TYRA

Corporate Deck

November 2024

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 for them to be first-in-class, and the potential safety and therapeutic benefits of our product candidates, our ability to commercialize our product candidates, if approved, 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: interim results of a clinical trial are not necessarily indicative of final results and one or more of the clinical outcomes may materially change as patient enrollment continues, following more comprehensive reviews of the data, as follow-up on the outcome of any particular patient continues and as more patient or final data becomes available, including the risk that unconfirmed responses may not ultimately result in confirmed responses to treatment after follow-up evaluations; the potential for proof-of-concept results to fail to result in successful subsequent development of TYRA-300: we are early in our development efforts, have only recently begun testing TYRA-300 and TYRA-200 for oncology 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, data readouts, 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; we may expend our limited resources to pursue a particular product candidate and/or indication and fail to capitalize on product candidates or indications with greater development or commercial potential; 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; 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; later developments with the FDA may be inconsistent with prior feedback from FDA, including with respect to the proposed initiation and design of our planned Phase 2 study of TYRA-300 in ACH; 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; we may not realize the benefits associated with orphan drug designation, including that orphan drug exclusivity may not effectively protect a product from competition and that such exclusivity may not be maintained or from the rare pediatric disease designation, including receipt of a Priority Review Voucher (PRV) or any value therefrom; 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; unstable market and economic conditions may adversely affect our business and financial condition and the broader economy and biotechnology industry; 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.

Here's a snapshot of 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 Sept 30, 2024: $360.1M

RECENT PROGRESS

TYRA-300^{ACH}: Ph 2 IND cleared by FDA

TYRA-300^{ONC}: interim clinical proof of concept established with TYRA-300 in mUC from SURF301; presented at ENA

TYRA-430^{ONC}: IND cleared by FDA

* ACH: Achondroplasia; ONC: Oncology
All TYRA small molecule inhibitors are for investigational use only

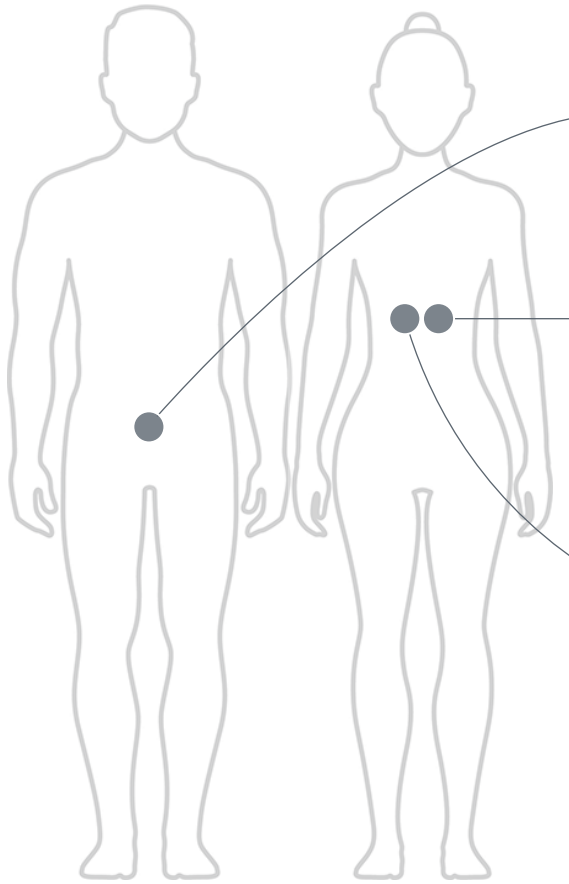
FGFR alterations are implicated in many cancers

FGFR1

2

3

4



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

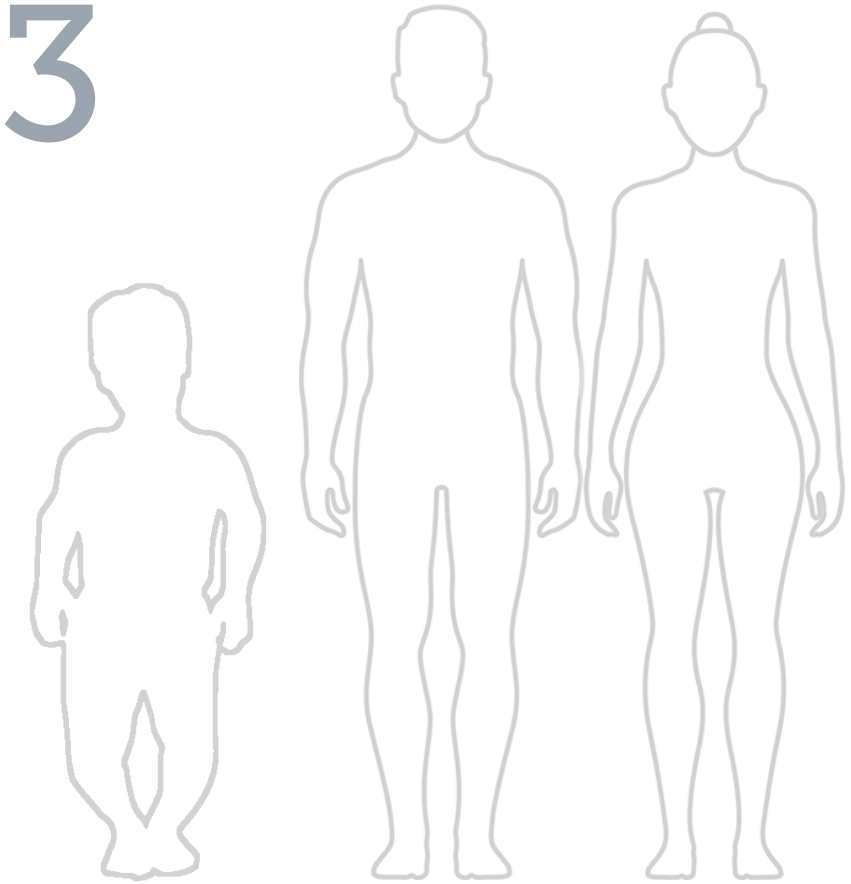
HEPATOCELLULAR CARCINOMA (HCC)
~30% FGFR4/3[1] | ~9,000/yr (US)

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

Oncology figures represent 2022 US incidence across all stages of the disease
1. Estimate of FGF19 IHC+ in HCC; Kim, 2019

FGFR3 drives two large market opportunities

FGFR3



ACHONDROPLASIA (ACH)
~99% FGFR3 | ~3,000/yr (US)

OTHER FGFR3-RELATED SKELETAL DYSPLASIA
~40,000/yr (US)

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

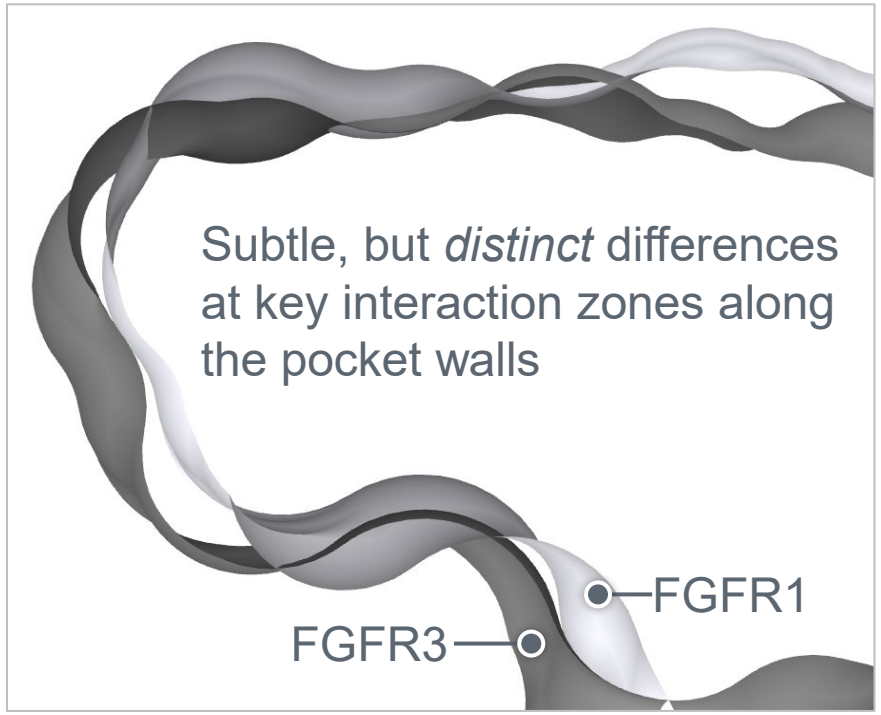
TYRA-300

Has the potential to address these indications

Oncology figures represent 2022 US incidence across all stages of the disease; skeletal dysplasias represent 2022 US pediatric prevalence
TYRA-300 is an investigational product currently in a Phase 1 clinical trial for oncology and has not been approved by any governmental body for any therapy or indication

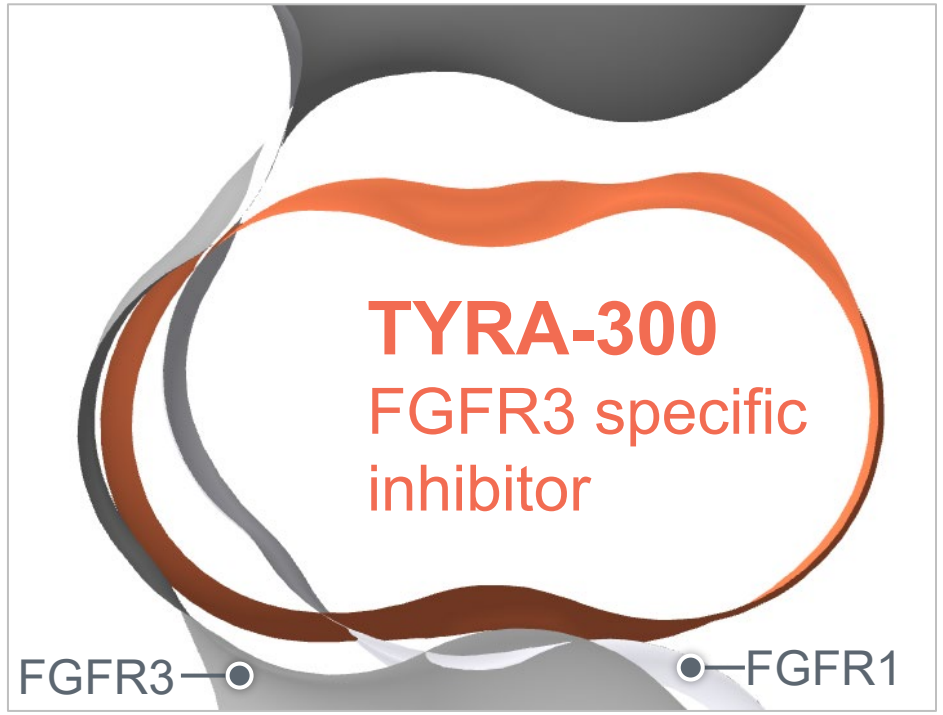
We have designed a potential solution to an intractable problem

FGFR isoform selectivity



MOLECULAR MODEL

dozens of high-resolution crystal structures

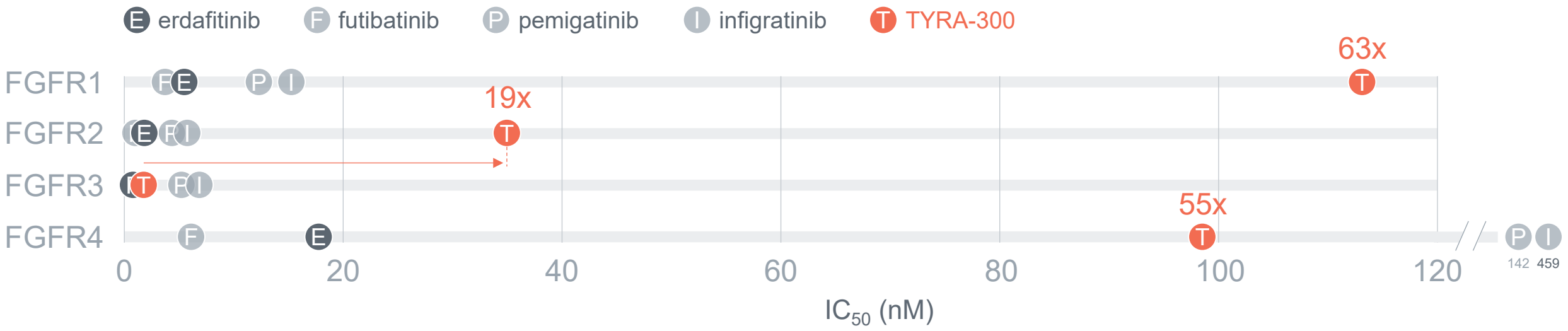


CRYSTALLOGRAPHY

3.0Å cross-sections

TYRA-300 is a potential first-in-class, highly selective FGFR3 inhibitor

Selectivity observed for TYRA-300 vs. approved or late-stage clinical compounds: *in vitro* Ba/F3 Cellular IC_{50} (nM)



	E	F	P	I
FGFR1	4.2x	4.9x	2.4x	2.2x
FGFR2	1.4x	1.3x	0.8x	0.8x
FGFR4	14x	7.6x	27x	67x

All experiments conducted under identical conditions, tested in duplicate.

Our expertise in FGFR biology creates a differentiated pipeline

GENETIC CONDITIONS	Estimated Annual US Addressable[1]	Discovery	IND-Enabling	Phase 1	Phase 2	Phase 3	Anticipated Milestone
FGFR3^{ACH}: TYRA-300	~3K			*Data from FGFR3^{ONC}*	BEACH 301 *IND cleared*		Dose 1st child in Q1 25

Potentially leading to additional skeletal dysplasia, including FGFR3-related conditions (HCH, SHOX), and pediatric short stature

ONCOLOGY							
FGFR3^{ONC}: TYRA-300	~40K			SURF 301			mUC: QD dose optimization NMIBC: submit Ph2 IND by YE 24
FGFR2^{ONC}: TYRA-200	~5.5K			SURF 201			Complete Ph1
FGFR4/3^{ONC}: TYRA-430	~9K			SURF 431			Dose first patient

TYRA retains an active FGFR3 discovery program.
1. Represents FGFR3/FGFR2/FGF19+ incidence and relapses for TYRA300/200/430, prevalence for ACH

TYRA

TYRA-300$^{A/HCH}$ — Potentially efficient development path in an attractive, rapidly-emerging market

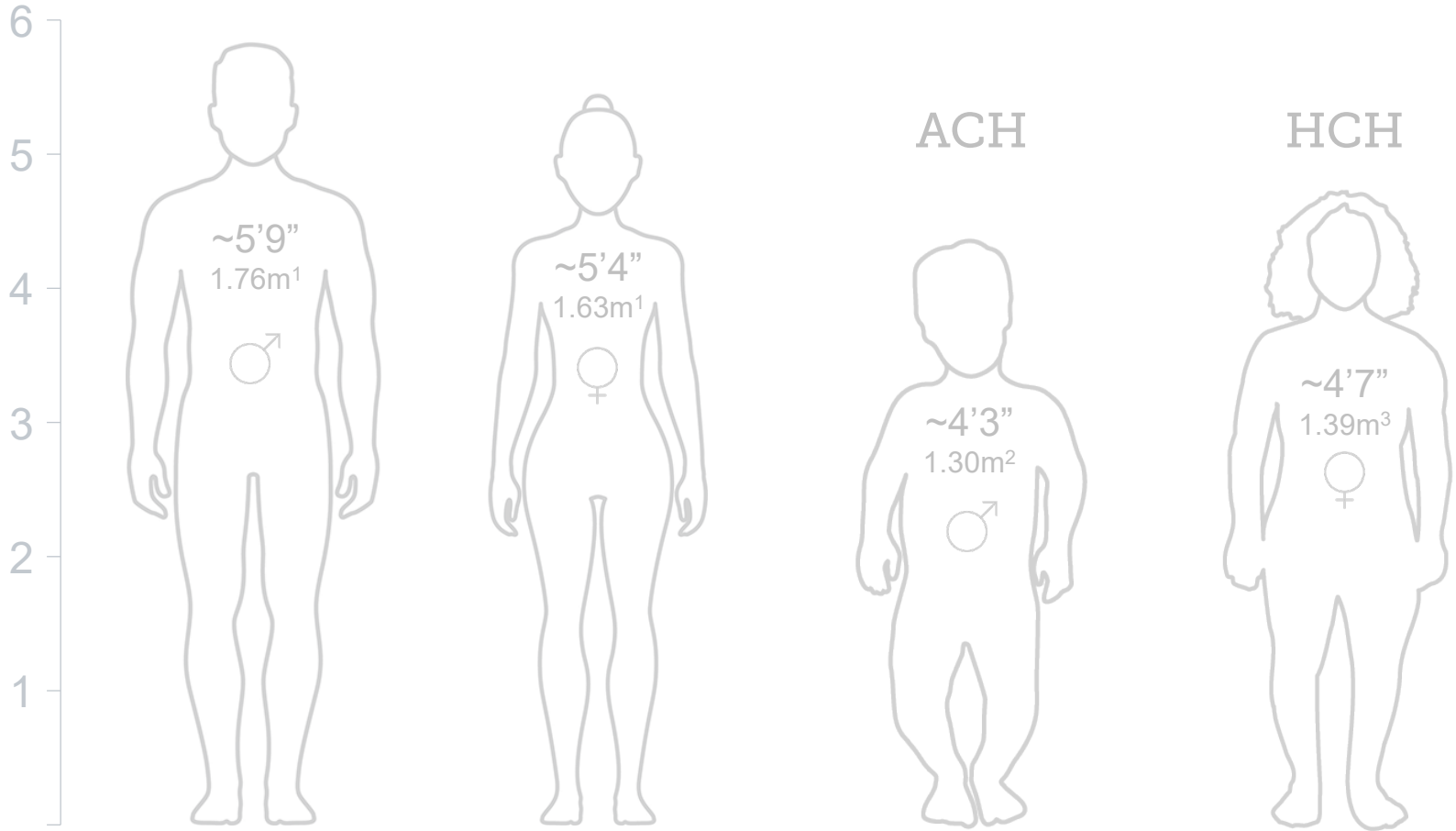
TYRA-300^{ONC}

SNÅP

Inhibiting FGFR3 may benefit people with skeletal dysplasia

FGFR3

Over-activation of this protein in bone growth plates underlies both ACH and HCH



1. CDC Vital and Health Statistics (represents average US male and female heights for ages 20-29); 2. Hoover-Fong, 2021 (represents average ACH adult male height);
3. Cheung, 2023 (represents average HCH female height at age 16)

ACHONDROPLASIA

ACH can result in serious clinical complications

ACH is the most common cause of disproportionate short stature

MECHANISM

FGFR3 G380R gain of function mutation accounts for ~99% of ACH[1,2]

FGFR3 inhibits chondrocyte proliferation and differentiation, resulting in decreased longitudinal bone growth[2]

