TYRA

Corporate Deck

May 2026

Disclaimers

Forward-looking statements and market data

We caution you that this presentation contains forward-looking statements. 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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.

Gearing up for potential pivotal trials in 3 blockbuster indications

Validated Target

FGFR3 alterations drive conditions with high unmet needs

Differentiated Molecule

Dabogratinib: the first oral, once-daily, highly selective FGFR3 inhibitor with clinical PoC (N > 100)

3 Potential Blockbuster Indications

	EXPECTED
IR NMIBC – Initial 3mo CR data	August ‘26
ACH – Initial results, safety sentinel cohort	4Q26
LG UTUC – Initial results	2027

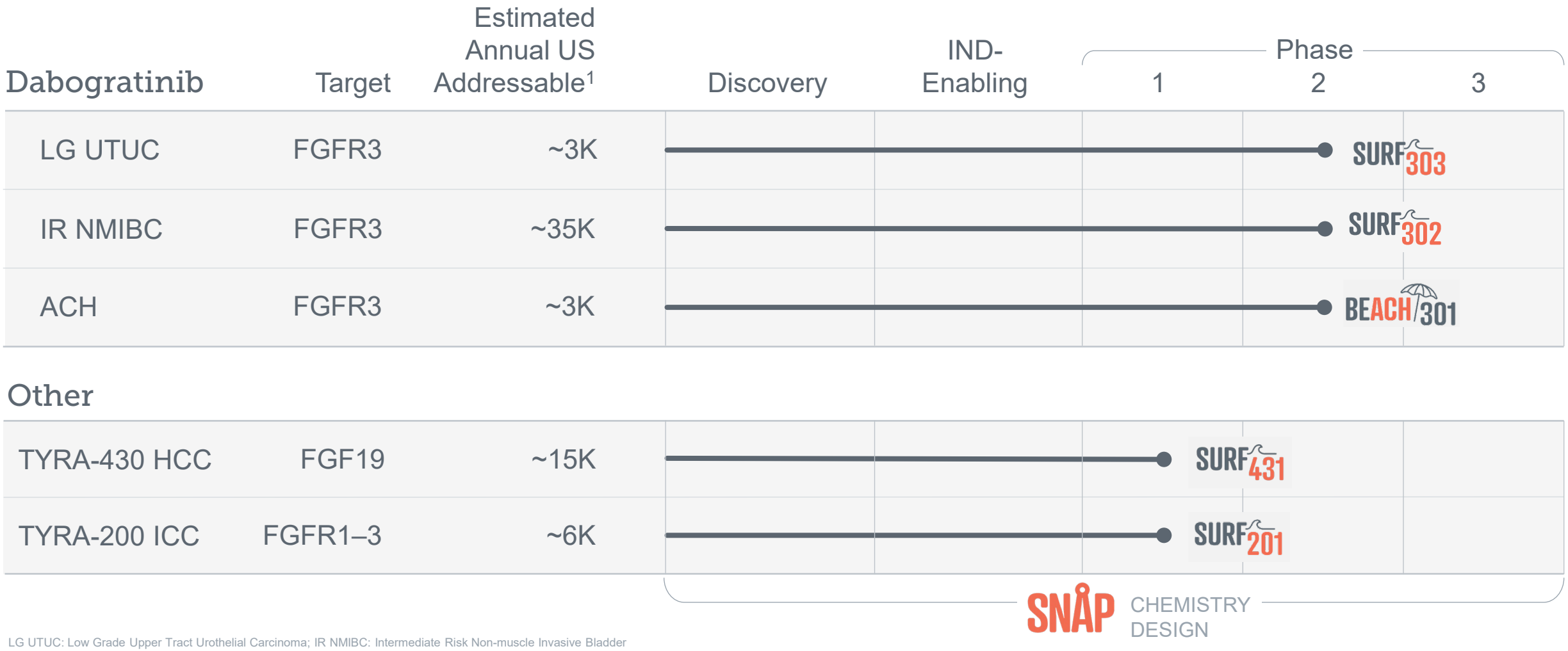
NASDAQ: TYRA

CASH: $383.5M (As of 1Q26)

DABO3^3

ACH: Achondroplasia; IR NMIBC: Intermediate Risk Non-Muscle-Invasive Bladder Cancer; LG UTUC: Low Grade Upper Tract Urothelial Carcinoma
All TYRA small molecule inhibitors are for investigational use only

Our expertise in FGFR biology creates a differentiated pipeline



LG UTUC: Low Grade Upper Tract Urothelial Carcinoma; IR NMIBC: Intermediate Risk Non-muscle Invasive Bladder Cancer; ACH: Achondroplasia; HCC: Hepatocellular Carcinoma; ICC: Intrahepatic Cholangiocarcinoma

1. Represents FGFR3/FGFR2/FGF19+ incidence and recurrences for ONC and prevalence for ACH FDA clearance for dabogratinib Phase 2 INDs in ACH, IR NMIBC and LG UTUC based on Ph1 data from SURF301

TYRA retains an active FGFR3 discovery program

TYRA

FGFR3 drives large unmet need

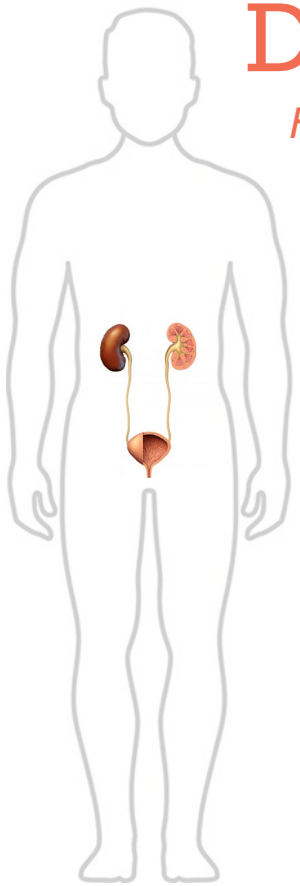
TYRA's highly selective FGFR3 inhibitor

Our development paths in Urology and ACH

FGFR3 drives unmet need in multiple large market opportunities

Dabogratinib

Has the potential to address these indications



TOTAL UROTHELIAL CARCINOMA (UC)

~45% FGFR3 | ~80,000/yr (US)

INTERMEDIATE RISK NMIBC

~70% FGFR3 | ~35,000/yr (US)

LOW GRADE UTUC

~85% FGFR3 | ~3,000/yr (US)

TOTAL FGFR3-RELATED SKELETAL DYSPLASIA

>40,000/yr (US)

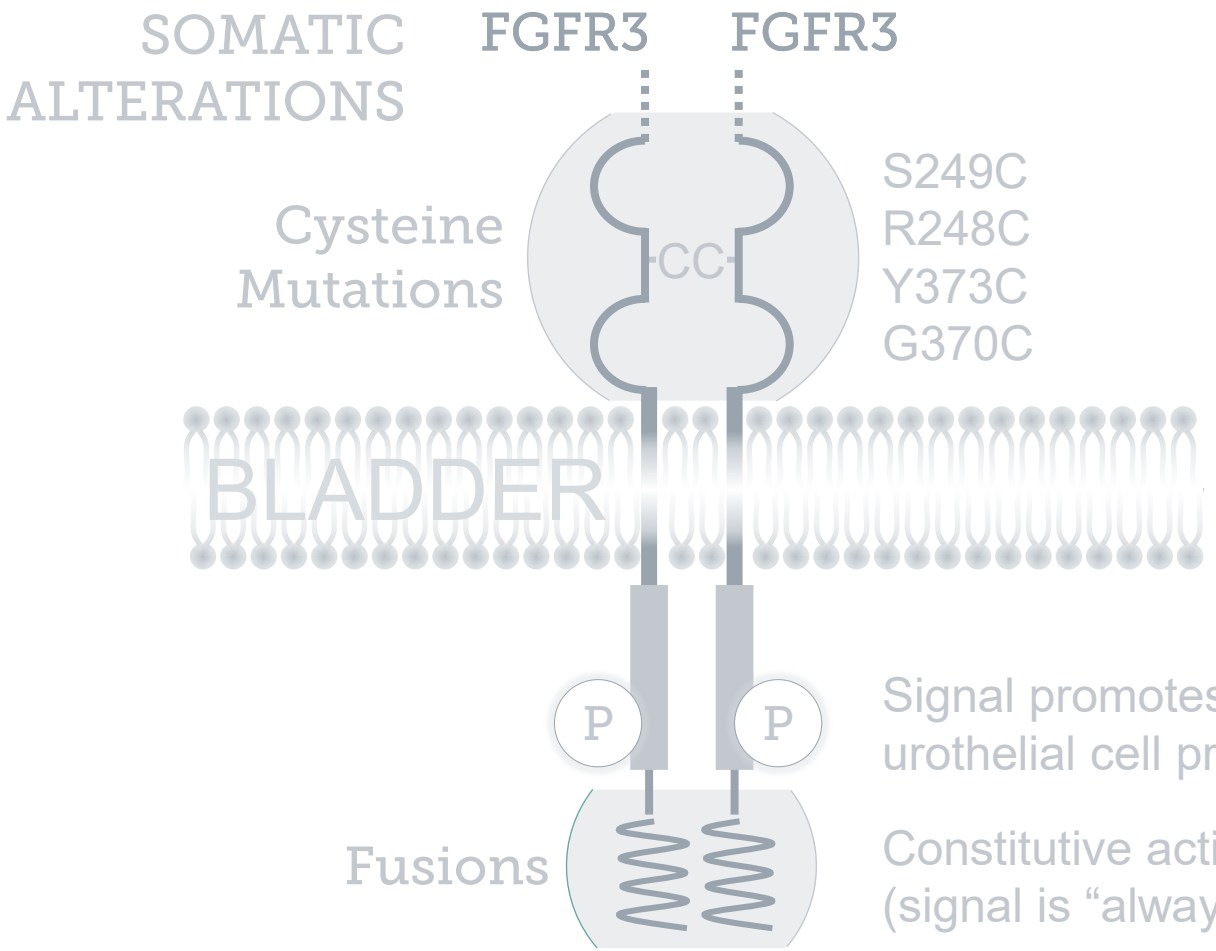
ACHONDROPLASIA (ACH)

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

Oncology figures represent US incidence and recurrences; skeletal dysplasias represent US pediatric prevalence; Sources: US 2024 Census; Vajo, 2000; van Rhijn, 2010; Mayr, 2022; Kacew, 2020; DR/Decision Resources LLC 2025; CancerMPact® Patient Metrics and Oracle Life Sciences analysis; Ravvaz, 2019, Caputo, 2020, Check, 2019, Ritch, 2020, Lyall, 2023; Vedder, 2014; Sfakianos, 2015, Moss, 2017; Nassar 2019

Dabogratinib 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

Pan-FGFR inhibitors have demonstrated activity in FGFR3+ UC

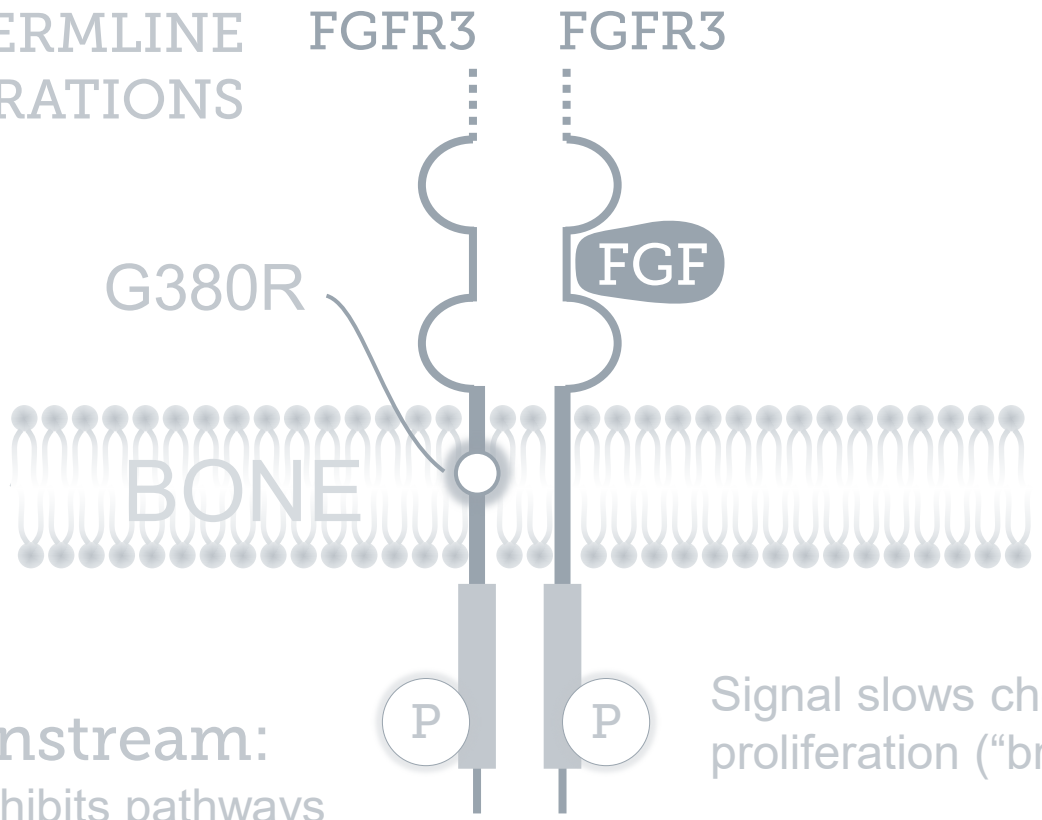


	mUC	IR NMIBC
FGFR3+	15-20%	>70%
Response Rate	35.3% ORR[1]	89% CR[2] (n=18)
Dose	Erdafitinib 8-9 mg	Erdafitinib 6 mg

Abbreviations: IR: Intermediate Risk; CR: Complete Response; ORR: Overall Response Rate

1. BALVERSA® (erdafitinib) prescribing information 01/2024; BLC3001 Cohort 1 data 2. Daneshmand, 2025 (European Urology)

FGFR inhibitors have accelerated bone growth in the clinic



Downstream:
CNP inhibits pathways activated by FGFR3

Signal slows chondrocyte proliferation (“brake”)

Over-activates FGFR3 signaling

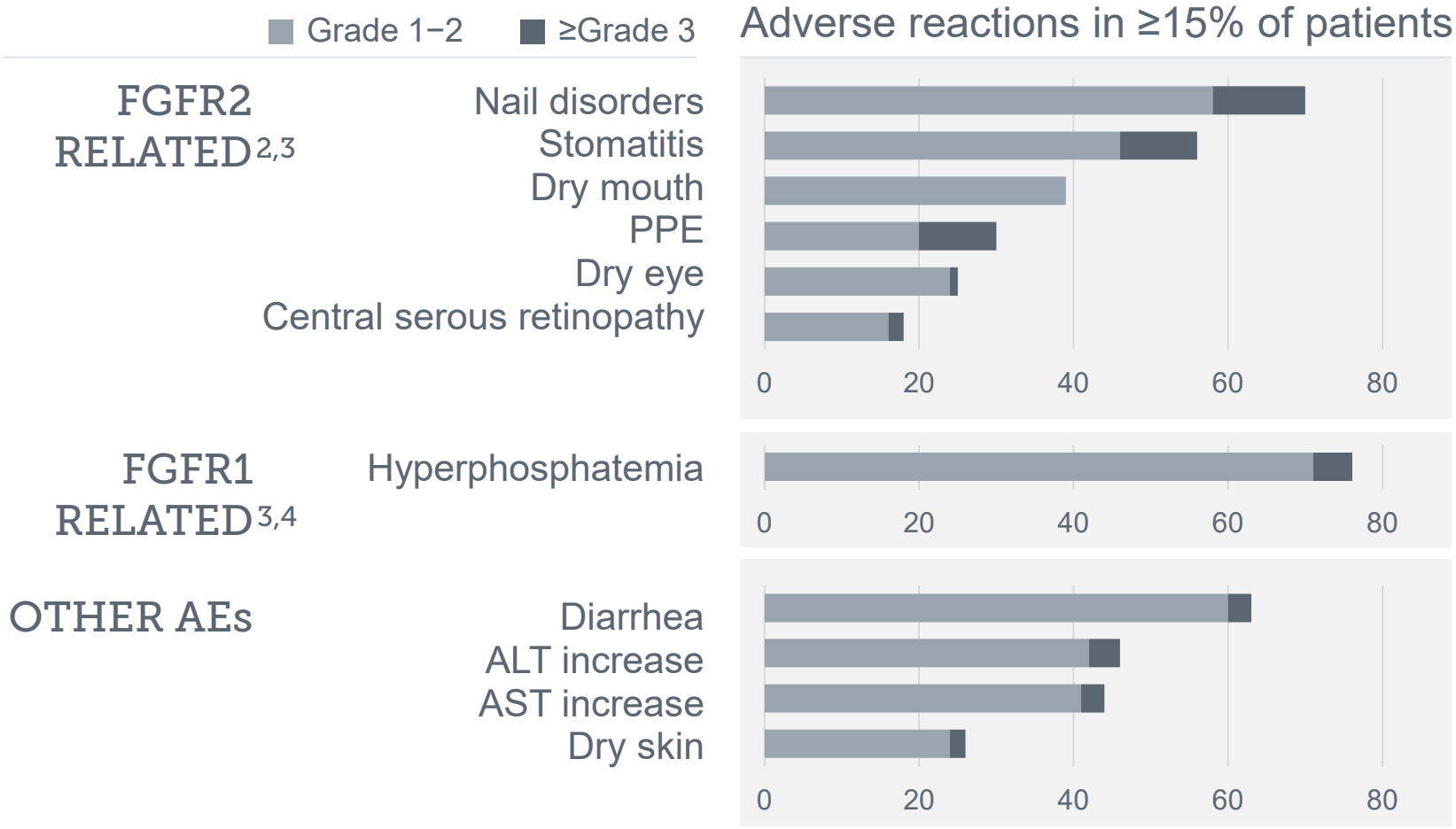
	ACH	PEDIATRIC[1]
AHV Untreated	4.0cm^2	7.6cm^3
AHV Treated	6.0 cm^4	19.2 cm^1
Dose	Low dose infigratinib	Oncology dose erdafitinib or Debio1347

1. Treated for FGFR-altered gliomas; associated with slipped capital femoral epiphyses (SCFE)

1. 19.2 represents AHV for three pediatric oncology case studies with available height velocity data; Sait, 2023; 2. Savarirayan, 2021 (5 to 14yrs); 3. Merck Manuals (12mo to 10yrs); 4. Ph2 Cohort 5 12mo data, Savarirayan, 2024

Pan-FGFR inhibition is associated with key FGFR1/2 toxicities

Adverse reactions in ≥15% of patients who received erdafitinib (n=135)[1]



DOSE REDUCTION[5]

