TYRA

Corporate Presentation

As of May 4, 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.

Why invest in TYRA?

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

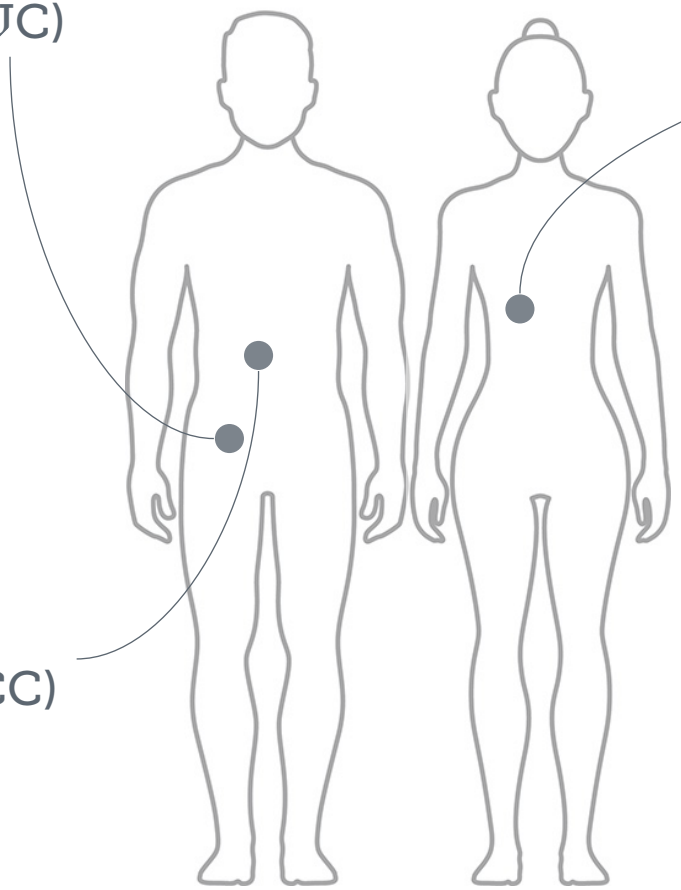
NASDAQ: TYRA

CASH:* $241.7M

*March 31, 2023

Alterations in the FGFR family: a major unmet need

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



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

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

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~40,000/yr (US)

INTRAHEPATIC CHOLANGIOCARCINOMA (ICC)
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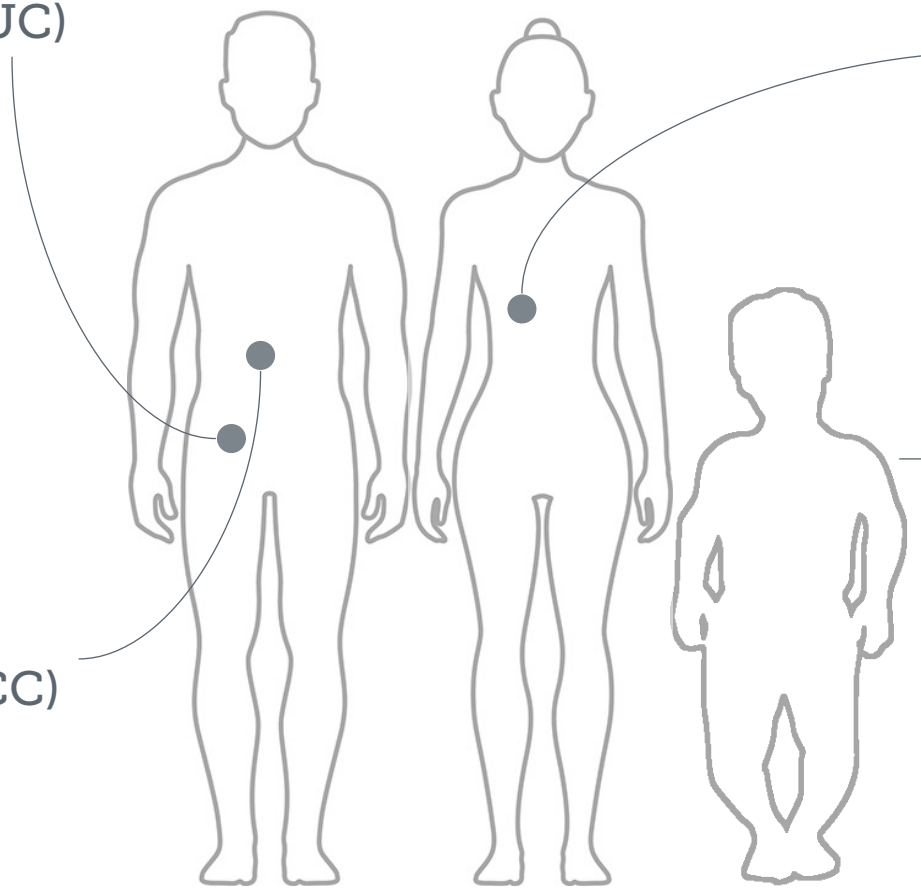


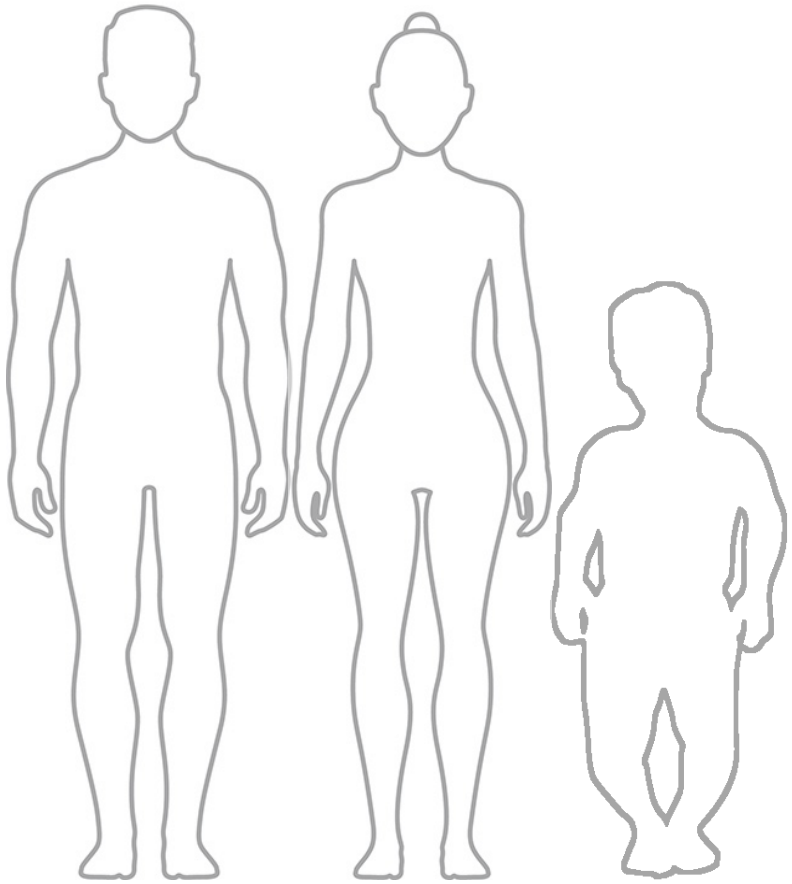
HEPATOCELLULAR CARCINOMA (HCC)
~30% FGF19 (FGFR4/3 ligand)
~9,000/yr (US)

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

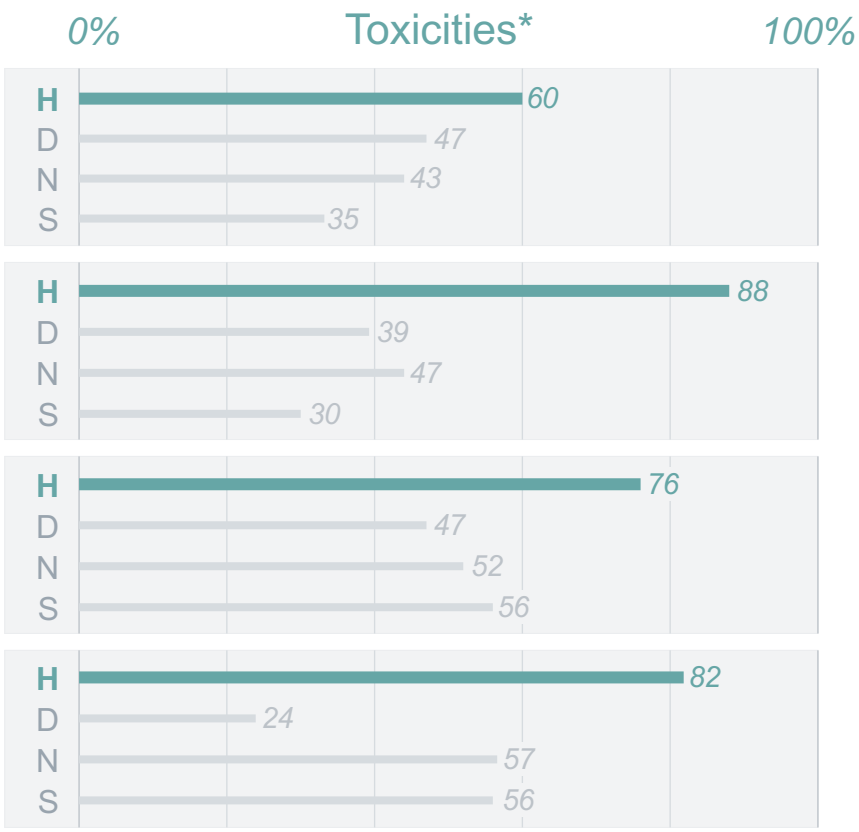
Liabilities

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

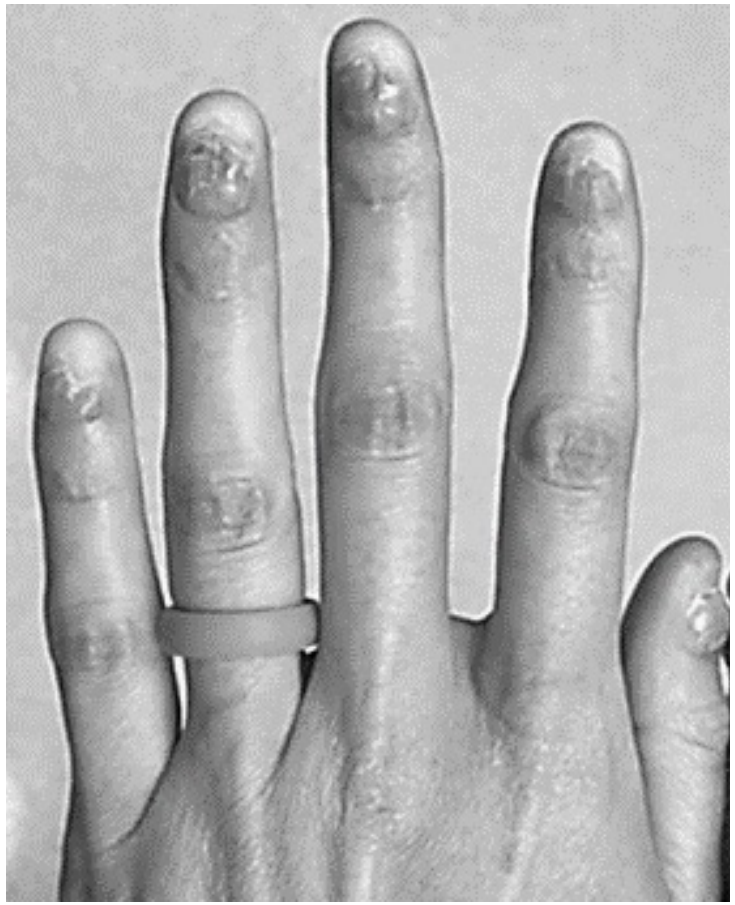
Source: product labels and websites

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

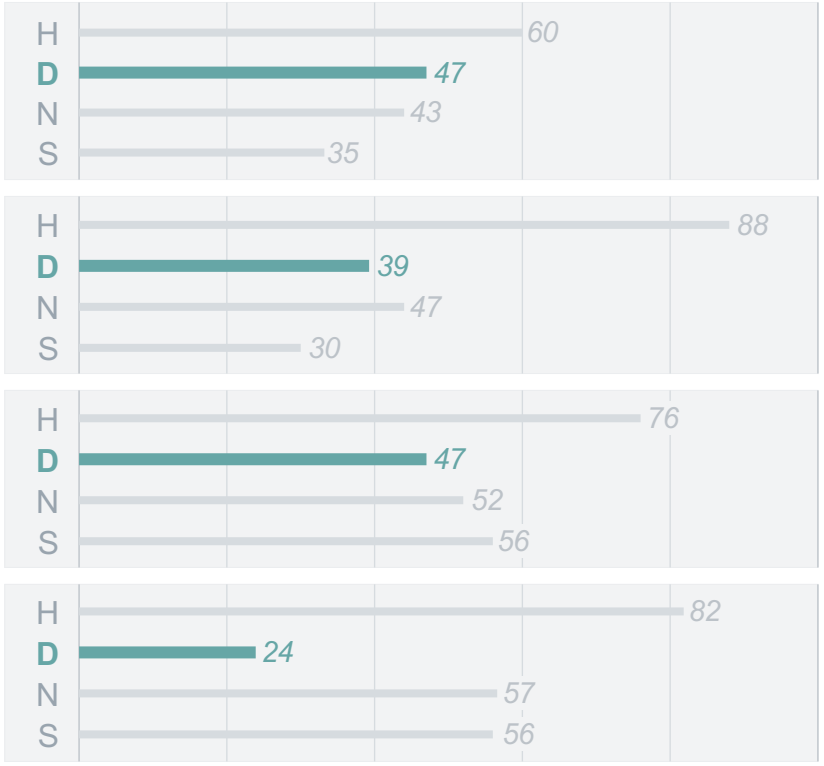
Source: product labels and websites

*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis



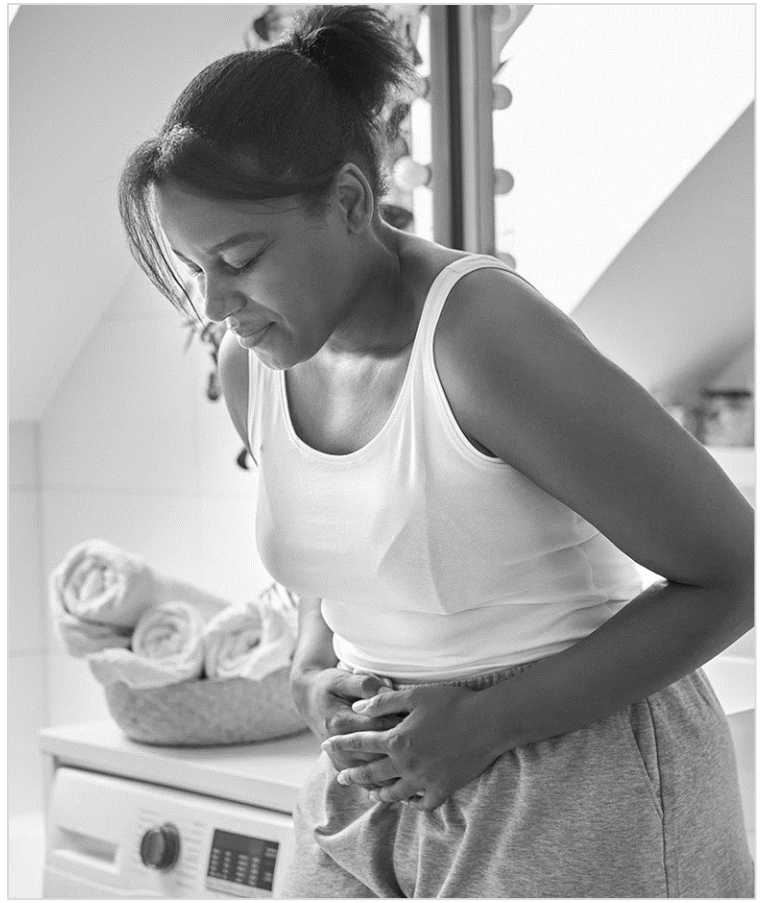
FGFR4 drives gastrointestinal and liver toxicity

