-phosphatemia (R1)
futibatinib	1–4	Covalent	H 88 D 39 N 47 S 30	63%	AST/ALT increase (R4)
erdafitinib	1–4	Reversible	H 76 D 47 N 52 S 56	66%	Hyper-phosphatemia (R1)
infigratinib	1–3	Reversible	H 82 D 24 N 57 S 56	75%	Hyper-phosphatemia (R1)

Source: product labels and websites

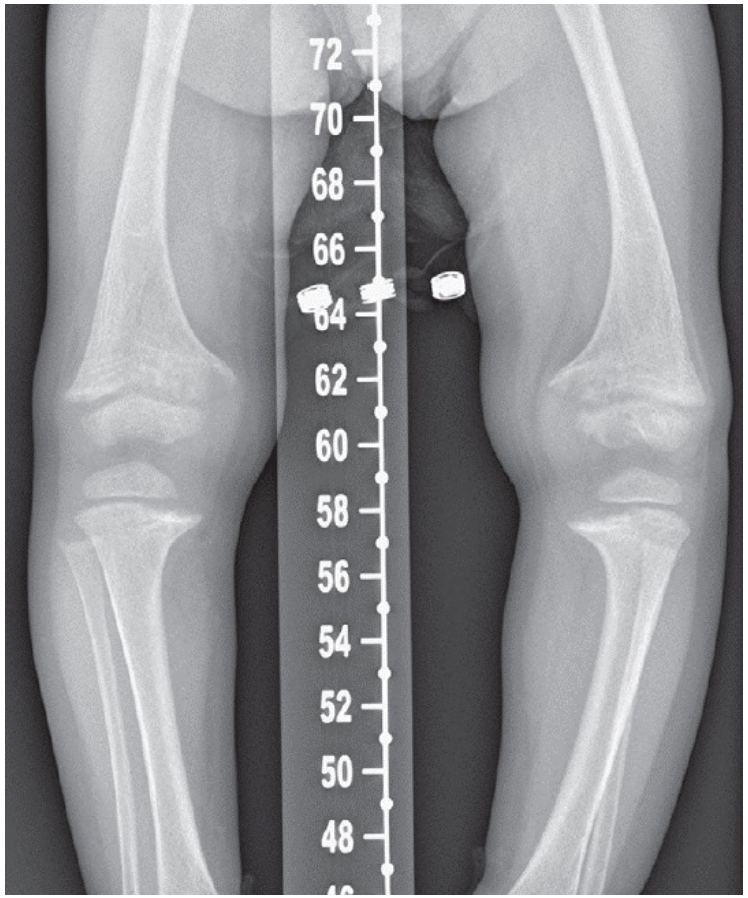
*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis

FGFR3 inhibition accelerates bone growth in children

	SELECTIVITY		SAFETY
	FGFR	Design	0% Toxicities* 100%
pemigatinib	1–3	Reversible	H 60, D 47, N 43, S 35
futibatinib	1–4	Covalent	H 88, D 39, N 47, S 30
erdafitinib	1–4	Reversible	H 76, D 47, N 52, S 56
infigratinib	1–3	Reversible	H 82, D 24, N 57, S 56