69%

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

1. Adapted from: Erdafitinib tablets, for oral use. Prescribing information 01/2024 (Study BLC3001) 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;
5. 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

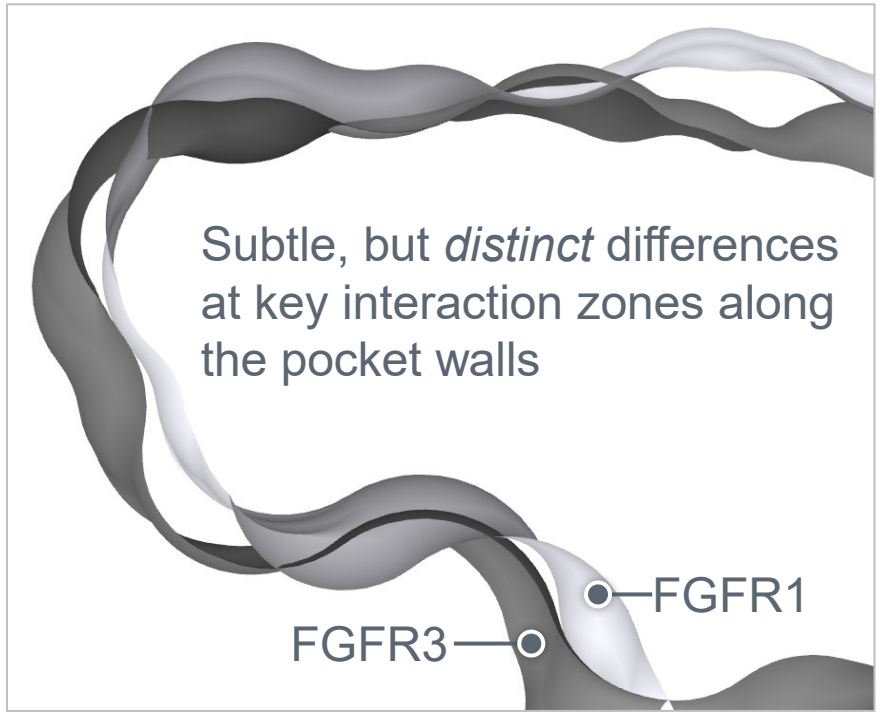
FGFR3 drives large unmet need

TYRA's highly selective FGFR3 inhibitor

Our development paths in Urology and ACH

Our SNAP platform was able to address an intractable problem

FGFR isoform selectivity



MOLECULAR MODEL

SNAP

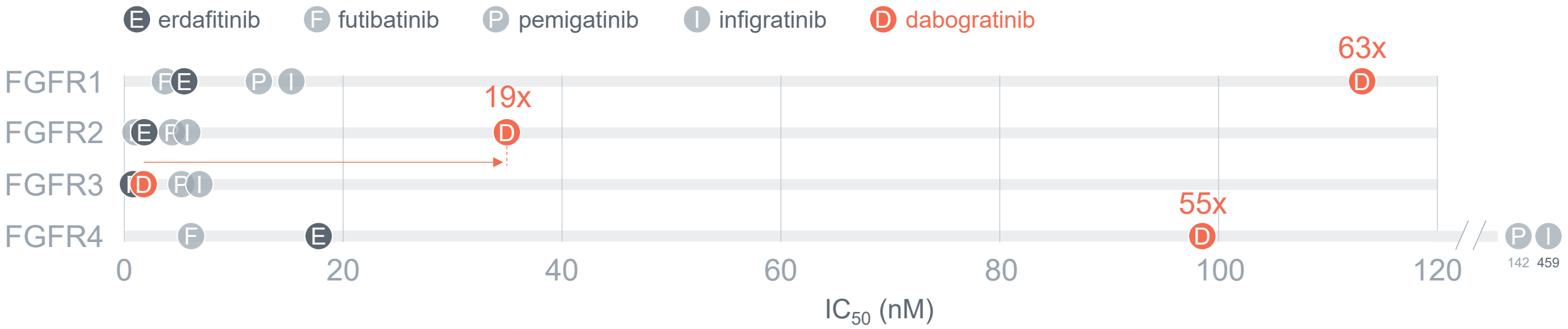
dozens of high-resolution crystal structures

CRYSTALLOGRAPHY

3.0Å cross-sections

Dabogratinib is a potential first-in-class, selective FGFR3 inhibitor

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



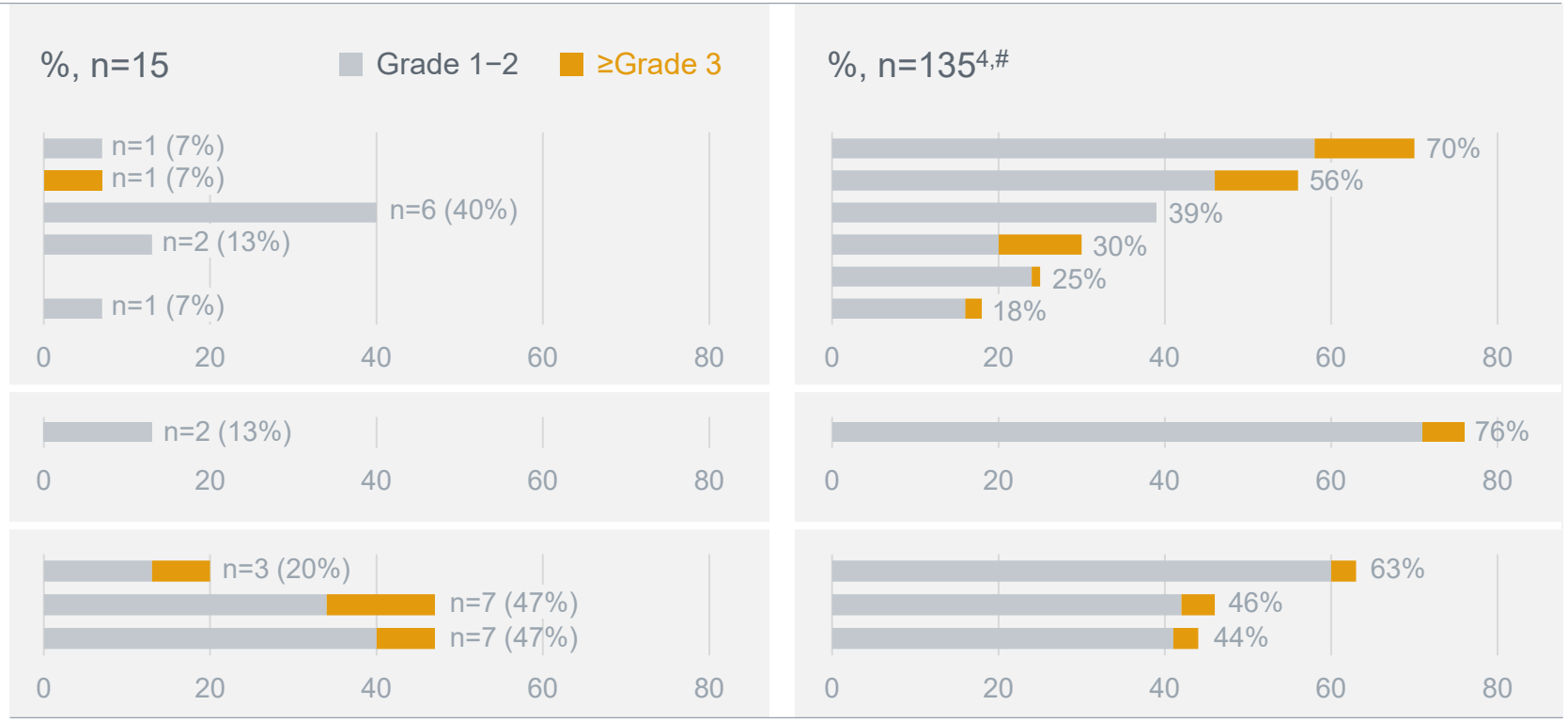
	E	F	P	I
FGFR1	4.2x	4.9x	2.4x	2.2x
FGFR2	1.4x	1.3x	0.8x	0.8x
FGFR4	14x	7.6x	27x	67x

Fold selectivity from FGFR3

All experiments conducted under identical conditions, tested in duplicate.
Note: Dabogratinib FGFR3 selectivity is defined by its activity against FGFR3 relative to its activity against the other individual FGFR isoforms.
Erdafitinib and futibatinib are FGFR1, 2, 3, 4 or Pan-FGFR inhibitors and infigratinib and pemigatinib are selective FGFR1, 2, 3 inhibitors (Loriot, 2023; Goyal, 2023; Savarirayan, 2024; Vogel, 2024). .

In Ph1, dabo showed activity and was well-tolerated at 90mg QD

FGFR3+ mUC Efficacy Data	Dabogratinib 90mg QD	Erdafitinib 8-9mg QD
	50% ORR (n=10)	35% ORR (n=136[4])



Treatment-Related Adverse Events (TRAEs)

As of August 15, 2024 Data Cutoff

[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

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

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

Information provided in the table above is for illustrative purposes only and is not a head-to-head comparison. Differences exist between study or trial designs and subject characteristics, and caution should be exercised when comparing data across studies.

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

No hyperphosphatemia at ≤60 mg

No discontinuations or reductions at ≤ 60 mg

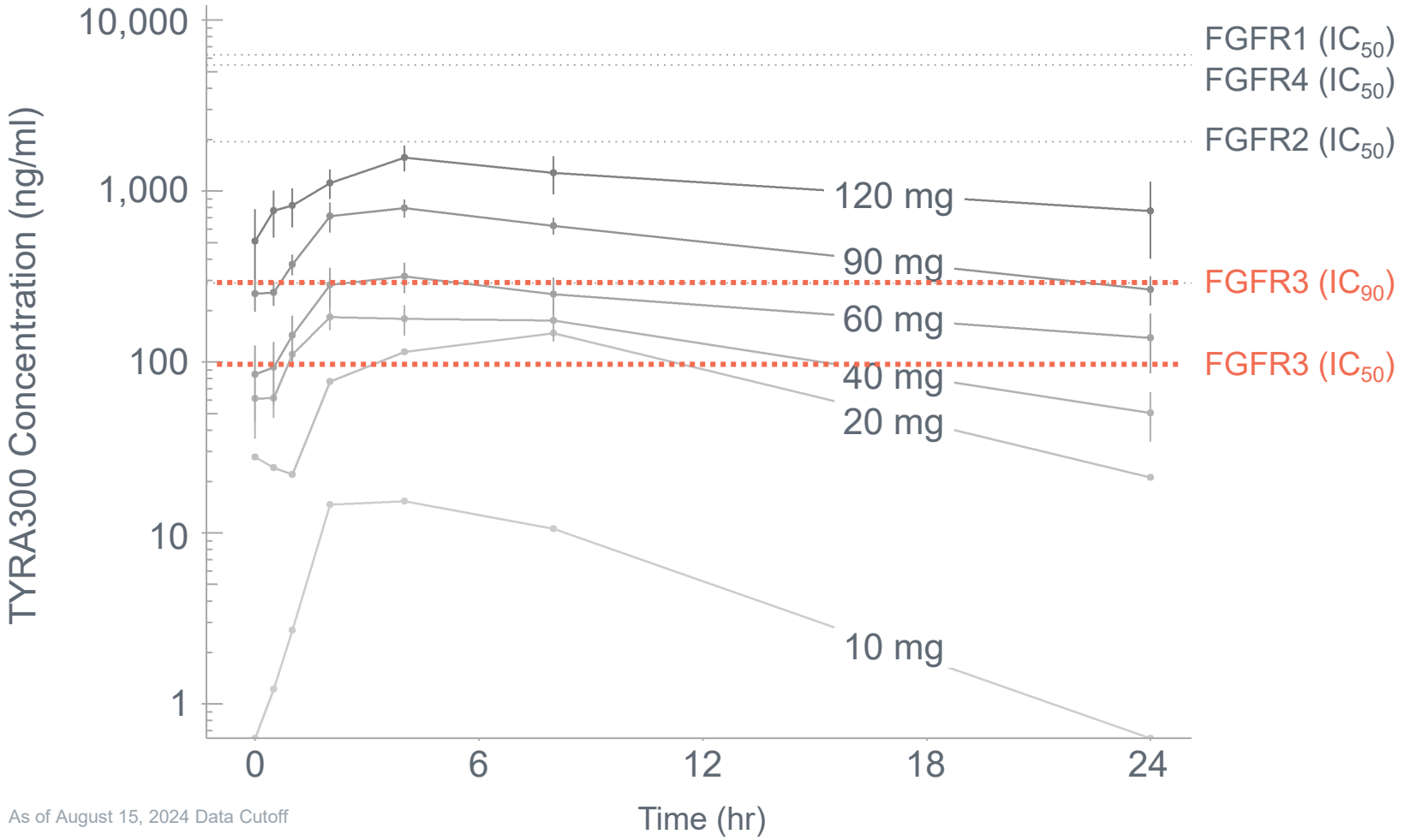
TRAEs in >10% of all participants, n (%)

	≤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)	—

As of August 15, 2024 Data Cutoff

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

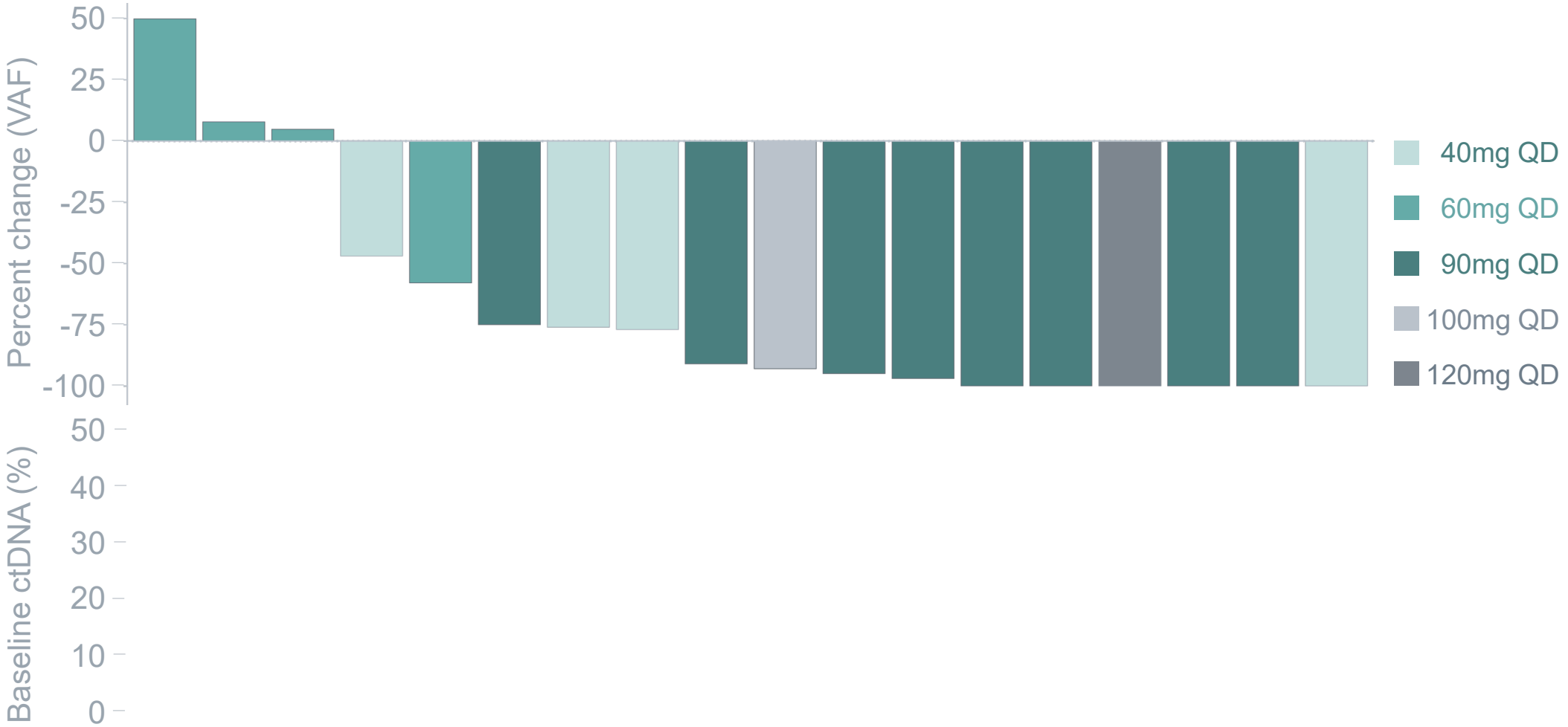
Exposure at doses ≥90 mg exceeded FGFR3 IC_{90} target coverage



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	

As of August 15, 2024 Data Cutoff

ctDNA changes in FGFR3+ mUC showed activity at doses >40mg



As of December 15, 2025 Data Cutoff, Presented at ASCO GU 2026
Four patients were ctDNA negative at screening and are excluded. One of the four had a positive ctDNA on treatment.