	SELECTIVITY		SAFETY
	FGFR	Design	0% Toxicities* 100%
pemigatinib	1–3	Reversible	
futibatinib	1–4	Covalent	
erdafitinib	1–4	Reversible	
infigratinib	1–3	Reversible	



*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis

Source: product labels and websites



FGFR1/2/4 toxicities limit dosing

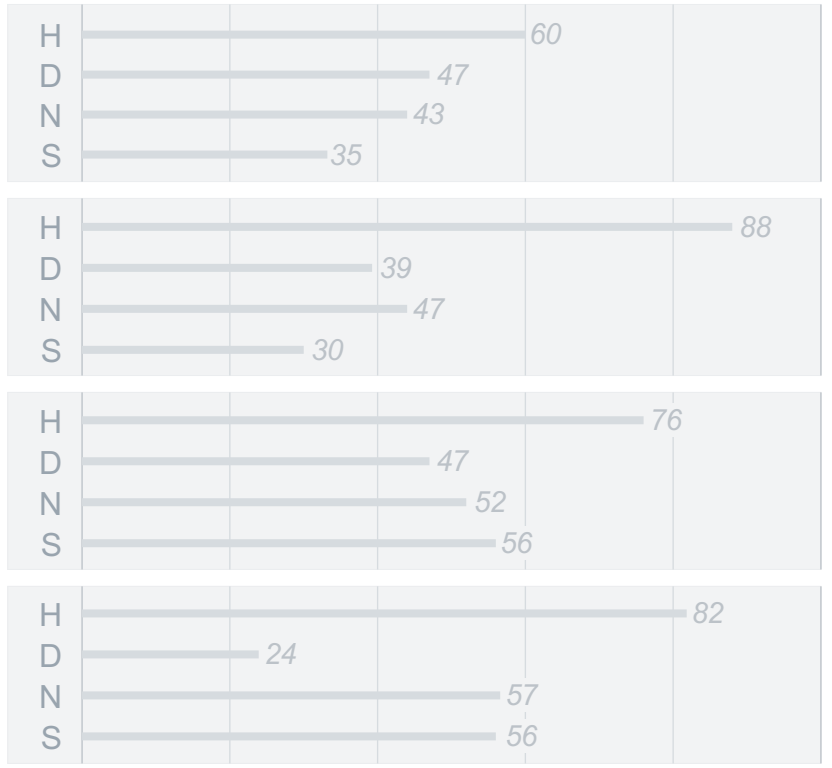
	SELECTIVITY		SAFETY		
	FGFR	Design	0% Toxicities* 100%	Discontinuation/ Reduction	DLT Driver
pemigatinib	1–3	Reversible	H 60, D 47, N 43, S 35	23%	Hyper-phosphatemia (R1)
futibatinib	1–4	Covalent	H 88, D 39, N 47, S 30	63%	AST/ALT increase (R4)
erdafitinib	1–4	Reversible	H 76, D 47, N 52, S 56	66%	Hyper-phosphatemia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

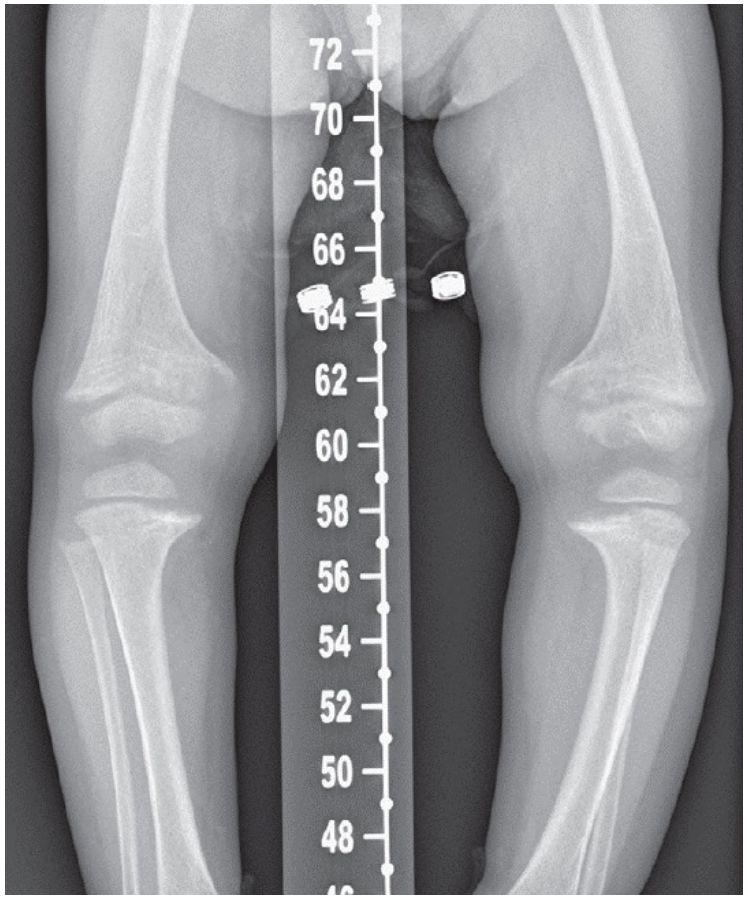
FGFR3 inhibition accelerates bone growth in children

	SELECTIVITY		SAFETY
	FGFR	Design	0% Toxicities* 100%
pemigatinib	1–3	Reversible	
futibatinib	1–4	Covalent	
erdafitinib	1–4	Reversible	
infigratinib	1–3	Reversible	



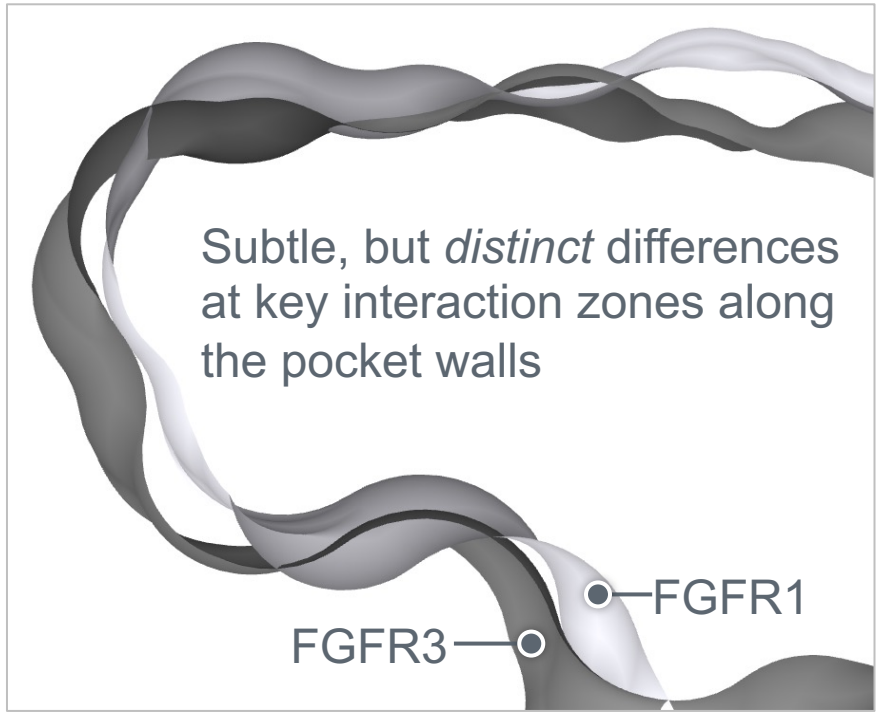
*Hyperphosphatemia *Diarrhea *Nail toxicity *Stomatitis

Source: product labels and websites



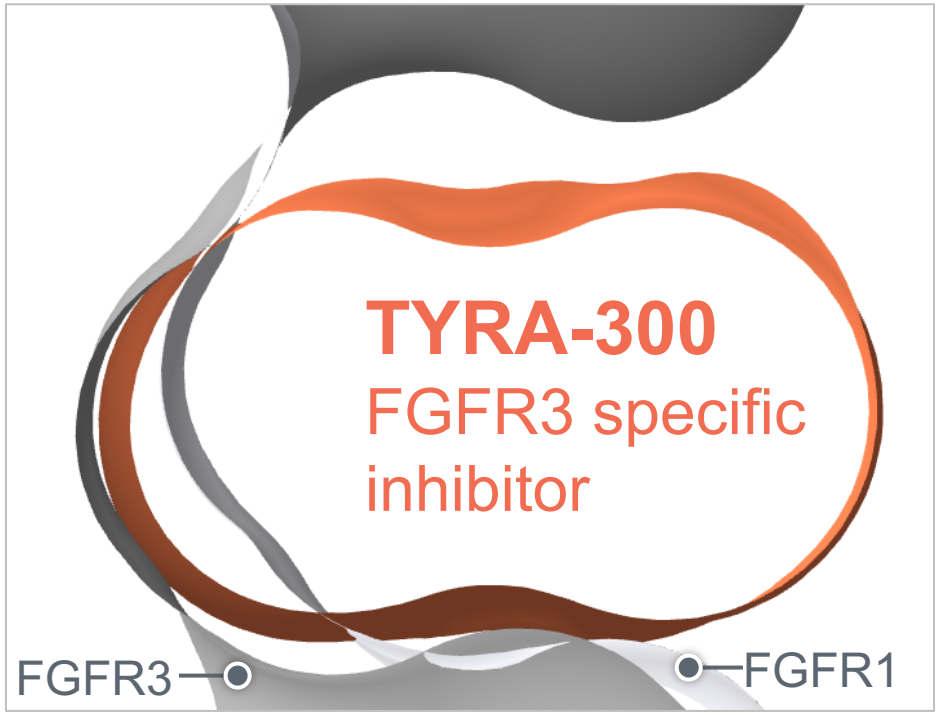
The challenge: FGFR family active sites are nearly identical

FGFR isoform selectivity



MOLECULAR MODEL

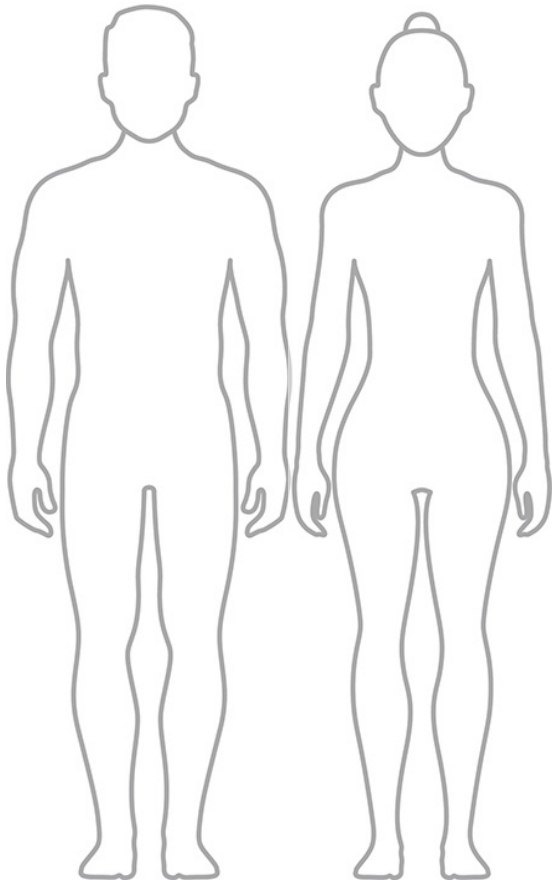
dozens of high-resolution crystal structures



CRYSTALLOGRAPHY

3.0Å cross-sections

Approved FGFR inhibitors have significant liabilities



Approved drugs are pan-FGFR inhibitors

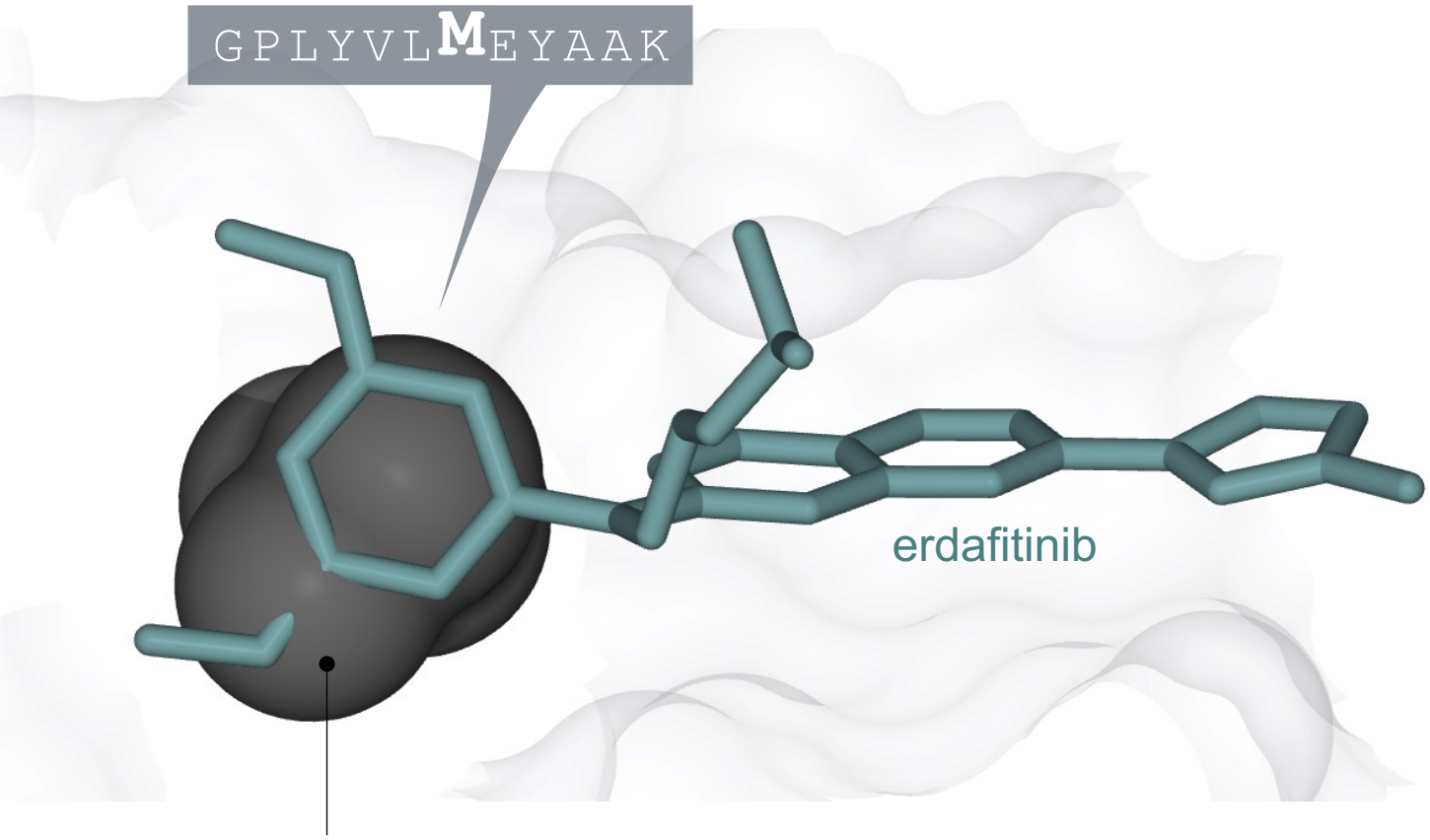
pemigatinib	erdafitinib
futibatinib	infigratinib