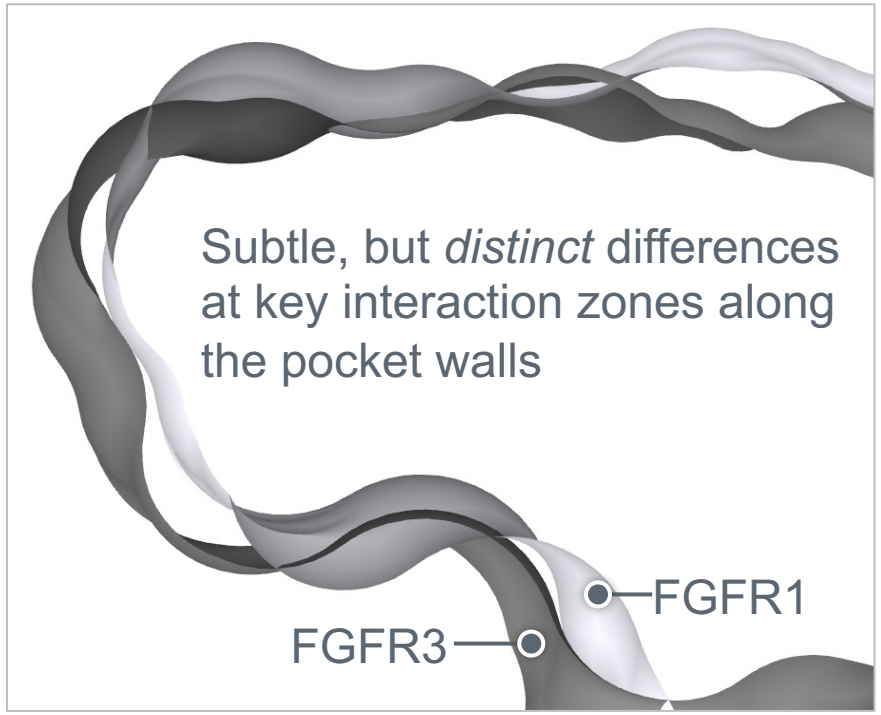
Source: product labels and websites

*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis



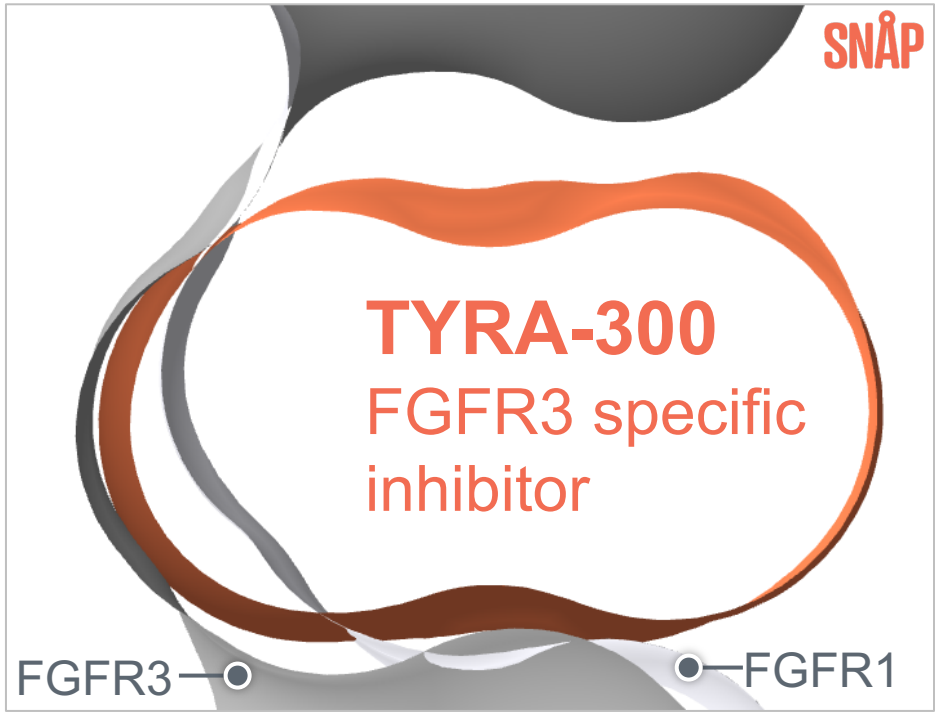
The challenge: FGFR family active sites are nearly identical

FGFR isoform selectivity



MOLECULAR MODEL

dozens of high-resolution crystal structures



CRYSTALLOGRAPHY

3.0Å cross-sections

TYRA

Introduction to Skeletal Dysplasias

Opportunity for next-gen FGFR3 inhibitors

Our Skeletal Dysplasias Program

Population

Selectivity

Pre-clinical

Clinical

TYRA-300^{ACH}

Achondroplasia (ACH)