TYRA

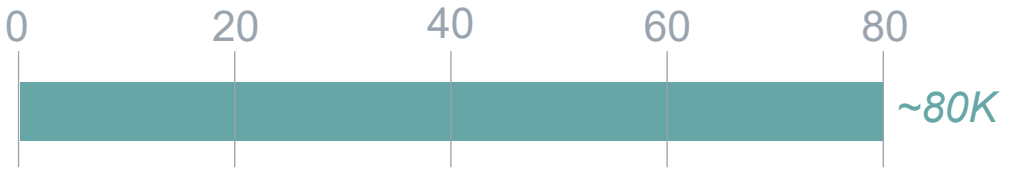
FGFR3 drives large unmet need

TYRA's highly selective FGFR3 inhibitor

Our development paths in Urology and ACH

High FGFR3+ rate drives an outsized opportunity in IR NMIBC

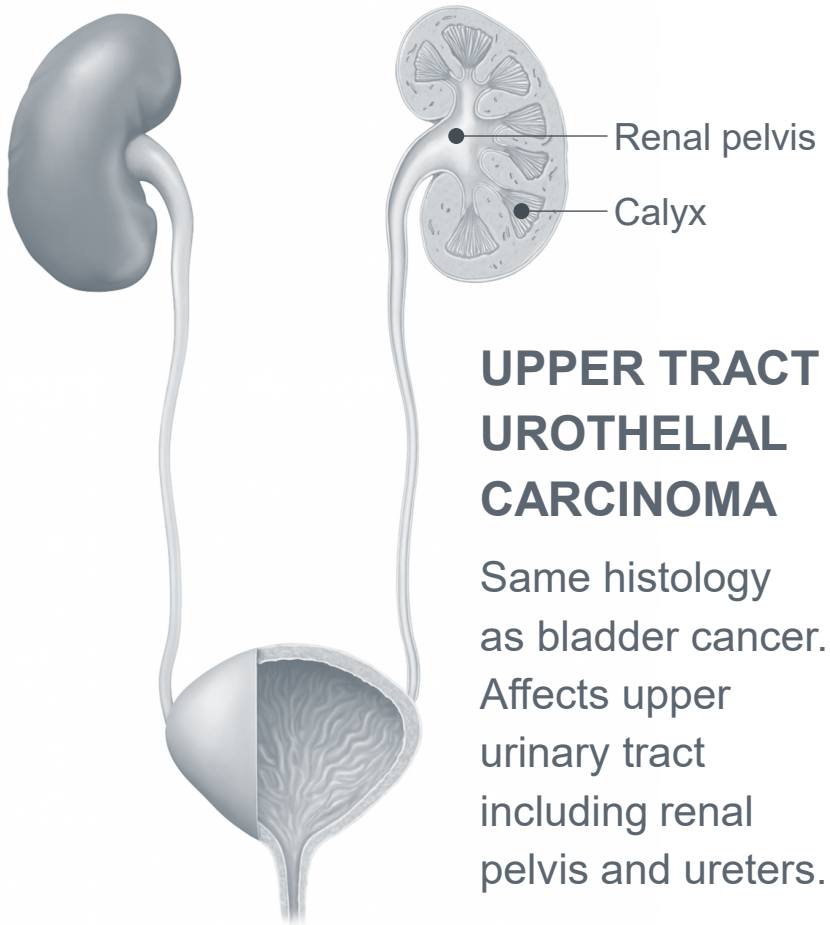
	Total seeking treatment each year	Percentage FGFR3+	FGFR3+ seeking new treatment
ALL BLADDER 790K+ people living with bladder cancer and UTUC in the US	~185K	~45%	~80K
LG UTUC No patient-friendly oral Tx to reduce risk of renal loss	~3.5K	~85%	~3.0K
IR NMIBC No patient-friendly oral Tx to reduce recurrences	~50K	~70%	~35K



FGFR3+ Rate Sources: van Rhijn, 2010; Mayr, 2022; Kacew, 2020; Sfakianos, 2015, Moss, 2017; Nassar 2019; MIBC, mUC; All Bladder Prevalence Source: SEER; MIBC and mUC Patients Seeking New Treatment Source: DR/Decision Resources LLC 2025 Epidemiology Figures; NMIBC and UTUC Patients Seeking New Treatment Source: CancerMPact® Patient Metrics and Oracle Life Sciences analysis; Ravvaz, 2019, Caputo, 2020, Check, 2019, Ritch, 2020, Lyall, 2023; Vedder, 2014

UTUC

Dabogratinib could address specific unmet needs in LG UTUC



UPPER TRACT UROTHELIAL CARCINOMA

Same histology as bladder cancer. Affects upper urinary tract including renal pelvis and ureters.

LOW GRADE UTUC

No high grade on biopsy or cytology

~85%
FGFR3+

~3,000
FGFR3+ seeking new treatment each year

UNMET NEED

ANATOMY

Ureter length, orientation, and intra-renal anatomy presents challenges with diagnosis and treatment

TREATMENT

Repetitive, burdensome surgeries and procedures with high rates of complications and potential for loss of kidney

Oncology figures represent US incidence and recurrences; Oracle Life Sciences analysis; Sfakianos, 2015, Moss, 2017; Nassar 2019

Current kidney-sparing endoscopic surgery SoC has drawbacks

SIGNIFICANT RISKS

MISSED TUMORS

≤25% missed due to challenging anatomy and ~30% multifocality

SURGERY

General anesthesia

Intra- and post-operative complications

POOR OUTCOMES

RECURRENCE AND PROGRESSION

ALL LG TUMORS

~35–50% recur within 3 years

LG TUMORS >2cm

90.5% recur (median 4.9 mo)

31.7% progress (median 26.3 months)

KIDNEY REMOVAL

After recurrence or progression, proceed to radical nephroureterectomy

(Can result in chronic dialysis, and increased CV morbidity, mortality)

Yamany, 2015; Shvero, 2021; Scotland, 2018; Cutress, 2012

Jelmyto, is more effective but comes with many limitations

Efficacy

58% CR rate

56% 12-month DoR

Eligibility

Renal pelvis (not ureters)

Tumors 5–15mm in size

Safety

58% Ureteric obstruction

41% Flank pain

34% Hematuria

34% UTI

25% Renal dysfunction

24% discontinuation

Administration

6 instillations:

- Ureteral catheter with fluoroscopy or antegrade nephrostomy tube
- Local anesthesia

Preparation

- >40 steps
- Refrigeration and reconstitution
- 45–60 minutes

JELMYTO package insert and website accessed September 5, 2025

First LG UTUC patient has been dosed in Dabogratinib Ph2 Study

SURF303

LG UTUC, Marker Lesion Study
Dabogratinib (TYRA-300)
NCT07265947

Key Eligibility Criteria

Adults (≥18 years)

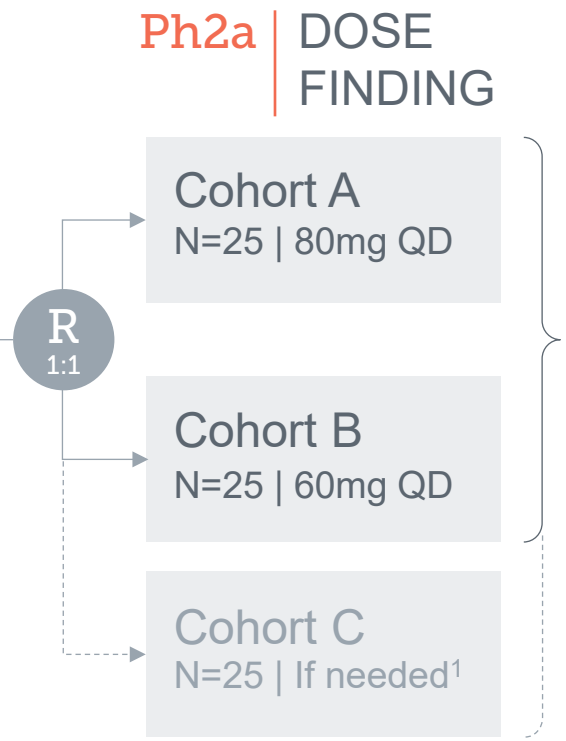
LR, LG UTUC based on AUA Guidelines, 2024

- No high-grade histology on biopsy or cytology
- No invasion or adenopathy on imaging

At least 1 marker lesion; total aggregate ≥5mm

Prescreening includes: urine sample for tumor genomics, tissue sample for retrospective sequencing for FGFR3-gene activating alterations, consent to provide prior tumor genomics report and consent to allow marker lesion(s) to remain *in situ*

Stratification by aggregate tumor baseline size prior to resection <1.5cm vs. ≥1.5cm



1. Optional if needed start after initial data review of 60 mg QD and 80 mg QD, as needed

Primary Endpoint

CR within 6 months in *FGFR3+* patients

Secondary Endpoints

- CR within 6 mos in all comers
- DOR
- Safety and tolerability
- Conversion of unresectable to resectable disease

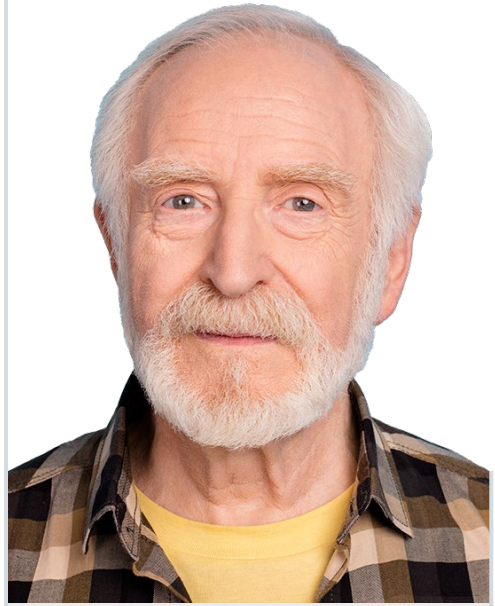
Q3 month disease assessments to 18 mo; Q6 month thereafter. Treatment allowed up to 24mo. Patients who do not achieve a CR at 3mo may continue to mo 6 if ML reductions at mo 3 are ≥50%

Abbreviations: LG, low grade; LR, low risk; UTUC, upper tract urothelial carcinoma; R, randomized; CR, complete response; DOR, duration of response; FGFR3, Fibroblast Growth Factor Receptor 3; QD, once daily; ML, marker lesion

IR NMIBC

Frequent, often lengthy clinic visits burden patients and urologists

PATIENT JOURNEY



Blood in urine

Elderly often face logistic challenges

>87%

Medicare patients live >30 min away from a urologist[1]

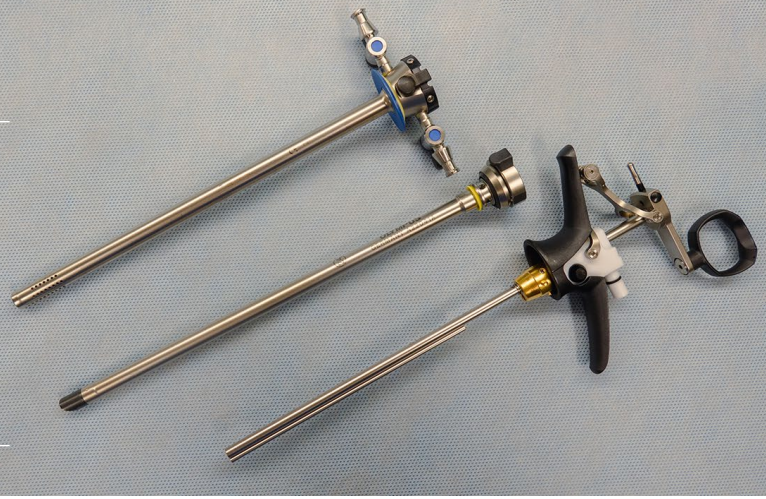
Urologist identifies lesion with cystoscope; schedules TURBT.

TURBT

Invasive

General anesthesia

AE risks such as bladder perforation

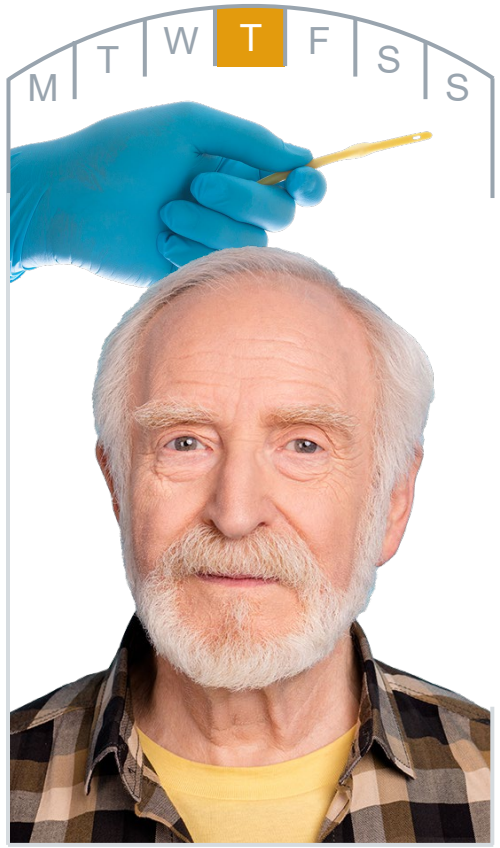


Biopsy pathology (~2 weeks)

RESULT: IR NMIBC, FGFR3+

DECISION: WAIT OR INTERVENE?

Intravesical therapies are uncomfortable and risk AEs



CHEMOTHERAPY (Current SOC)

Weekly for 6 weeks — Monthly for 24 months

Urethral installations: ~30

Recurrence Rate
30% at 12 mo
40% at 24 mo

EMERGING THERAPIES

ZUSDURI (Mitomycin hydrogel)

Weekly for 6 weeks

6

CRETOSTIMOGENE (Oncolytic adenovirus)

Weekly for 6 weeks — Quarterly 1–3x

14

TAR-210 (erdafitinib "pretzel")

Quarterly for 12 months

8

High rates of local side effects
e.g., UTI, dysuria, hematuria

Long dwell times and admin limit clinic throughput

Oral dabo could transform NMIBC therapy for patients and HCPs

Urethral installations: 0

At low dose, erdafitinib showed positive efficacy and durability

THOR-2 TRIAL IR NMIBC

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

Design allowed for up to 2 years of Tx

BEST OVERALL RESPONSE

CR: 89% CR (16/18)[1]

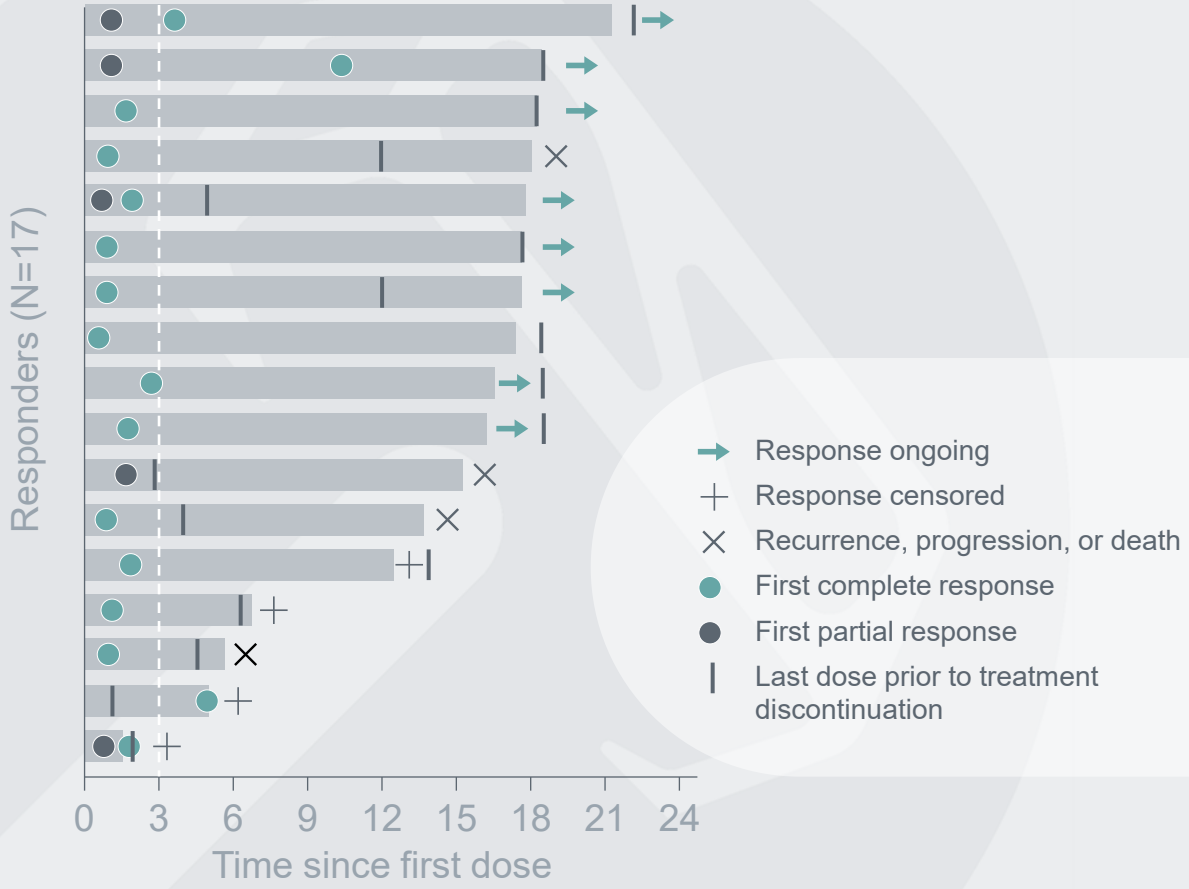
ORR: 94% ORR (17/18)

DURATION OF RESPONSE

DOR: NR *(95% CI 13.4mo-NE)*

12-mo Duration: 100%

For those that remained on study drug[2]



Daneshmand, 2025 (European Urology)

1. 4 of 5 PRs converted to CRs with continued treatment; 2.12-month Landmark CR

Note: the oral Erdafitinib trial was stopped early due to slow accrual in part due to concerns regarding systemic toxicities (Catto, ESMO 2023)

While erdafitinib efficacy was favorable, tolerability was challenging

THOR-2 TRIAL IR NMIBC

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

Design allowed for up to 2 years of Tx

DOSE INTENSITY AND DURATION OF THERAPY

Dose reductions: 61%

Dose interruptions: 78%

Tx duration: 12 mo median

10/18 willing to stay on therapy
>12 months, despite tolerability issues

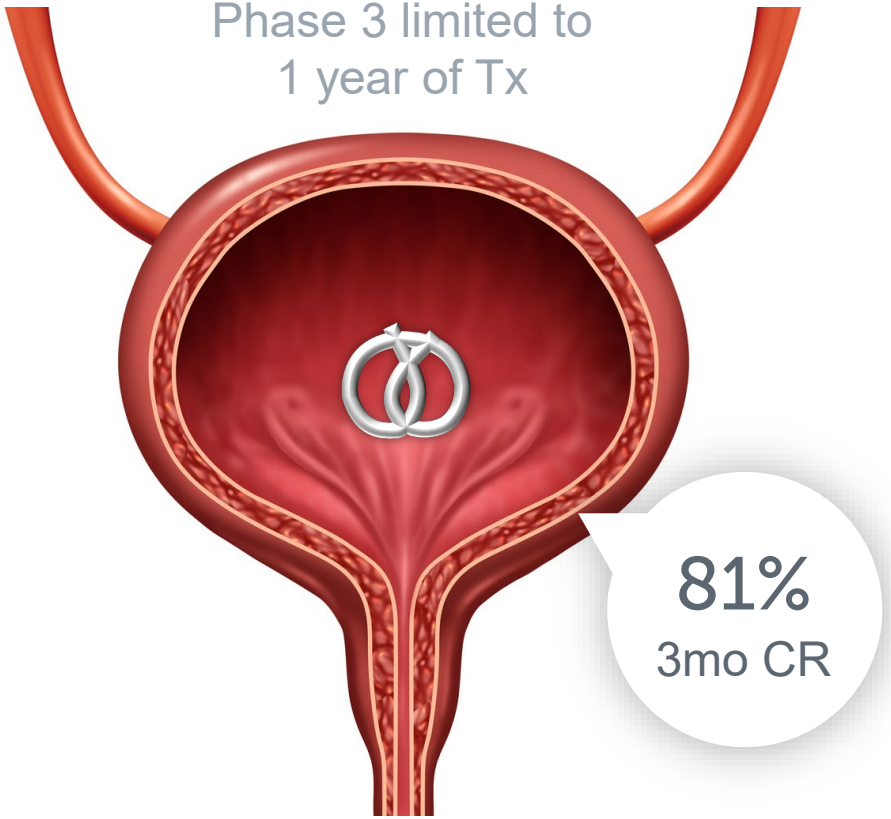
Most common AEs	ANY GRADE %	≥ GRADE 3 %
>1 AE	**100**	**22**
≥1 TRAE	**100**	**17**
Hyperphosphatemia	100	
Dry mouth	72	
Diarrhea	61	6
Dysgeusia	56	
Dry skin	39	
PPE syndrome	39	
Nail disorder	28	
Stomatitis	28	
AST / ALT Increase	17	