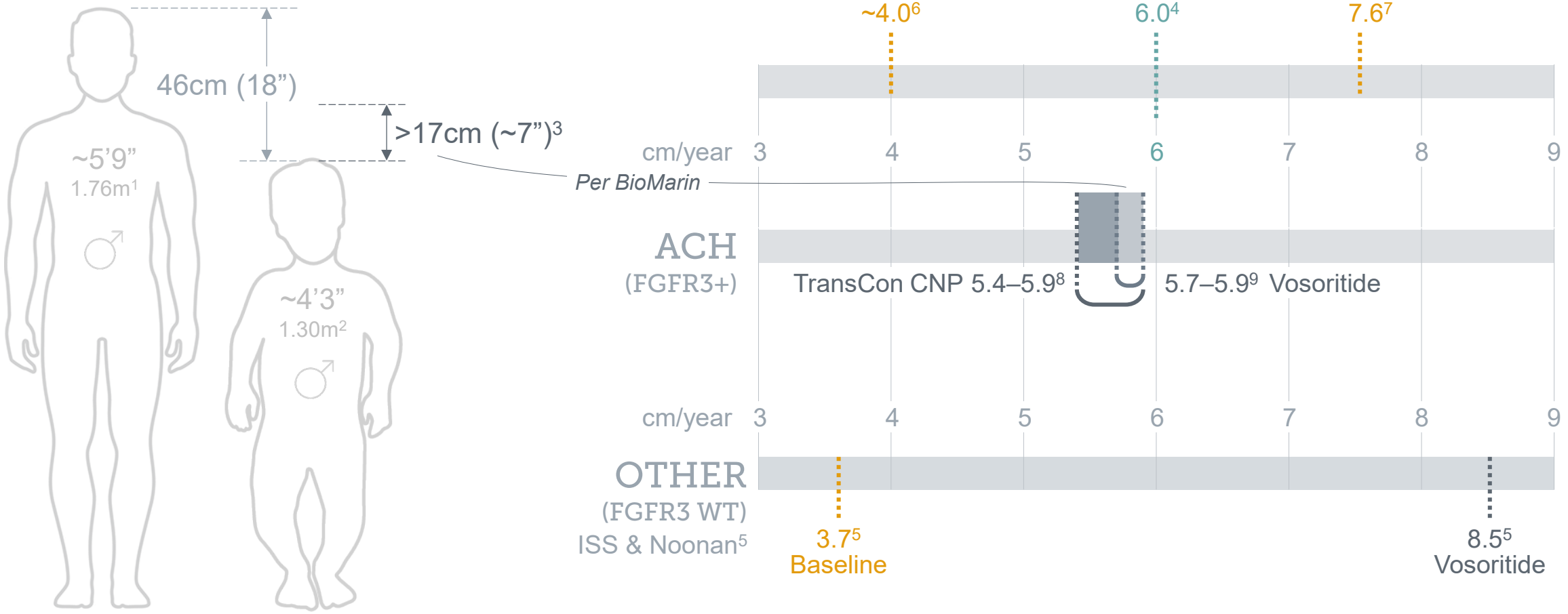
COMPLICATIONS

Infants can face serious complications related to critical foramen magnum stenosis[1,3]

Additionally: Pain, multiple surgeries, and functional limitations (e.g., reach, stride)

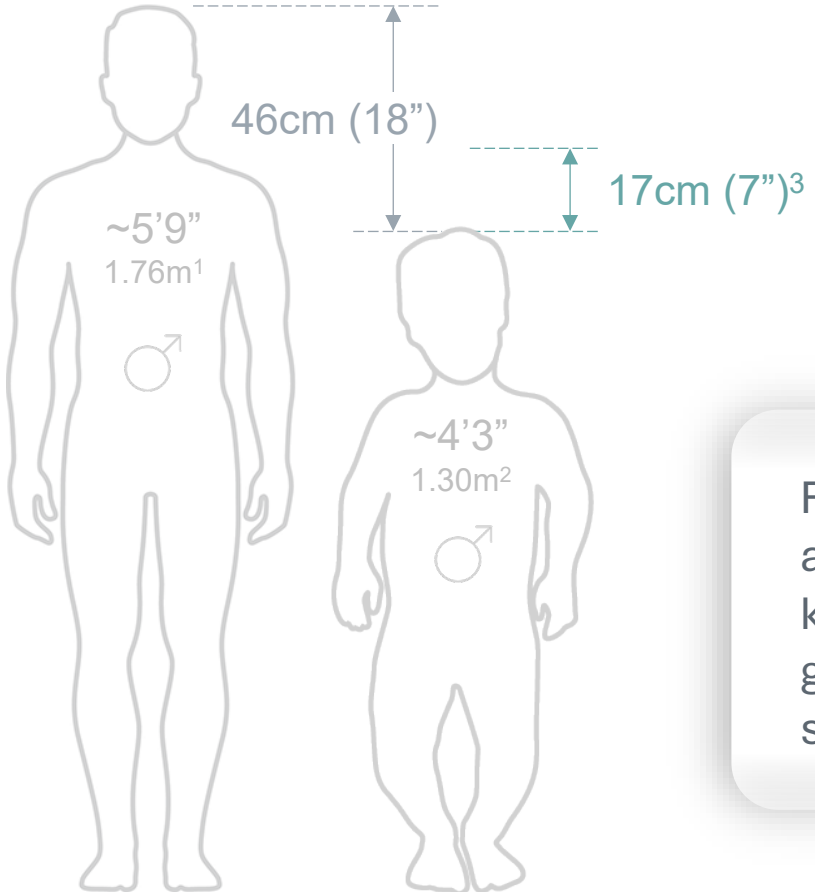
1. Pauli et al., 2019; 2. Ornitz and Legeai-Mallet, 2017; 3. Hecht et al., 1987

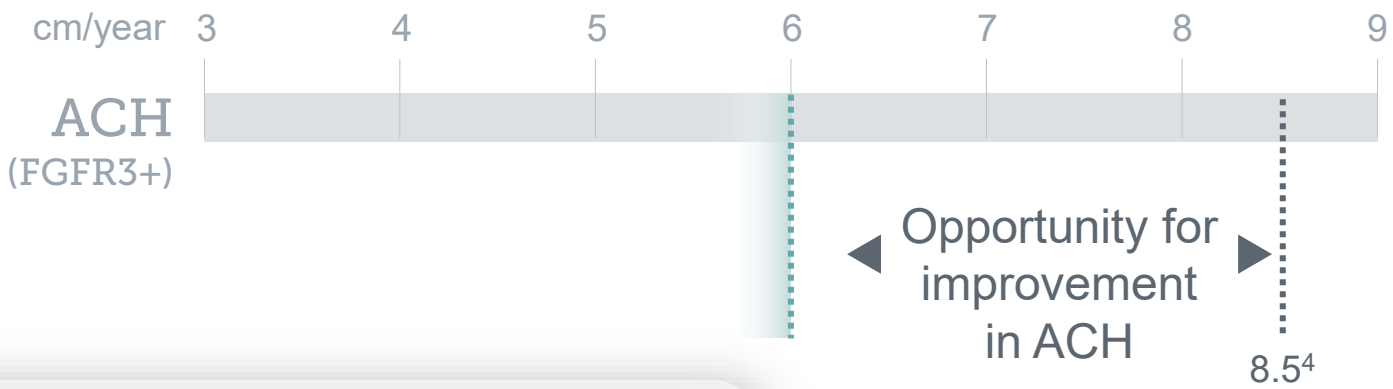
~6cm AHV is potentially leaving efficacy on the table



1. CDC Vital and Health Statistics (represents average US male and female heights for ages 20-29); 2. Hoover-Fong, 2021 (represents average ACH adult male height); 3. Potential total height gain from VOXZOGO BMRN 9/4/24 Investor Day Presentation 4. Ph2 Cohort 5 12mo data, Savarirayan, 2024; 5. BioMarin 05/04/24 press release; 12mo data from Ph2 (n=8) Investigator initiated study in ISS (ACAN Deficiency, NPR2 Mutation) and Noonan Syndrome; 6. Savarirayan, 2021 (5 to 14yrs); 7. Merck Manuals (12mo to 10yrs); 8. 5.4 from Ph2 100 µg/kg/week cohort 6mo data (n=11) Ascendis 11/14/22 Corporate Presentation; 5.9 based on Ph3 data (n=57); Ascendis 9/16/24 Corporate Presentation 9. 5.9 from Ph2 Cohort 3 12mo Data (n=10), Savarirayan, 2019; 5.7 based on VOXZOGO prescribing information 10/2023 (n=60)

Clinicians and families hope for more effective therapies

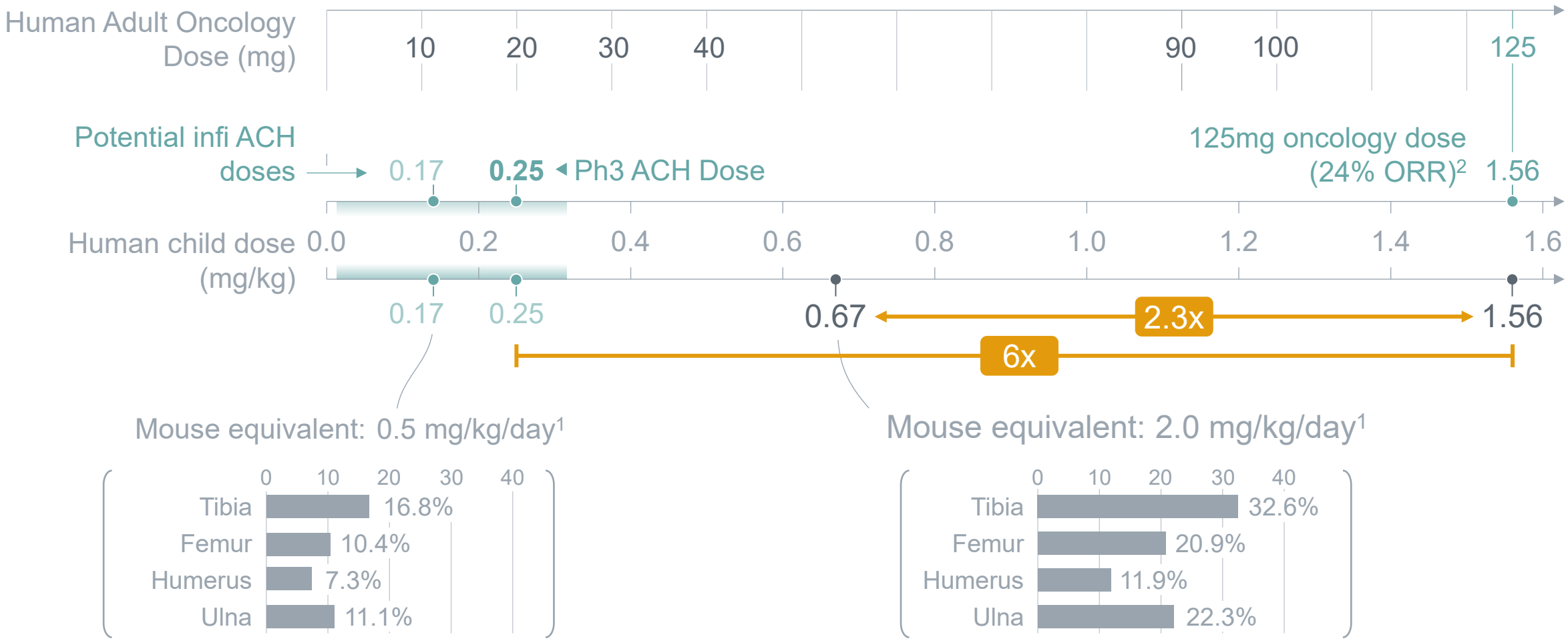




Further engaging FGFR3 and achieving a higher AHV could potentially benefit key clinical sequelae like reach and gait, plus reduce spinal stenosis / surgeries.

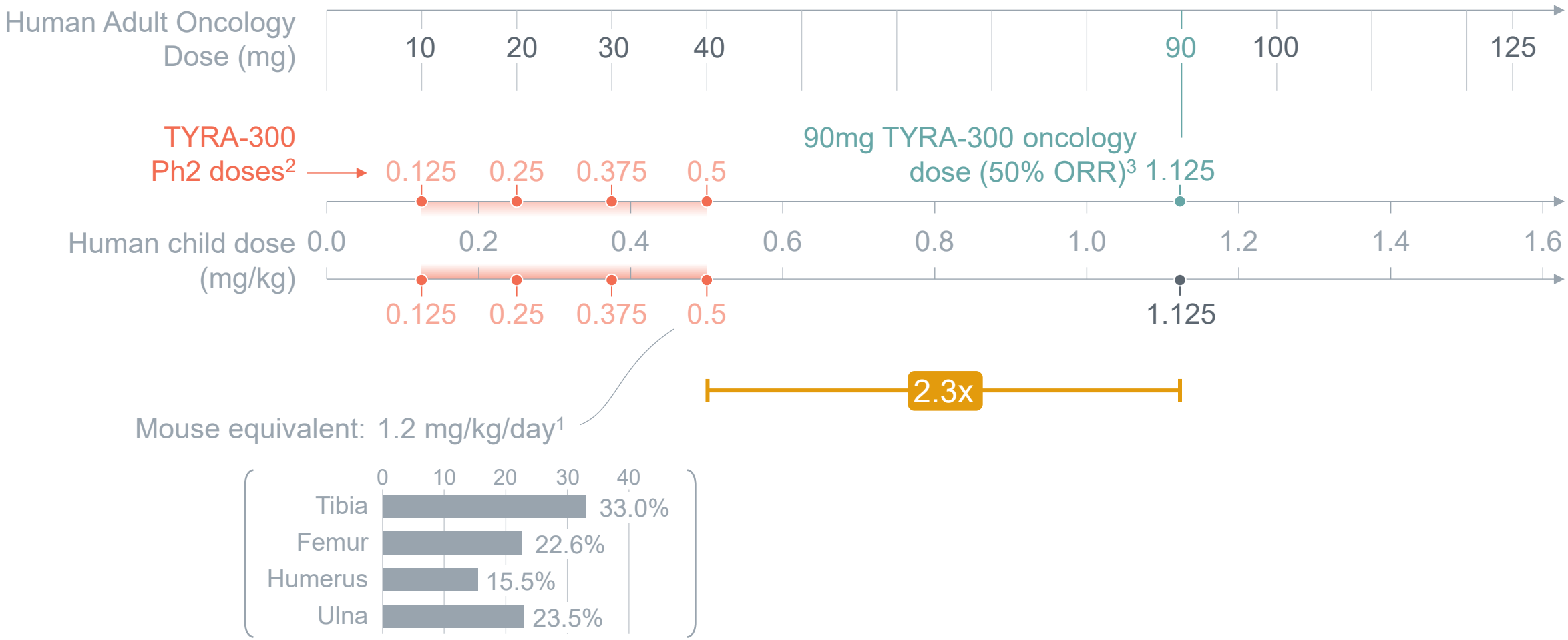
1. CDC Vital and Health Statistics (represents average US male and female heights for ages 20-29); 2. Hoover-Fong, 2021 (represents average ACH adult male height); 3. Potential total height gain from VOXZOGO BMRN 9/4/24 Investor Day Transcript 4. BioMarin 05/04/24 press release; 12mo data from Ph2 (n=8) Investigator initiated study in ISS (ACAN Deficiency, NPR2 Mutation) and Noonan Syndrome

Infigratinib Ph3 ACH dose ~6x lower than oncology dose



1. 2.0mg/kg data in Komla-Ebri D, et al. J Clin Invest 2016; Potential infigratinib ACH doses and mouse to human dose extrapolation highlighted in Demuynck et. al. (ASHG, 2019)
2. Converted to mg/kg dosing based on 80kg adult weight

In Ph2 we plan to explore doses predicted to be active in ACH



1. Mouse subcutaneous dose 2. Low dose predicted to cover lowest exposure where physis thickening was observed in a preclinical model 3. Converted to mg/kg dosing based on 80kg adult weight; represented 90mg confirmed ORR for n=10 patients

Cleared to proceed with Ph2 BEACH301 for TYRA-300 in ACH

BEACH 301

Safety Sentinel Cohort

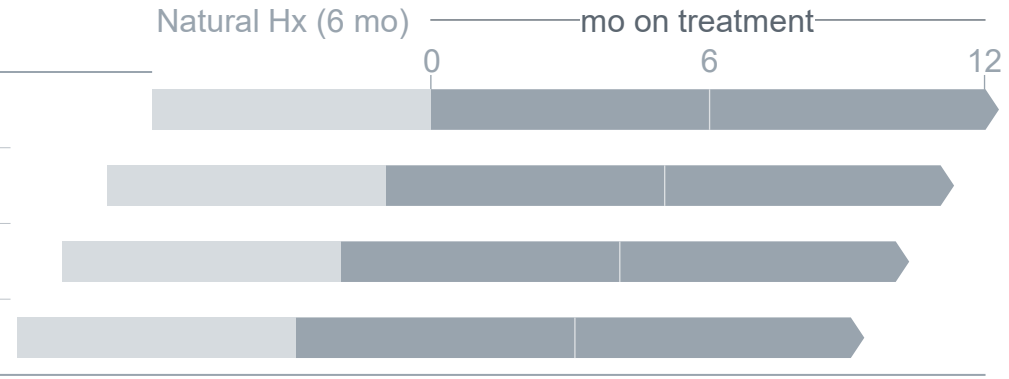
ACH age 5-10, treatment naïve

N=	DOSE (mg/kg)
3	0.50
3	0.375
3	0.25
3	0.125

Cohort 1

Treatment naïve
ACH age 3-10

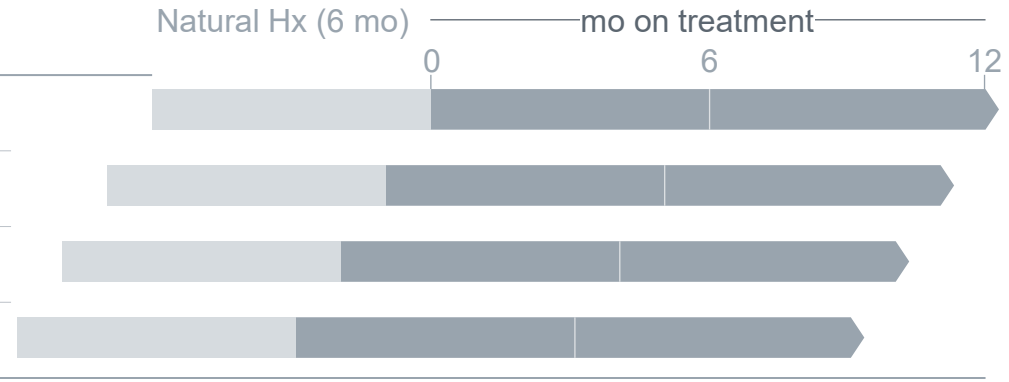
N=*	DOSE (mg/kg)
6	0.50
6	0.375
6	0.25
6	0.125



Cohort 2

Received prior growth-accelerating therapy
ACH age 3-10

N=*	DOSE (mg/kg)
6	0.50
6	0.375
6	0.25
6	0.125



*Dose decisions based on 6 participants. Additional participants may be assigned at discretion of Sponsor

HYPOCHONDROPLASIA

Like ACH, HCH results in disproportionate long bones

The proximal long bones are more affected than distal bones*

**Arm: humerus*
Leg: femur

MECHANISM

FGFR3 inhibits chondrocyte proliferation and differentiation

Over-activation of FGFR3 decreases longitudinal bone growth in ACH and HCH[1]