FGFR3-selective

Daily oral

Rationale for additional indications

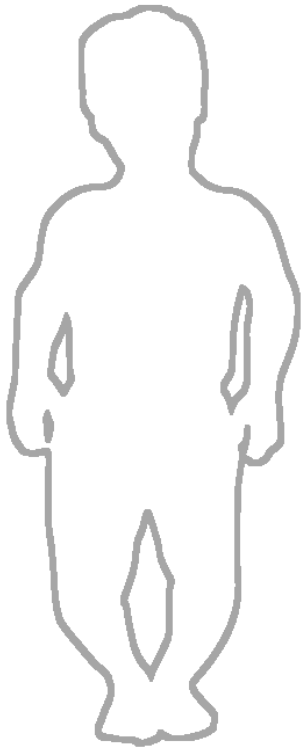
FGFR3 aberrations drive >97% of pediatric achondroplasia (ACH)

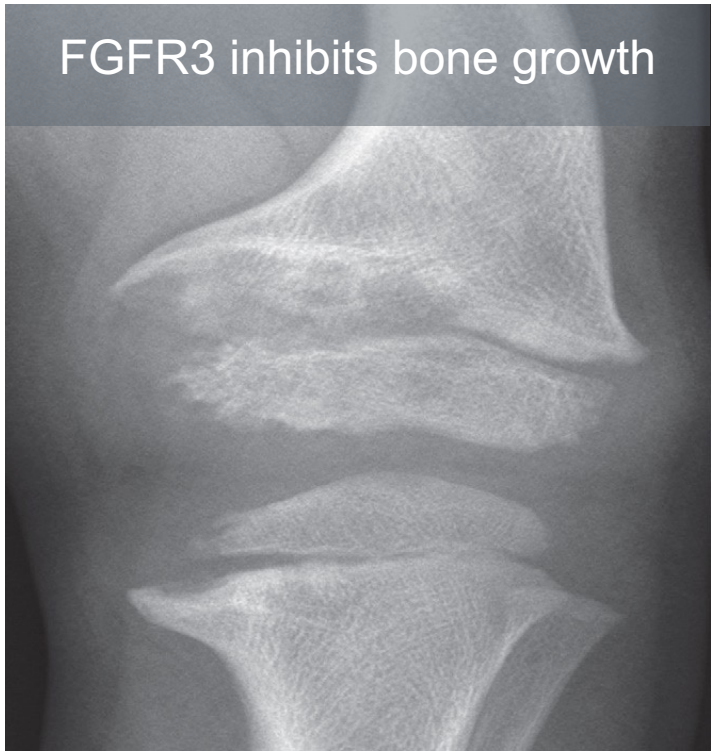
- Population
- Selectivity
- Pre-clinical
- Clinical

ACH

Prevalence 2–5,000/year[1]

>97% FGFR3 mutations





1. US pediatric prevalence: Vajo et al. 2000; US census

VOXZOGO was approved based on growth acceleration

Population

Selectivity

Pre-clinical

Clinical

ACH

Prevalence 2–5,000/year[1]

>97% FGFR3 mutations

LEAD OPTION	UNMET NEEDS
Height: VOXZOGO 1.57 cm/year Annual Height Velocity (AHV) Baseline Increase vs placebo	Disproportionate growth Formulation / administration
Other: Surgeries / supportive care	Includes cranial or spinal stenosis, hydrocephalus and sleep apnea

Daily Injectable
VOXZOGO

1. US pediatric prevalence: Vajo et al. 2000; US census

Oral pan-FGFR inhibitor infigratinib increased AHV in pediatric ACH

Population

Selectivity

Pre-clinical

Clinical

	Annualized Height Velocity (AHV) baseline increase	
BIOMARIN® **VOXZOGO** SubQ CNP Analog On market	1.57 cm/year vs placebo	Phase 3 Data / Label 15µg/kg Daily SubQ 52 wks, Ages 5-15 N=60
bridgebio **infigratinib** Oral FGFR1-3 Inhibitor Mid-phase 2	1.52 cm/year	Phase 2 Cohort 4 Data 0.128mg/kg daily oral 26.9 wks, Ages 5-10 N=11