Daneshmand, 2025 (European Urology)
Note: the oral Erdafitinib trial was stopped early due to slow accrual in part due to concerns regarding systemic toxicities (Catto, ESMO 2023)

TAR-210's high efficacy comes with intravesical tolerability issues

TAR-210 PRETZEL

Phase 3 limited to
1 year of Tx



SAFETY SUMMARY

Total n=62

Patients with events	%
Grade ≥3 TRAE	5
Discontinuations	10
Interruptions	5
≥1 TRAE	63
Hematuria	29
Dysuria	19
Pollakiuria	18
Micturition urgency	15
UTI	15
Bladder Spasm	11

Urinary tract issues

Source: Vilaseca Cabo, 2026 (EAU); CR Rate and safety summary represents IR NMIBC Data
TAR-210 pretzel representation for illustrative purposes only

IVe package inserts detail patient burden and systemic AEs

BEHAVIOR MODIFICATIONS (Inlexzo & Zusduri)

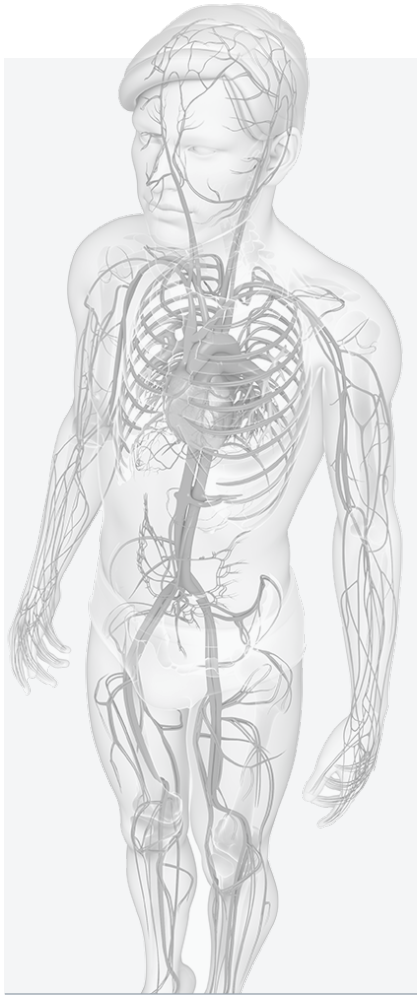
Avoid urine contact with skin

Wash genitals after *every* urination

Launder urine-soiled clothing separately

Inlexzo only

- MRI precaution
  (metal can heat up to +2ºC)
- Prophylactic antibiotic optional
- Drink 1500 mL fluids (daily for 1 year)



	Inlexzo	Zusduri
Interruptions	41%	10%
SAEs	24%	12%
Discontinuations	7%	~3%
Frequency	48%	<10%
UTI	44%	12%
Dysuria	42%	23%
Hematuria	24%	10%
Diarrhea	11%	<10%
Hemoglobin ↓	-31%	-17%
Lymphocytes ↓	-24%	-14%
Lipase ↑	+28%	
Creatinine ↑	+24%	+29%
Potassium ↑	+22%	+26%
AST/ALT ↑	+16–17%	+15%

Adapted from: Inlexzo Prescribing Information, Inlexzo Instructions for Use, Zusduri Prescribing Information accessed 11/2025

N>20 enrolled in Dabogratinib Phase 2 Study in LG IR NMIBC

SURF302

LG IR NMIBC, Marker Lesion Study
Dabogratinib (TYRA-300)
NCT06995677

Key Eligibility Criteria

Adults (≥18 years)

FGFR3 activating gene alterations confirmed by urine or tissue test

IR NMIBC based on AUA Guidelines, 2024

- Recurrence w/in 1 year, LG Ta
- Solitary LG Ta >3cm
- LG Ta, multifocal
- LG T1

At least 1 marker lesion;
total aggregate ≥3mm ≤12 mm

R
1:1

Cohort A
N=30 | 60mg QD

Cohort B
N=30 | 50mg QD

Cohort C
N=30 | If needed[1]

CR at 3 mo
Continue on study treatment up to 24 mo.

No CR at 3 mo
May continue to mo 6 if ML reductions at mo 3 are >50%

Primary Endpoint
CR at 3 months

Secondary Endpoints
- Time to recurrence
- Median duration of response
- Recurrence free survival (RFS)
- Progression free survival (PFS)
- Safety and tolerability

Enrollment as of May 13, 2026

Prescreening includes: urine sample for tumor genomics and/or consent to provide prior tumor genomics report or archival tissue and consent to allow marker lesion(s) to remain *in situ*

1. Optional if needed, start after initial data review of 50 mg QD and 60 mg QD
2. Q3 month disease assessments to 18 mo; Q6 month thereafter

Abbreviations: LG, low grade; IR NMIBC, intermediate risk non-muscle invasive bladder cancer; R, randomized; CR, complete response; DOR: duration of response; FGFR3, Fibroblast Growth Factor Receptor 3; QD, once daily; ML, marker lesion

Why have KOLs advised to set the Ph2 CR bar at 70% or better?

PATIENT UNMET NEED

Low risk of progression but high risk of recurrence by 24 months

AVOID REPEATED

- Urethral violation
- Lengthy office visits
- Surgery with general anesthesia

PROMISE OF AN ORAL OPTION

THOR-2

Erdafitinib
NMIBC PoC

100% 12 mo duration for those that remained on study drug[1]

Development discontinued
Poor tolerability

Daily pressure on FGFR3

SURF302

Dabogratinib
NMIBC PoC

70% 3 MONTH CR
TARGET SET BY KOLS[2]

Urologists support potential modest tradeoff in 3 mo CR for:

1) Tolerability leading to durability
2) Fewer urethral violations, visits, and surgeries

Up to 24 mo of daily pressure on FGFR3

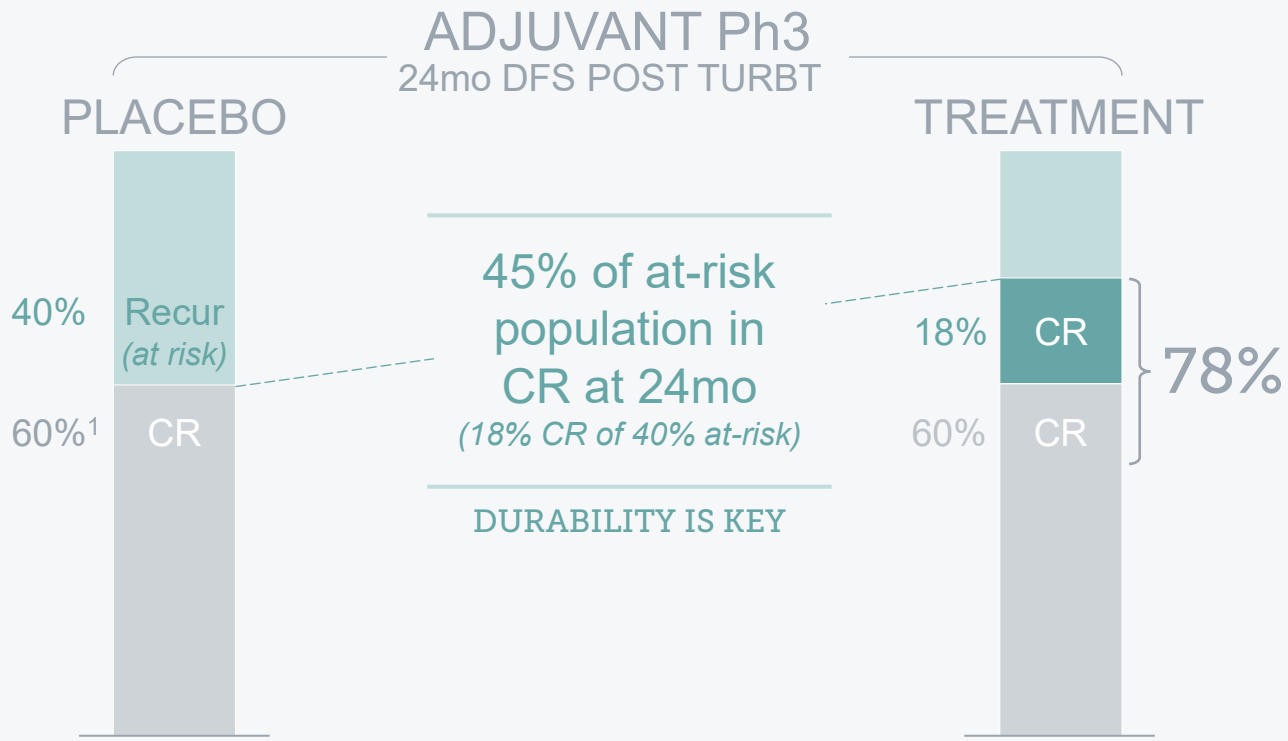
1. Daneshmand, 2025; 12-month Landmark CR 2. internal company analysis and market research on file

A 70% 3mo CR in Ph2 could support a successful adjuvant Ph3

Ph3 Scenario
ILLUSTRATIVE

Compelling Hazard Ratio 0.5

Resulting in 24 mo DFS 78%



Ph2 Objective

ABLATIVE Ph2

SURF302 Target 3mo CR[2] 70%

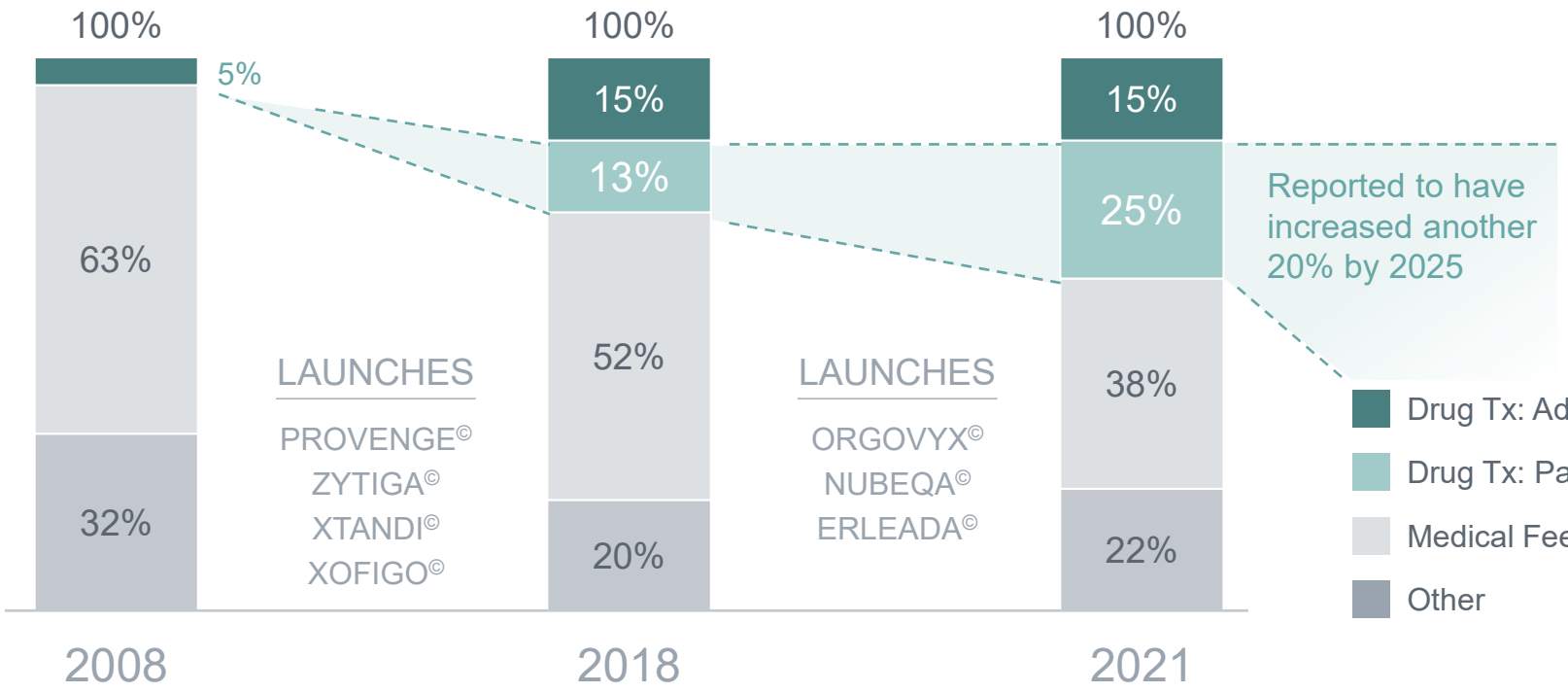
24mo CR to achieve 0.5 HR 45%

70% well above 45% 24mo CR threshold

1. Matulewicz, 2020; 2. internal company analysis and market research on file

Community urology revenue growth largely driven by drug therapy

Private Urology Practice Revenue by Category 2008-2021



~50% of urologists are a part of private practices

~80% of private practices are members of UroGPO[1], which supports In Office Dispensing

1. UroGPO is A Group Purchasing Organization which is part of Cardinal Health

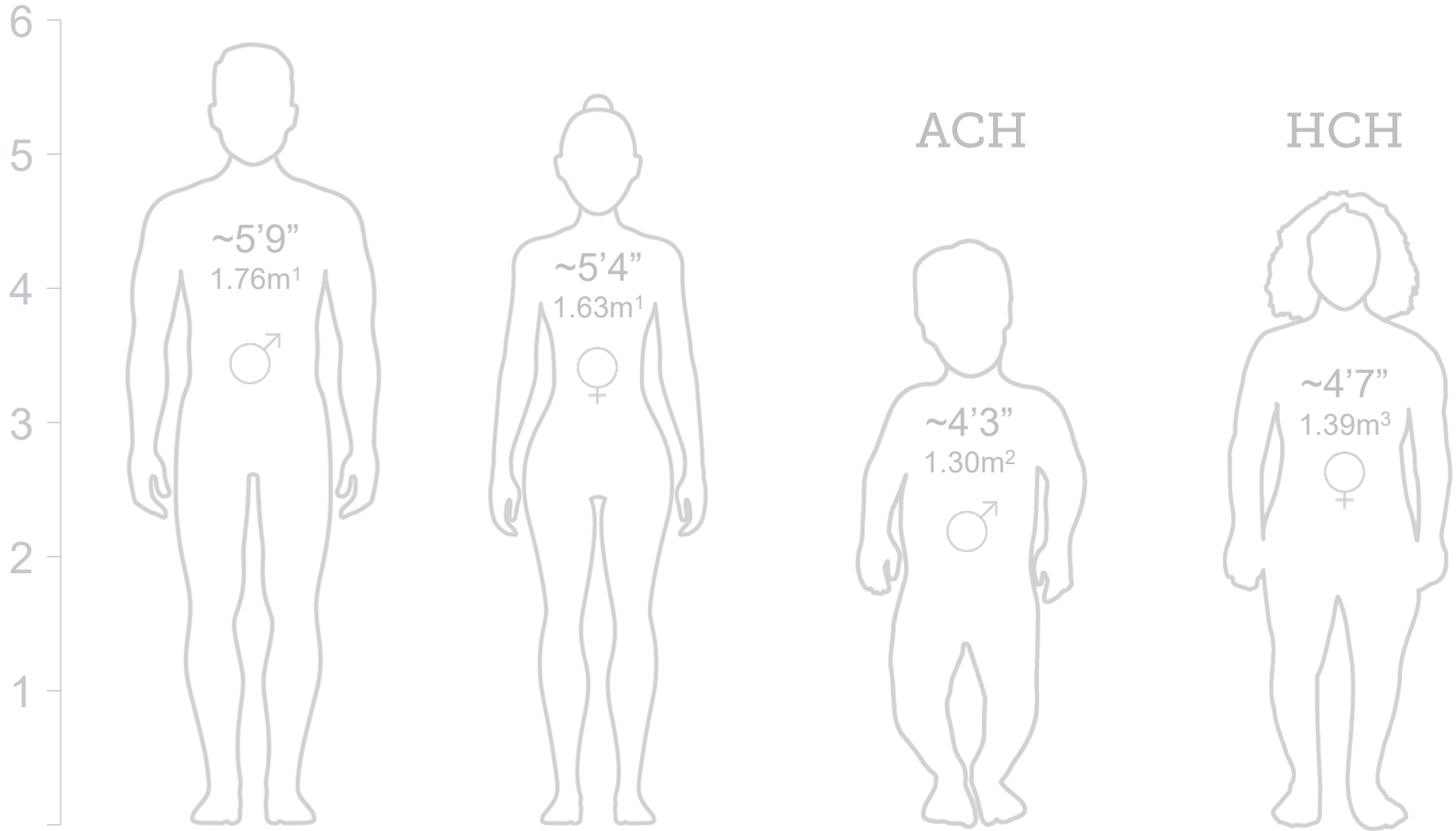
Source: data from Specialty Networks Urology Practice Survey; AUA 2023 annual census

ACHONDROPLASIA

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)

ACH can result in serious clinical complications

ACH is the most common cause of disproportionate short stature

MECHANISM

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

FGFR3 inhibits chondrocyte proliferation and differentiation, resulting in disorganized growth plates and dysregulated bone growth[2]

COMPLICATIONS

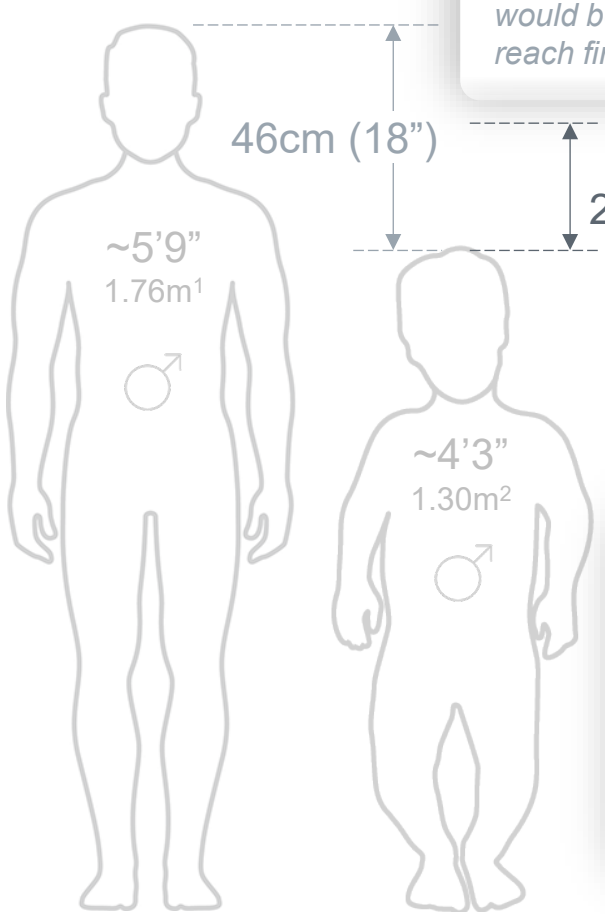
Infants can face serious complications related to critical foramen magnum stenosis[1,3]