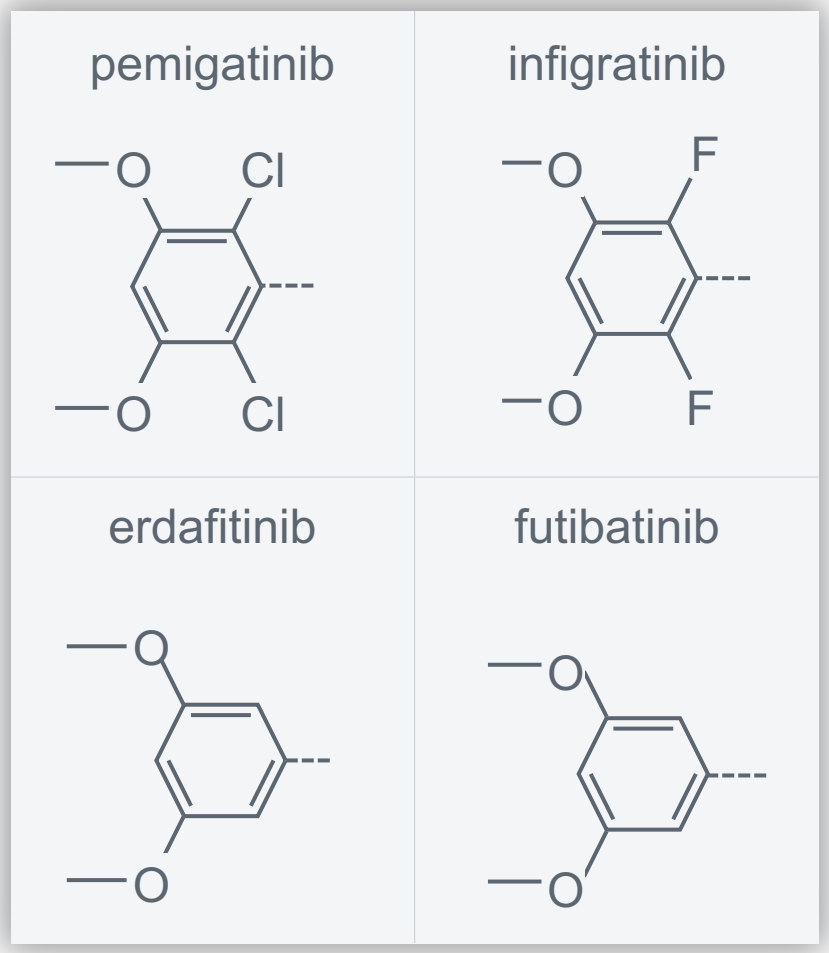
Liabilities

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

Acquired resistance is driven by sequence and structure

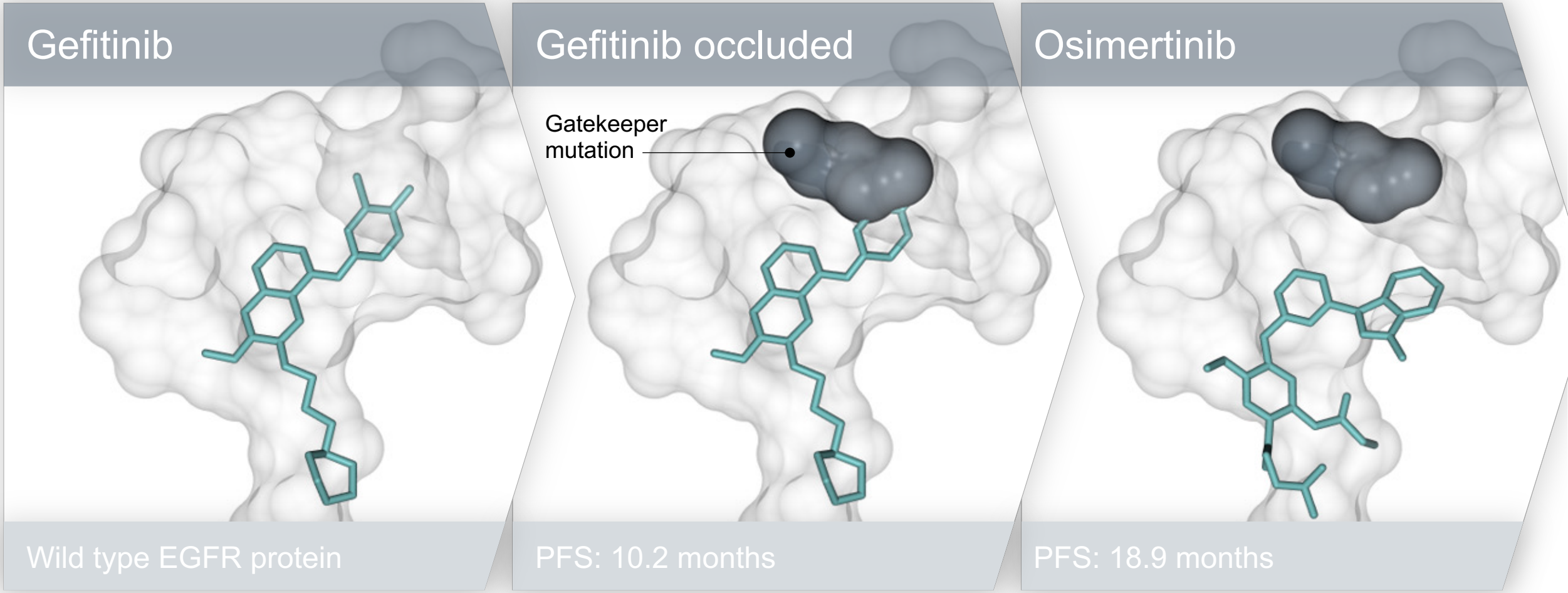
FGFR3 Example



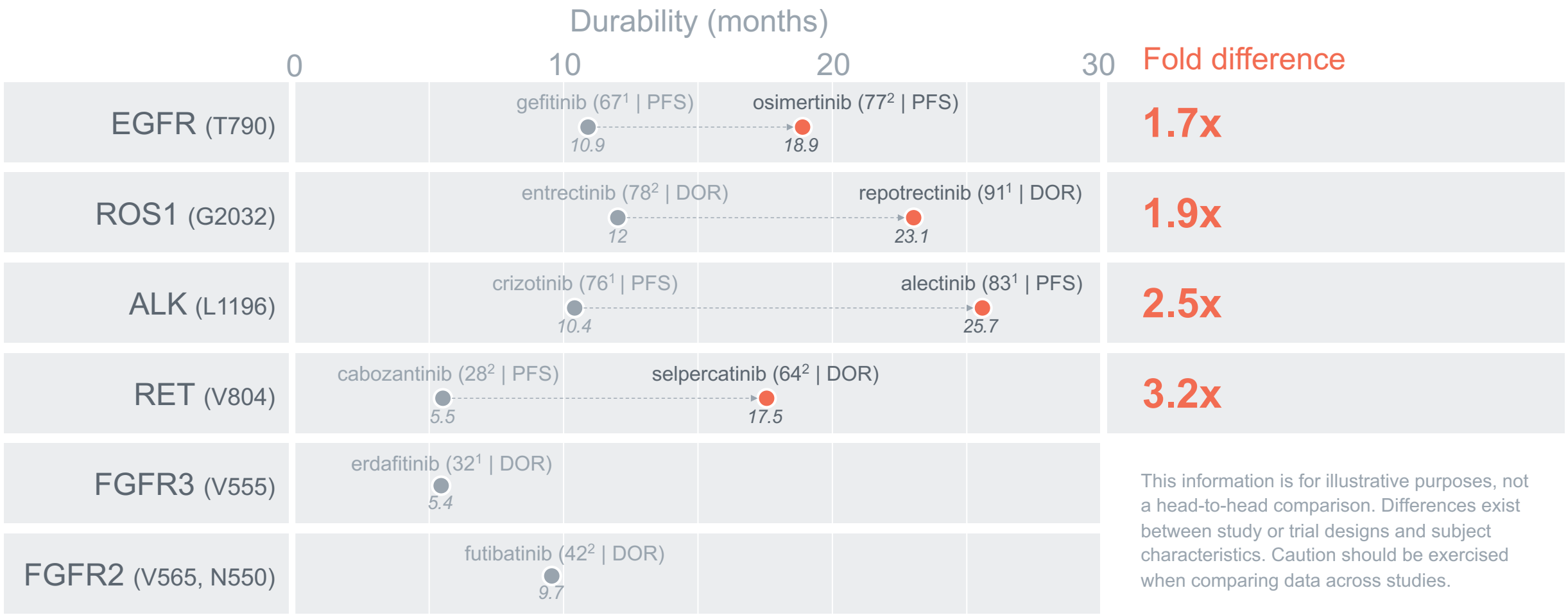


Structural insights provide a rational path to address recurrence

EGFR Example



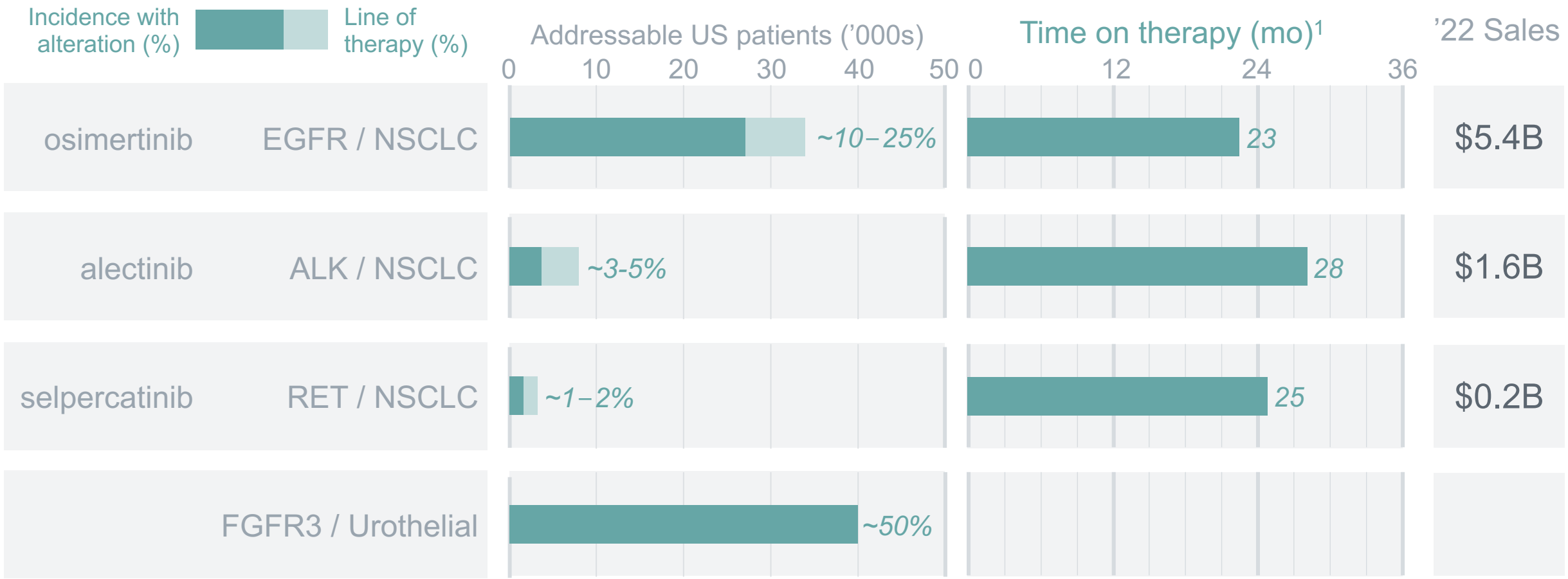
Next gen drugs extend progression free survival



This information is for illustrative purposes, not a head-to-head comparison. Differences exist between study or trial designs and subject characteristics. Caution should be exercised when comparing data across studies.

1. Objective Response Rate (%); 2. Overall Response Rate (%)

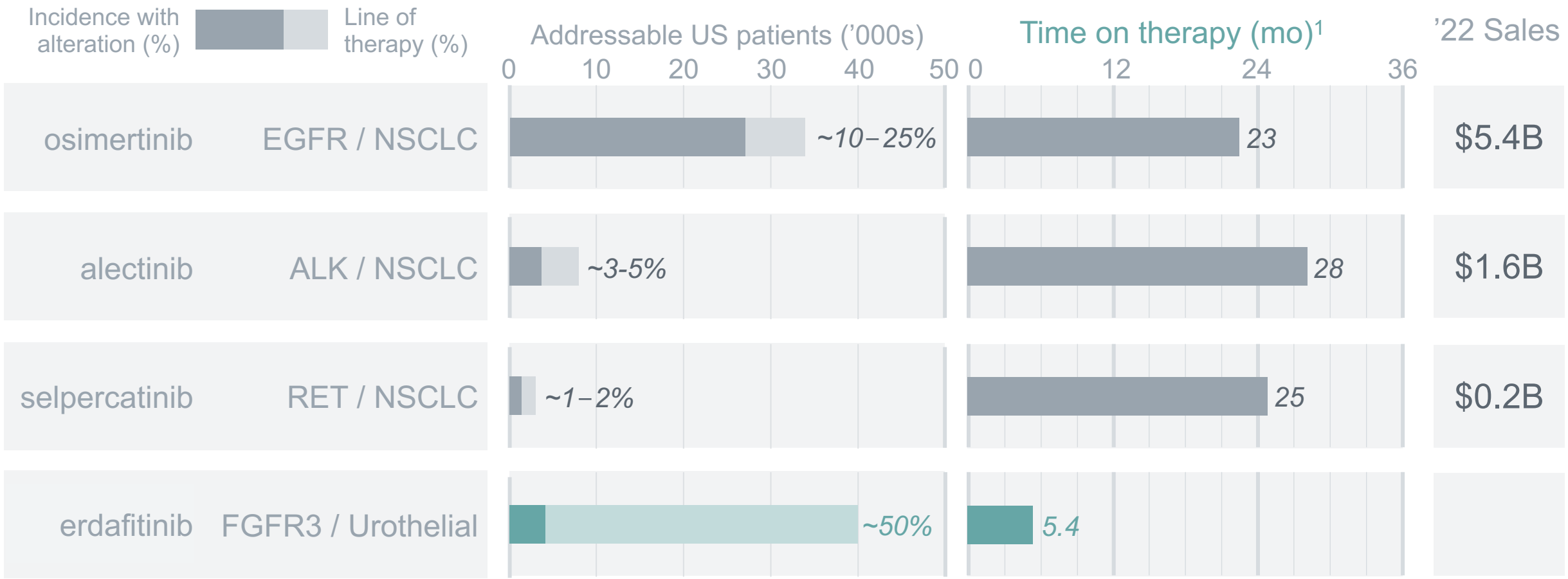
FGFR3 positive urothelial cancer is an outsized opportunity



1. Median duration of exposure for earliest line study

Source: SEER, ACS, NCCN; UpToDate; Company filings; GlobalData; Wu et al, 2020; Zhang et al, 2016; Graham et al., 2018; Mok et al, 2020; Subbiah et al, 2020; Drilon et al, 2023; Kacew et al, 2020; Matulewicz, 2020; Rizzo, 2022; Necchi, 2020