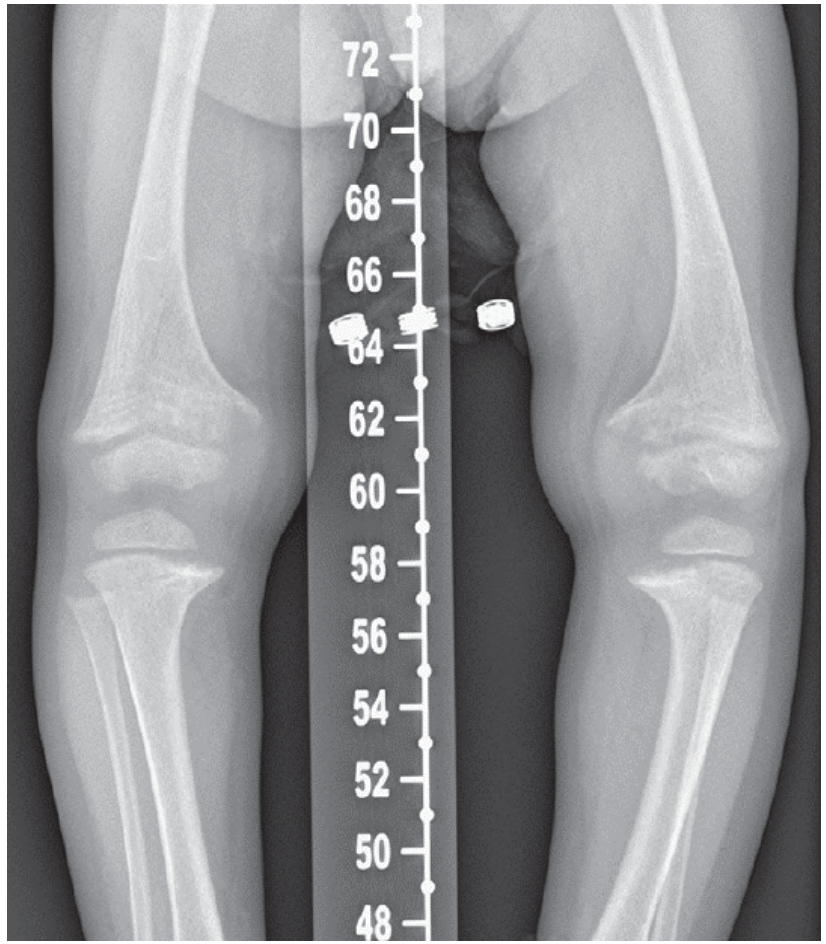
CHALLENGES

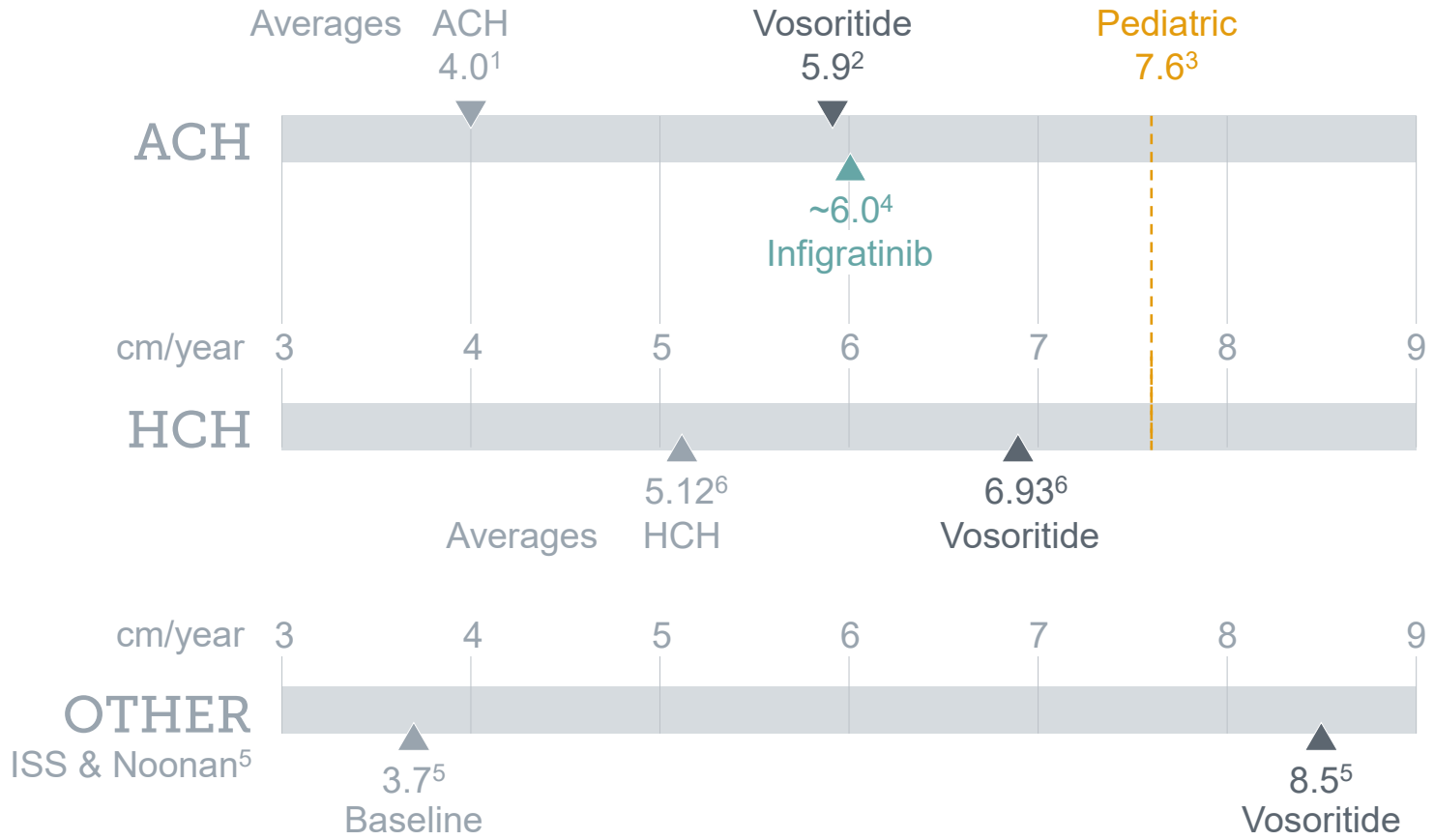
Skeletal features and functional limitations similar to those seen in ACH, but milder[2]

Additional (examples): orthopedic surgery, sleep apnea, increased frequency of ear infections

1. Ornitz and Legeai-Mallet, 2017; 2. Bober et al., 1999

There is a strong need for an oral therapy selective for FGFR3

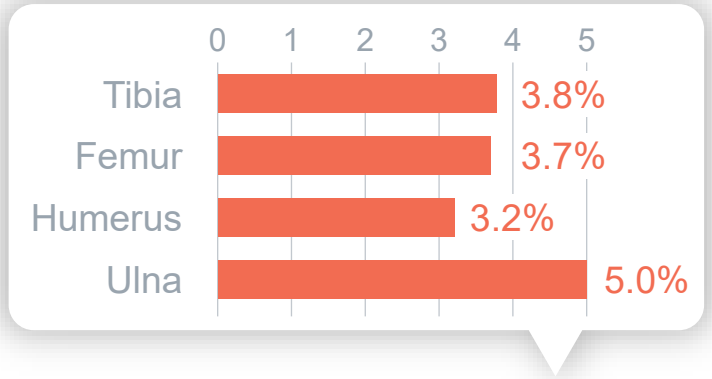




1. Savarirayan, 2021 (5 to 14yrs); 2. Ph2 Cohort 3 (n=10) 12mo Data, Savarirayan, 2019; 3. Merck Manuals (12mo to 10yrs); Presentation 4. Ph2 Cohort 5 12mo data, Savarirayan, 2024; 5. BioMarin 05//04/24 press release; 12mo data from Ph2 (n=8) Investigator initiated study in ISS (ACAN Deficiency, NPR2 Mutation) and Noonan Syndrome; 6. Dauber et al., 2024; 12mo data for baseline and vosoritide from Ph2 study in HCH

TYRA-300 improved long bone growth at 1.8 mg/kg/day

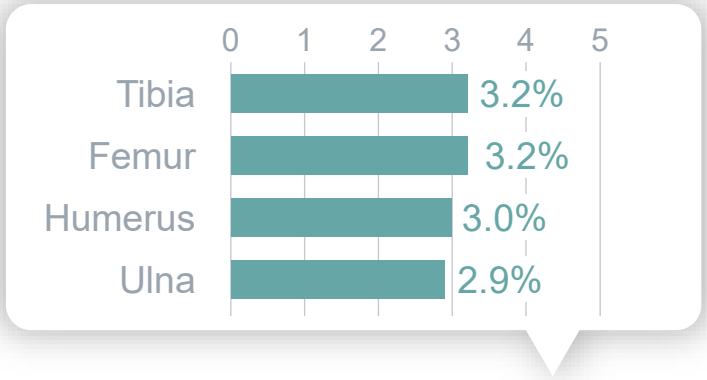
1.8
mg/kg/day



Data generated from an experiment conducted in the lab of Dr. Laurence Legeai-Malei

QED communicated intent to maintain 0.25 mg/kg dose for HCH

1.0
mg/kg/day



Dambkowski et. al. (ENDO, 2022); 06/04/2024 PROPEL 2 Phase II Trial Results Call transcript

There are many development opportunities beyond ACH and HCH

FGFR3 GERMLINE MUTATIONS

SKELETAL DYSPLASIA

Achondroplasia (~3K) ← *ODD & RPD granted*

Hypochondroplasia (~2K)

Thanatophoric dysplasia

SADDAN syndrome

Double dominant ACH

CRANIOSYNOSTOSIS

Crouzon syndrome with acanthosis nigricans

Muenke syndrome

OTHER GENETIC SHORT STATURE

Leri-Weill dyschondrosteosis (~26K)

Turner syndrome (~10K)

Osteogenesis imperfecta (~4K)

Mucopolysaccharidoses IVA and VI

Laron syndrome (Growth Hormone Insensitivity)

PEDIATRIC SHORT STATURE

Idiopathic short stature (~700K[1])

Addressable US pediatric population; Source: company research

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

TYRA

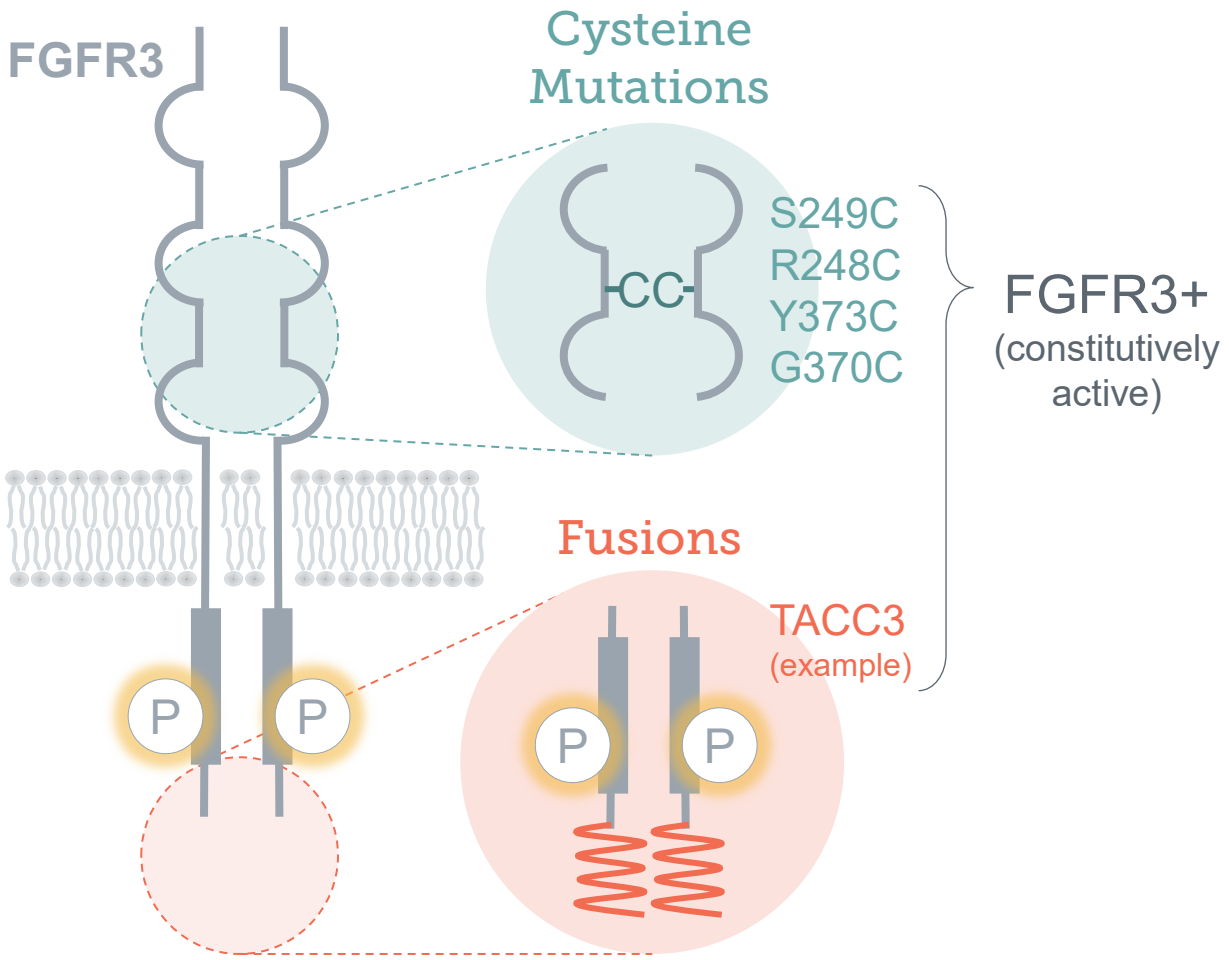
TYRA-300$^{A/HCH}$

TYRA-300^{ONC}

SNÅP

Designed to potentially improve tolerability and durability over current standards of care

FGFR3 oncogenic alterations are common in bladder cancer

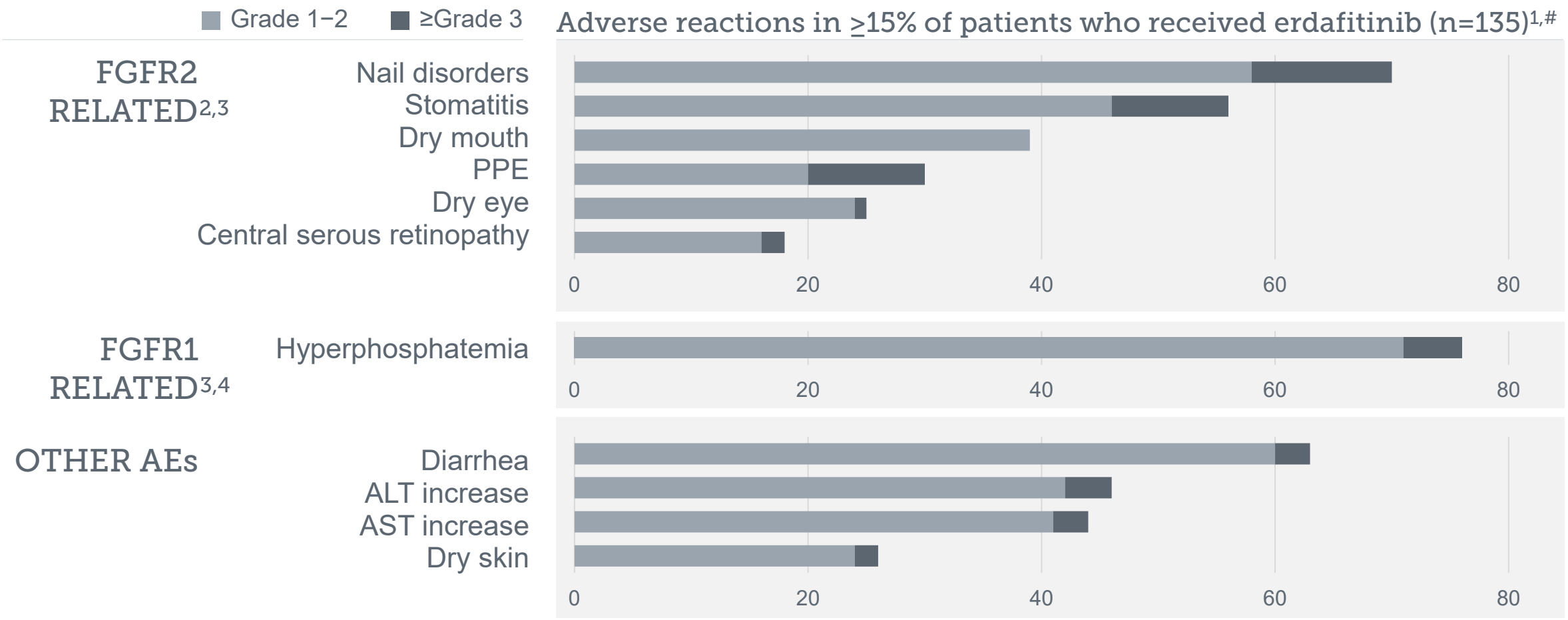


	mUC	NMIBC
FGFR3+	10–20%[1]	40–80%[2]
Oral erdafitinib	35.3% ORR, 12.1mo OS[3] (n=136)	IR: 83% CR[4] (n=18) HR(Cis): 73% CR[5] (n=11)
infigratinib	24% ORR[6] (n=54)	HR (Pap): 75% CR[7] (n=4)
pemigatinib	23% ORR[8] (n=103)	

Abbreviations: IR, Intermediate Risk; HR, High Risk

1. Weickhardt 2022 2. Mayr, 2022; Kacew, 2020; Knowles, 2020 3. BALVERSA® (erdafitinib) prescribing information 01/2024; BLC3001 Cohort 1 data 4. Daneshmand, 2023 (SUO) 5. Catto, 2023 (SUO) 6. Lyou, 2022 7. Cha, 2020 (ASCO GU) 8. Necchi, 2023; represents ORR for continuous dosing cohort

Pan FGFR inhibition is associated with key on-target toxicities



[1]Adapted from: Erdafitinib tablets, for oral use. Prescribing information 01/2024. https://www.accessdata.fda.gov/drugsatfda_docs/label/2024/212018s007s008s009lbl.pdf. Accessed 06 October 2024. [2]Lacouture ME et al. Oncologist. 2021. [3]Subbiah V, Verstovsek S. Cell Rep Med. 2023. [4]Kommalapati A, et al. Cancers. 2021. [#]Study BLC3001

Adverse reactions have occurred requiring dosage modifications of erdafitinib

Adverse reactions resulting in dose adjustments in patients who received erdafitinib (n=135)[1]

INTERRUPTION

72%

Nail disorders	22
Stomatitis	19
Eye disorders	16
PPE	15
Diarrhea	10
Hyperphosphatemia	7
Increased AST	6
Increased ALT	5

REDUCTION

69%

Nail disorders	27
Stomatitis	19
Eye disorders	17
PPE	12
Diarrhea	7
Dry mouth	4.4
Hyperphosphatemia	4.4

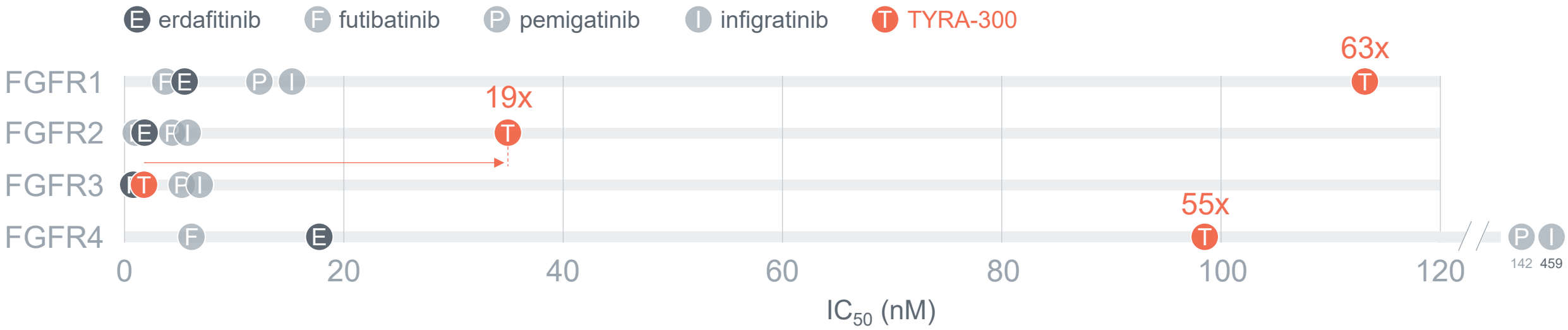
DISCONTINUATION

14%

1. BALVERSA (erdafitinib) prescribing information 01/2024, BLC3001 Cohort 1 data. Adverse reactions leading to dosage interruptions or reductions of erdafitinib in >4% of patients.