Children and adults may experience 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

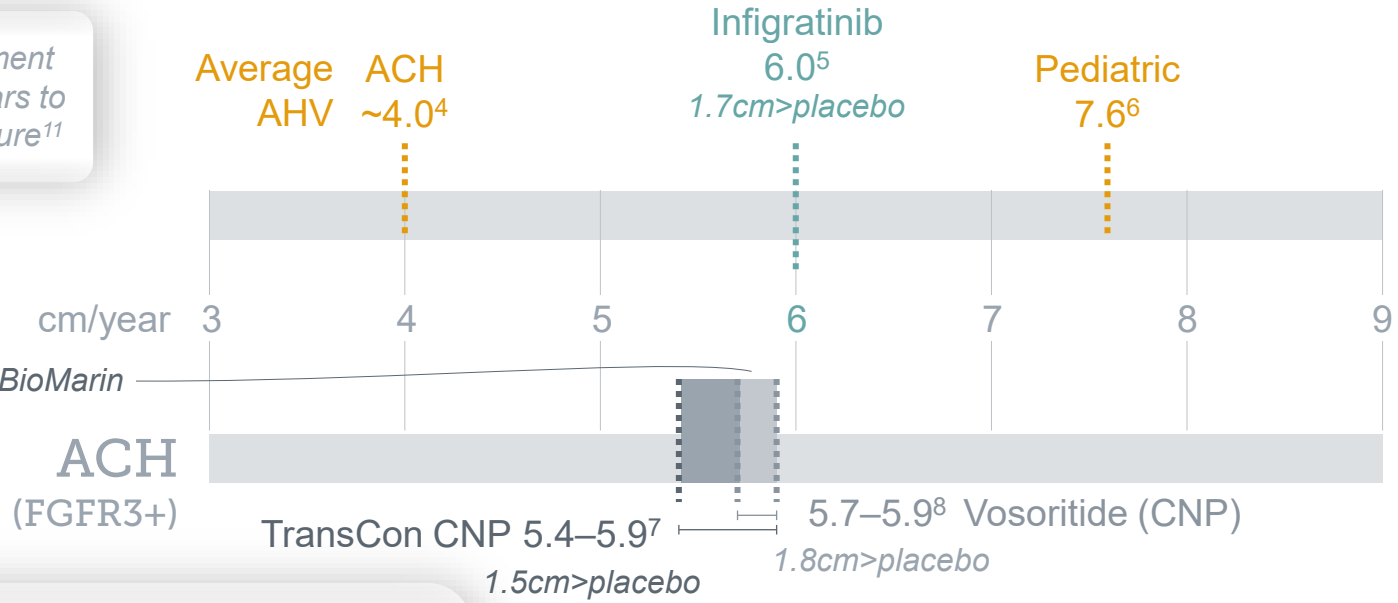
~6cm AHV is potentially leaving efficacy on the table

~2.7cm/yr avg AHV improvement would be needed over 17 years to reach final average male stature[11]



26 cm (10”)[3]

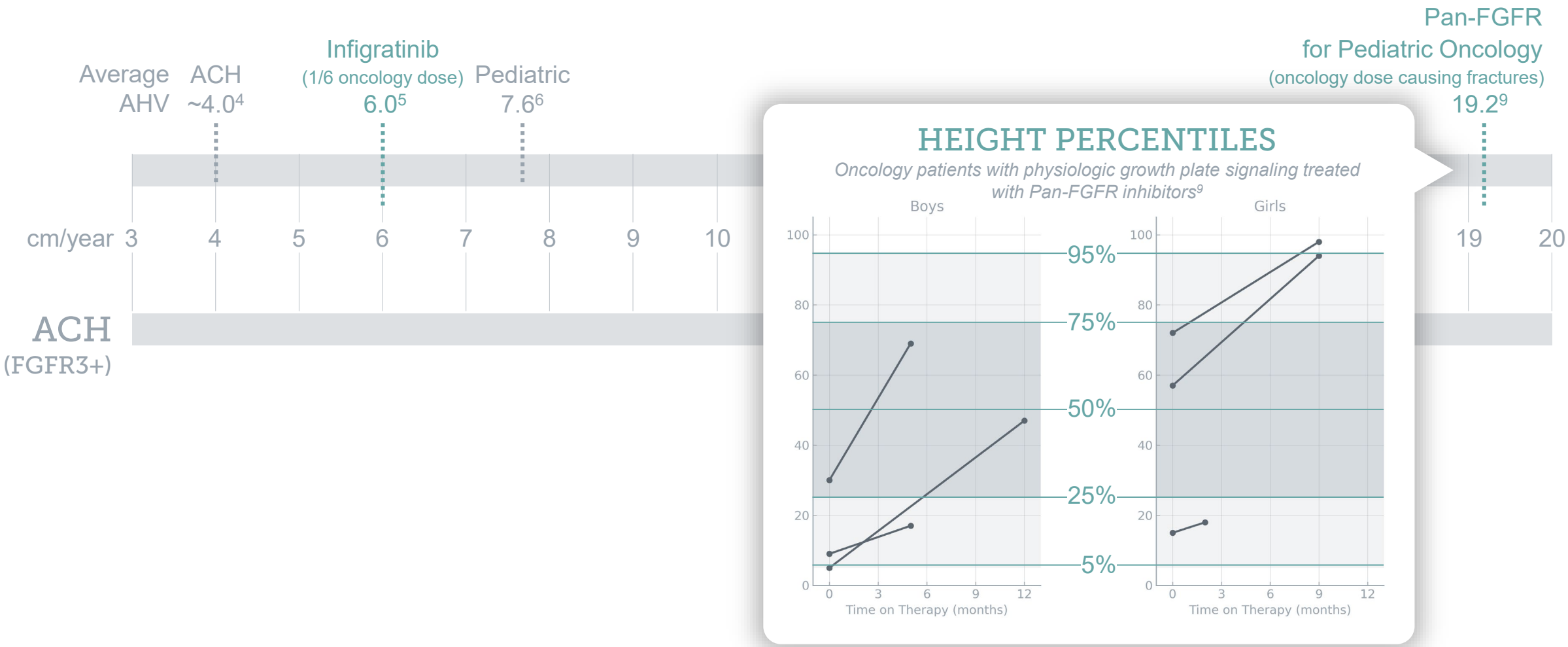
Per BioMarin



Further engaging FGFR3 and achieving a higher AHV could potentially benefit key sequelae like reach and gait, and potentially 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 5/12/25 Press Release; 4. Savarirayan, 2021 (5 to 14yrs); 5. 6.0 AHV from Ph2 Cohort 5 12mo data, Savarirayan, 2024; 6.0 LS mean AHV and 1.7 LS mean absolute AHV improvement vs placebo data based on Ph3 study (n=113), BBIO 2/12/26 Corporate Presentation; infigratinib showed 1.7cm LS mean change from baseline AHV improvement over placebo; 6. Merck Manuals (12mo to 10yrs); 7. 5.4 AHV from Ph2 100 µg/kg/week cohort 6mo data (n=11) Ascendis 11/14/22 Corporate Presentation; 5.9 LS mean AHV and 1.5 LS mean absolute AHV improvement vs placebo data based on Ph3 study (n=84), Savarirayan, 2025; 8. 5.9 from Ph2 Cohort 3 12mo Data (n=10), Savarirayan, 2019; 5.7 LS mean and 1.8 LS mean absolute AHV improvement vs placebo data based on VOXZOGO prescribing information 10/2023 (n=121); VOXZOGO showed 1.6cm LS mean change from baseline AHV improvement over placebo; 9. 19.2 represents AHV for three pediatric oncology case studies with available height velocity data; Sait, 2023; 10. BioMarin 05/04/24 press release; 12mo data from Ph2 (n=8) Investigator initiated study in ISS (ACAN Deficiency, NPR2 Mutation) and Noonan Syndrome 11. Calculation assumes male growth plate closure at 17 years of age, Sarafoglou, 2009

FGFR3 inhibition has been shown to accelerate height velocity

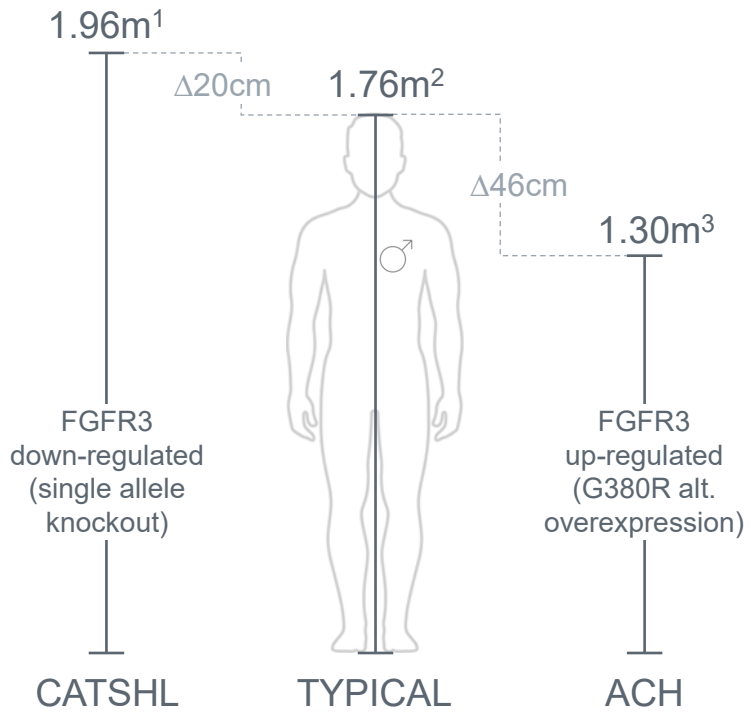


1. CDC Vital and Health Statistics (represents average US male and female heights for ages 20-29); 2. Hoover Fong, 2021 (represents average ACH adult male height); 3. Potential total height gain from VOXZOGO BMRN 5/12/25 Press Release; 4. Savarirayan, 2021 (5 to 14yrs); 5. 6.0 AHV from Ph2 Cohort 5 12mo data, Savarirayan, 2024; 6.0 LS mean AHV and 1.7 LS mean absolute AHV improvement vs placebo data based on Ph3 study (n=113), BBIO 2/12/26 Corporate Presentation; infigratinib showed 1.7cm LS mean change from baseline AHV improvement over placebo; 6. Merck Manuals (12mo to 10yrs); 7. 5.4 AHV from Ph2 100 µg/kg/week cohort 6mo data (n=11) Ascendis 11/14/22 Corporate Presentation; 5.9 LS mean AHV and 1.5 LS mean absolute AHV improvement vs placebo data based on Ph3 study (n=84), Savarirayan, 2025; 8. 5.9 from Ph2 Cohort 3 12mo Data (n=10), Savarirayan, 2019; 5.7 LS mean and 1.8 LS mean absolute AHV improvement vs placebo data based on VOXZOGO prescribing information 10/2023 (n=121); VOXZOGO showed 1.6cm LS mean change from baseline AHV improvement over placebo; 9. 19.2 represents AHV for three pediatric oncology case studies with available height velocity data; Sait, 2023; 10. BioMarin 05/04/24 press release; 12mo data from Ph2 (n=8) Investigator initiated study in ISS (ACAN Deficiency, NPR2 Mutation) and Noonan Syndrome 11. Calculation assumes male growth plate closure at 17 years of age, Sarafoglou, 2009

Evidence points to FGFR3 inhibition potential beyond ACH

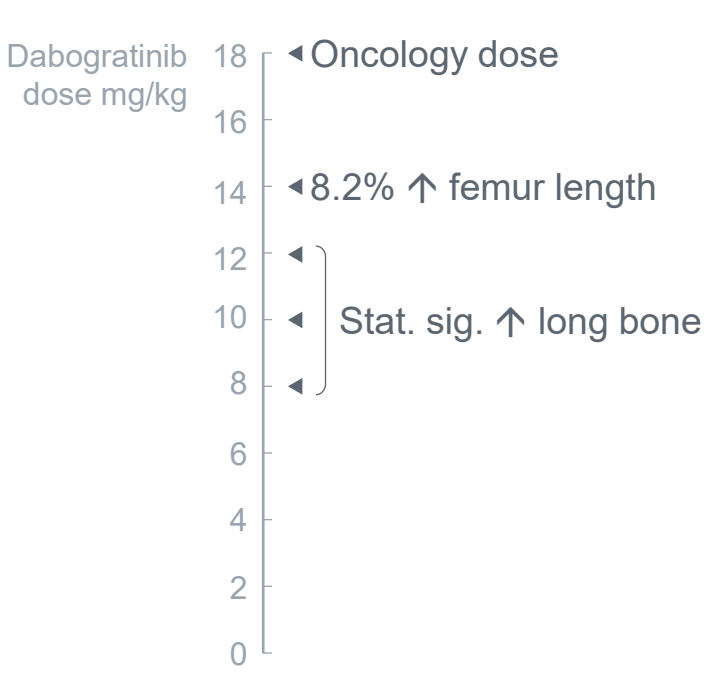
GENETIC

FGFR3 mutation phenotypes point to a key role in regulating bone growth.



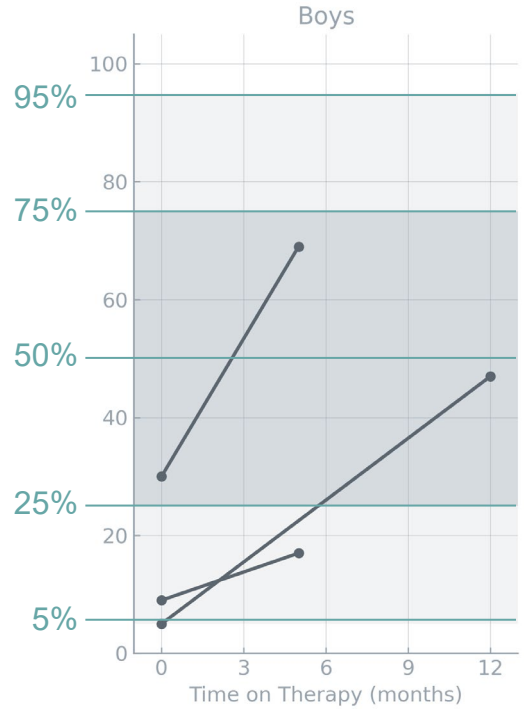
PRECLINICAL

FGFR3 inhibition below an IC_{90} drove hyper-typical growth in WT mice.[4]



CLINICAL

Oncology doses of pan-FGFR drive hyper-typical growth in pediatric cancers.[5]

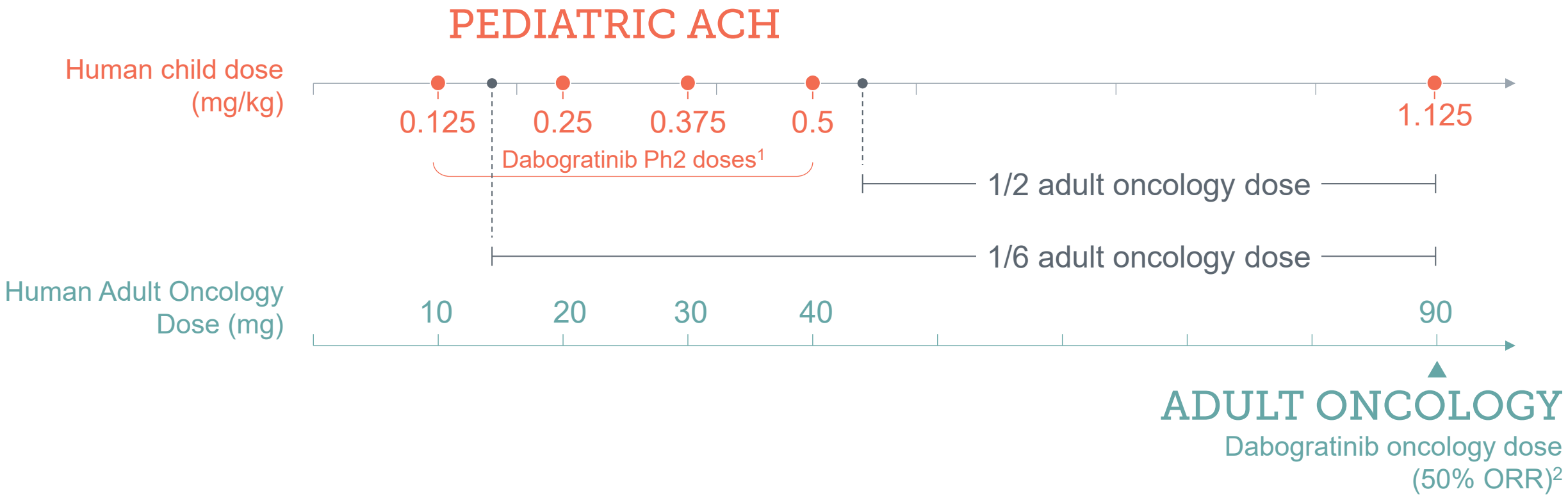


HEIGHT PERCENTILES

Pediatric oncology patients with physiologic growth plate signaling treated with Pan-FGFR inhibitors[3]

1. Toydemir, 2026; 2. CDC Vital and Health Statistics (represents average US male and female heights for ages 20-29); 3. Hoover-Fong, 2021 (represents average ACH adult male height); 4. Starett, 2025; 5. Sait, 2023