Despite a large opportunity, pan-FGFR inhibitors fall short



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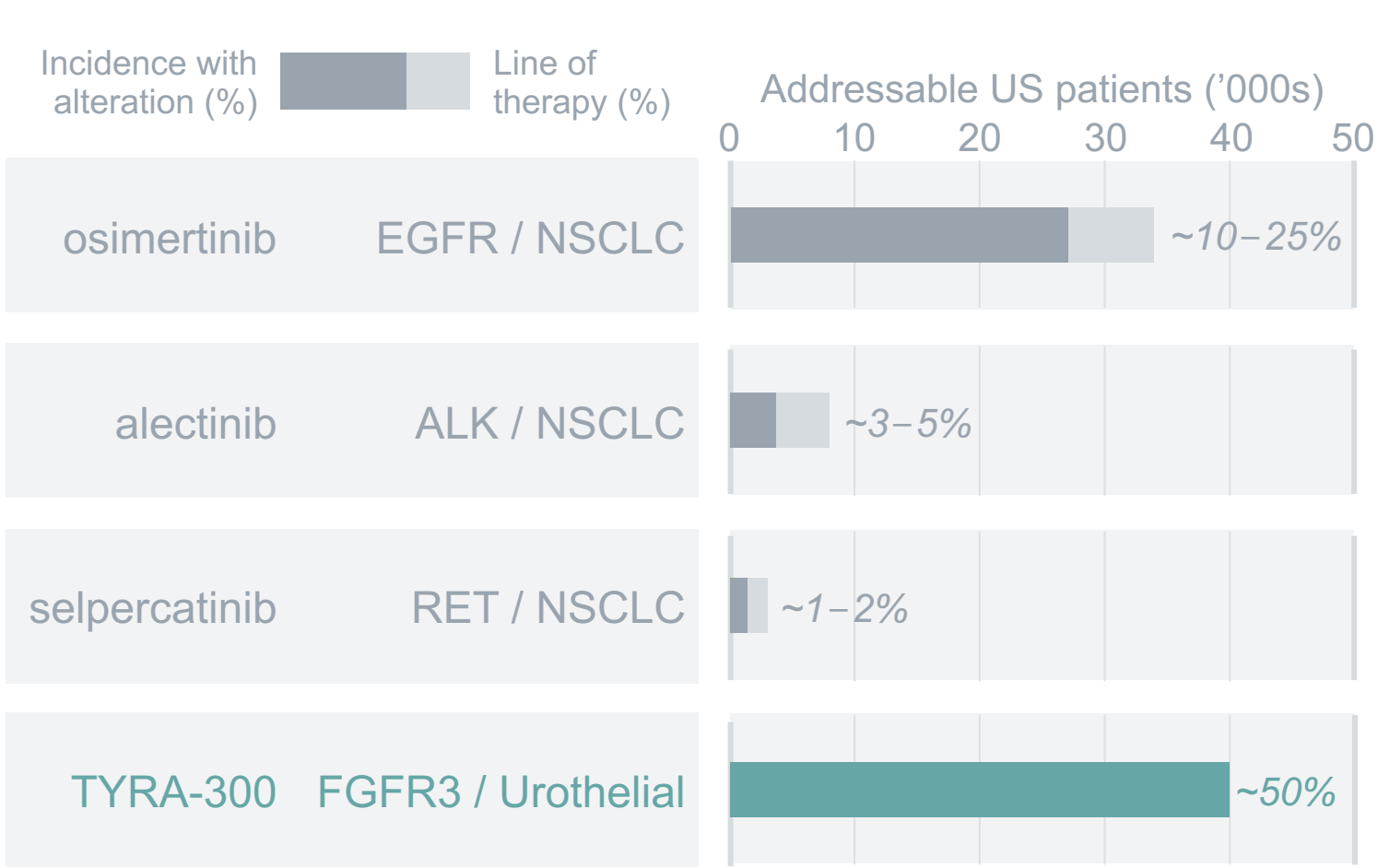
TYRA-300 is designed to address the unmet need



NEXT GEN IMPROVEMENT	
Mutation	**Selectivity**
Gatekeeper	Mutant selectivity
Gatekeeper	ALK selectivity
Gatekeeper	RET selectivity
Gatekeeper	FGFR isoform selectivity

1. Median duration of exposure for earliest line study

Source: SEER, ACS, NCCN; UpToDate; Company filings; GlobalData; Wu et al, 2020; Zhang et al, 2016; Graham et al., 2018; Mok et al, 2020; Subbiah et al, 2020; Drilon et al, 2023; Kacew et al, 2020; Matulewicz, 2020; Rizzo, 2022; Necchi, 2020

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

TYRA

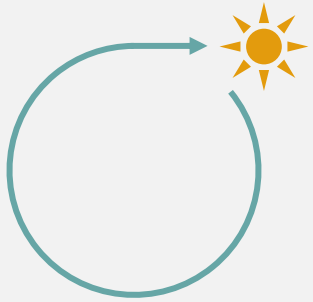
Accelerated design

Differentiated assets

Our unconventional approach accelerates discovery

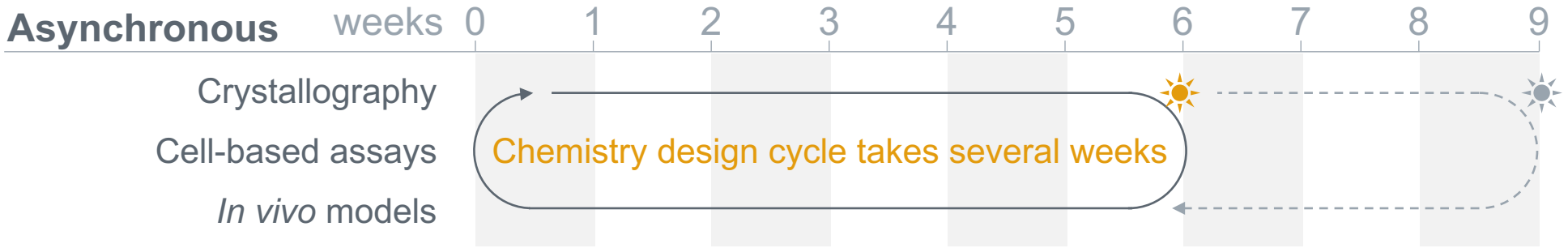
DESIGN CYCLE

Chemistry design

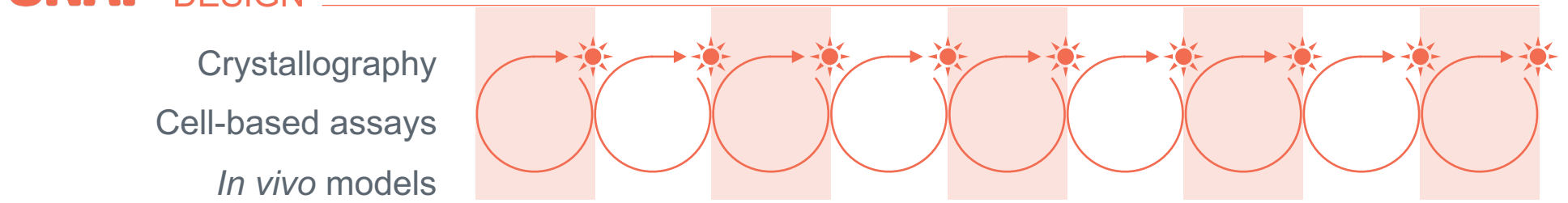


Crystallography
Cell-based assays
In vivo models

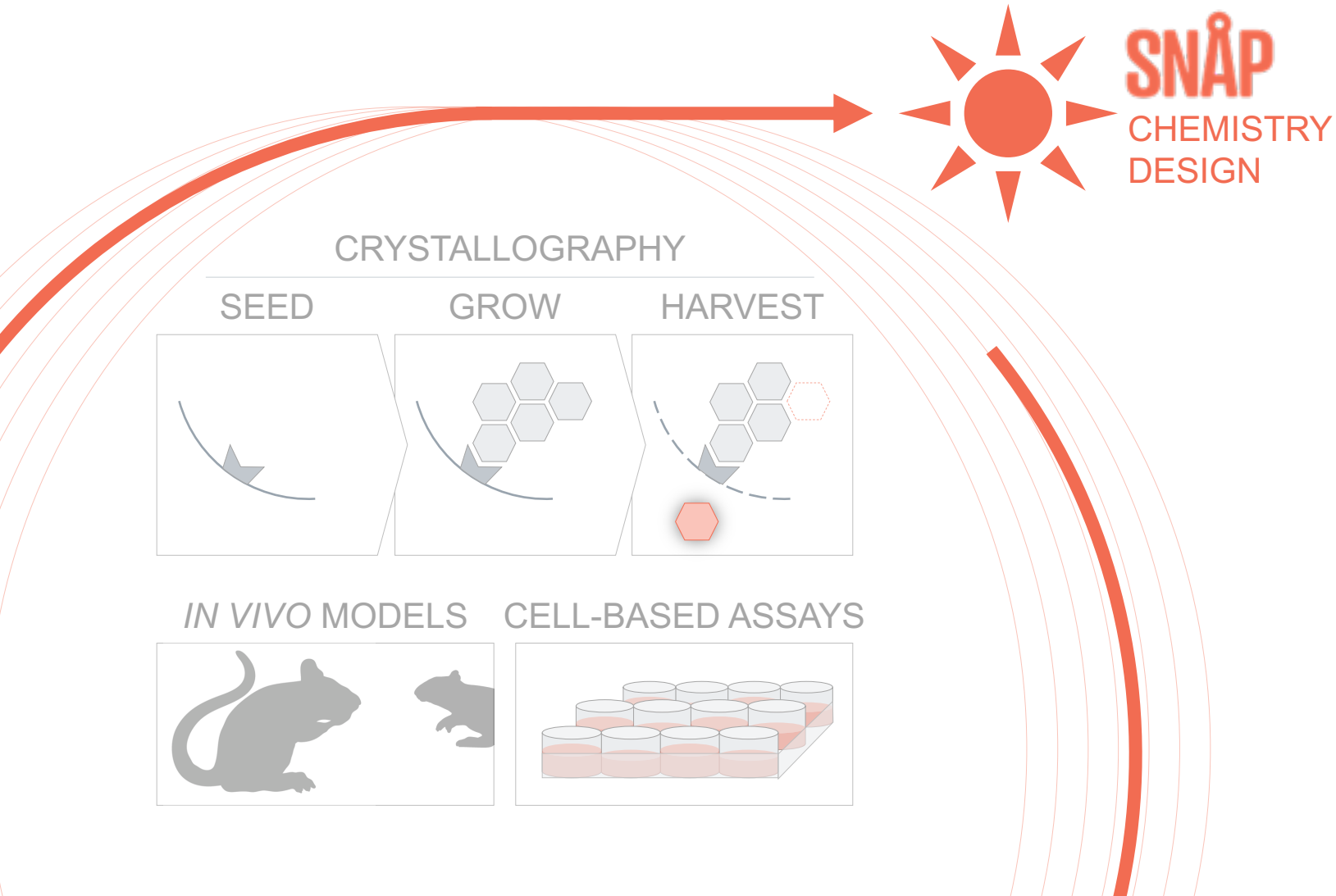
Asynchronous



SNÅP CHEMISTRY DESIGN



We’ve optimized the drug design cycle in-house



CRYSTALLOGRAPHY

New compound to structure in as little as 3 days

CELL-BASED ASSAYS

New compound to cellular data in as little as 2 days

IN VIVO MODELS

New compound to initial PD readout in as little as 5 Days

TYRA

Accelerated design

Differentiated Assets

Selectivity

Resistance

Pre-clinical

Clinical

Population

TYRA-300^{Onc}
Urothelial carcinoma (UC)

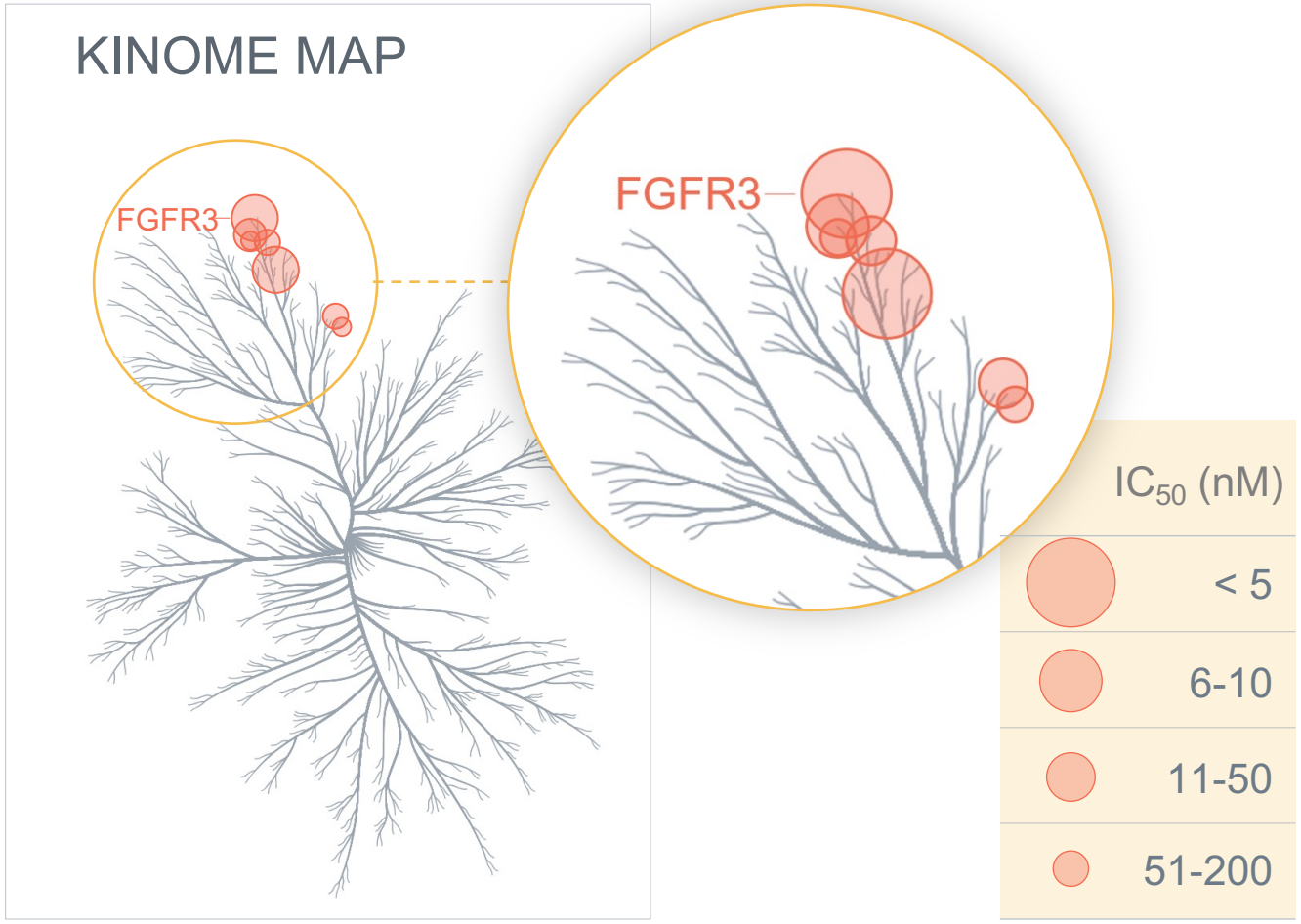
TYRA-300^{ACH}
Achondroplasia (ACH)

TYRA-200
Intrahepatic cholangiocarcinoma (ICC)

- FGFR3-selective
- Gatekeeper agnostic
- Daily oral
- Large population

TYRA-300 is more potent for FGFR3 than other FGFR isoforms

- **Selectivity**
- Resistance
- Pre-clinical
- Clinical
- Population



	TYRA-300	FGFR3 selectivity
FGFR3	1.6	1.0x
FLT4	2.1	1.3x
FGFR2	6.5	4.0x
FGFR4	11.0	6.9x
JAK2	35.5	22x
LTK	65.1	41x
FGFR1	108	68x
FLT1	201	126x
JAK3	206	129x

TYRA-300 was profiled in a scanMAX^{SM} (KINOMEscan) screen, IC_{50} data generated by Reaction Biology Inc.