TYRA-300 is a potential first-in-class, highly selective FGFR3 inhibitor

Selectivity observed for TYRA-300 vs. approved or late-stage clinical compounds: *in vitro* Ba/F3 Cellular IC_{50} (nM)



	E	F	P	I
FGFR1	4.2x	4.9x	2.4x	2.2x
FGFR2	1.4x	1.3x	0.8x	0.8x
FGFR4	14x	7.6x	27x	67x

All experiments conducted under identical conditions, tested in duplicate.

Our Phase 1 explored QD* dose escalation and expansion

Illustrative

Dose Escalation	**PART A** (all comers) Dose QD*	All solid tumor types[1] FGFR+/-	SURF301
Dose Expansion	**PART B** (FGFR3+ only) Dose QD	Solid tumors with focus on mUC[1] FGFR3+ only	

*1x daily

1. Previously treated patients, including FGFRi, allowed

We dose escalated to 120mg QD and then expanded up to 90mg

*1x daily

SURF301

INTERIM PHASE 1 RESULTS

ENA 2024

Data Cutoff: August 15, 2024

TYRA-300 is the world's first oral, selective FGFR3 inhibitor with the potential to deliver benefit to cancer patients with a tolerable safety profile

SURF301

	BENCHMARK[1]	READOUT
ACTIVITY	On par with erdafitinib label in FGFR3+ mUC patients at active dose levels	54.5% (6/11) confirmed PRs vs. 35.3% ORR erdafitinib label
PK/PD	Dose-dependent activity	Anti-tumor activity observed in all FGFR3+ mUC ≥90 mg QD
SAFETY	Safety profile with improved tolerability in FGFR1/2/4-driven toxicities compared to pan-FGFRis	Generally well-tolerated with infrequent FGFR2- and FGFR1-associated toxicities

1. This comparison is solely based on BALVERSA® (erdafitinib) prescribing information as of January 2024 and not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities and differences. The values shown in the cross-study comparisons are directional and may not be directly comparable.

The study population was older and heavily pre-treated

n=41		
MEDIAN AGE	(range 34–84)	66 (yrs)
		n (%)
SEX AT BIRTH	Male	30 (73)
ECOG PS	0	14 (34)
	1	27 (66)
FGFR3 ALTERATION	Mutation	17 (41)
	Fusion	15 (37)
	None	10 (24)

		n (%)
TUMOR TYPE	mUC	25 (61)
	Lung	3 (7)
	Head and Neck	4 (10)
	Other	9 (22)
PRIOR LINES OF THERAPY	0	5 (12)
	1	7 (17)
	2	11 (27)
	≥3	18 (44)

Abbreviations: mUC, metastatic urothelial cancer
Safety analysis set, n=41

The study population was older and heavily pre-treated

n=41		
MEDIAN AGE	(range 34–84)	66 (yrs)
		n (%)
SEX AT BIRTH	Male	30 (73)
ECOG PS	0	14 (34)
	1	27 (66)
FGFR3 ALTERATION	Mutation	17 (41)
	Fusion	15 (37)
	None	10 (24)

		n (%)
TUMOR TYPE	mUC	25 (61)
	Lung	3 (7)
	Head and Neck	4 (10)
	Other	9 (22)
PRIOR LINES OF THERAPY	0	5 (12)
	1	7 (17)
	2	11 (27)
	≥3	18 (44)

76%
of mUC patients had ≥3 prior lines of therapy

Abbreviations: mUC, metastatic urothelial cancer
Safety analysis set, n=41

Preliminary data suggests TYRA-300 is generally well tolerated

n=41	Any Grade	≥ Grade 3
Any TRAEs, n (%)	32 (78)	8 (20)
TRAEs in >10% of participants, n(%)		
ALT increase#	10 (24)	2 (5)
Diarrhea*	9 (22)	1 (2)
Dry mouth	9 (22)	
AST increase	8 (20)	1 (2)
Dry skin	6 (15)	
Fatigue	5 (12)	

1 DLT — 90 mg QD, Gr. 3 diarrhea*

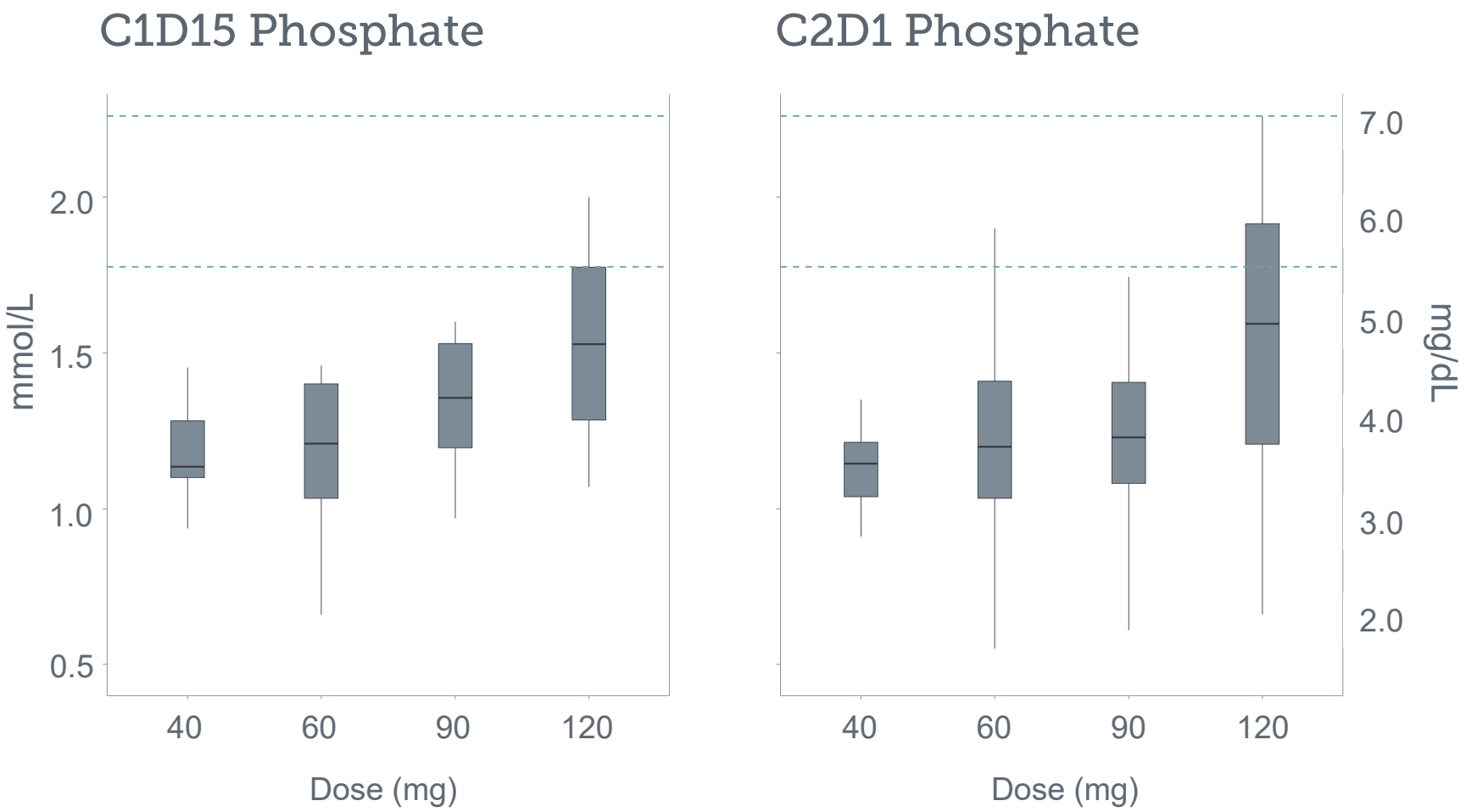
1 Drug-related discontinuation — 90 mg QD, Gr. 3 ALT elevation#

4 Related SAEs — Related to TYRA-300

0 ≥Grade 4 SAE — No drug-related events leading to death

#Drug-related discontinuation, Grade 3 ALT elevation 90 mg QD; *DLT, Grade 3 diarrhea 90 mg QD
Abbreviations: TRAE, treatment-related adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; DLT, dose-limiting toxicity; SAE, serious adverse event
Safety analysis set, n=41

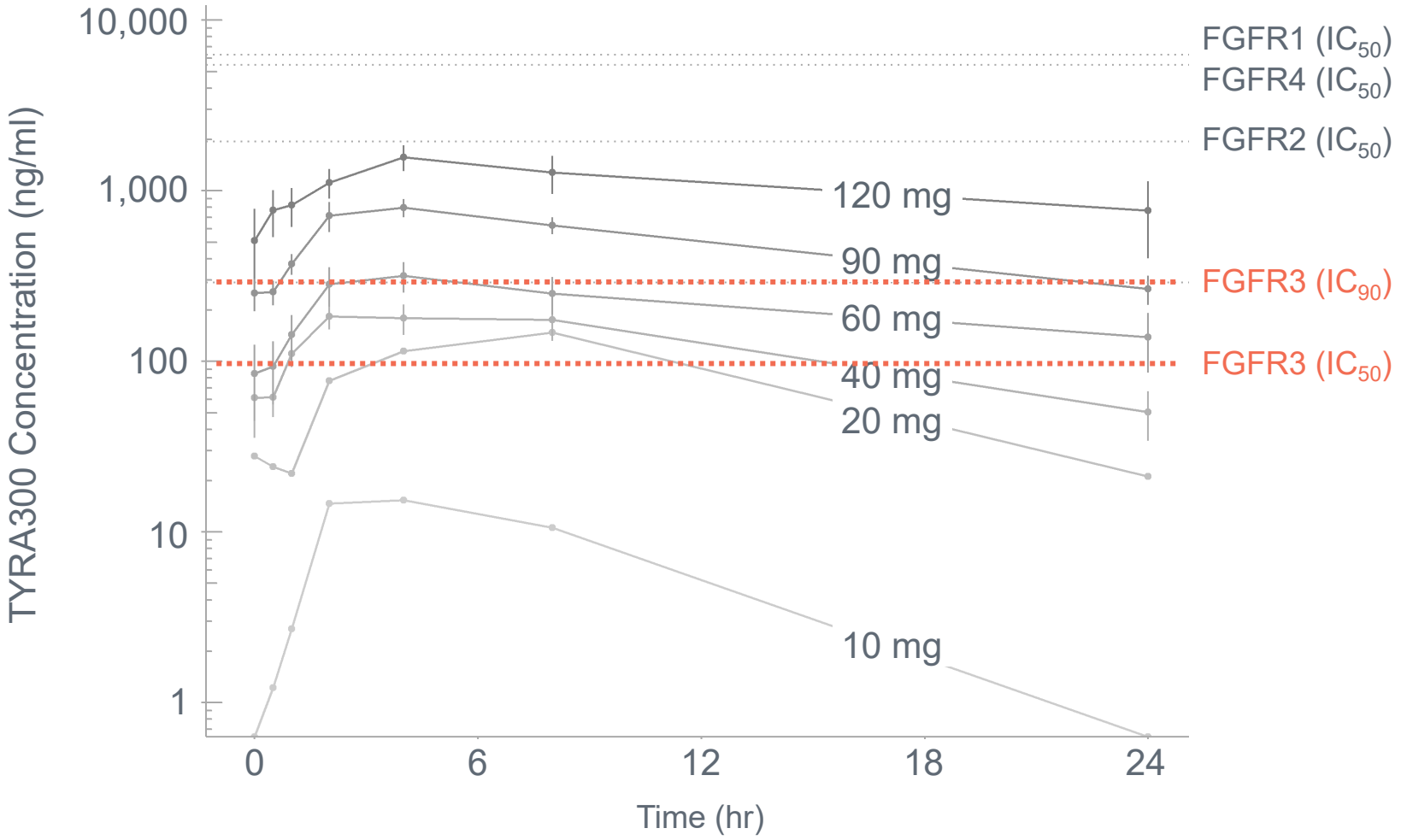
Minimal changes in phosphate at ≤90 mg QD observed



Phosphate binder was used to manage treatment-related hyperphosphatemia in one patient (90 mg QD).

Minimal impact in phosphate at ≤ 90 mg QD. Dashed lines denote 5.5 and 7 mg/dL used by Loriot et al. where 5.5- 6.9 mg/dL was defined as Grade 1 and 7.0-8.9 mg/dL as Grade 2.

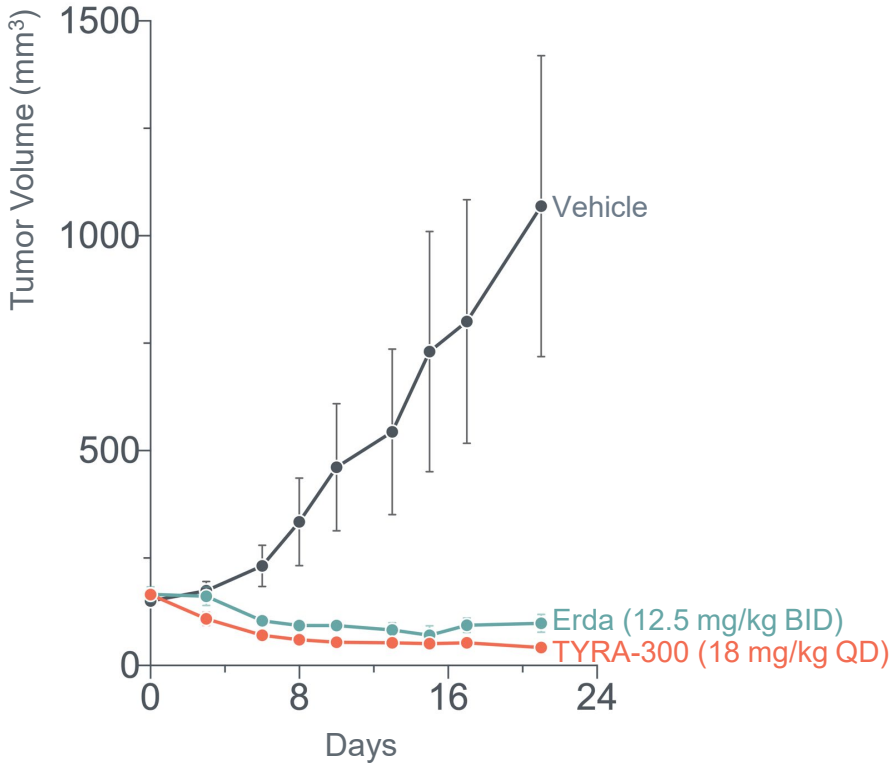
Exposure at doses ≥90 mg exceeded FGFR3 IC_{90} target coverage



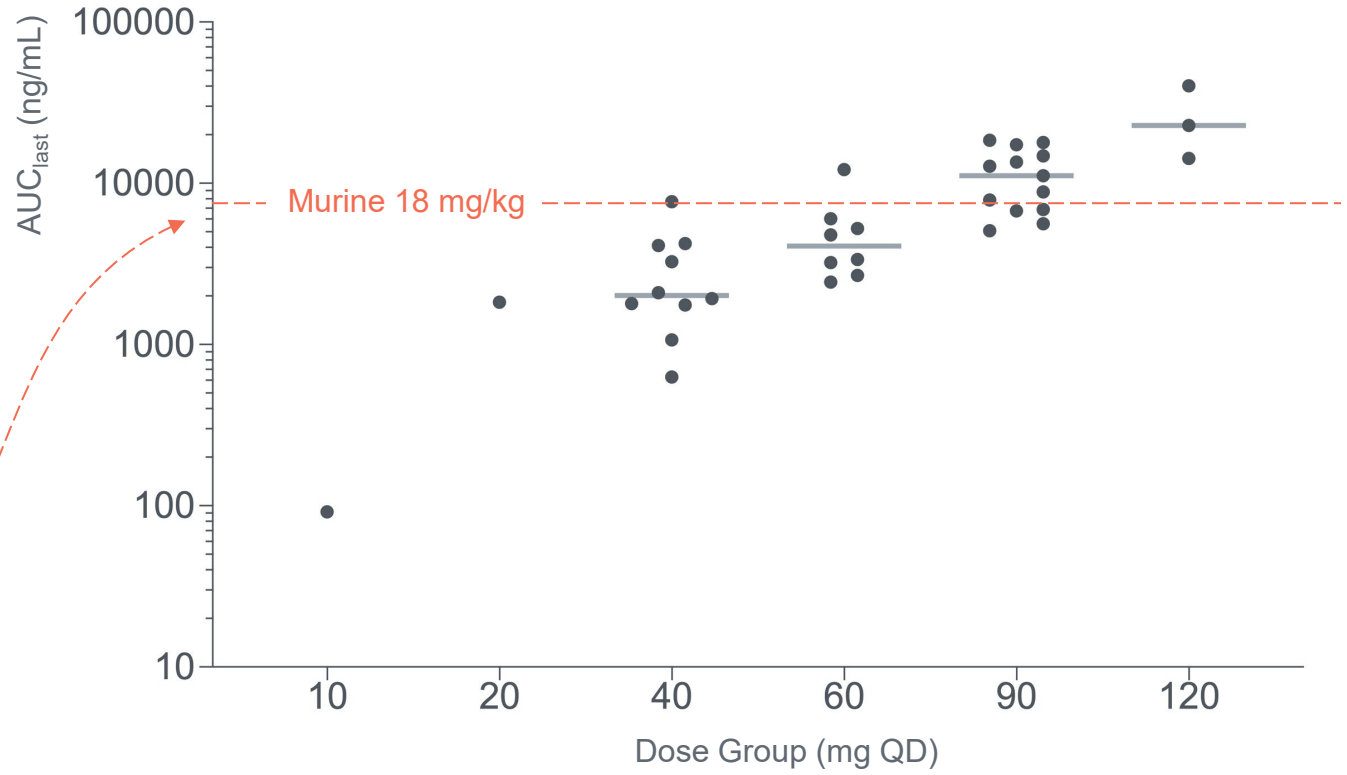
Dose (mg)	N (C1D15)	AUC (ng*h/mL)	
120	3	23,578	2.3x
90	13	10,300	2.4x
60	8	4,360	1.9x
40	10	2,270	
20	1	1,830	
10	1	91.5	