In Ph2 we are exploring doses predicted to be active in ACH



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

Dabogratinib cleared safety sentinel cohort 4 in Ph2 BEACH301

BEACH 301

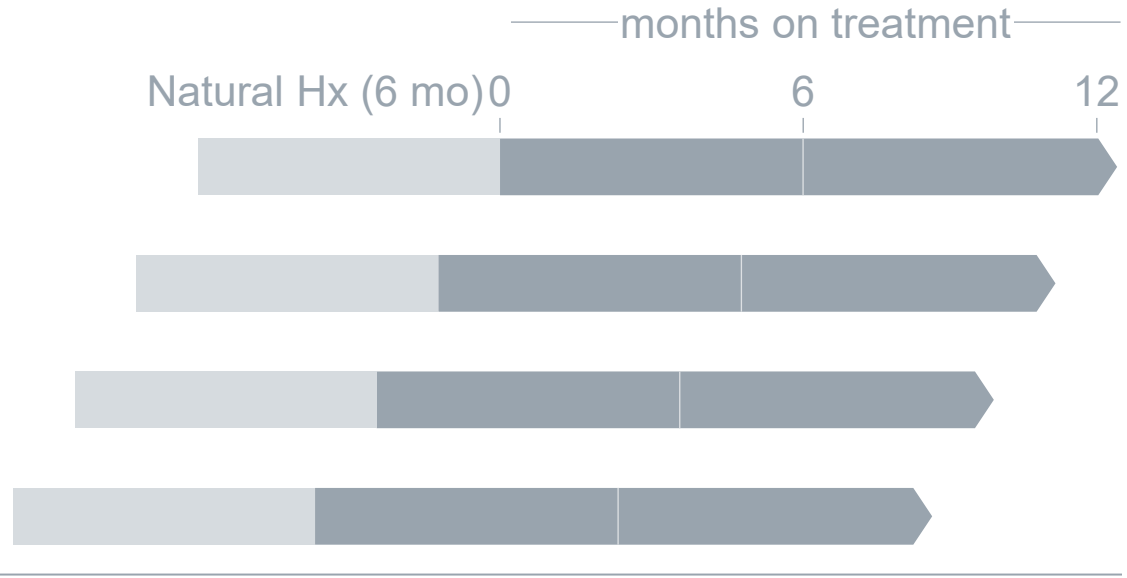
Sentinel Safety Cohort

ACH age 5-10
No run-in

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

Cohorts 1 and 2*

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



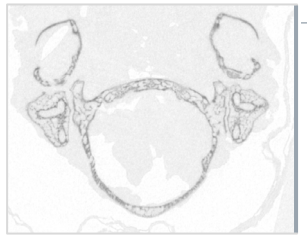
*Cohort 1 (6-mo run-in)
Treatment naïve;
ACH age 3-10

*Cohort 2 (6-mo run-in)
Received prior growth-accelerating therapy; ACH age 3-10

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

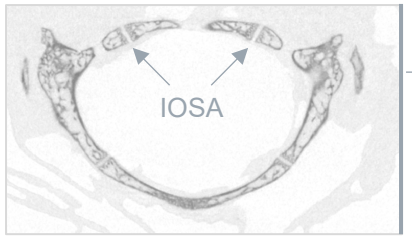
In mice, prenatal dosing reduced premature synchondroses fusion

Fgfr3$^{Y367C/+}$
ACH Model



Foramen Magnum cross-section

Fgfr3$^{+/+}$
Wild-type

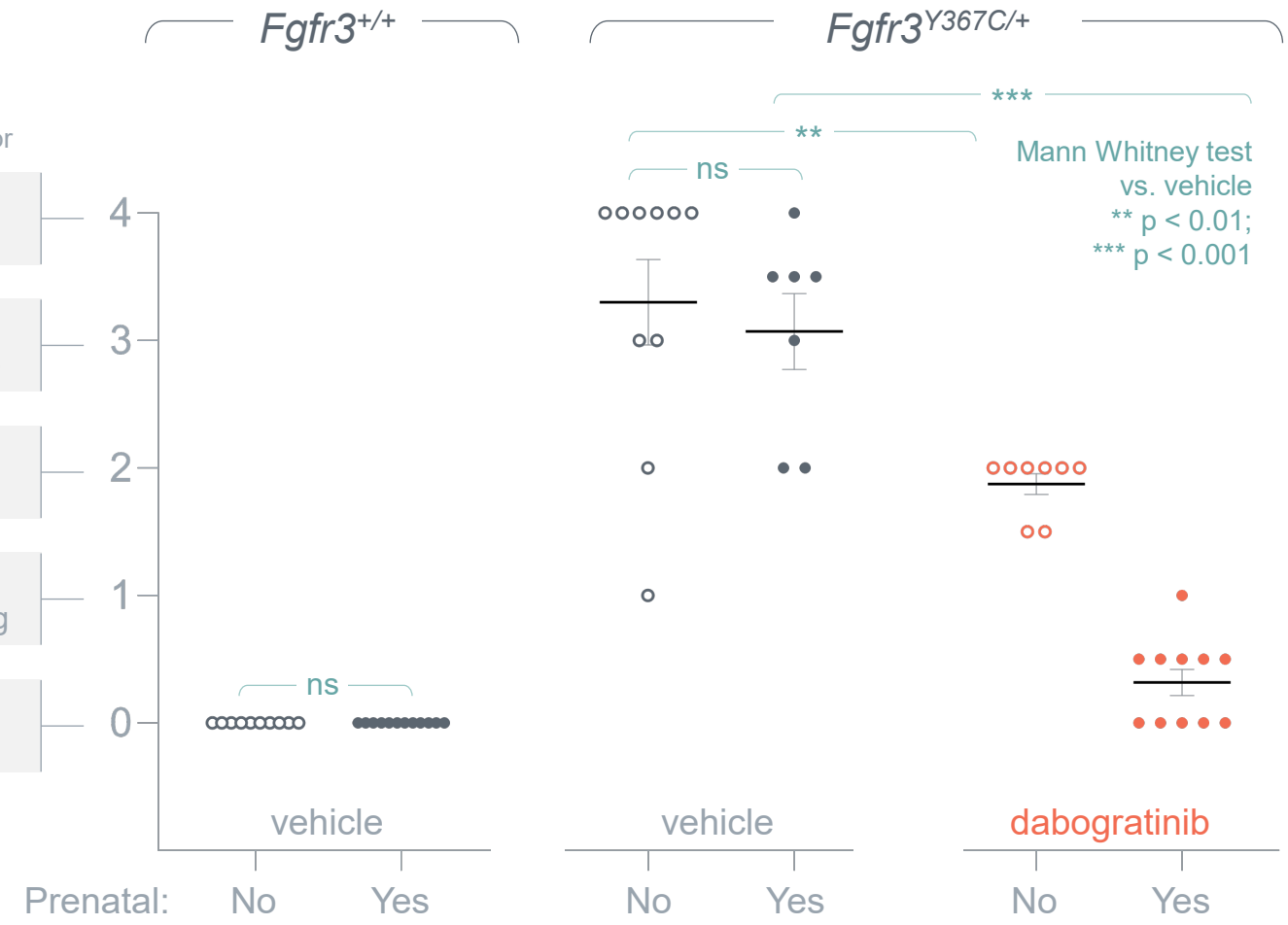


Foramen Magnum cross-section

IOSA GRADE

Intraoccipital synchondroses anterior

4	completely fused synchondroses
3	completely fused with remnants of margin (cartilage)
2	bony bridge between the two borders
1	clear separation with some areas of possible bone bridging
0	borders of synchondroses completely separated



Prenatal "No" (○): Starrett et al., JCI Insight, 2025;
Prenatal "Yes" (●): unpublished data from Legeai-Mallet Lab in which pregnant mice were treated with 2.7 mg/kg SC from E14.5 to birth (prenatal), then neonatal mice were treated with 1.2 mg/kg SC from P1 to P16

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

Genetic and 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

Gearing up for potential pivotal trials in 3 blockbuster indications

Validated Target

FGFR3 alterations drive conditions with high unmet needs

Differentiated Molecule

Dabogratinib: the first oral, once-daily, highly selective FGFR3 inhibitor with clinical PoC (N > 100)

3 Potential Blockbuster Indications

	EXPECTED
IR NMIBC – Initial 3mo CR data	August ‘26
ACH – Initial results, safety sentinel cohort	4Q26
LG UTUC – Initial results	2027

NASDAQ: TYRA

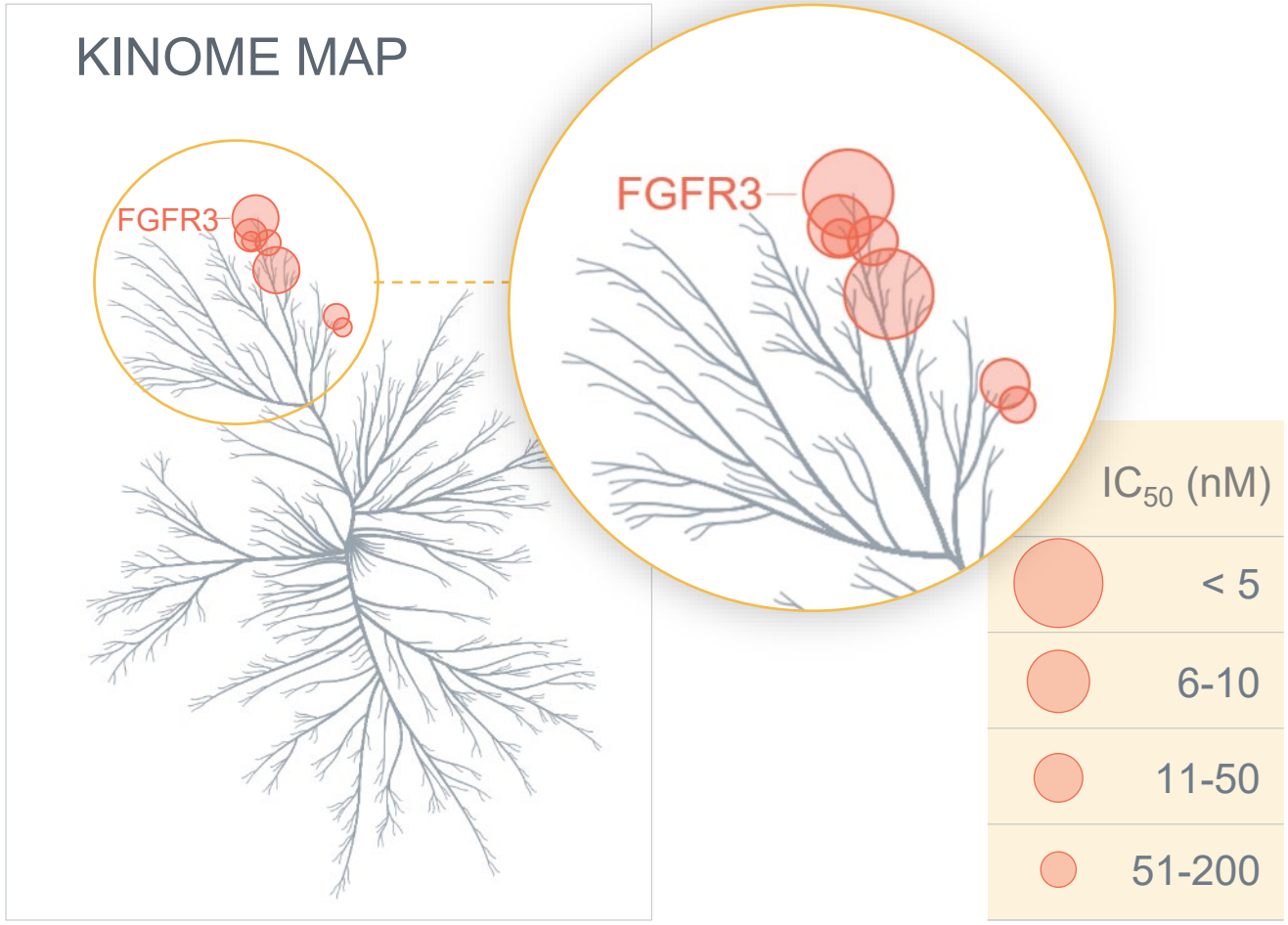
CASH: $383.5M (As of 1Q26)

DABO3^3

ACH: Achondroplasia; IR NMIBC: Intermediate Risk Non-Muscle-Invasive Bladder Cancer; LG UTUC: Low Grade Upper Tract Urothelial Carcinoma
All TYRA small molecule inhibitors are for investigational use only

APPENDIX

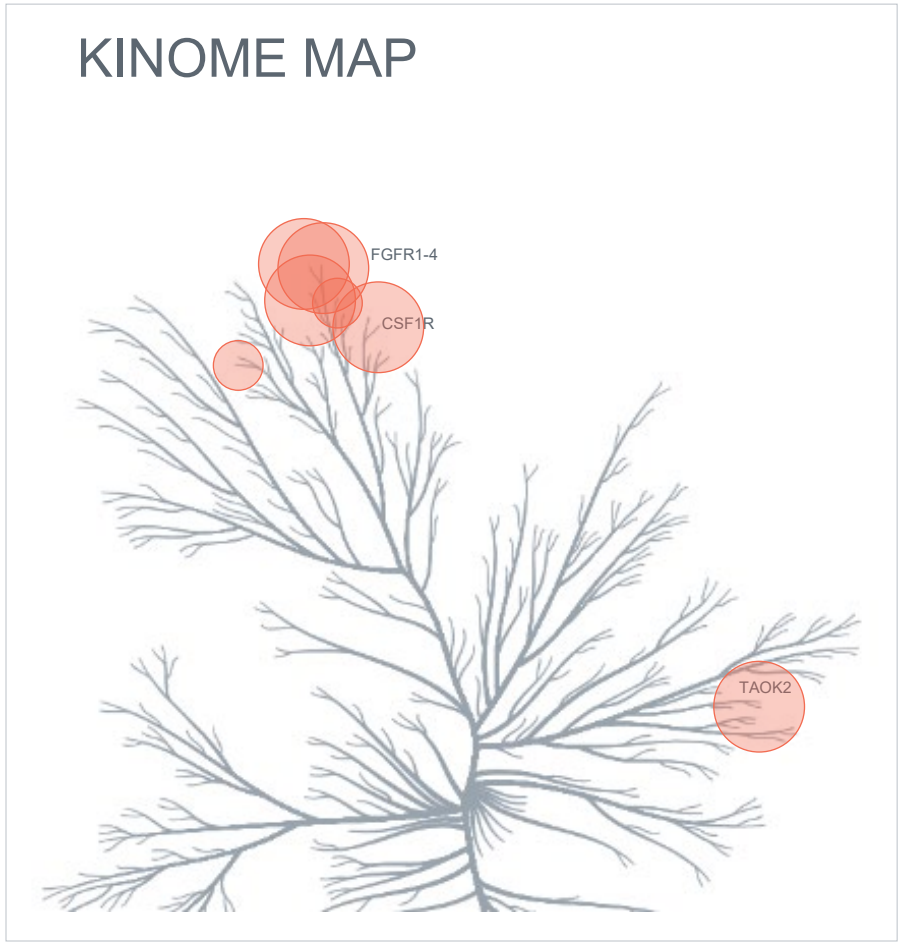
Dabogratinib had greater potency for FGFR3 than other isoforms



	Dabo-gratinib	Fold selectivity from top target (FGFR3)	
FGFR3	1.6	1.0x	*Most potent inhibition*
FLT4/VEGFR-3	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	

Dabogratinib was profiled in a scanMAX℠ (KINOMEscan), kinases with ≥90% inhibition were followed up for enzymatic IC_{50} data generation in triplicate at 100 micromolar ATP by Reaction Biology Inc.

Infigratinib top kinome hits include FGFR3



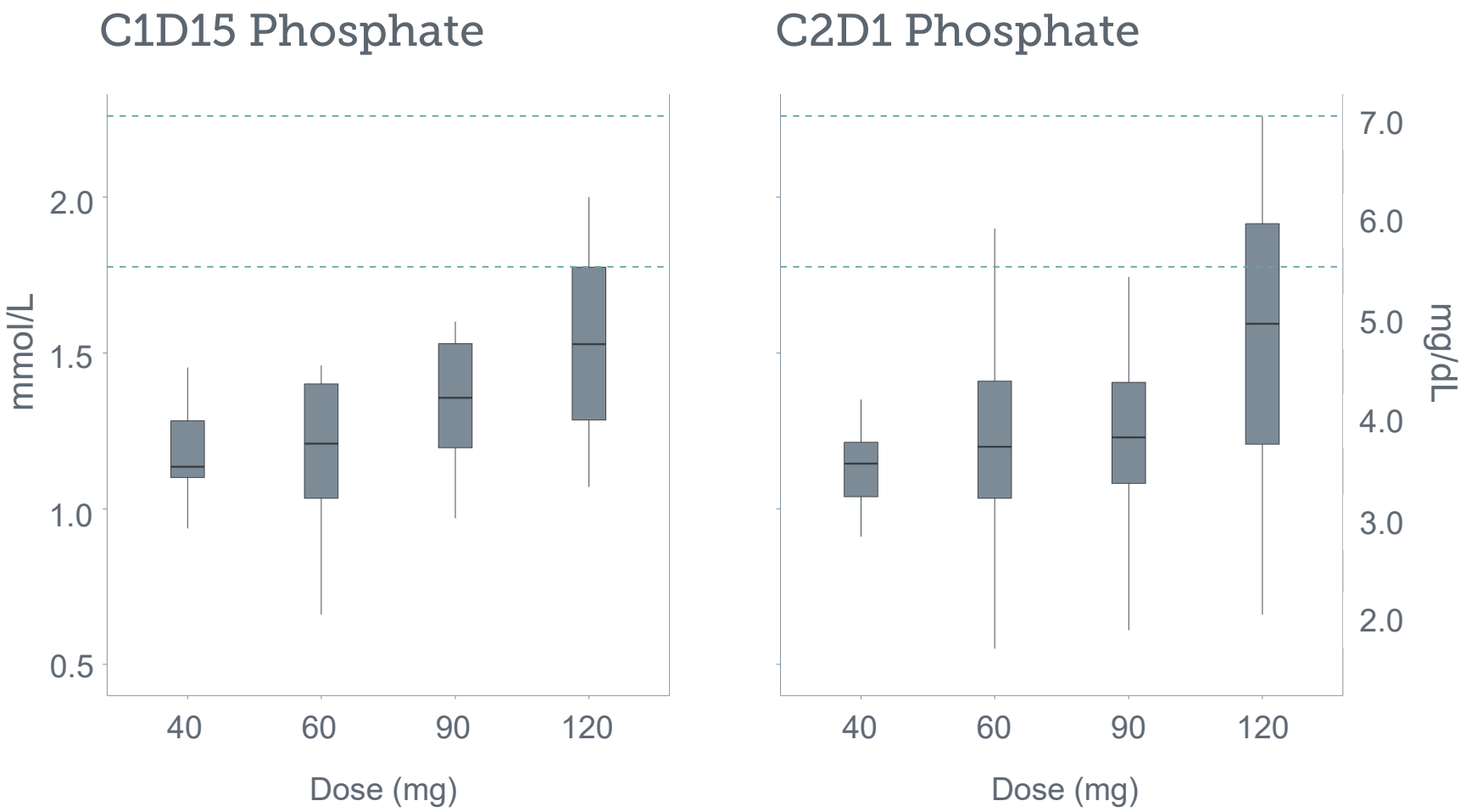
IC_{50} (nM)
< 5
6-10
11-50
51-200

	Infigratinib	Fold selectivity from top target (FGFR1/2)
FGFR1	0.063	1.0x
FGFR2	0.065	1.0x
CSF1R	0.70	11x
FGFR3	1.6	25x
TAOK2	2.9	46x
FGFR4	16.2	257x
DDR1	96.2	1527x

Most potent inhibition

Infigratinib was profiled in a scanMAX℠ (KINOMEscan), kinases with ≥90% inhibition were followed up for enzymatic IC_{50} data generation in triplicate at 100 micromolar ATP by Reaction Biology Inc.