TYRA-300 is the first FGFR3-selective inhibitor in the clinic

Selectivity

Resistance

Pre-clinical

Clinical

Population

TYRA-300 selectivity vs. approved or late-stage clinical compounds: Ba/F3 Cellular IC_{50} (nM)

	erdafitinib	futibatinib	pemigatinib	infigratinib	TYRA-300
FGFR1	5.5	3.9	12.3	15.3	113
FGFR2	1.8	1.0	4.3	5.8	34.9
FGFR3	1.3	0.8	5.2	6.9	1.8
FGFR4	17.7	6.1	142	459	98.4

Fold Selectivity for FGFR3

FGFR1	4.2x	4.9x	2.4x	2.2x	63x
FGFR2	1.4x	1.3x	0.8x	0.8x	19x
FGFR4	14x	7.6x	27x	67x	55x

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

TYRA-300 did not elevate phosphate relative to erdafitinib

Selectivity

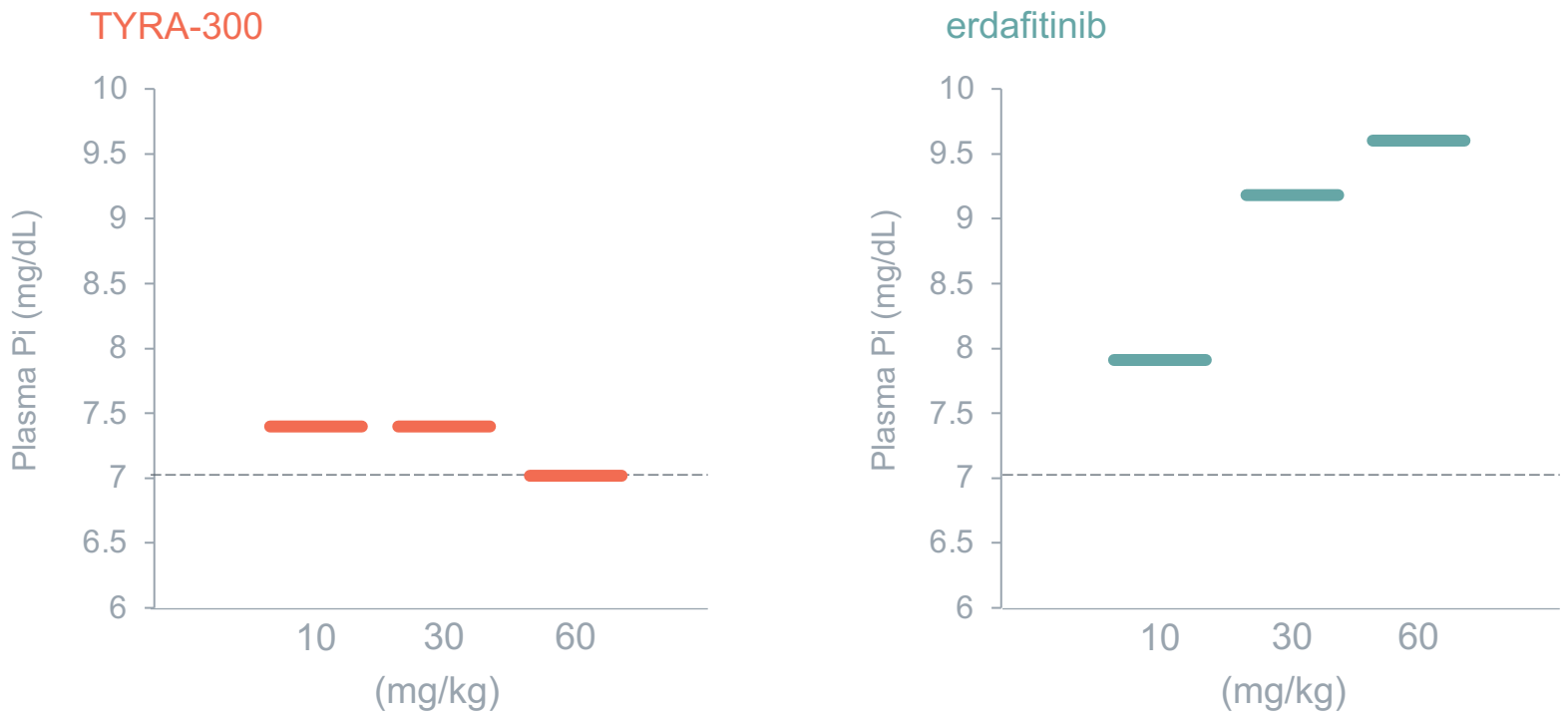
Resistance

Pre-clinical

Clinical

Population

Rat plasma phosphate at 24 hours after single dose[1]



1. N=4 per group, pooled rat plasma; dotted line = pre-dose phosphate value of 3 dose groups

TYRA-300 retains potency against FGFR3 gatekeeper mutations

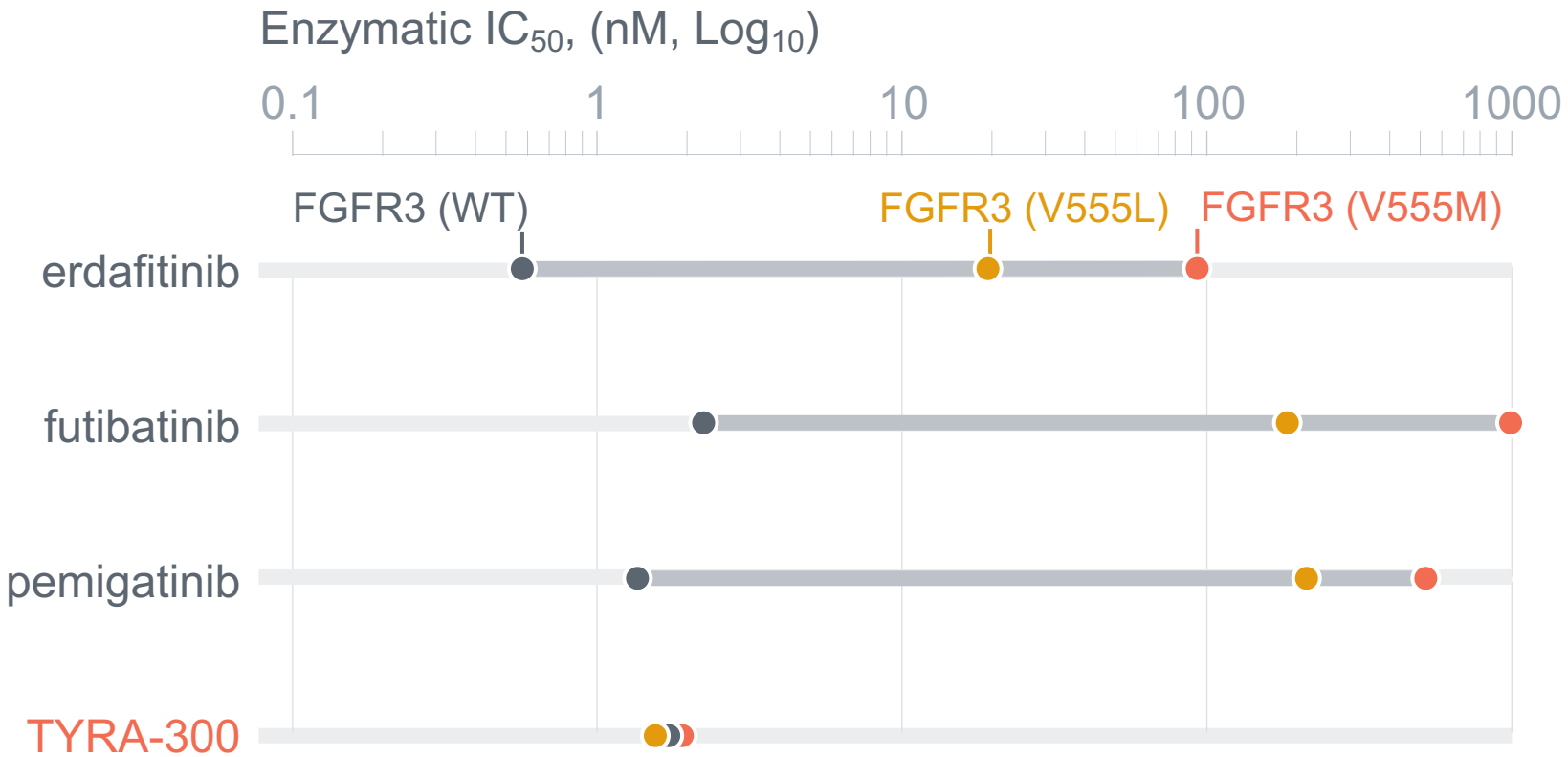
Selectivity

Resistance

Pre-clinical

Clinical

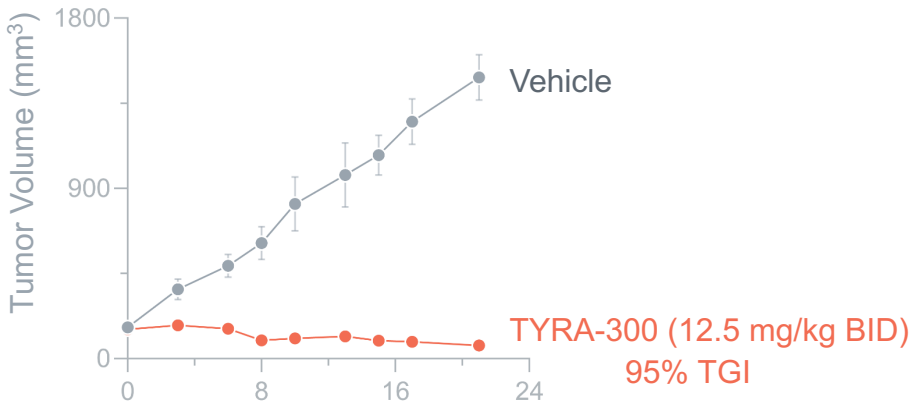
Population

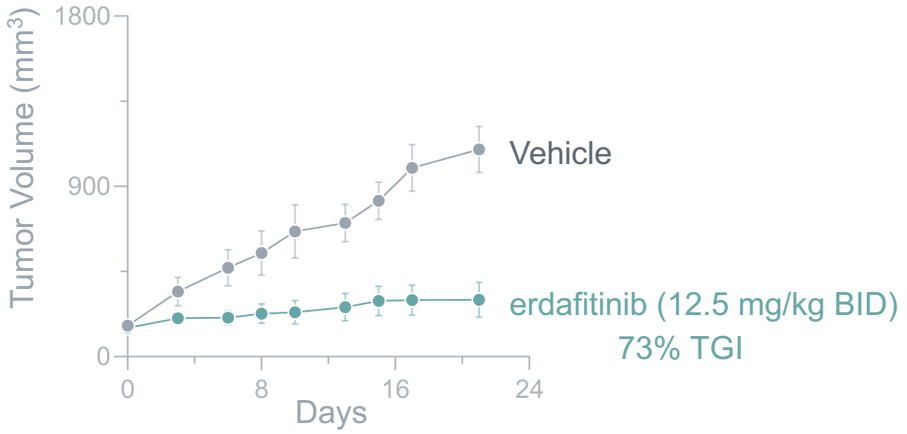


TYRA-300 is active *in vivo* in bladder cancer models

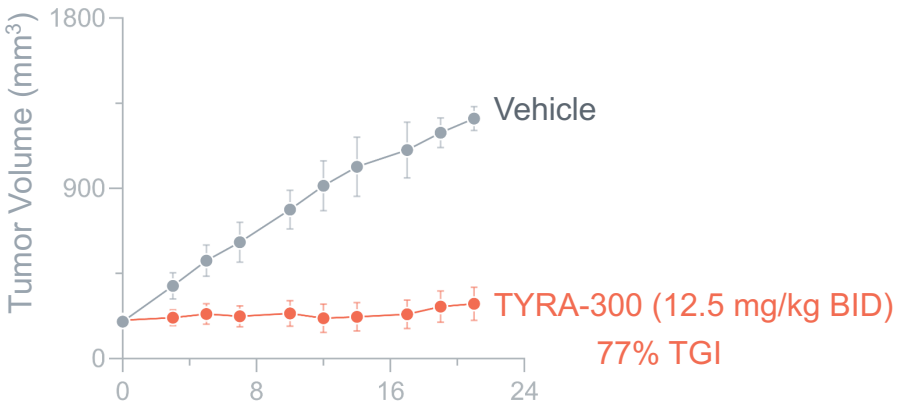
- Selectivity
- Resistance
- **Pre-clinical**
- Clinical
- Population