Sources: VOXZOGO label, BridgeBio press release July 26, 2022
Note: Infigratinib 0.25mg/kg dose cohort 5 currently enrolling

TYRA-300 is more selective for FGFR3 than infigratinib

Population

Selectivity

Pre-clinical

Clinical

TYRA-300 selectivity vs. infigratinib: Ba/F3 Cellular IC_{50} (nM)

	infigratinib	TYRA-300
FGFR1	15.3	113
FGFR2	5.8	34.9
FGFR3	6.9	1.8
FGFR4	459	98.4

Fold Selectivity for FGFR3

FGFR1	2.2x	63x
FGFR2	0.8x	19x
FGFR4	67x	55x

TYRA-300 shows significant isoform selectivity for FGFR3 over other FGFR isoforms

TYRA-300 increased bone growth in FGFR3$^{Y367C/+}$ model

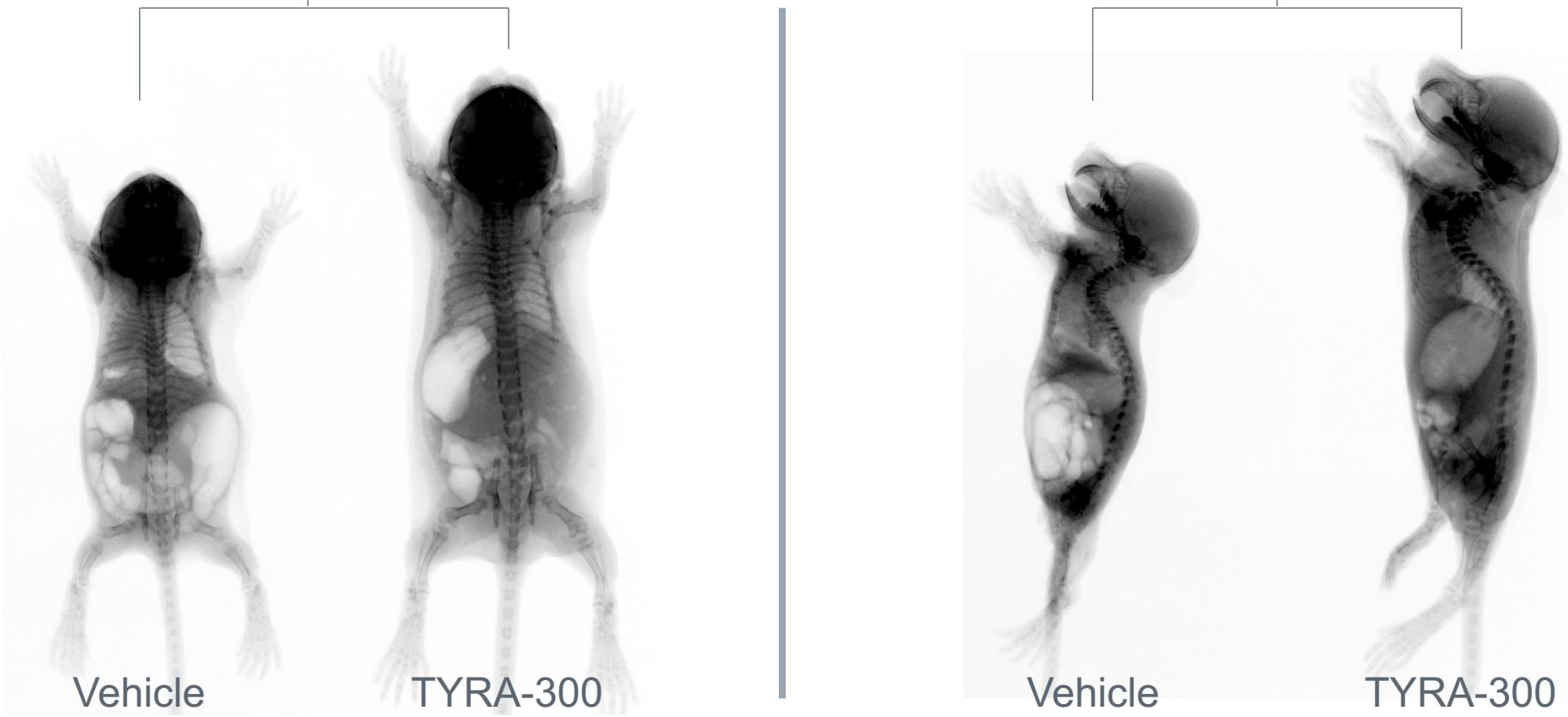
Population

Selectivity

Pre-clinical

Clinical

17.6% increase in naso-anal length*



Experiment conducted in the lab of Laurence Legeai-Mallet at Imagine Institute in Paris, France
Daily subcutaneous injection of TYRA-300 at 1.2 mg/kg/day for 15 days starting at Day 1; *p<0.0001

TYRA-300 increased bone growth in FGFR3$^{Y367C/+}$ model

Population

Selectivity

Pre-clinical

Clinical

Increase in length compared to vehicle-treated Y367C/+ mouse[1]

	Dose (mg/kg/day)	Femur	Tibia	L4-L6
TYRA-300	1.2	24.4%*	38.3%*	23.9%*
infigratinib[2]	2.0[2]	20.9%	32.6%	12.1%
infigratinib[3]	0.5[3]	10.4%	16.8%	N/R

*p<0.0001

Experiment conducted in the lab of Laurence Legeai-Mallet at Imagine Institute in Paris, France; data reflects separate experiments for TYRA-300 and infigratinib
1. 15 days subQ starting at day one; 2. Data from Komra-Ebri et al 2016 (Legai-Mallet lab); 3. Demuynck, 2019; 0.667 mg/kg human equivalent dose for 2.058mg/kg; 0.167 mg/kg human equivalent dose for 0.514mg/kg