Predicted exposure was achieved in human doses ≥90 mg QD

FGFR3+ UM-UC-14 Xenograft

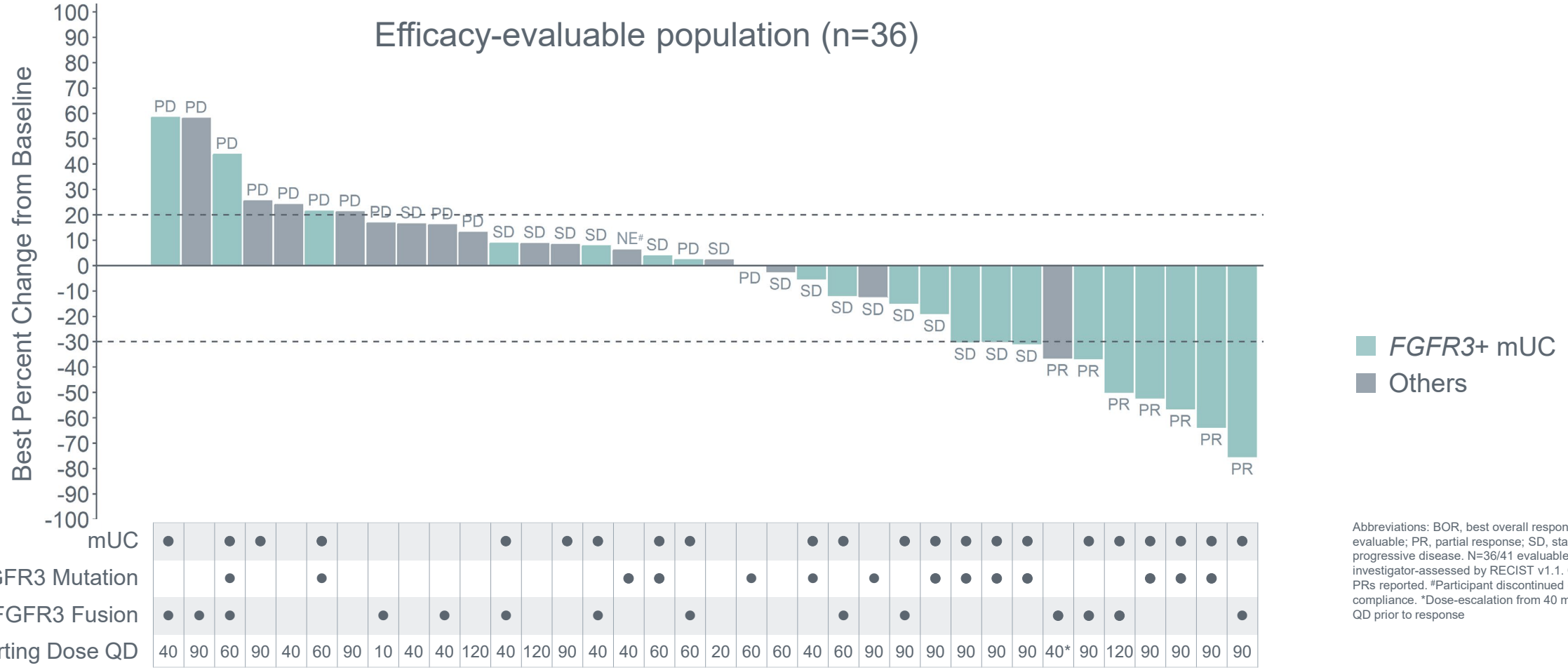


Individual Steady State AUCs in Patients



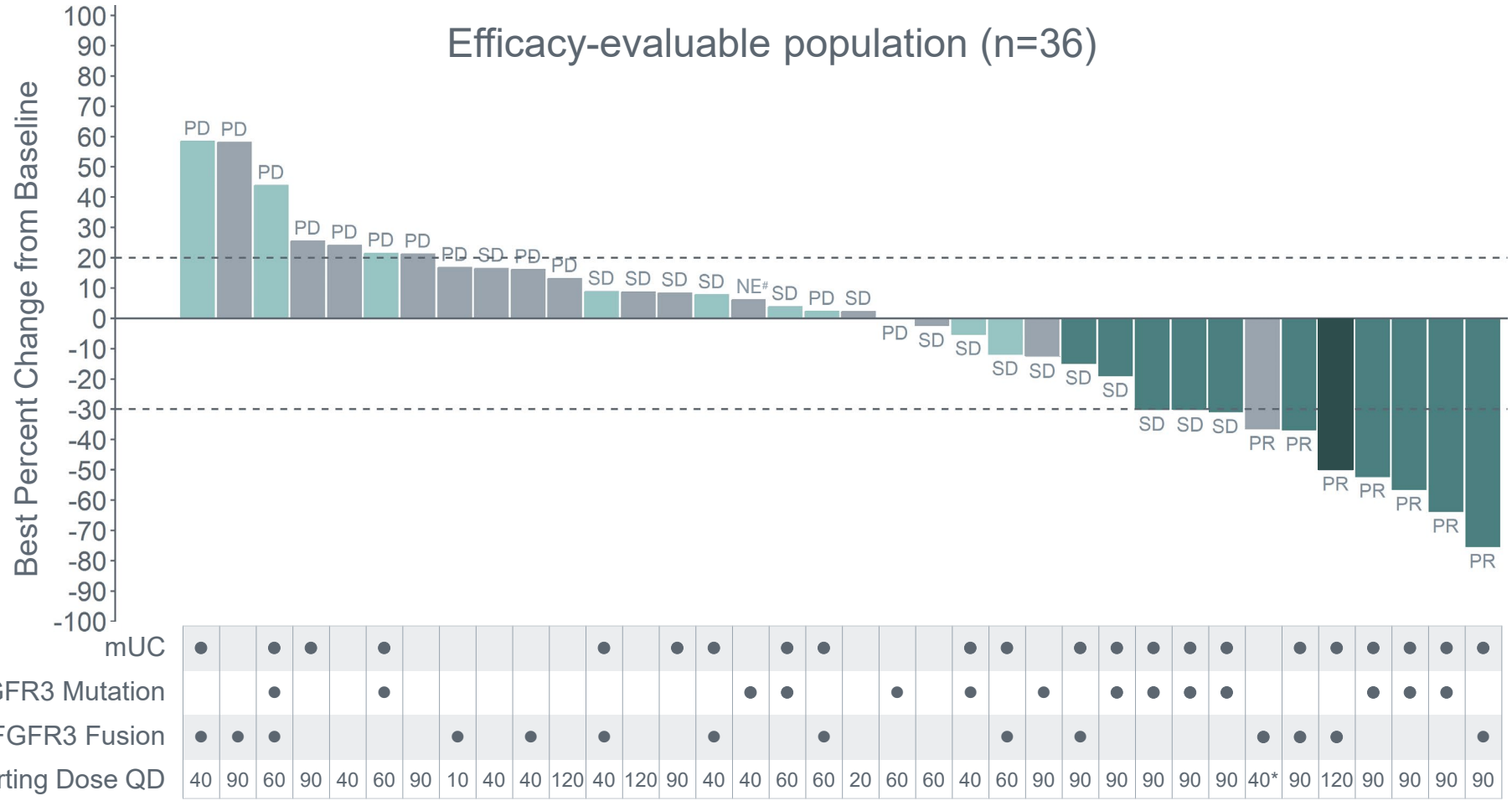
Data on File.

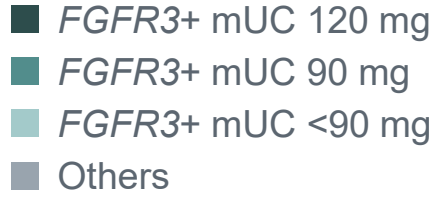
Radiographic tumor response assessment in all evaluable patients



Abbreviations: BOR, best overall response; NE, non-evaluable; PR, partial response; SD, stable disease; PD, progressive disease. N=36/41 evaluable patients. BOR by investigator-assessed by RECIST v1.1. Only confirmed PRs reported. #Participant discontinued due to non-compliance. *Dose-escalation from 40 mg QD to 60 mg QD prior to response

Anti-tumor activity observed in all *FGFR3*+ mUC ≥90 mg QD





Abbreviations: BOR, best overall response; NE, non-evaluable; PR, partial response; SD, stable disease; PD, progressive disease. N=36/41 evaluable patients. BOR by investigator-assessed by RECIST v1.1. Only confirmed PRs reported. #Participant discontinued due to non-compliance. *Dose-escalation from 40 mg QD to 60 mg QD prior to response

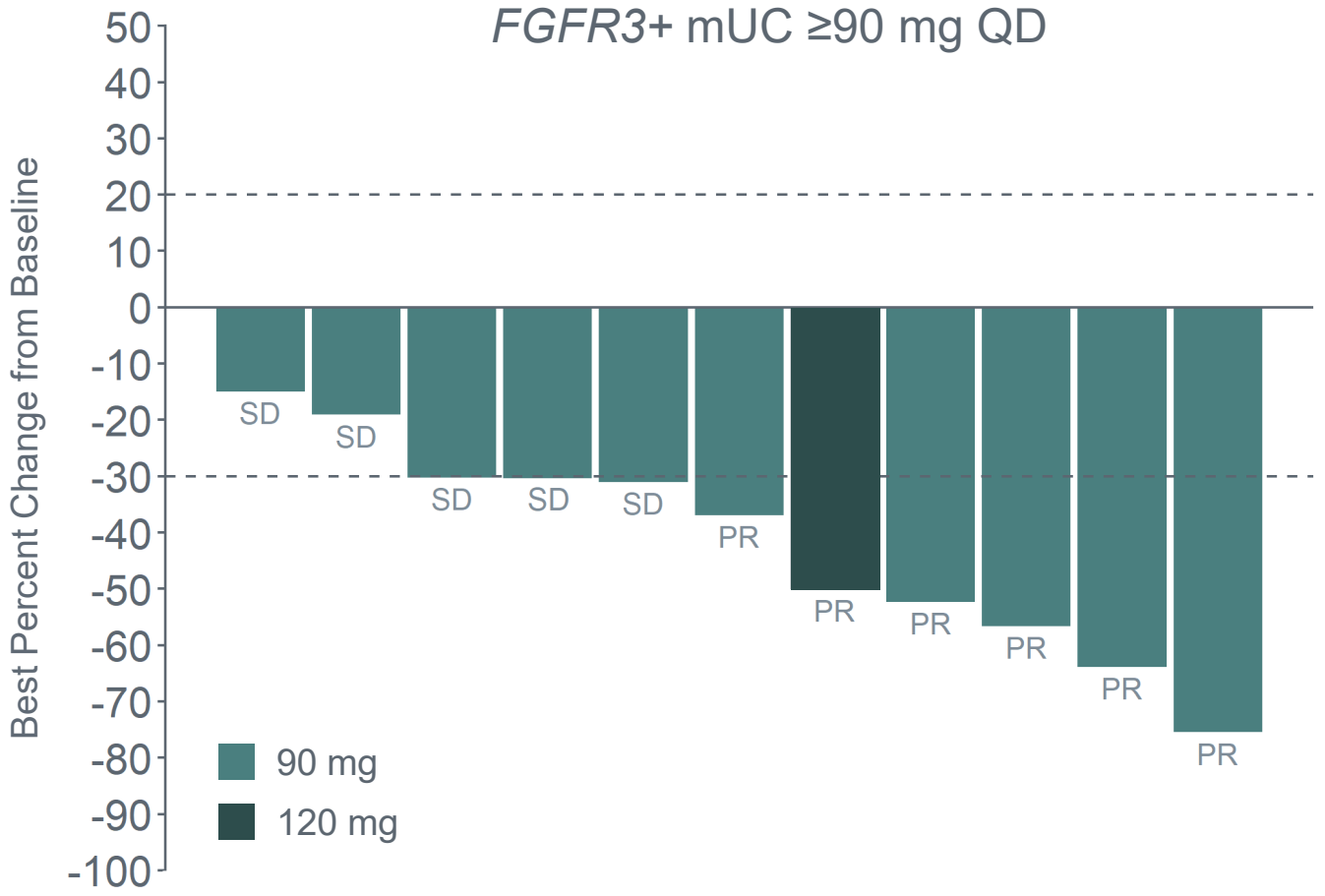
Anti-tumor activity observed in all *FGFR3*+ mUC ≥90 mg QD

Investigator-assessed radiographic BOR by RECIST v1.1 (n=11)

6 (54.5%) confirmed PRs at ≥90 mg QD (n=11)

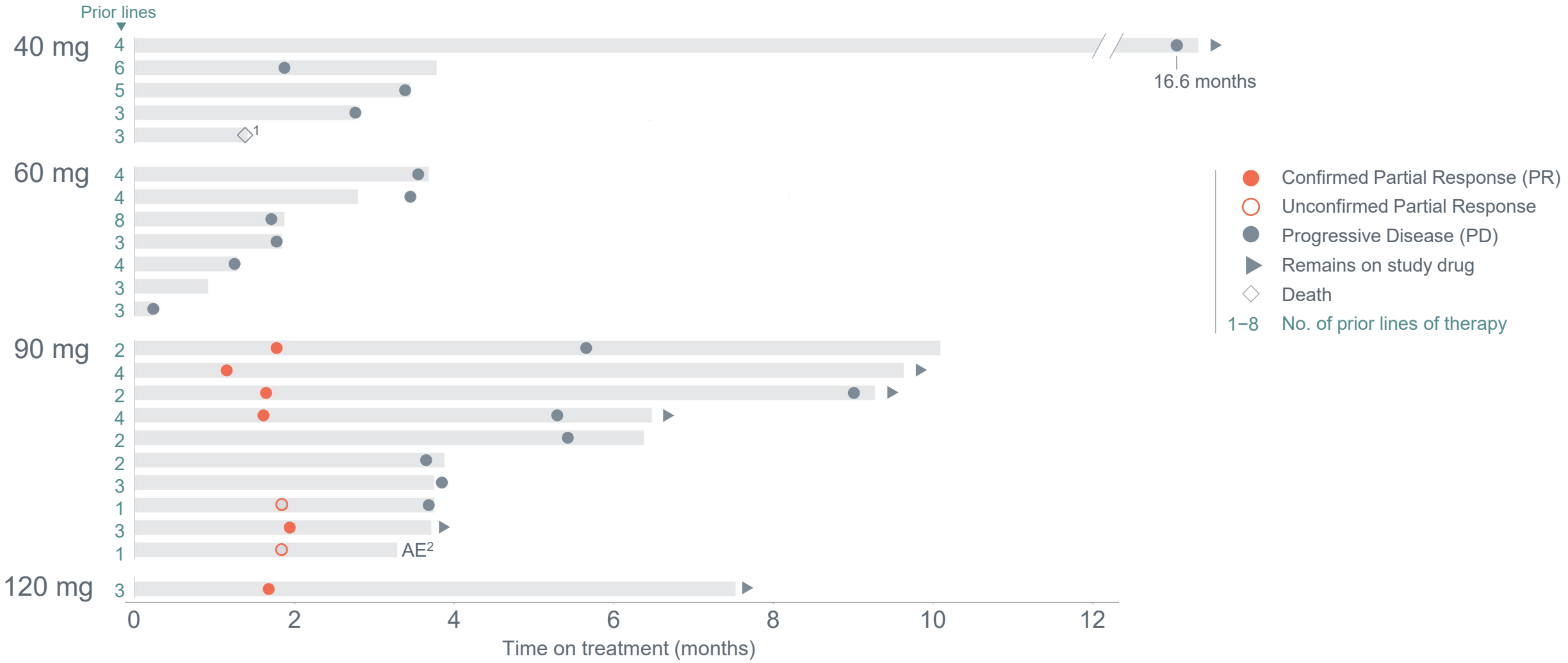
- 5 confirmed PRs at 90 mg QD (n=10)
- 1 confirmed PR at 120 mg QD (n=1)

100% Disease Control Rate



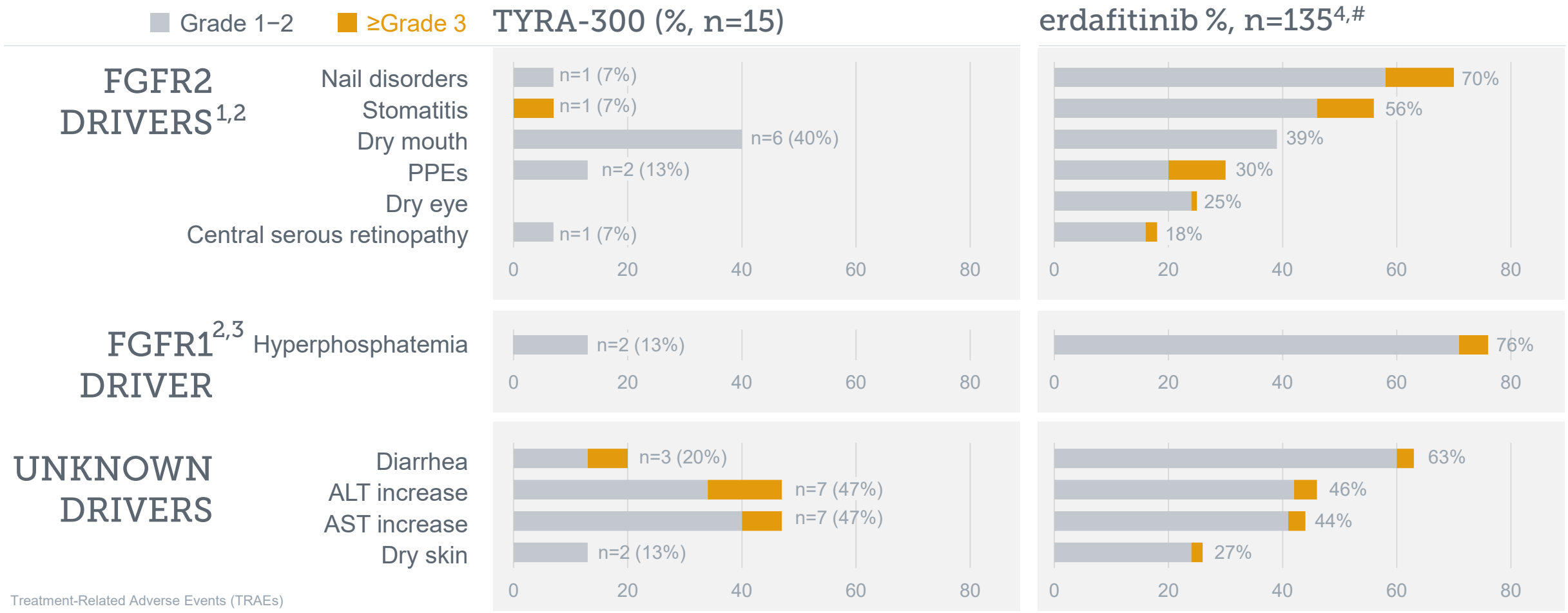
Abbreviations: BOR, best overall response; CR, complete response; PR, partial response; SD, stable disease. Only confirmed PRs reported.
Disease Control Rate: CR+PR+SD

Time on treatment for target population, *FGFR3*+ mUC



1. Death unrelated to study drug (Respiratory Syncytial Virus)
2. AE refers to adverse event

At 90mg QD, improved tolerability observed compared to erdafitinib



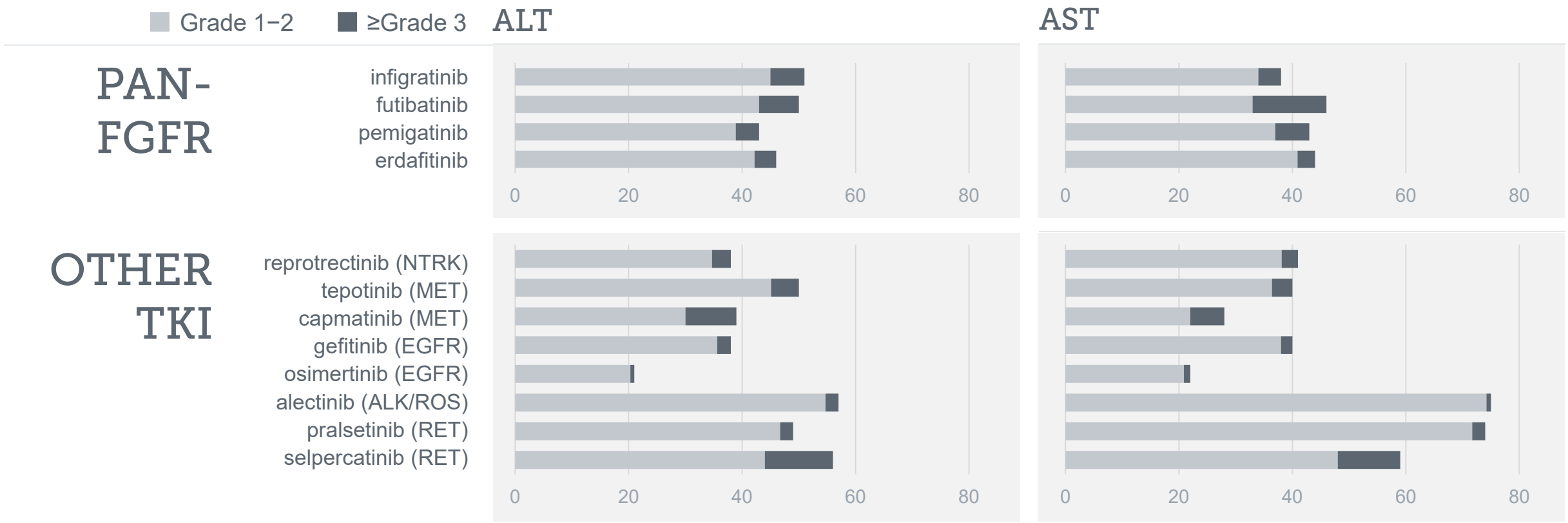
Treatment-Related Adverse Events (TRAEs)