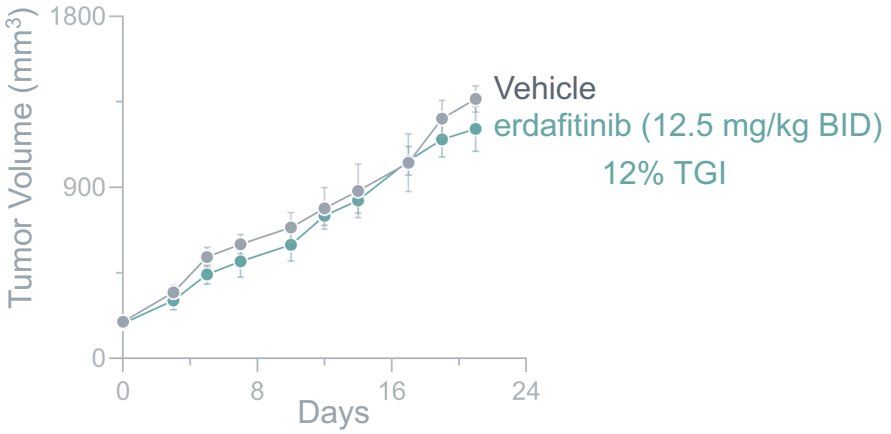
Bladder Cancer Xenograft *FGFR3:TACC3*





Bladder Cancer Xenograft *FGFR3:TACC3-V555M*





TYRA-300 regresses tumors in key urothelial xenografts

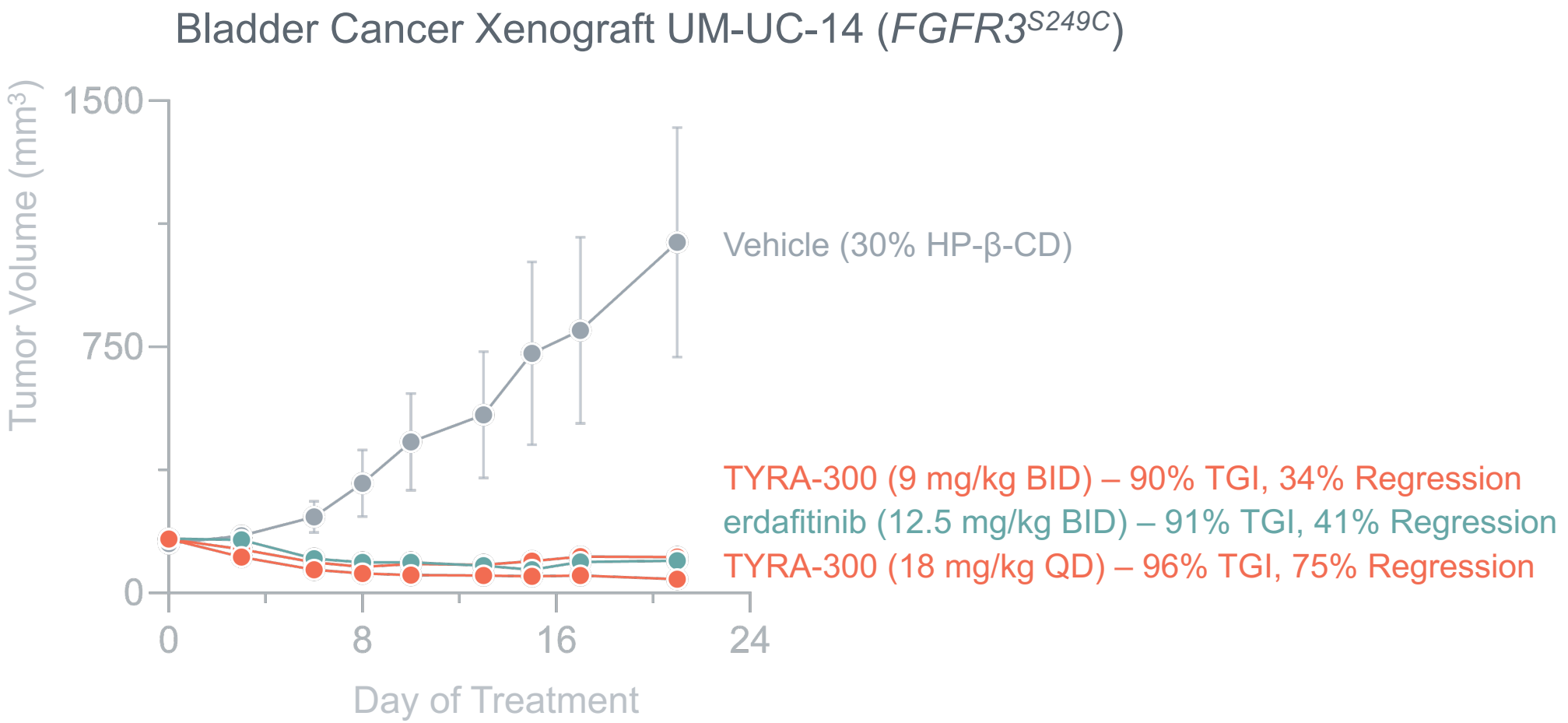
Selectivity

Resistance

Pre-clinical

Clinical

Population



Our Phase 1 trial is designed to identify the optimal RP2D

Selectivity

Resistance

Pre-clinical

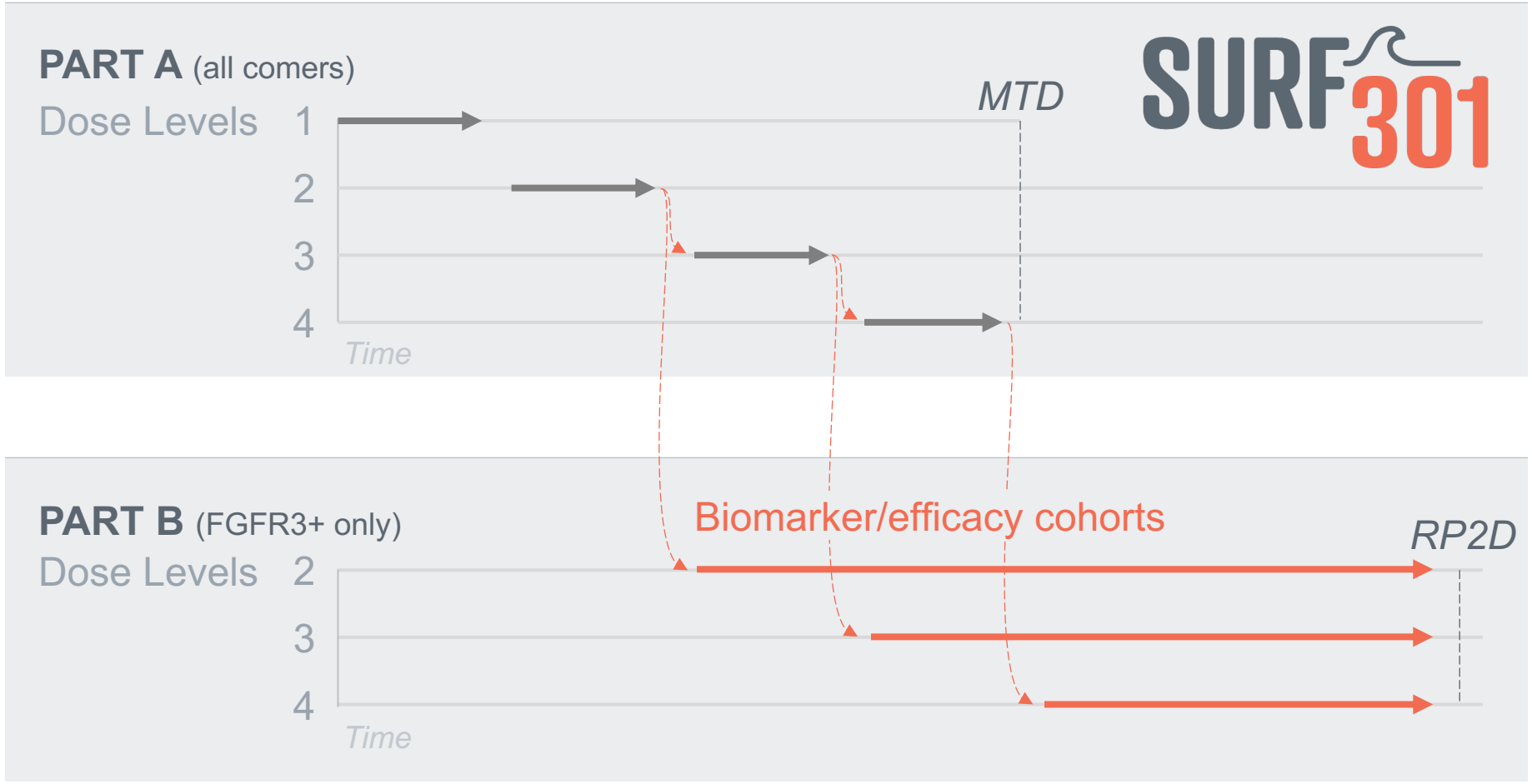
Clinical

Population

What is the MTD?

What is the optimal dose?

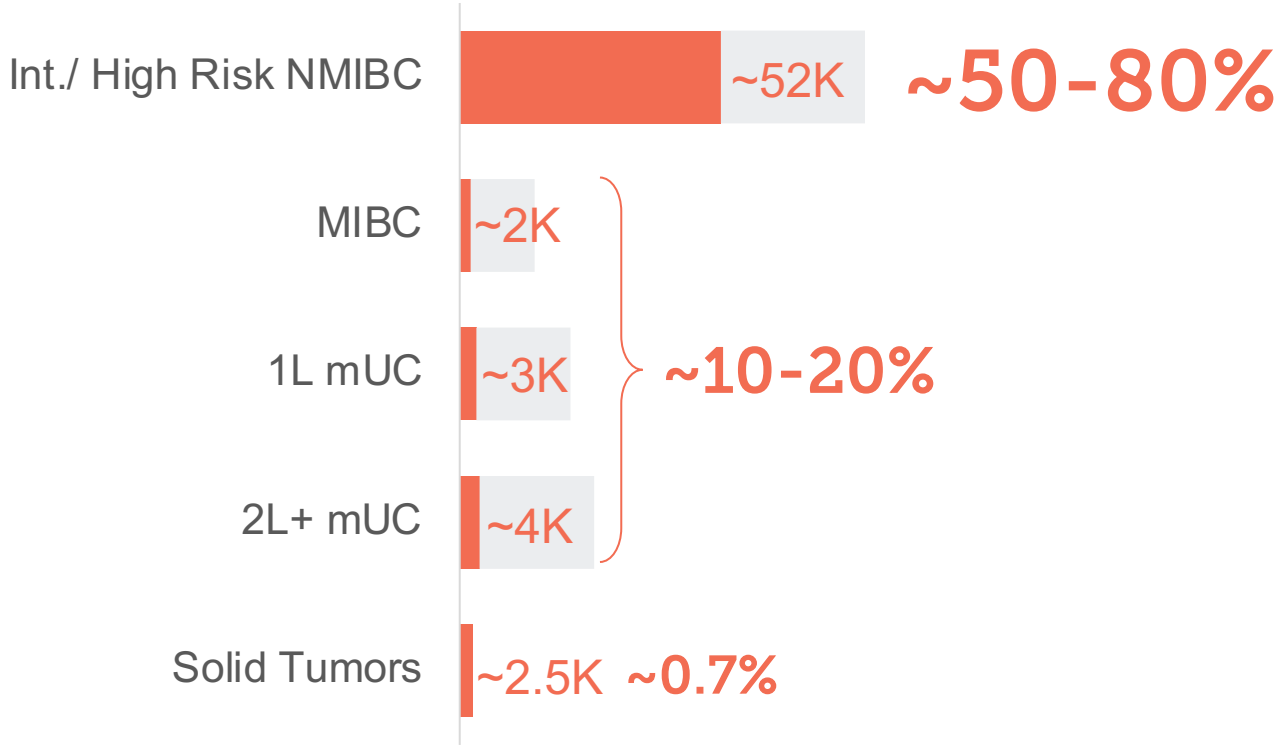
Illustrative



The addressable FGFR3 patient population is large

- Selectivity
- Resistance
- Pre-clinical
- Clinical
- **Population**

Estimated 2022 US FGFR3+ Addressable Population[1]



DRIVER MUTATIONS

S249C,
R248C,
Y373C,
G370C,
FGFR3-TACC3 fusion

CDx

Liquid Biopsy
NGS
Fusion detection

1. Bladder figures represent potential annual diagnosed incident and recurrent case estimates by addressable disease stage; Solid tumors figure based on annual deaths
Source: Clarivate Analytics; Kacew, 2020; Knowles, 2014; Murugesan, 2022

There are high unmet needs in all stages of bladder cancer

Selectivity

Resistance

Pre-clinical

Clinical

Population

ADDRESSABLE (US)[1]		LEAD TX OPTION	UNMET NEED
Int. / High Risk NMIBC	52K	IVE Chemo or BCG	25%–30% recur ≤1yr
BCG Res. NMIBC		Immunotherapy	~30–50% relapse to mUC
MIBC	2K	Neo/adjuvant chemo	
1L mUC	3K	Chemo or PD1 (+ ADC)	Tolerability
2L/3L mUC	4K	erdafitinib or ADC	

UROLOGY

CYSTECTOMY

ONCOLOGY

1. Represents potential annual diagnosed incident and recurrent case estimates by addressable disease stage
Source: Matulewicz, 2020; Mari et al, 2018

Erdafitinib has demonstrated high response rates in UC

Selectivity
Resistance
Pre-clinical
Clinical
Population

	ERDAFITINIB DATA[1]
Int. / High Risk NMIBC	75% 3mo CR[2] (n=8)
BCG Res. NMIBC	100% 3mo CR[3] (n=9)
MIBC	N/A
1L mUC	68% ORR with PD-1[4] (n=19)
2L/3L mUC	32-41% ORR[5]

UNMET NEED

Tolerability

UROLOGY

CYSTECTOMY

ONCOLOGY

1. Data from FGFR3+ mutation or fusion patients 2. Interim data from intermediate risk patients 3. Interim data from BCG-unresponsive patients 4. Preliminary data from cis-ineligible patients in NORSE trial 5. Range from overall population to FGFR3 point mutation population in Erdafitinib label
Source: Daneshmand, 2023 (ASCO GU); Catto, 2023 (ASCO GU); Powles, 2021 (ESMO); Erdafitinib label; Matulewicz, 2020; Mari et al, 2018

Population

Selectivity

Pre-clinical

Clinical

TYRA-300^{Onc}
Urothelial carcinoma (UC)

TYRA-300^{ACH}
Achondroplasia (ACH)

TYRA-200
Intrahepatic cholangiocarcinoma (ICC)

FGFR3-selective

Daily oral

Rationale for additional indications

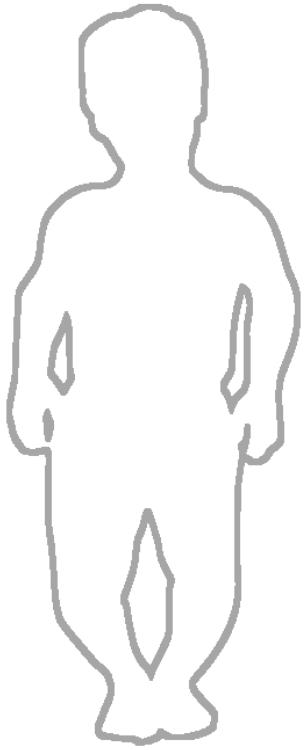
FGFR3 aberrations drive >97% of pediatric achondroplasia (ACH)

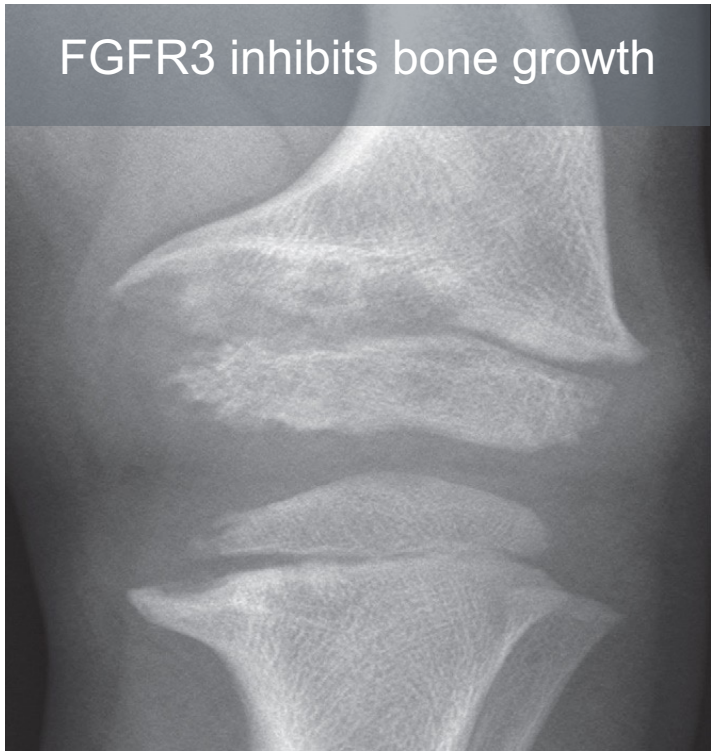
- Population
- Selectivity
- Pre-clinical
- Clinical

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	Cranial or spinal stenosis, hydrocephalus, CV events and sleep apnea

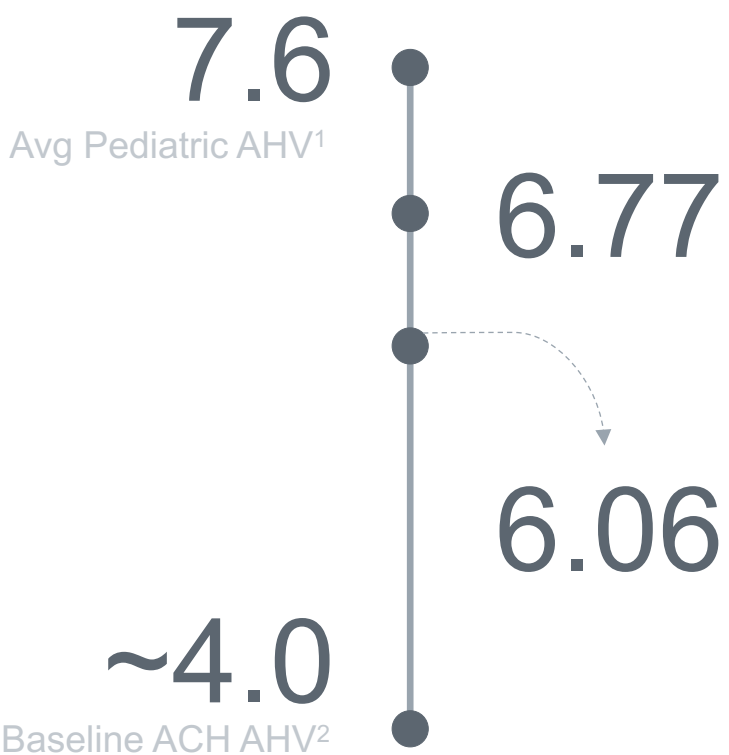
Daily Injectable
VOXZOGO

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

Mean Annualized Height Velocity (AHV) in cm/year



bridgebio

infigratinib
Oral FGFR1-3 Inhibitor
Phase 3 Initiated

Phase 2 Cohort 5 Data[4]
0.250mg/kg daily oral
26 wks, Ages 5-11
N=10

BIOMARIN®

VOXZOGO
SubQ CNP Analog
On market

Phase 2 Cohort 3 Data[3]
15µg/kg Daily SubQ
26 wks, Ages 6-11
N=10

1. Merck Manuals 2. P2 baseline AHV ranges from 3.73 in infigratinib P2 cohort 5, to 4.01 in infigratinib P2 cohort 4, to 4.04 in vosoritide P2 cohort 3; P3 cohort ranges from 4.06 (placebo arm) to 4.26 (vosoritide arm) in VOXZOGO label; 3. Savarirayan, 2019; 4. BridgeBio corporate presentation March 6, 2023;