Minimal changes in phosphate at ≤90 mg QD observed



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

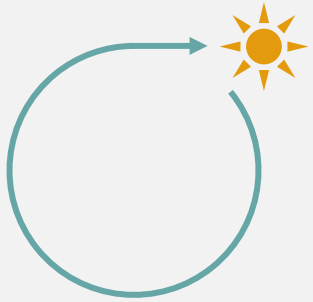
As of August 15, 2024 Data Cutoff

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.

Our unconventional approach accelerates discovery

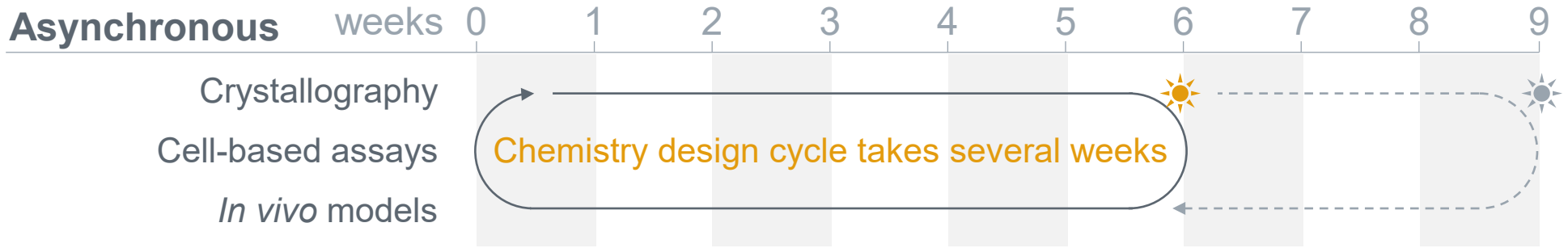
DESIGN CYCLE

Chemistry design

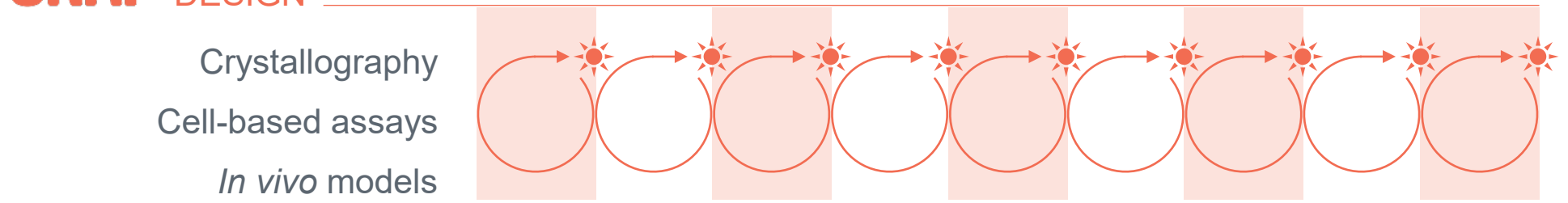


Crystallography
Cell-based assays
In vivo models

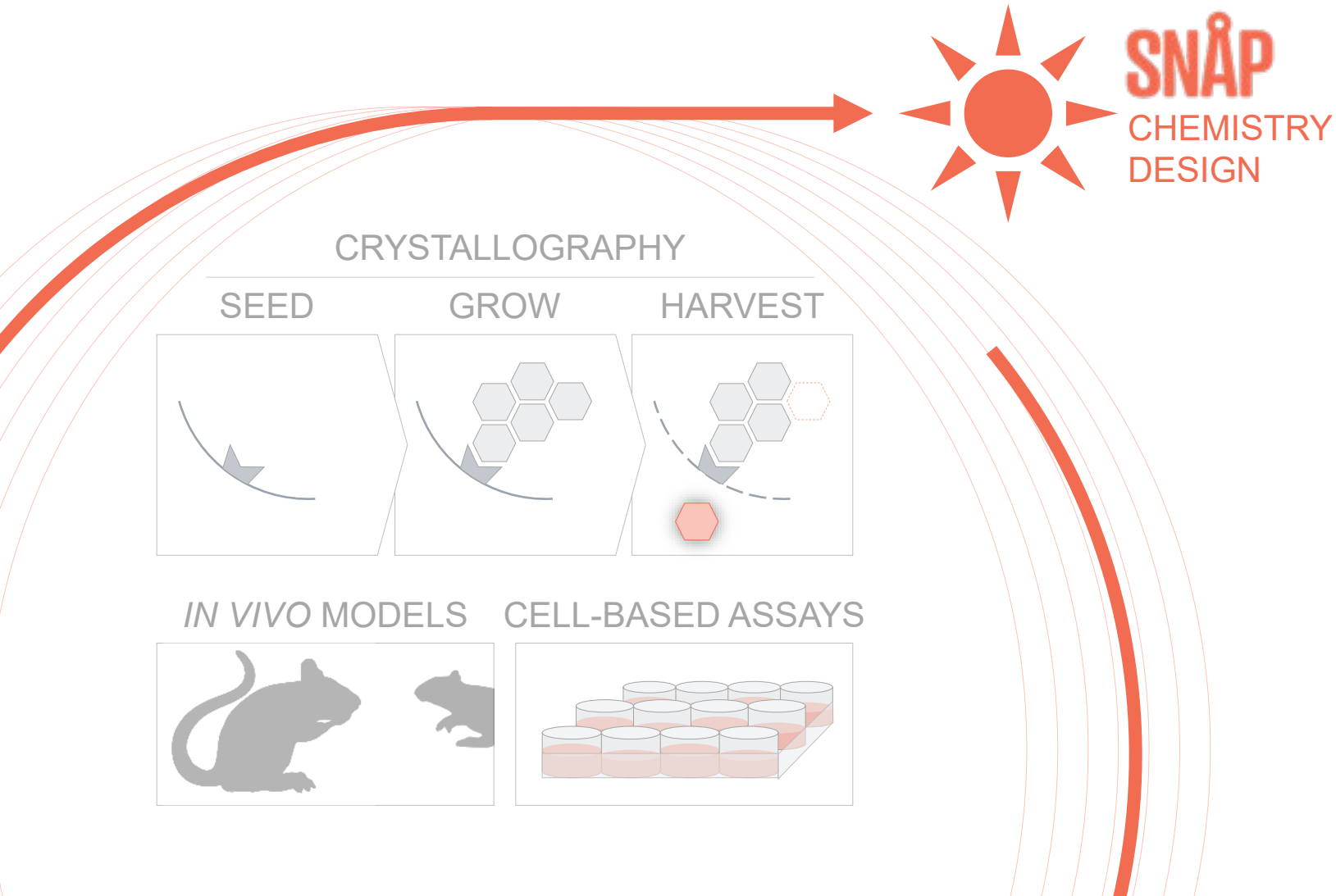
Asynchronous



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)

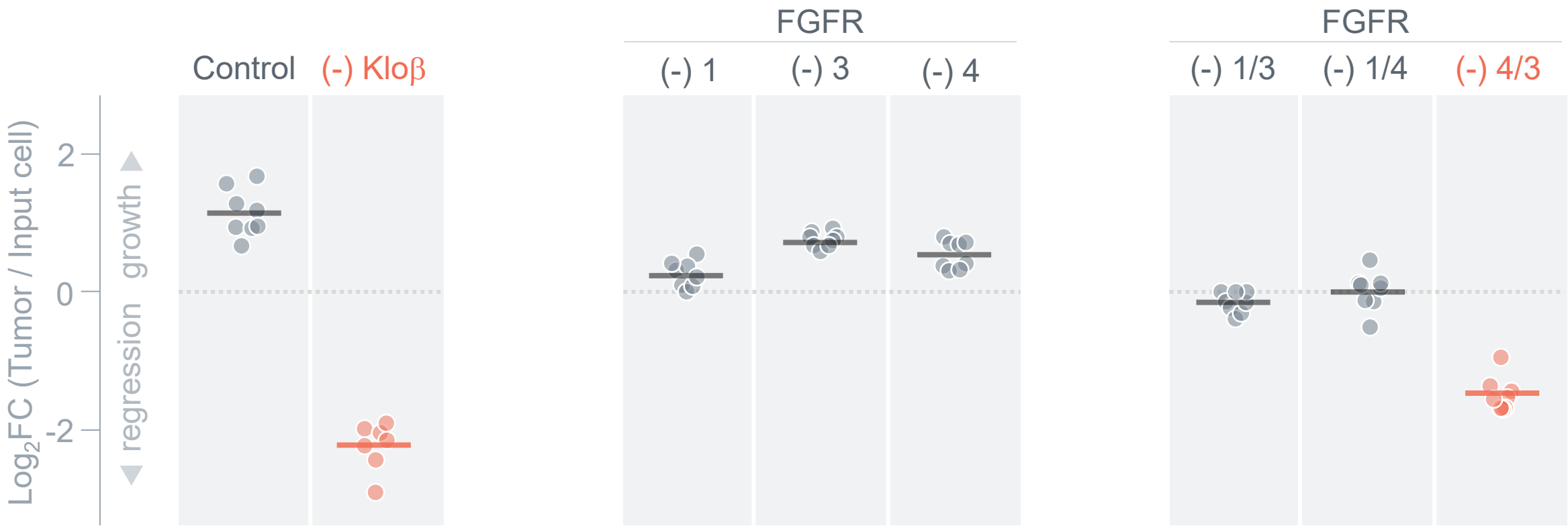
	FGF19 Positive	FGF19 Negative
BLU-554[1]	17% ORR 5.3 month mDoR 3.3 month mPFS (2.1-3.7)	0% ORR NO mDoR 2.3 month mPFS (1.8-5.5)
FGF-401[2]	21% ORR	3% ORR

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.

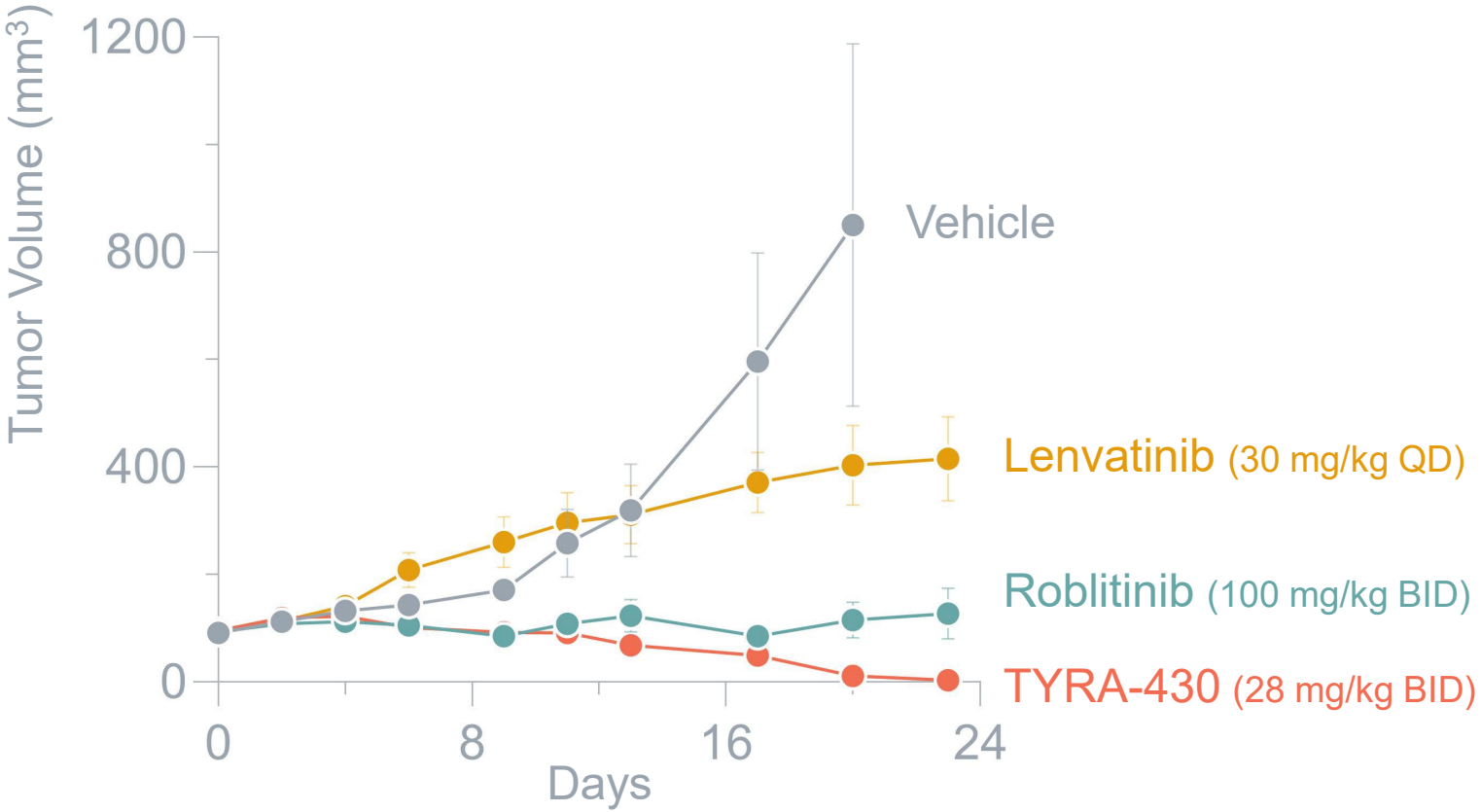
TYRA-430 demonstrated a strong bias for FGFR3 and FGFR4

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 2025 US FGF19+ Addressable Population

Kim, 2019; DR/Decision Resources LLC 2025 Epidemiology Figures for treatable advanced and intermediate stage HCC; GlobalData

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

	ADDRESSABLE (US)	LEAD OPTION	UNMET NEED
1st Line	~9.7K	Atezo/Bev Other PD1 combos	Response rate and PFS improvement (27% and 6.8 months for Atezo/Bev)
2nd Line	~4.3K	Sorafenib/Lenvatinib Other MKIs	ORR and PFS improvement (4-7% and 2.8-5.2 months for 2L MKIs)
3rd Line	~1.4K	Cabozantinib Other MKIs	No 3L approvals or de facto SOC

No biomarker-driven, targeted therapy available

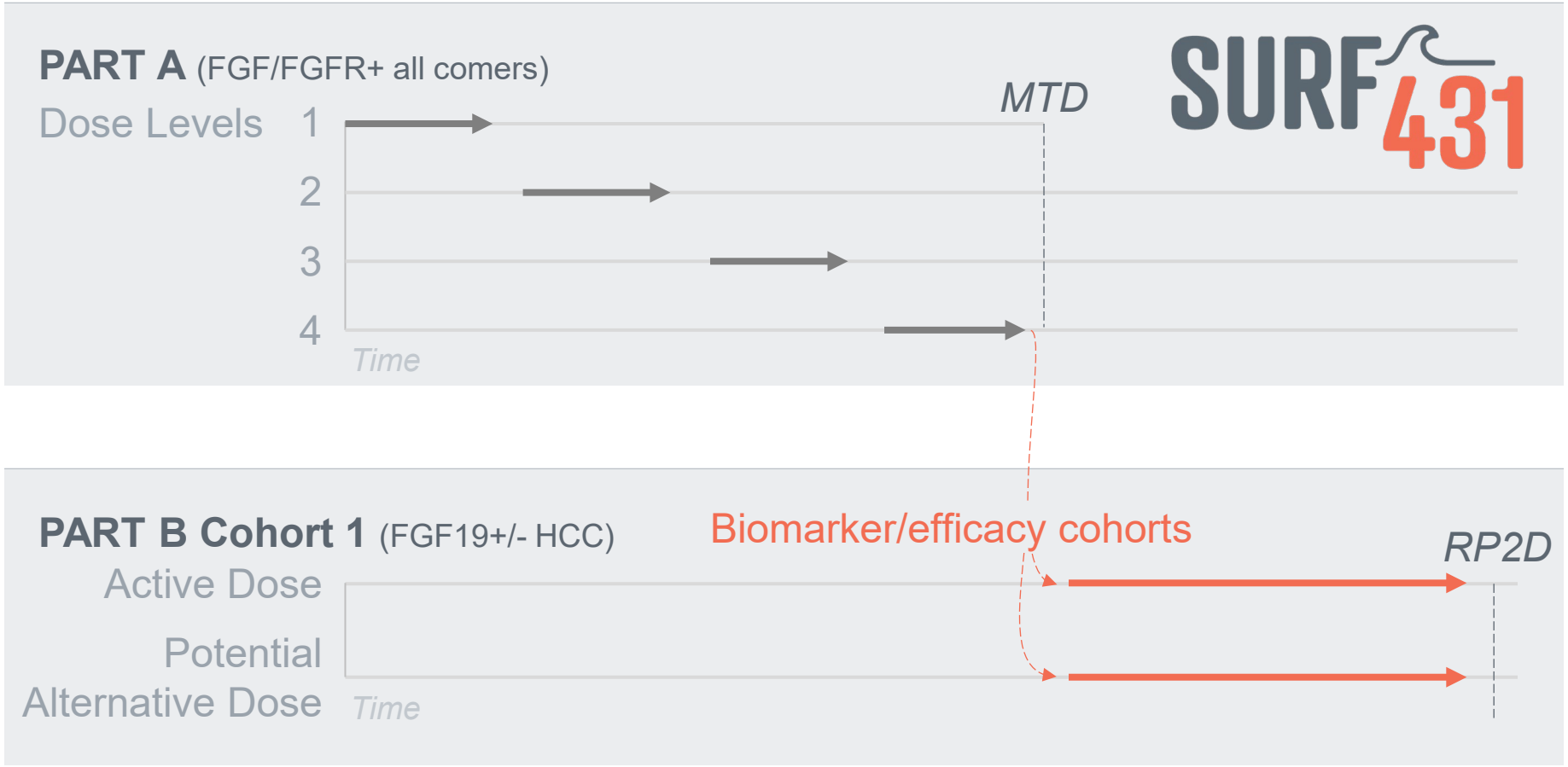
Kim, 2019; DR/Decision Resources LLC 2025 Epidemiology Figures for treatable advanced and intermediate stage HCC; GlobalData; Tecentriq label, Cabometyx label, Stivarga label, Cyramza label

Our Phase 1 trial will evaluate FGF19+ HCC

Illustrative

What is the MTD?

What is the RP2D?