We plan to initially file a Phase 2 IND in Achondroplasia in 2024

Population

Selectivity

Pre-clinical

Clinical

FGFR3 GERMLINE MUTATIONS

Achondroplasia (~3K)

Hypochondroplasia (~2K)

Craniosynostosis (~2.5K)

Muenke syndrome (~1.4K)

Thanatophoric dysplasia (~0.3K)

Crouzon syndrome with acanthosis nigricans (~0.3K)

SADDAN syndrome (~0.06K)

OTHER GERMLINE MUTATIONS

Leri-Weill Dyschondrosteosis (~30K)

Recessive multiple epiphyseal dysplasia (~0.7K)

Laron Syndrome (Growth Hormone Insensitivity) (~0.2K)

PEDIATRIC SHORT STATURE

Genetic Short Stature (~90K[1])

Idiopathic short stature (~700K[2])

Addressable US pediatric population; Source: company research

1. Represents children ages 4-17 under 3 standard deviations from mean height
2. Represents children ages 4-17 under 2.25 standard deviations from mean height

We’re building a pipeline of differentiated assets

Program	Resistance alteration	Annual US incidence	Lead Optimization	IND-Enabling	Phase 1	Phase 2	Phase 3	Anticipated Milestone
FGFR3: TYRA-300^{Onc}	V555^{GK}	~42K[1]						Complete Phase 1
FGFR3: TYRA-300^{ACH}	G380R[2]	~3K[3]						Submit Phase 2 IND in 2024
FGFR1/2/3: TYRA-200	V565^{GK} N550^{MB}	~6K[1]						Dose first patient in 2H23
FGF19+ / FGFR4	V550^{GK} C552^{CYS}	~9K						Nominate lead candidate
RET	V804^{GK} G810^{SF}	~5K						Nominate lead candidate

SNÅP CHEMISTRY DESIGN

ACH: Achondroplasia, GK: Gatekeeper, CYS: Cysteine Mutant, SF: Solvent Front, MB: Molecular Brake

1. Represents incidence for lead indication and deaths for other solid tumors across all stages of the disease 2. Key activating mutation for ACH
3. Number represents US ACH prevalence rather than incidence