TYRA-300 is more selective for FGFR3 than infigratinib

Population

Selectivity

Pre-clinical

Clinical

TYRA-300 selectivity vs. approved or late-stage clinical compounds: Ba/F3 Cellular IC_{50} (nM)

	erdafitinib	futibatinib	pemigatinib	infigratinib	TYRA-300
FGFR1	5.5	3.9	12.3	15.3	113
FGFR2	1.8	1.0	4.3	5.8	34.9
FGFR3	1.3	0.8	5.2	6.9	1.8
FGFR4	17.7	6.1	142	459	98.4

Fold Selectivity for FGFR3

FGFR1	4.2x	4.9x	2.4x	2.2x	63x
FGFR2	1.4x	1.3x	0.8x	0.8x	19x
FGFR4	14x	7.6x	27x	67x	55x

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

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

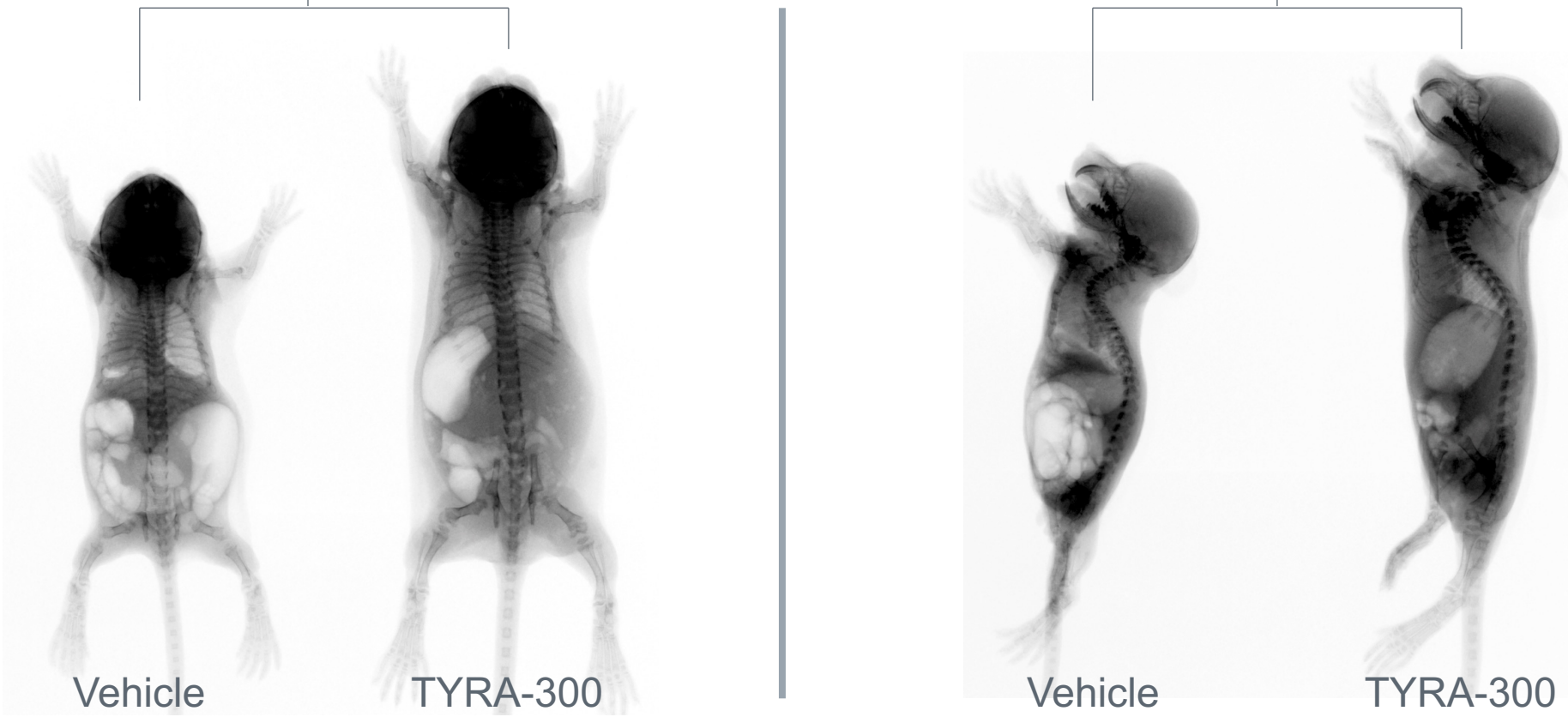
Population

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

Selectivity

Resistance

Pre-clinical

Population

Clinical

TYRA-300^{Onc}
Urothelial carcinoma (UC)

TYRA-300^{ACH}
Achondroplasia (ACH)

TYRA-200
Intrahepatic cholangiocarcinoma (ICC)

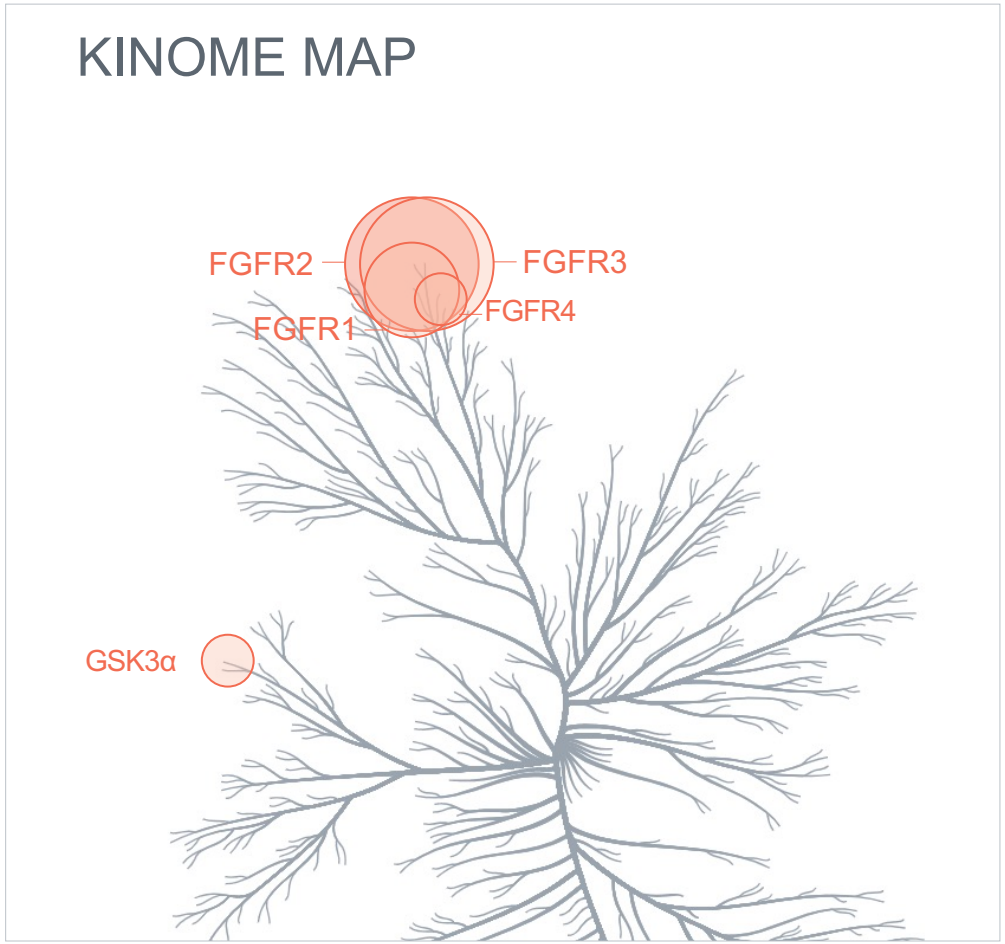
Gatekeeper and molecular brake agnostic

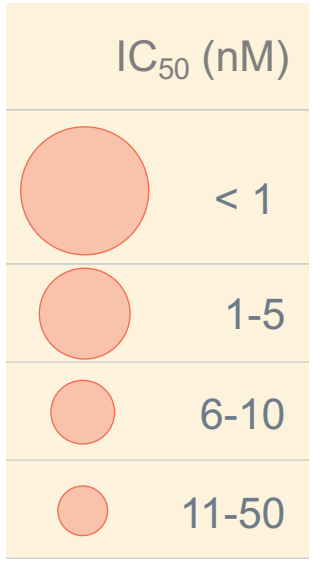
FGFR4-sparing

Gateway to additional solid tumor indications

TYRA-200 is highly selective for FGFR1/2/3, and spares FGFR4

- Selectivity
- Resistance
- Pre-clinical
- Population
- Clinical





	TYRA-200	FGFR2 selectivity
FGFR2	0.47	1.0x
FGFR3	0.66	1.4x
FGFR1	1.8	3.8x
FGFR4	30.5	65x
GSK3α	35.6	76x

TYRA-200 was profiled in a scanMAX℠ (KINOMEscan) screen, IC50 data generated by Reaction Biology Inc.

Polyclonal acquired drug resistance occurs often in FGFR2

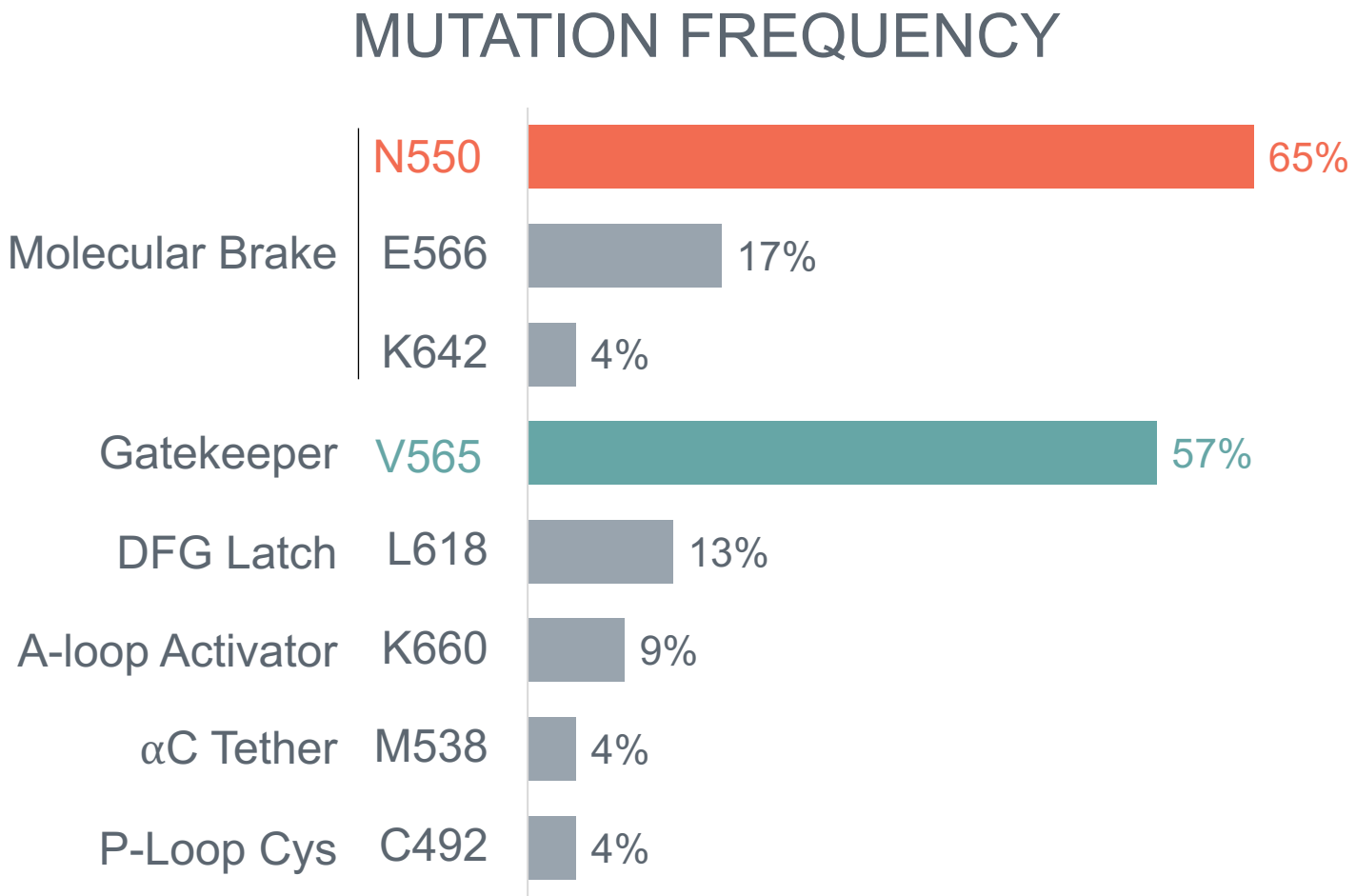
Selectivity

Resistance

Pre-clinical

Population

Clinical



Data presented at EORTC (October 2020); N=46. Evaluable FGFR2-fusion Patients (ICC) on FGFR therapy w/ post-progression biopsy

TYRA-200 retains potency for key acquired resistance mutations

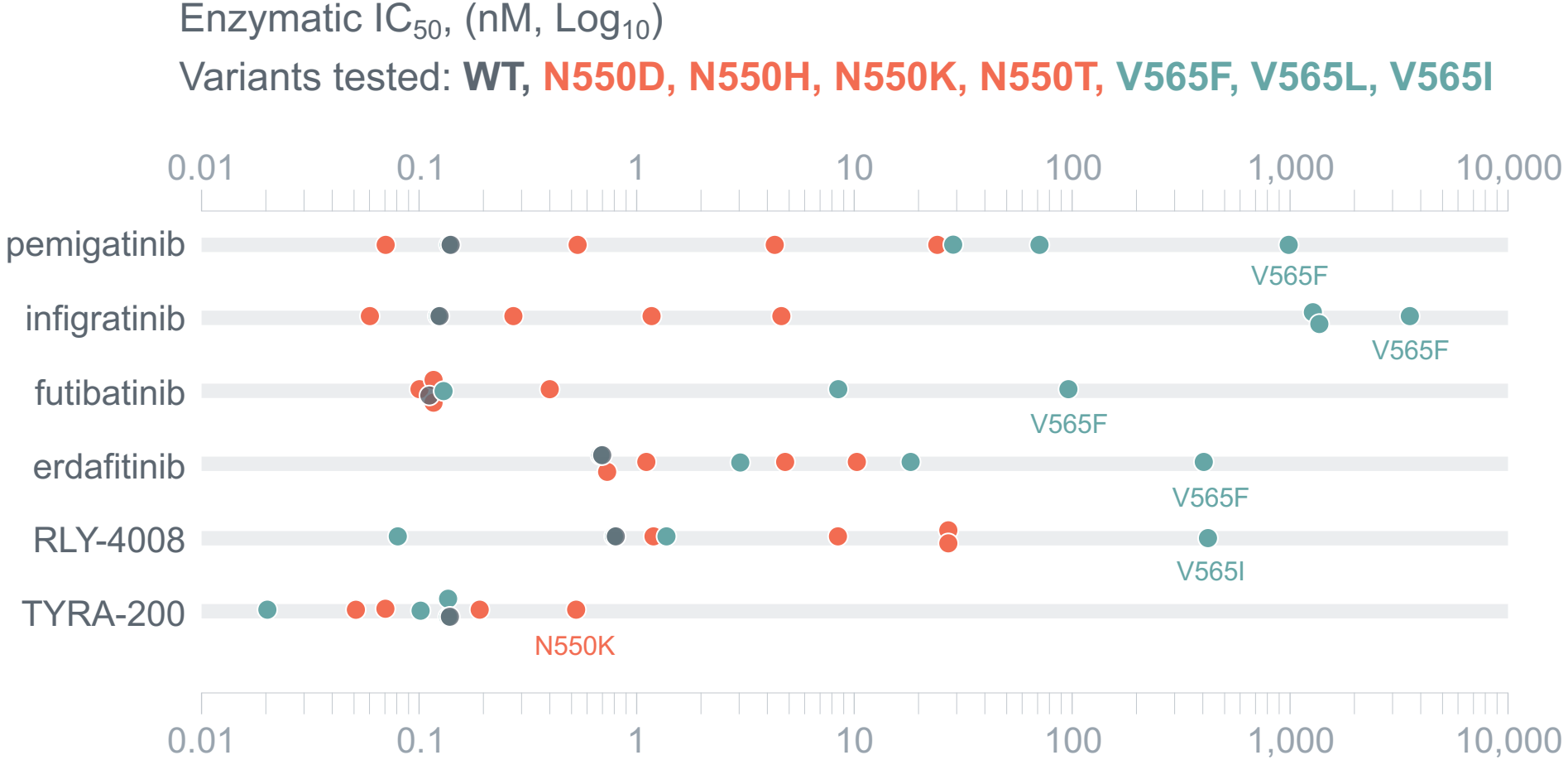
Selectivity

Resistance

Pre-clinical

Population

Clinical



Enzymatic IC50 measurements generated at Reaction Biology Corp using Tyra enzymes.
All experiments conducted under identical conditions, tested in duplicate.