[1]Lacouture ME, et al. Oncologist. 2021. [2]Subbiah V, Verstovsek S. Cell Rep Med. 2023. [3]Kommalapati A, et al. Cancers. 2021. [4]BALVERSA® (erdafitinib) prescribing information 01/2024; [#]BLC3001 Cohort 1 data, Adverse reactions leading to dosage interruptions or reductions of erdafitinib in >4% of patients

Results are preliminary based on the emerging data from the ongoing Phase 1 portion of the SURF301 study

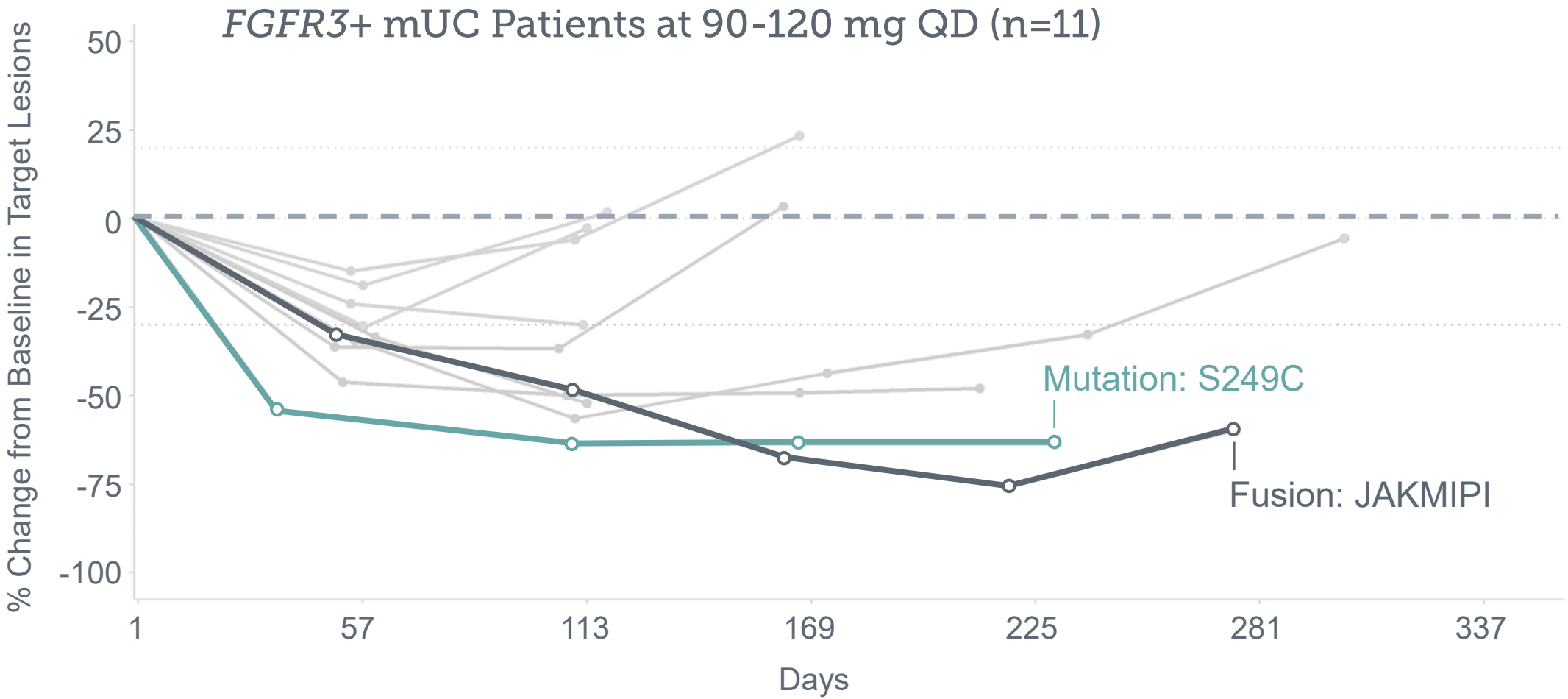
90 mg QD: Dose reduction, n = 4; Discontinuation, n = 1

As a class, TKIs for solid tumors have high rates of LFT increases[1]



[1]Liver function tests (LFTs) including alanine aminotransferase (ALT) and aspartate aminotransferase (AST) from current labels of tyrosine kinase inhibitors (TKIs) as found on https://www.accessdata.fda.gov/scripts/cder/daf/ (11/25/24)

Radiographic regression seen at first imaging



Based on Investigator Assessment; RECIST v 1.1

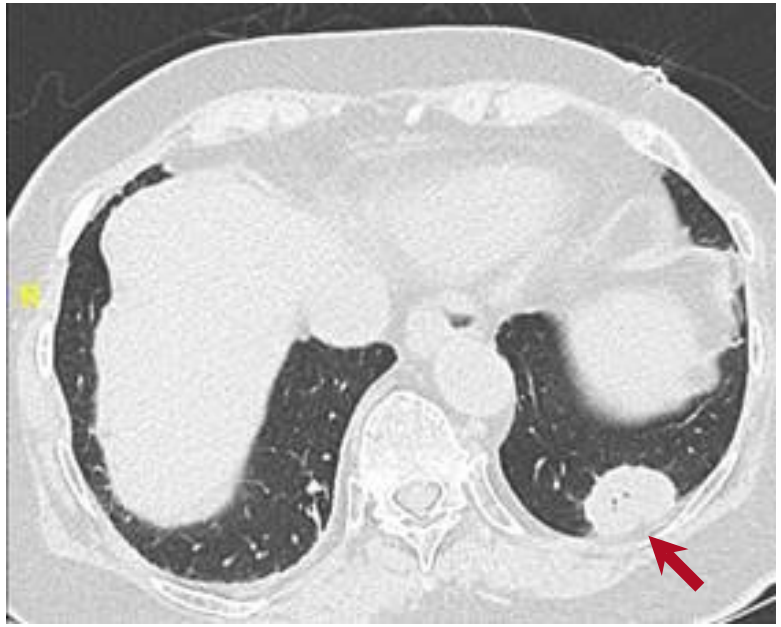
Case study: mUC with activating *FGFR3*^{S249C} mutation

Mutation

Age/sex:	84-year-old female
Alteration:	FGFR3 S249C
Prior tx:	4 prior lines
Target lesions:	Lung
NTL:	Lung, bone
BOR:	-64% (cPR)
Treatment:	90 mg QD, 11 mo.*

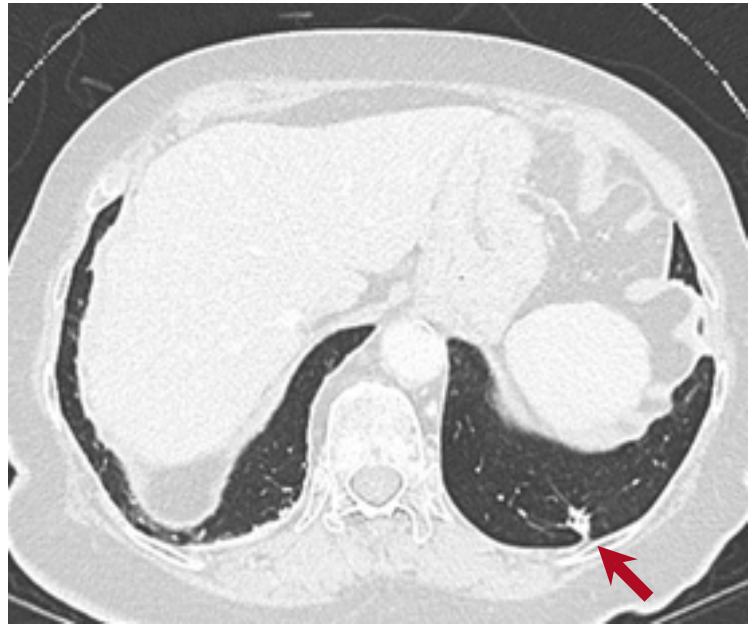
LUNG TARGET LESION

SEPT 25, 2023



Baseline

JAN 15, 2024



Confirmed PR

* Treatment ongoing at time of data cut
BOR: Best Overall Response; cPR: Confirmed Partial Response; NTL: Non-Target Lesion; tx: Therapy

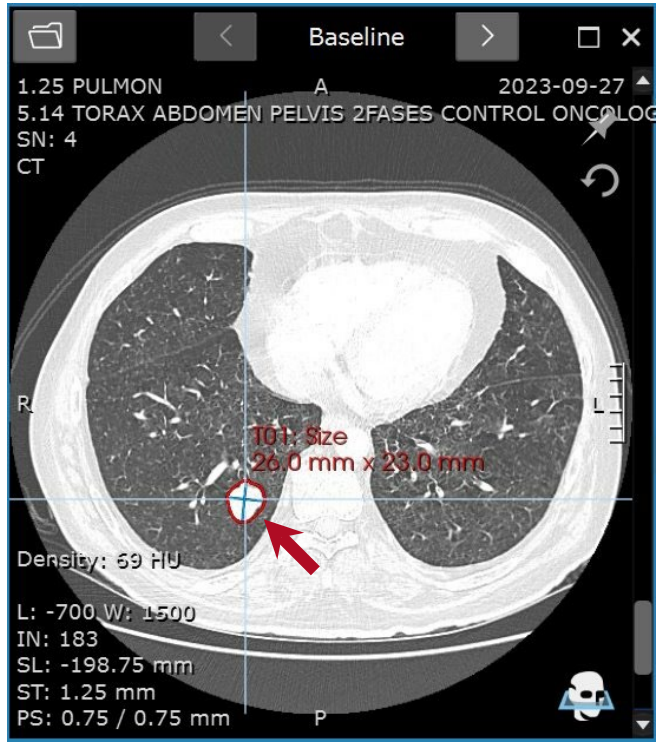
Case study: mUC with activating *FGFR3-JAKMIPI* fusion

Fusion

Age/sex:	64-year-old male
Alteration:	FGFR3 JAKMIPI
Prior tx:	2 prior lines
Target lesions:	lung (x2), LN (x2)
NTL:	lung (x2) and LN (x2)
BOR:	-75% (cPR)
Treatment:	90 mg QD, 11 mo.*

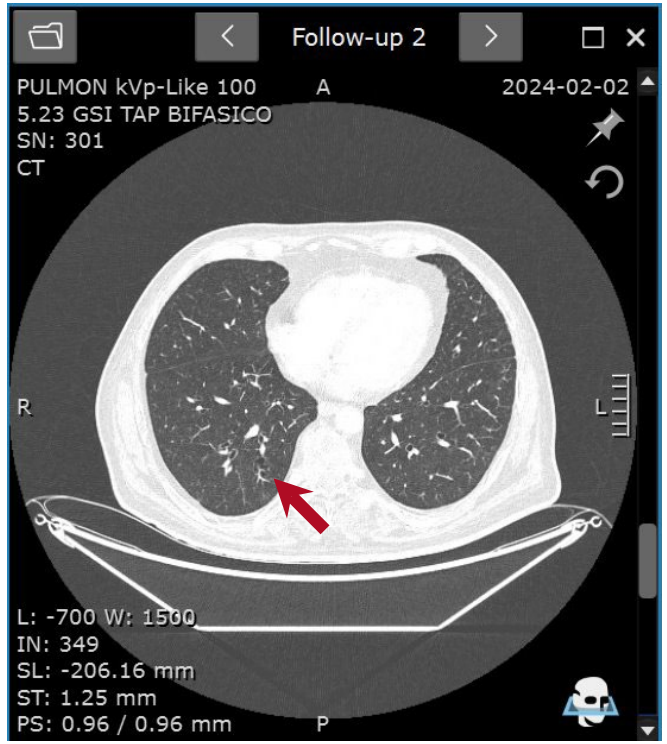
LUNG TARGET LESION

SEPT 27, 2023



Baseline

FEB 2, 2024



Confirmed PR

* Treatment ongoing at time of data cut

BOR: Best Overall Response; cPR: Confirmed Partial Response; NTL: Non-Target Lesion; tx: Therapy

Interim clinical proof-of-concept established with TYRA-300 in mUC

1. Preliminary data from SURF301 suggest TYRA-300 to be generally well-tolerated, with infrequent FGFR2- and FGFR1-associated toxicities.

2. TYRA-300 plasma concentrations indicate adequate target coverage at ≥90 mg QD; further pharmacokinetic characterization is ongoing.

3. Preliminary anti-tumor activity of TYRA-300 in heavily pre-treated patients is encouraging, especially at doses ≥90 mg QD.

Phase 1 is ongoing and the MTD was not reached; the optimal dose is yet to be determined. Emerging data warrants continued development in mUC, prioritizing QD dosing.

LOOKING AHEAD: TYRA-300

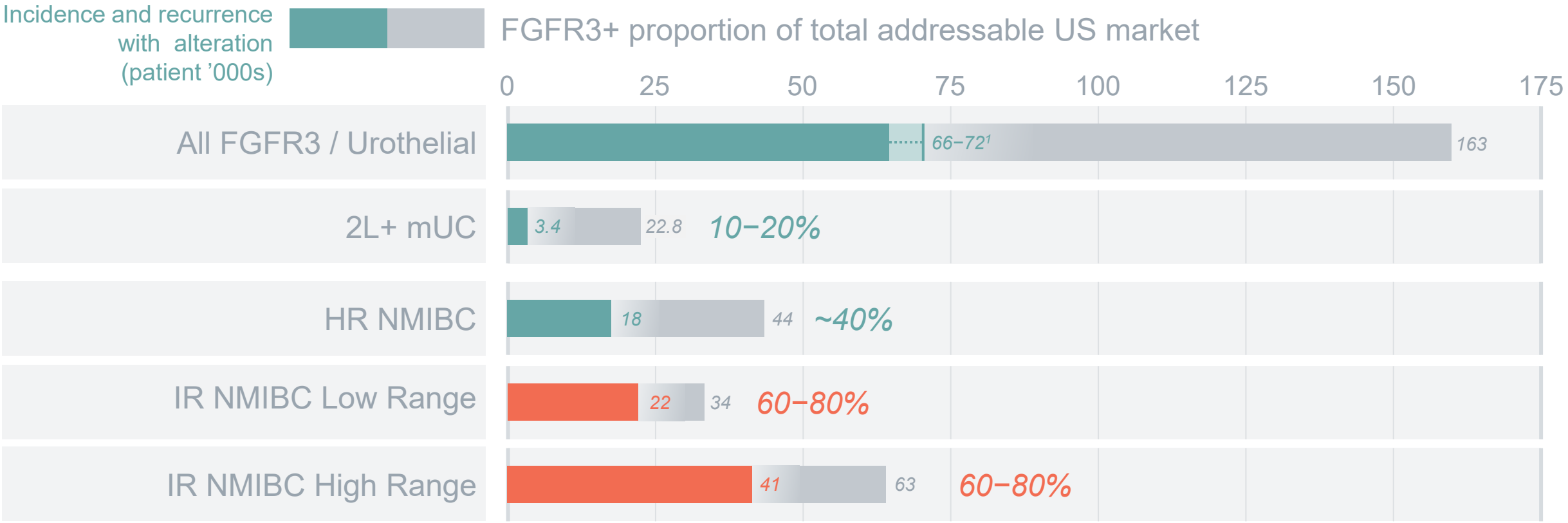
mUC	Improved toxicity profile
NMIBC	Patient-friendly oral
ACH	Differentiated efficacy

Attractive market opportunities for TYRA-300

	2L+ mUC	NMIBC	ACH
Total addressable (incident and recurrent) FGFR3+ Market Size	US: ~3.4K	IR US: ~22−41K	US: ~3K Global: ~20K
Unmet Needs	Less FGFR1/2/4-related and other toxicities	Reduction in recurrence Oral administration vs TURBT + chemotherapy	Differentiated efficacy Oral administration vs daily injection
SURF301 Dataset Read Through	Improved tolerability	Generally tolerable at exposures ~2/3[1] of the mUC dose	Generally tolerable at <50%[2] of the mUC dose

1. Estimated based on 6mg oral erdafitinib dose studied in THOR-2 for NMIBC
2. Estimated based on infigratinib preclinical 2mg/kg model demonstrating efficacy at a dose translating to <50% of dose tested in mUC

High FGFR3+ rate drives an outsized opportunity in IR NMIBC



1. All FGFR3/Urothelial includes Low Risk NMIBC, which is not included in addressable population lines below

FGFR3+ Rate Sources: Knowles, 2020; Mayr, 2022; Kacew, 2020, Weickhardt 2022; MIBC, mUC; MIBC and mUC Epidemiology Source: DR/Decision Resources LLC 2023 Epidemiology Figures

NMIBC Epidemiology Source: CancerMPact® Patient Metrics, Oracle Life Sciences. Available from cancermpact.lsapps.oracle.com. Accessed 18 Sep 2024; Low range driven by Ravvaz, 2019, Caputo, 2020, Check, 2019, Ritch, 2020, Lyall, 2023; High Range driven by Vedder, 2014

Erdafitinib demonstrated efficacy… but also toxicity at a lower dose

THOR-2 TRIAL IR NMIBC

Erdafitinib 6mg (vs. 8 or 9mg in mUC trial)

Design allowed for up to 2 years of Tx

Cohort 3 n=18

CR Rate: 83% (15 of 18)

DOR: 12.7 months (median)

Tx duration: 7.1 months (median)

	ANY GRADE	≥ GRADE 3
Most common AEs	%	%
>1 AE	**100**	**22.2**
≥1 TRAE	**100**	**16.7**
Hyperphosphatemia	100	
Dry mouth	72.2	
Diarrhea	61.1	5.6
Dysgeusia	50	
Dry skin	38.9	
PPE syndrome	33.3	
Fatigue	33.3	
Abdominal pain	16.7	5.6
Gastritis	5.6	5.6

Source: Daneshmand, 2023 (SUO)

Safety readthrough at lower doses: FGFR-related toxicities were infrequent

No hyperphosphatemia at ≤60 mg

No discontinuations or reductions at ≤ 60 mg

TRAEs in >10% of all participants, n (%)

	≤60 mg (n=22)		90 mg (n=15)		120 mg (n=4)		All (n=41)	
	Gr. 1-2	*≥Gr. 3*	*Gr. 1-2*	*≥Gr. 3*	*Gr. 1-2*	*≥Gr. 3*	*Gr. 1-2*	*≥Gr. 3*
ALT increase	1 (5)	—	5 (33)	2 (13)	2 (50)	—	8 (20)	2 (5)
Diarrhea	4 (18)	—	2 (13)	1 (7)	2 (50)	—	8 (20)	1 (2)
Dry mouth	3 (14)	—	6 (40)	—	—	—	9 (22)	—
AST increase	—	—	6 (40)	1 (7)	1 (25)	—	7 (17)	1 (2)
Dry skin	2 (9)	—	2 (13)	—	2 (50)	—	6 (15)	—
Fatigue	2 (9)	—	2 (13)	—	1 (25)	—	5 (12)	—