TYRA-200 regresses tumors with gatekeeper mutations

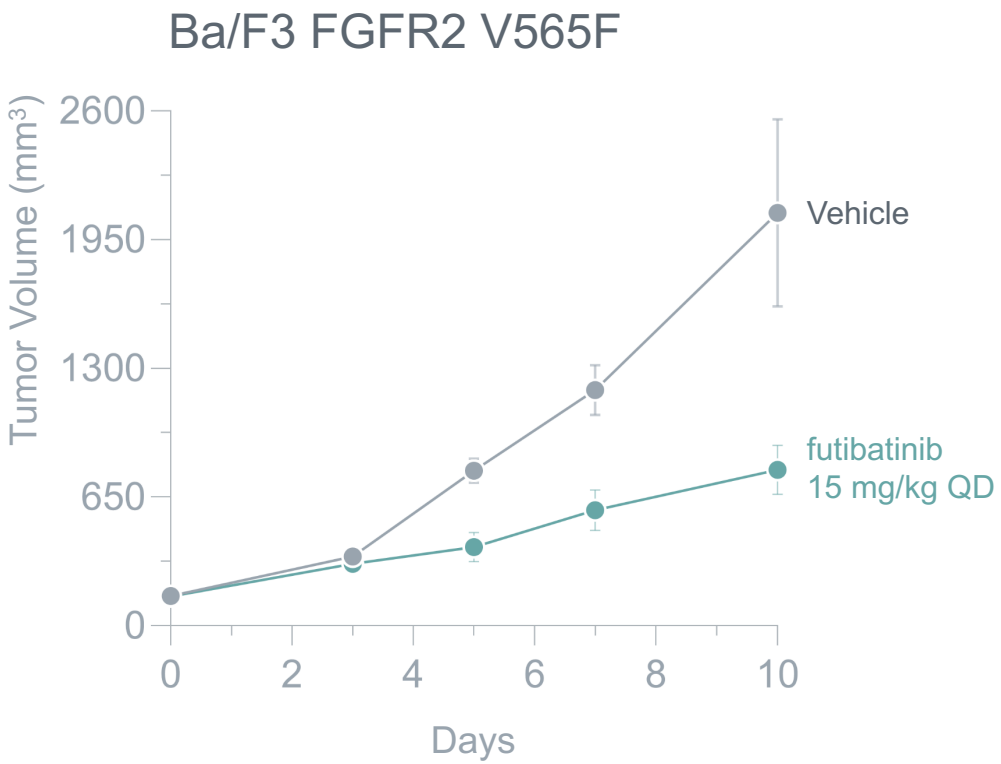
Selectivity

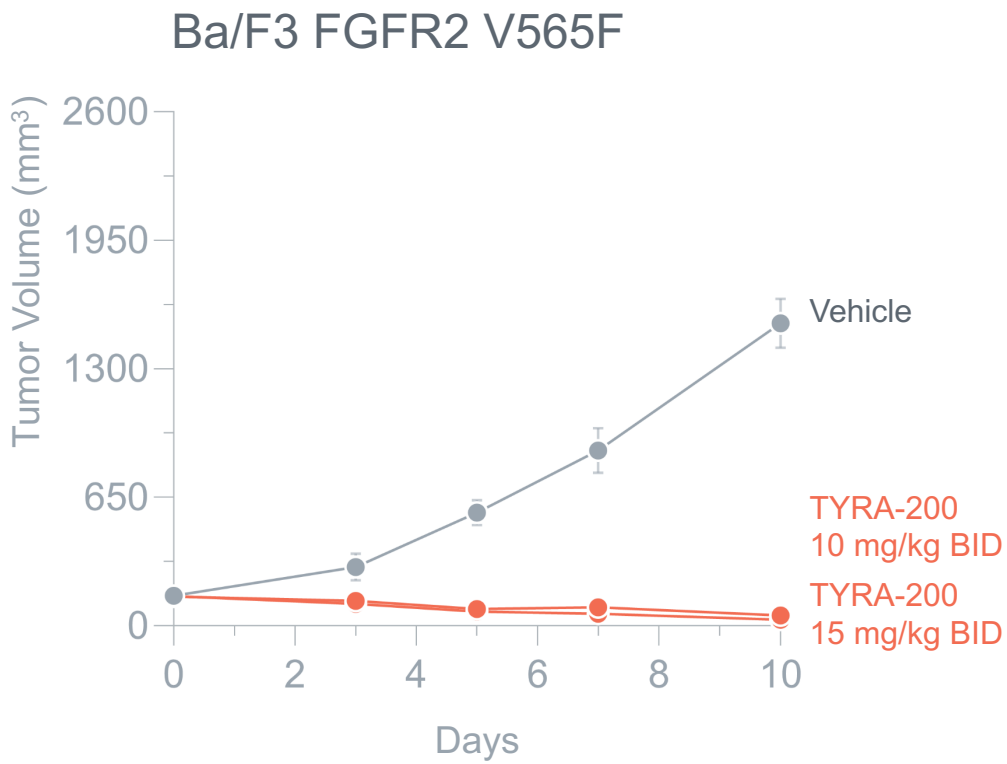
Resistance

Pre-clinical

Population

Clinical





TYRA-200 has multiple opportunities across FGFR2+ solid tumors

Selectivity

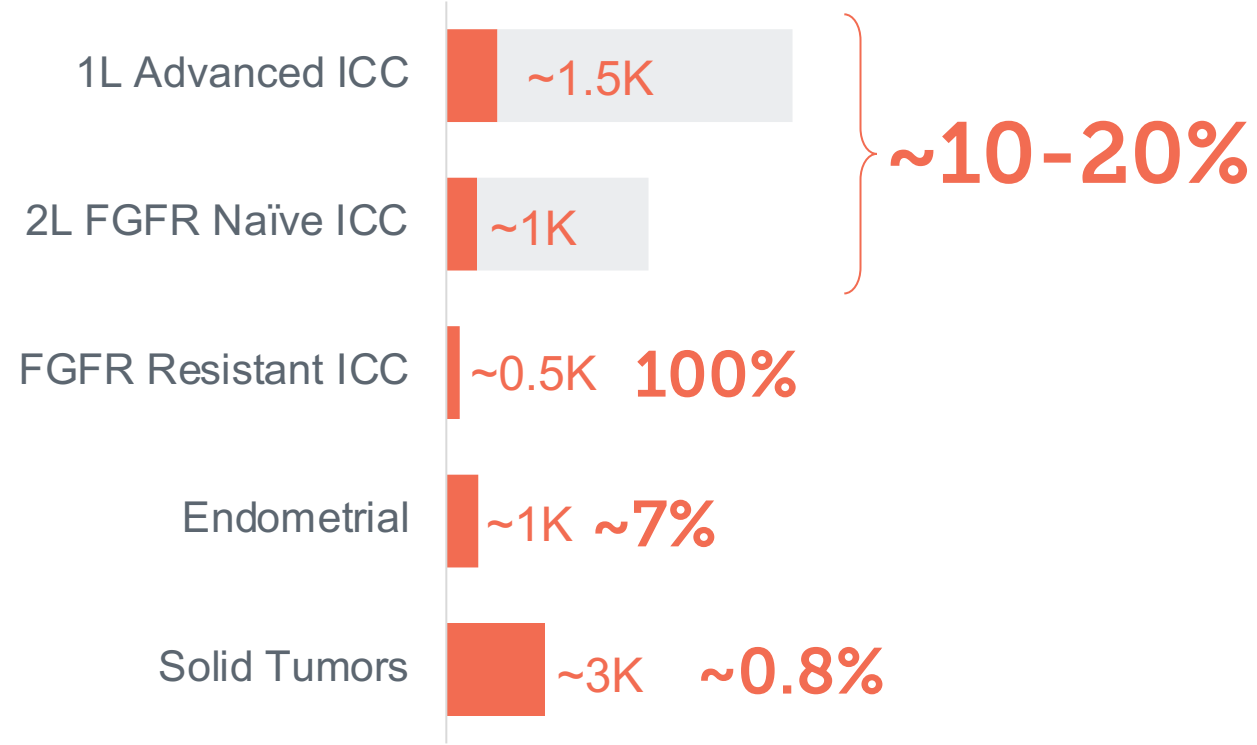
Resistance

Pre-clinical

Population

Clinical

Estimated Potential Annual US FGFR2+ Addressable Population[1]



Driver mutations

Rearrangement+,
$N550^{MB}$,
K650E,
S252W,
Y375C,
C382R

1. ICC figures represent potential annual incident and recurrent case estimates by addressable disease stage; Solid tumors figure based on annual deaths
Source: SEER; Cleary, 2021; Conway, 2022; Oh, 2022; Goyal, 2020; Murugesan, 2022; Company Research

Acquired resistance is a key unmet need in FGFR2+ ICC

Selectivity

Resistance

Pre-clinical

Population

Clinical

	ADDRESSABLE (US)[1]	LEAD OPTION	UNMET NEED
1st Line	~1.5K	CPI + Chemo	Only ~27% of patients respond; Increased PFS (Durva+Gem/Cis: 7.2mo[2])
2nd Line	~1K	FGFR2 Inhibitors	Increased PFS (futibatinib: 8.9mo[3]) ~67% of FGFR2i responders relapse with resistance mutations[4]
3rd Line	~0.5K	Chemo or palliative	Polyclonal resistance; need for gatekeeper and molecular brake-agnostic approach

1. Represents estimated potential annual incident and recurrent case estimates by addressable disease stage 2. Oh et al, 2022; 3. Data presented at at ASCO (June 2022); N=103; 4. Data presented at EORTC (October 2020); N=46. Evaluable FGFR2-fusion Patients (ICC) on FGFR therapy w/ post-progression biopsy

Our Phase 1 trial will focus on FGFR resistance patients

Selectivity

Resistance

Pre-clinical

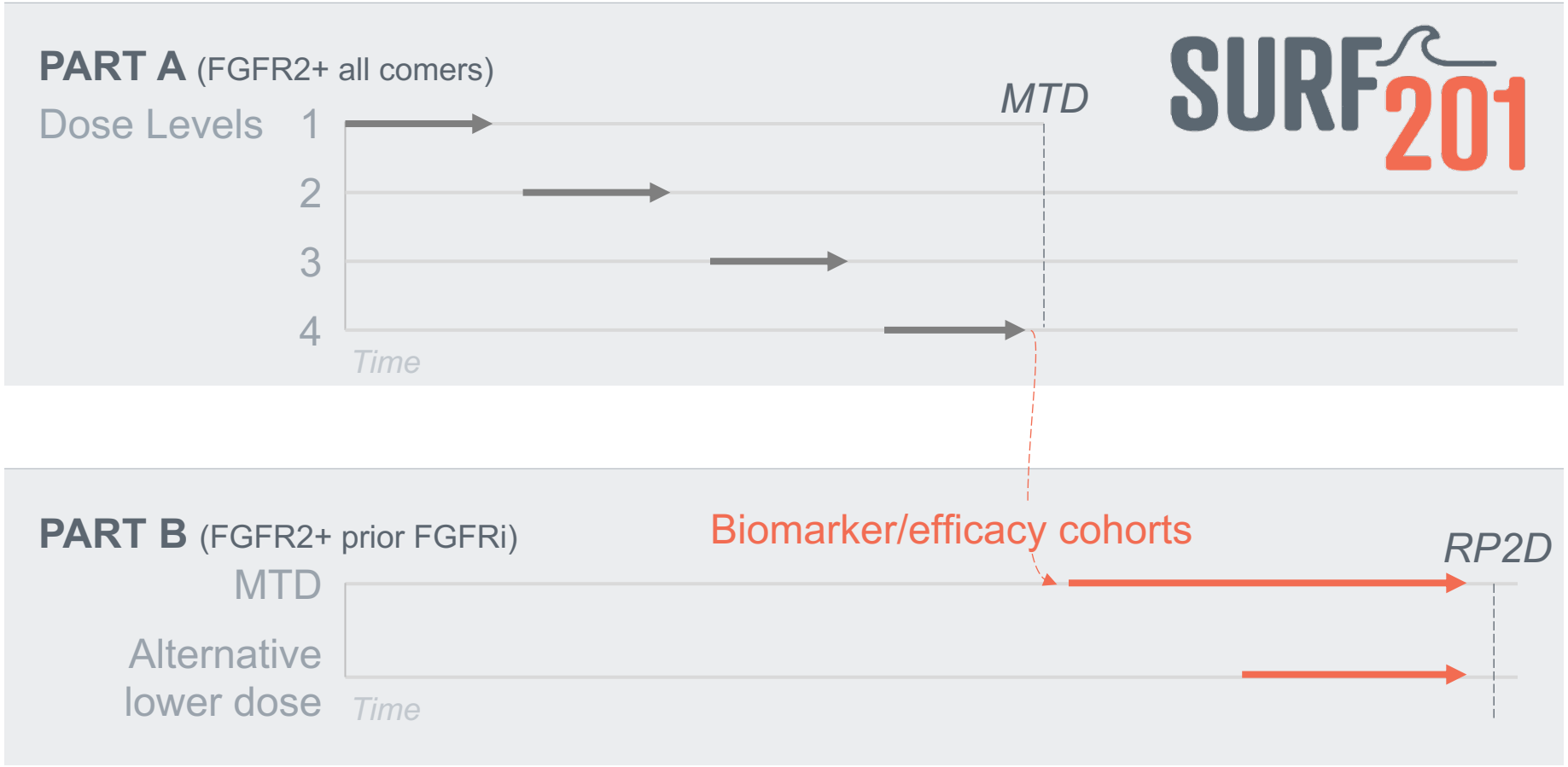
Population

Clinical

What is the MTD?

What is the optimal dose?

Illustrative



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

Why invest in TYRA?

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:* $241.7M

*March 31, 2023