Results are preliminary based on the emerging data from the ongoing Phase 1 portion of the SURF301 study.
ALT, alanine aminotransferase; AST aspartate aminotransferase; TRAE, treatment-related adverse event

We are continuing QD dose optimization and advancing toward P2

SURF301

Dose Escalation

Phase 1 Part A i3+3 design

- Advanced solid tumors (must have exhausted all standard therapies)
- Prior FGFRi allowed

Dose Expansions

Phase 1 Part B

- Advanced solid tumors with activating *FGFR3* alterations; focus on mUC
- Prior FGFRi allowed

QD (mg)	
n= 4	120
n= 5	90
n= 3	60
n= 3	40
n= 1	20
n= 1	10

QD (mg)	
	120
n= 10	90
n= 7	60
n= 7	40

ADDING 100mg QD COHORT

UP NEXT

Phase 1

- Testing 100mg QD

Phase 2

- Advancing regulatory interaction to open Phase 2 portion of the study
- Planning to test 90mg and 1 additional dose to satisfy Project Optimus

Our goals for TYRA-300 in mUC, NMIBC and ACH

mUC	Improved tolerability profile for 2L+ mUC in larger Phase 2 study	*Further QD dose optimization*
NMIBC	A patient-friendly oral alternative to IVE therapies for NMIBC	*Submit Phase 2 IND by YE 24*
ACH	AHV changes leading to differentiated final adult height and functional improvements	*First child dosed in Q1 2025*

TYRA

TYRA-300^{ACH}

TYRA-300^{ONC}

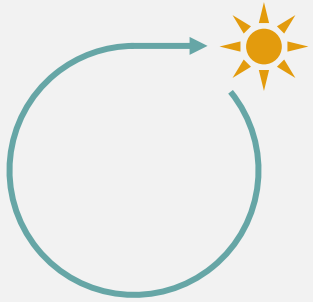
SNÅP

Our rapid approach to discovery continues to generate promising candidates such as TYRA-200

Our unconventional approach accelerates discovery

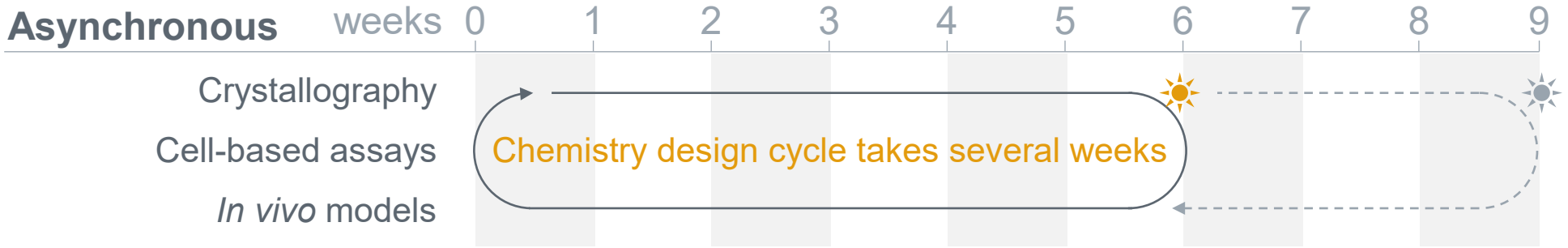
DESIGN CYCLE

Chemistry design

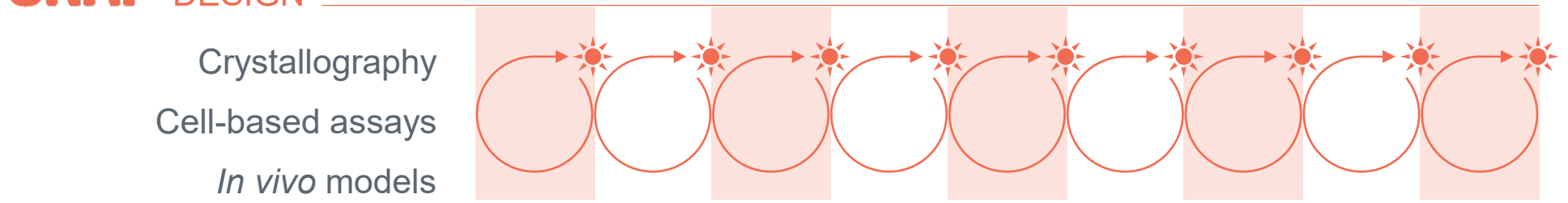


Crystallography
Cell-based assays
In vivo models

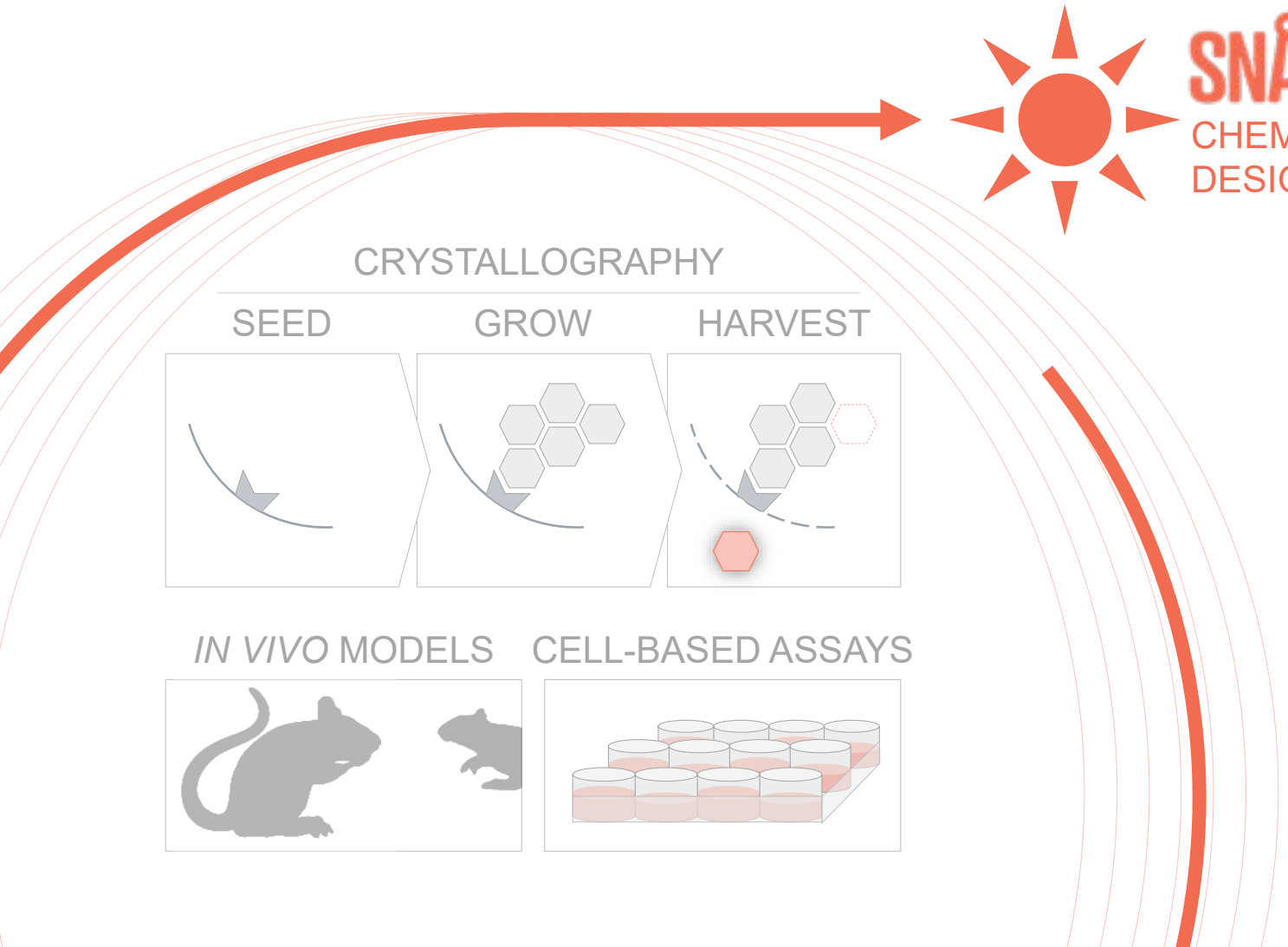
Asynchronous



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

EXAMPLE

TYRA-430

Hepatocellular Carcinoma (HCC)

Biomarker-driven, targeted therapeutic approach for HCC

Designed to be FGFR4/3 biased and gatekeeper- and cysteine-mutant agnostic

Prior FGFR4 approaches show promise but limited durability

Hepatocellular carcinoma (HCC)

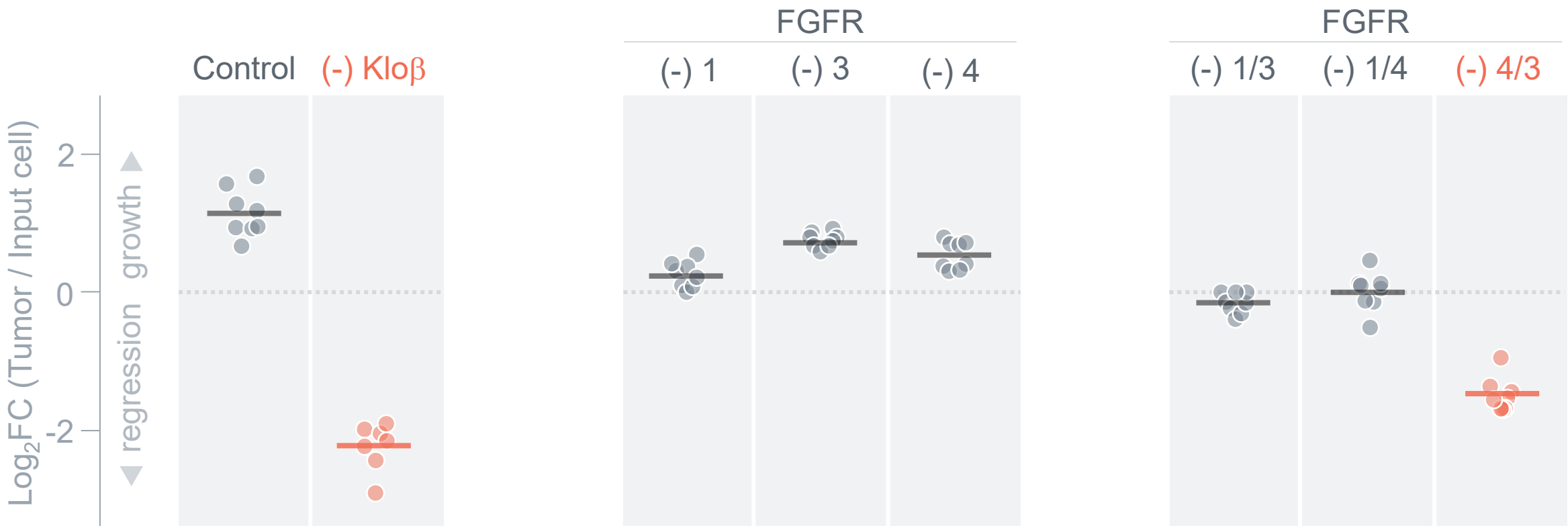
	FGF19 Positive	FGF19 Negative
BLU-554[1]	17% ORR 5.3 month mDoR 3.3 month mPFS (2.1-3.7)	0% ORR NO mDoR 2.3 month mPFS (1.8-5.5)
FGF-401[2]	21% ORR	3% ORR

FGF-19 status determined by IHC
Both agents demonstrated dose-limiting toxicities from ALT and AST increases

1. Kim et al, 2019 2. Chan et al, 2022
Note: FGF19 status determined by IHC

Recent data supports the need for dual FGFR4/3 inhibition

Cell-Derived Xenograft Model
(Adapted from Tao et al. PNAS 2022)



Tao et al. Proc Natl Acad Sci U S A. 2022 Oct 4;119(40):e2208844119.

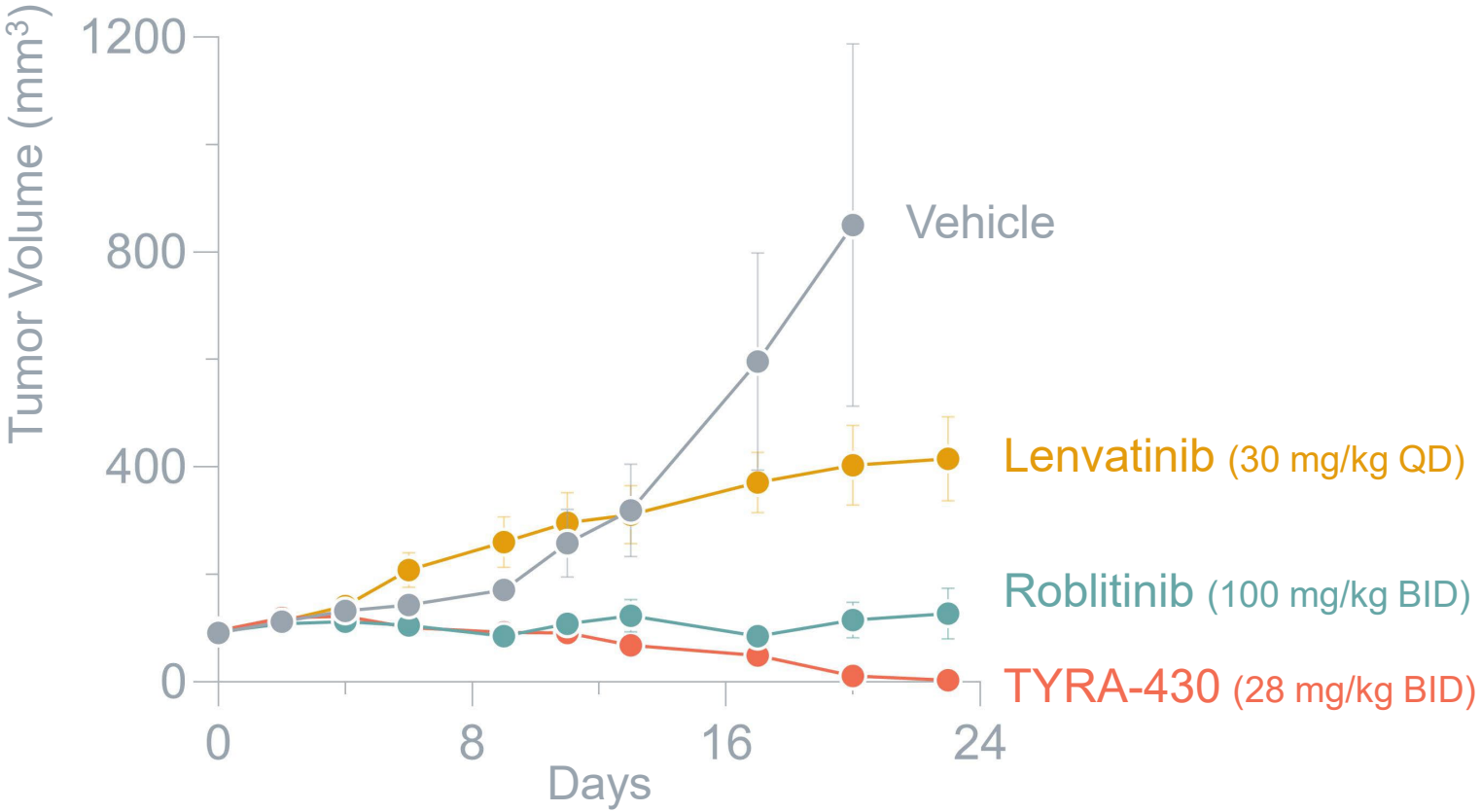
TYRA-430 demonstrated a strong bias for FGFR3 and FGFR4

Selectivity observed for TYRA-430 vs. late-stage clinical compounds: *in vitro* Ba/F3 Cellular IC_{50} (nM)

All experiments conducted under identical conditions, tested in duplicate (average n>10).

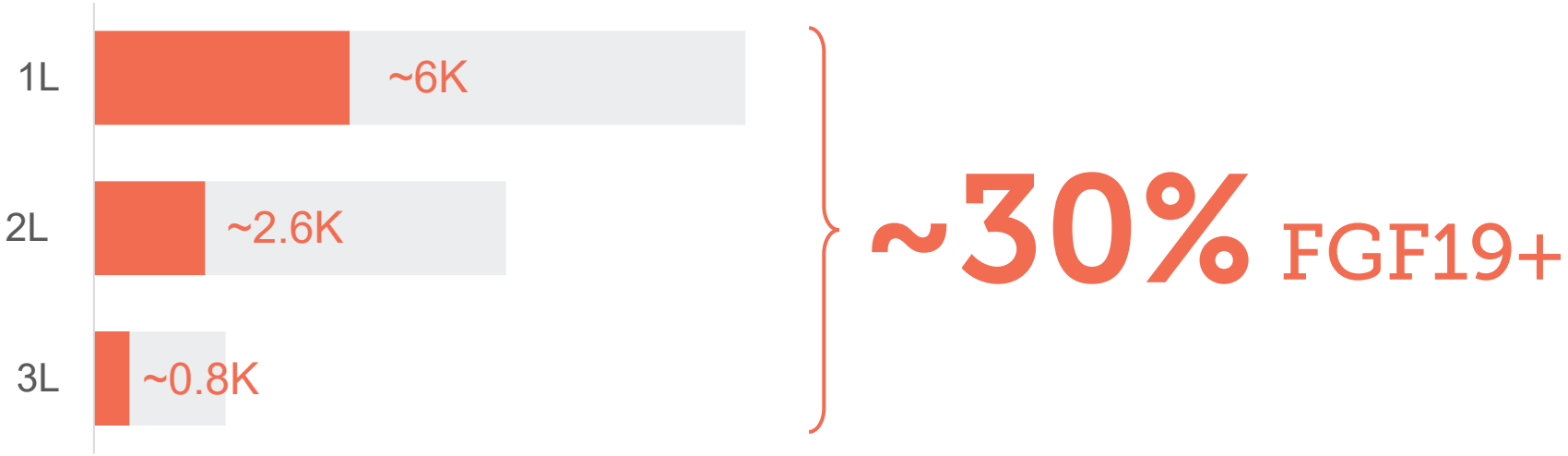
TYRA-430 led to tumor regression in HCC FGF19+ xenograft

JHH7 FGF19+ Hepatocellular Xenograft



~30% of patients with HCC have high FGF19

Estimated 2022 US FGF19+ Addressable Population



Kim, 2019; Clarivate Analytics; GlobalData

There is no biomarker-driven, targeted therapy approved in HCC

	ADDRESSABLE (US)	LEAD OPTION	UNMET NEED
1st Line	~6K	Atezo/Bev Other PD1 combos	Response rate and PFS improvement (27% and 6.8 months for Atezo/Bev)
2nd Line	~2.5K	Sorafenib/Lenvatinib Other MKIs	ORR and PFS improvement (4-7% and 2.8-5.2 months for 2L MKIs)
3rd Line	~0.8K	Cabozantinib Other MKIs	No 3L approvals or de facto SOC

No biomarker-driven, targeted therapy available

Kim, 2019; Clarivate Analytics; GlobalData; Tecentriq label, Cabometyx label, Stivarga label, Cyramza label

Here's a snapshot of 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 Sept 30, 2024: $360.1M

RECENT PROGRESS

TYRA-300^{ACH}: Ph 2 IND cleared by FDA

TYRA-300^{ONC}: interim clinical proof of concept established with TYRA-300 in mUC from SURF301; presented at ENA

TYRA-430^{ONC}: IND cleared by FDA

* ACH: Achondroplasia; ONC: Oncology
All TYRA small molecule inhibitors are for investigational use only

APPENDIX

EXAMPLE

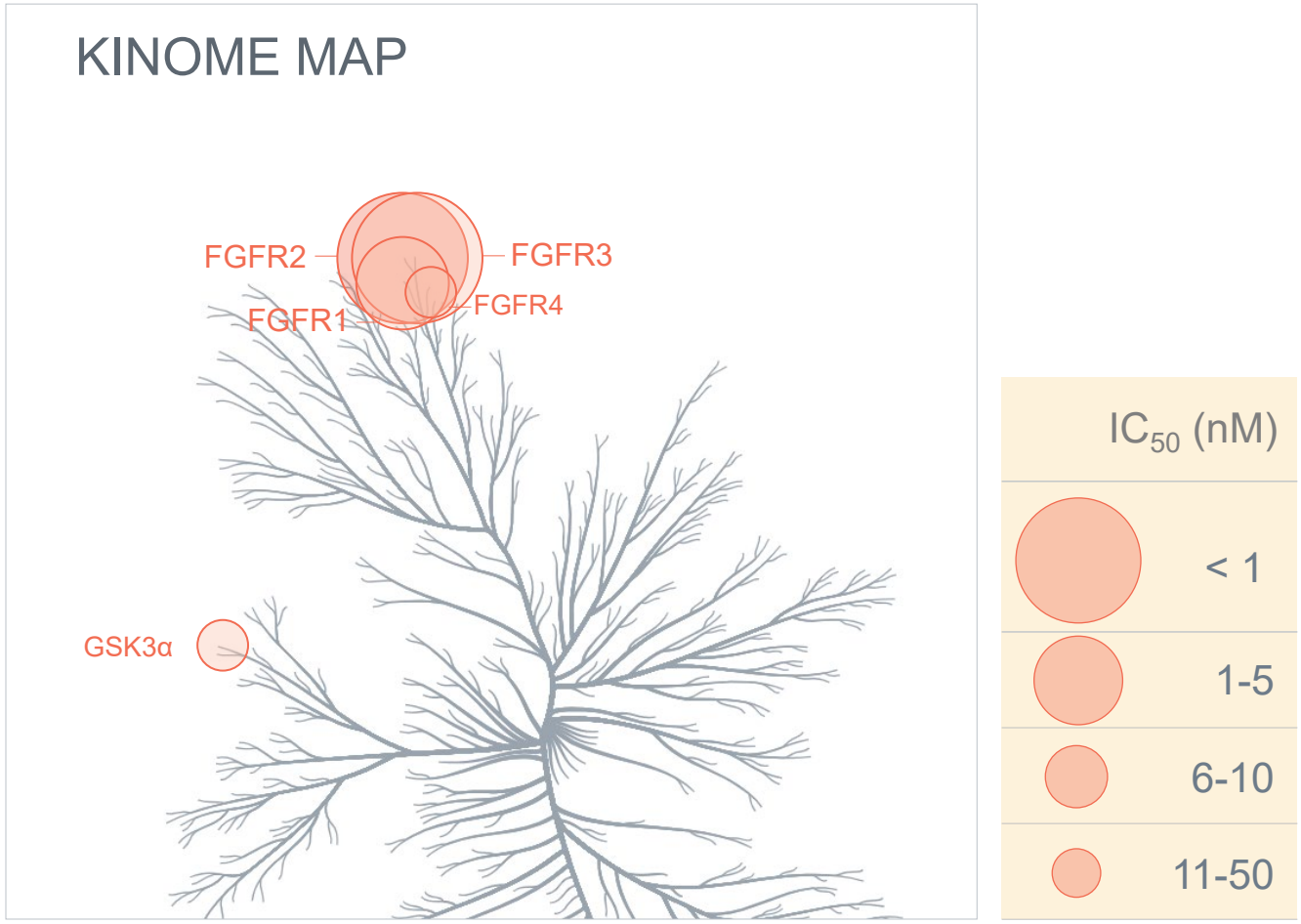
TYRA-200

Intrahepatic cholangiocarcinoma (ICC)

Designed to be gatekeeper- and molecular brake-agnostic and FGFR4-sparing

Potential gateway to additional solid tumor development programs

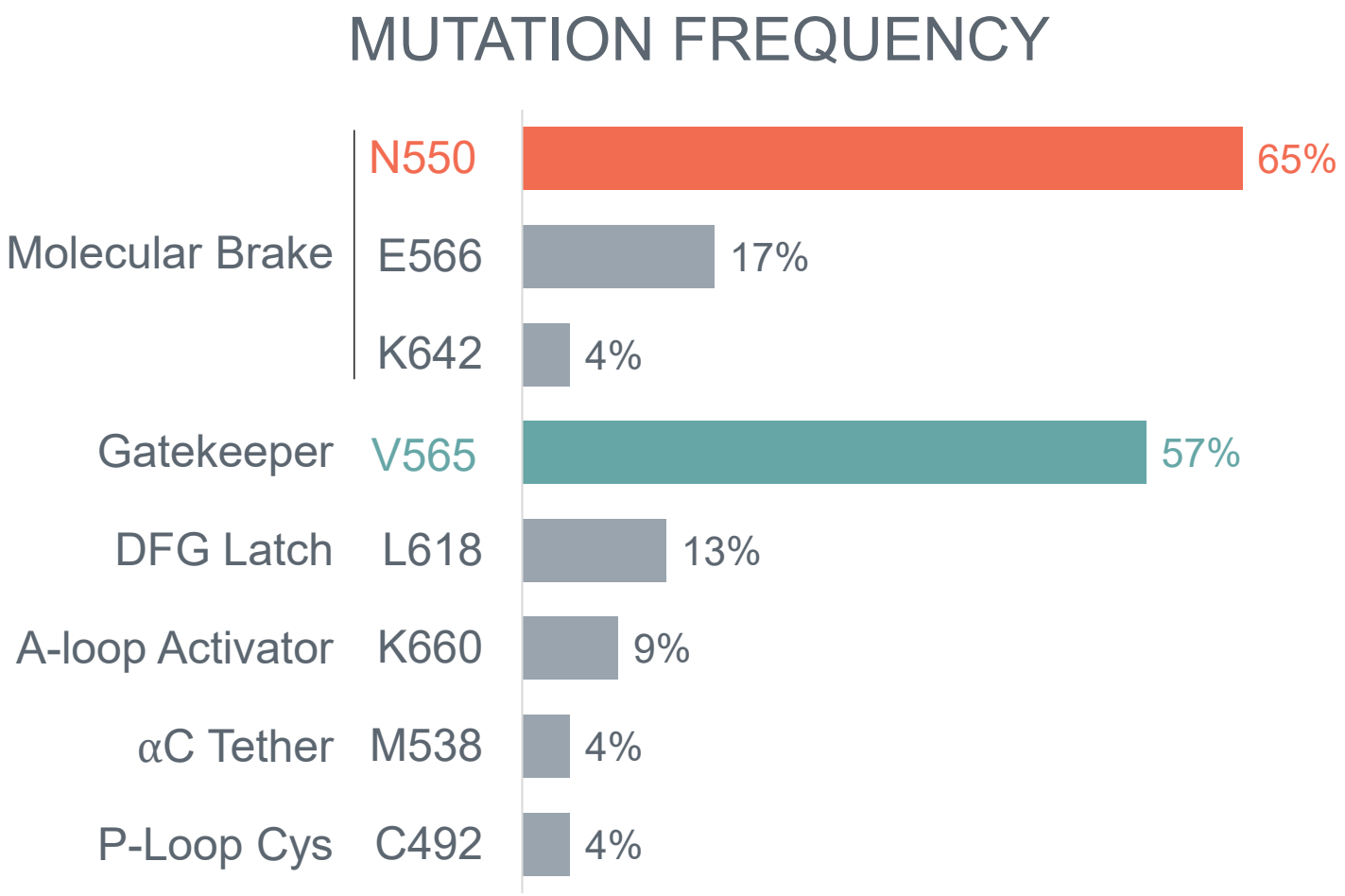
TYRA-200 showed high selectivity for FGFR1/2/3, sparing FGFR4



	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 scanMAXSM (KINOMEscan) screen, IC50 data generated by Reaction Biology Inc.

Polyclonal acquired drug resistance occurs often in FGFR2



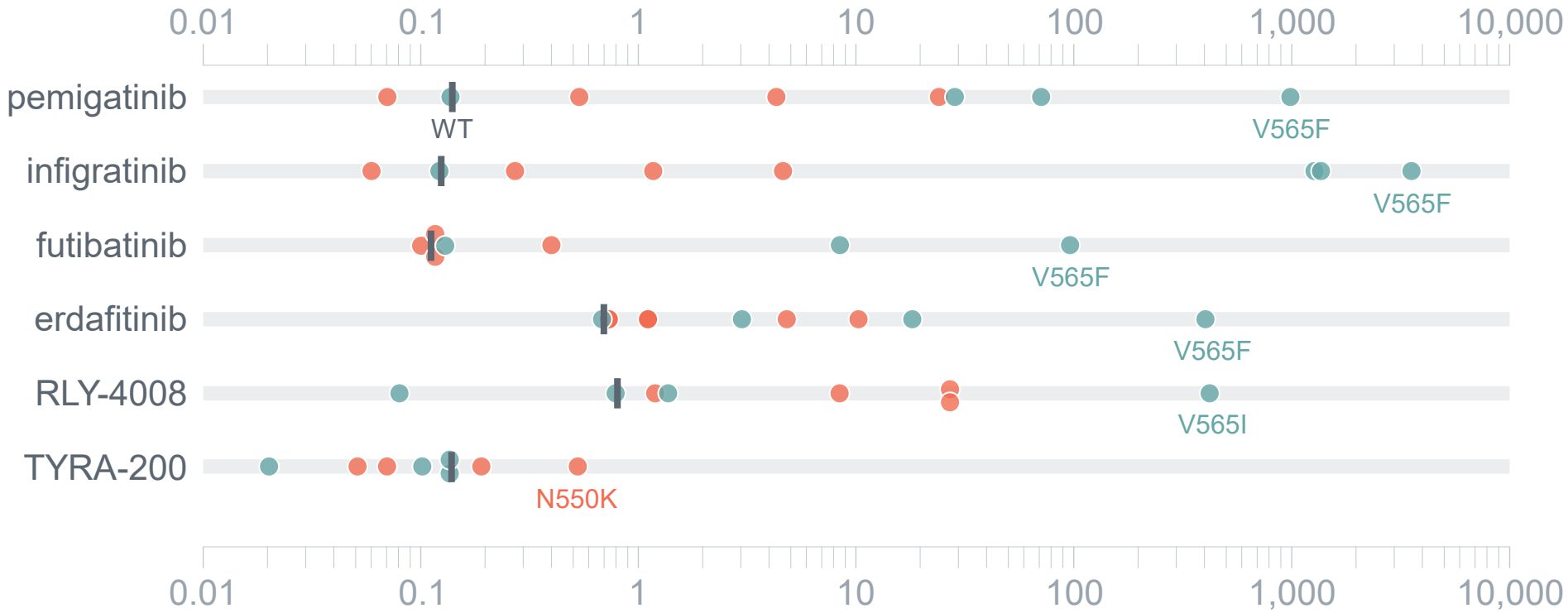
Over 50% of patients who recur on FGFR therapy have gatekeeper and molecular brake mutations

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

TYRA-200 retained potency for key acquired resistance mutations

In Vitro Enzymatic IC_{50}, (nM, Log_{10})

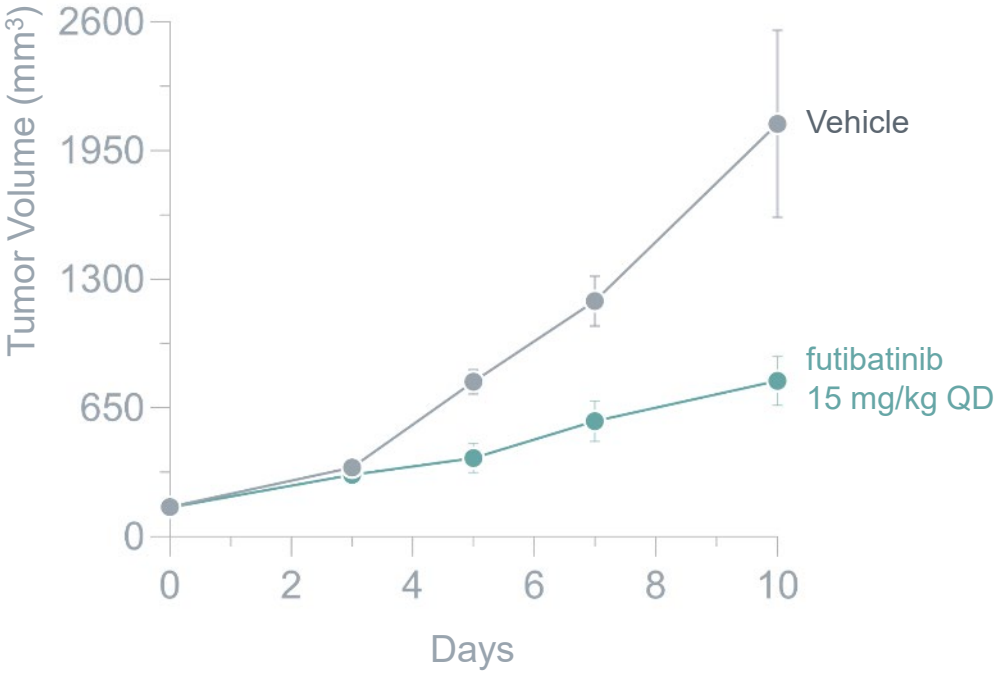
Variants tested: **WT, N550D, N550H, N550K, N550T, V565F, V565L, V565I**



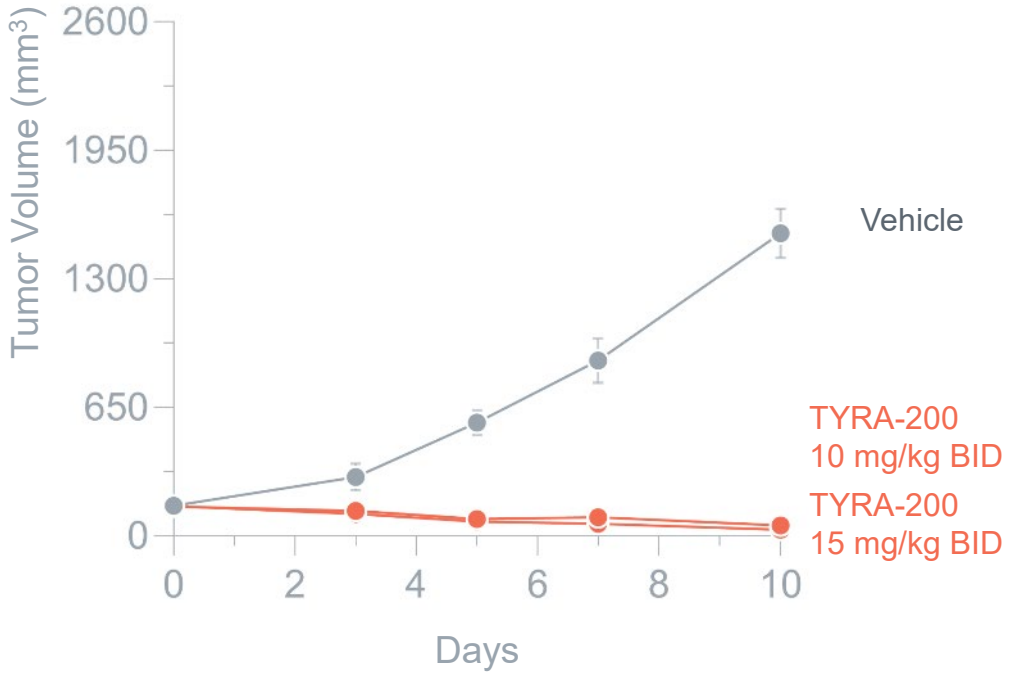
Enzymatic IC50 measurements generated at Reaction Biology Corp using Tyra enzymes.
All experiments conducted under identical conditions, tested in duplicate.

TYRA-200 regressed tumors with gatekeeper mutations

Ba/F3 FGFR2 V565F Allografts

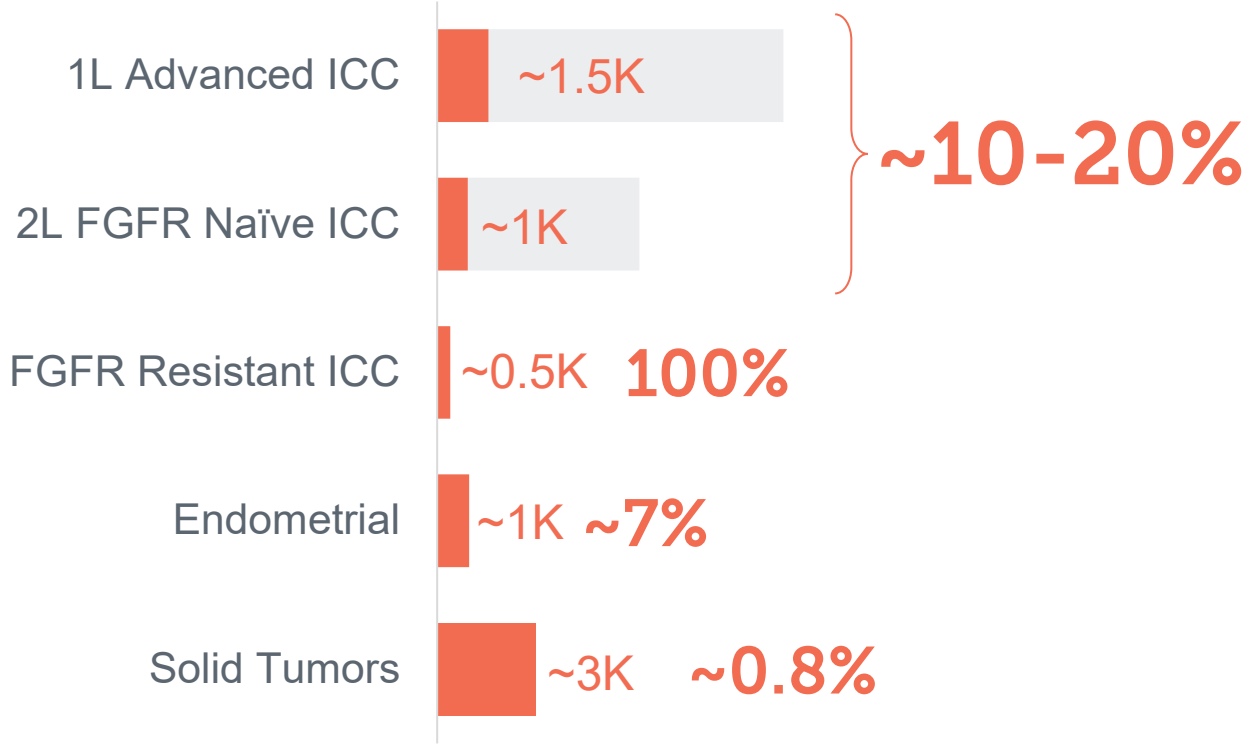


Ba/F3 FGFR2 V565F Allografts



TYRA-200 has multiple opportunities across FGFR2+ solid tumors

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

	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 focuses on FGFR resistance patients

Illustrative

What is the MTD?

What is the RP2D?