EXAMPLE

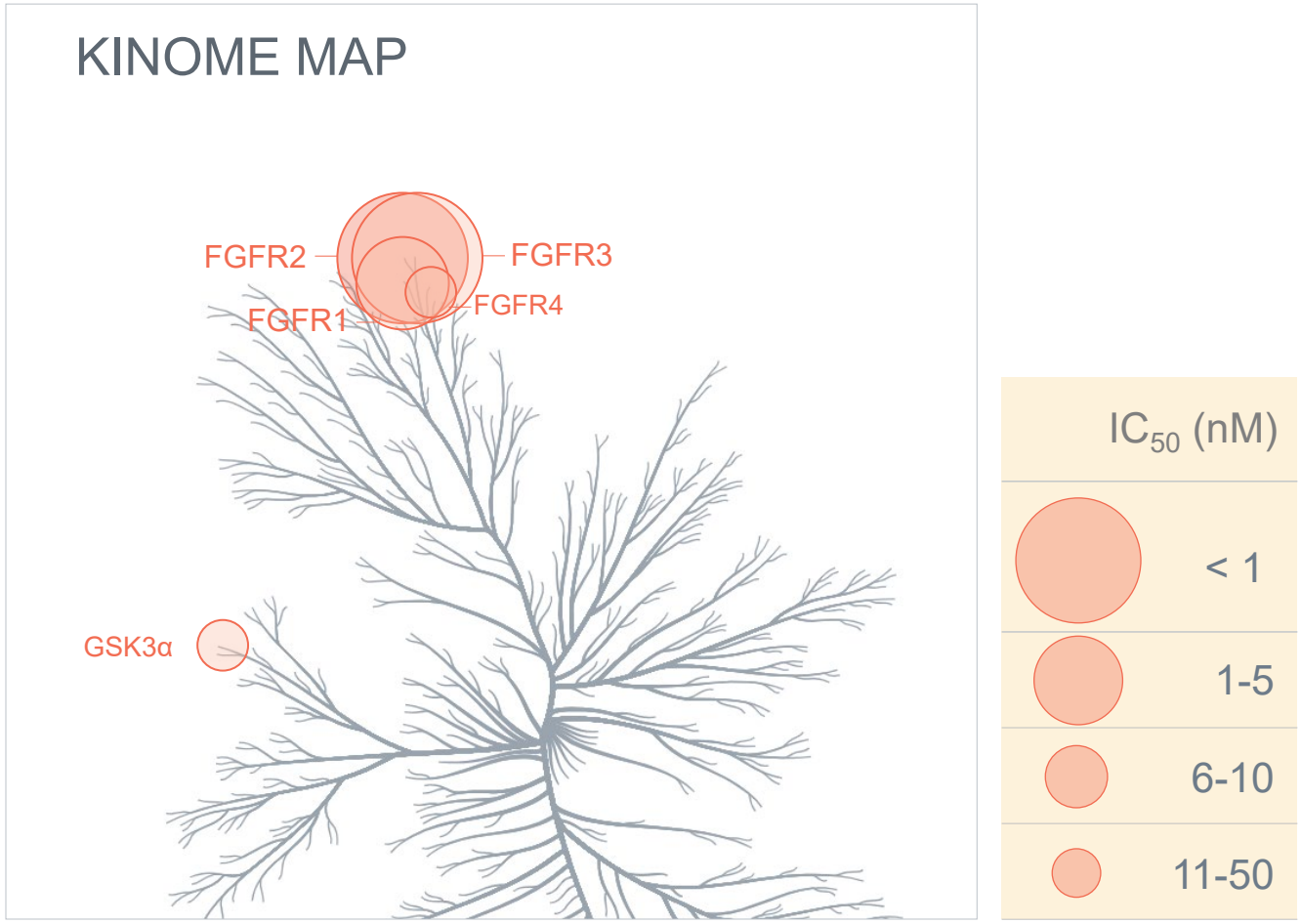
TYRA-200

Intrahepatic cholangiocarcinoma (ICC)

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

Potential gateway to additional solid tumor development programs

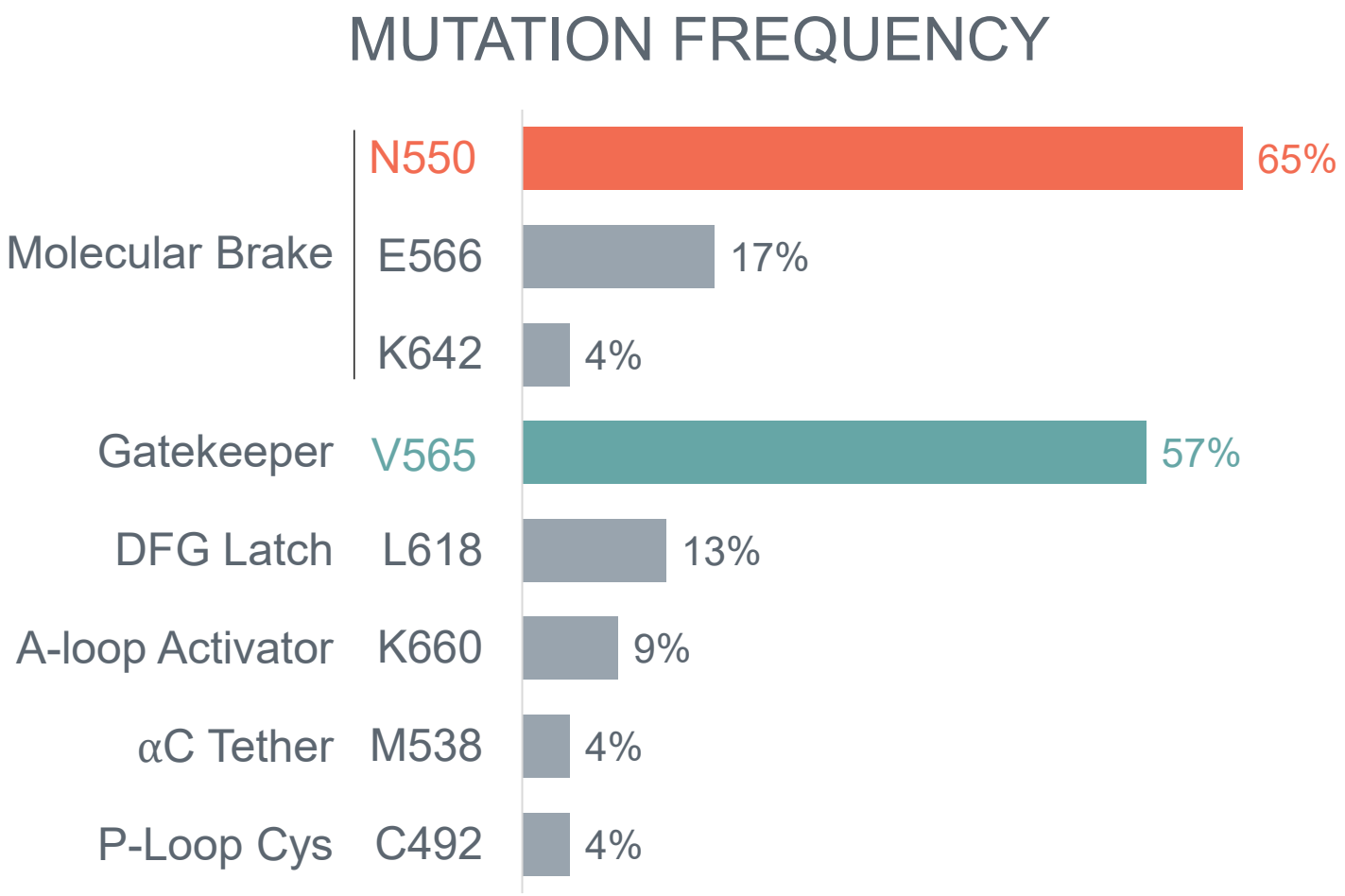
TYRA-200 showed high selectivity for FGFR1/2/3, sparing FGFR4



	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



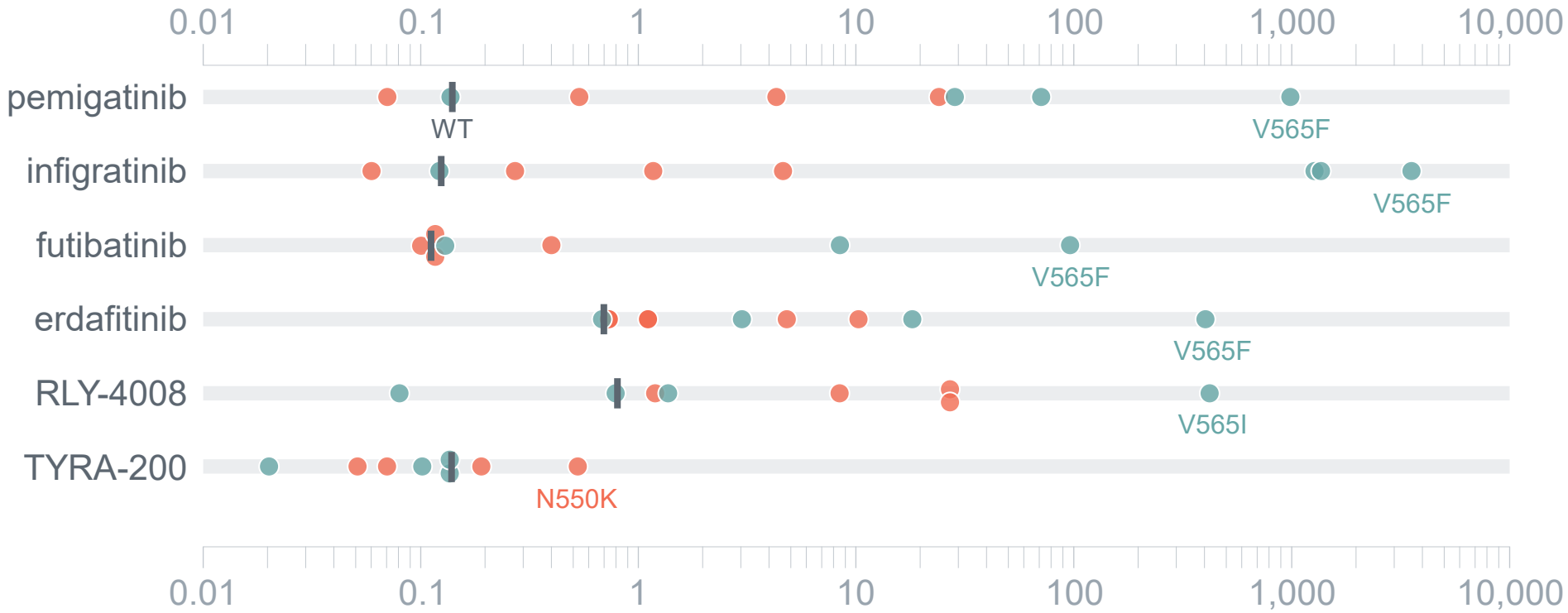
Over 50% of patients who recur on FGFR therapy have gatekeeper and molecular brake mutations

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})

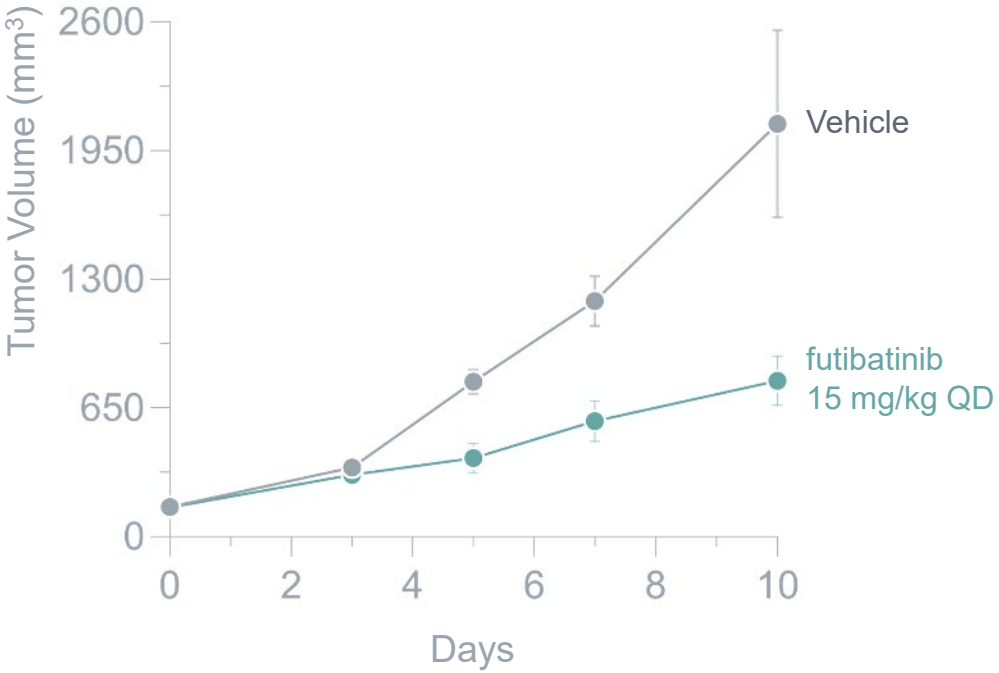
Variants tested: **WT, N550D, N550H, N550K, N550T, V565F, V565L, V565I**



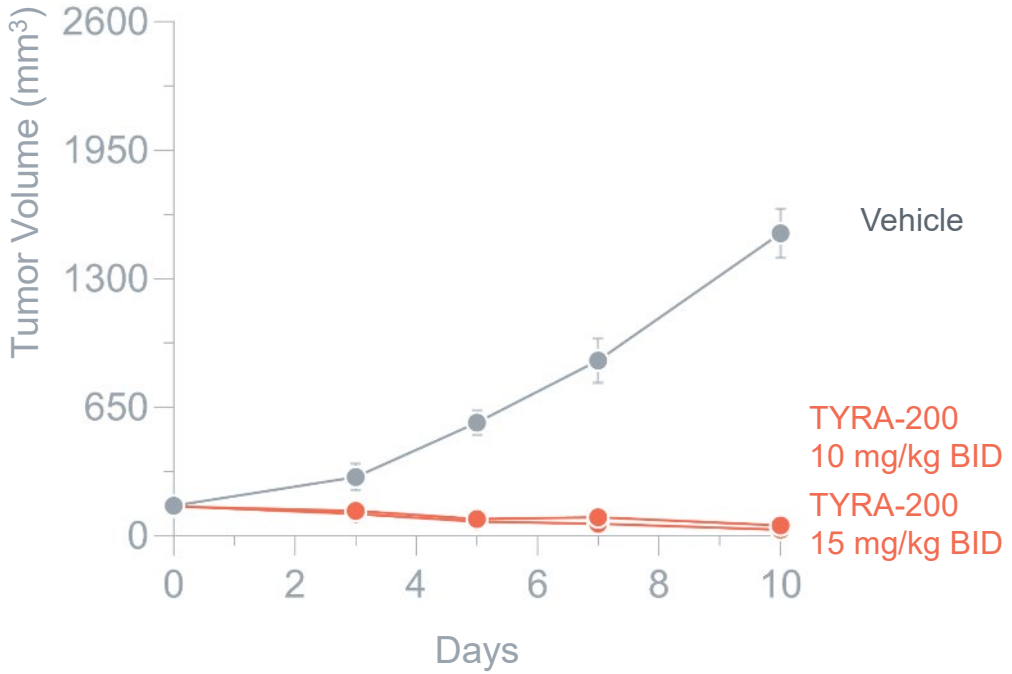
Enzymatic IC50 measurements generated at Reaction Biology Corp using Tyra enzymes.
All experiments conducted under identical conditions, tested in duplicate.

TYRA-200 regressed tumors with gatekeeper mutations

Ba/F3 FGFR2 V565F Allografts

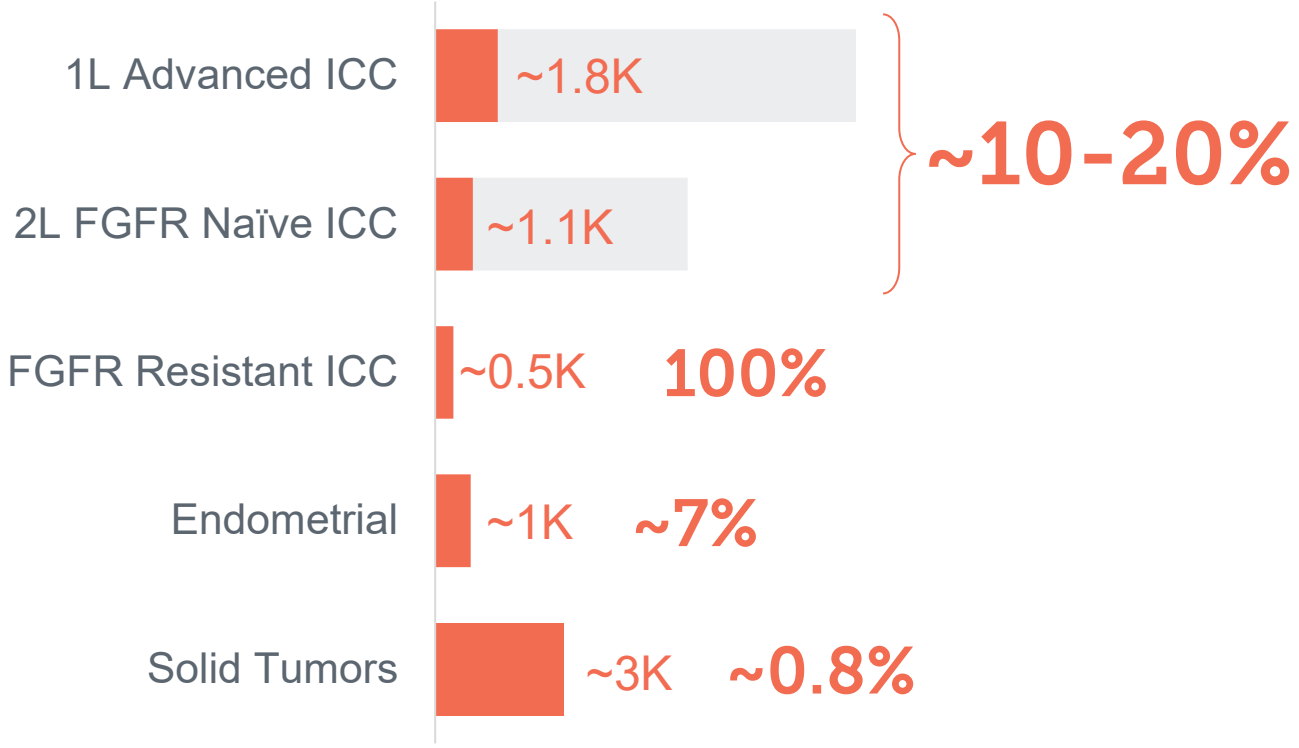


Ba/F3 FGFR2 V565F Allografts



TYRA-200 has multiple opportunities across FGFR2+ solid tumors

Estimated 2025 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 2025; Seigel, 2025; 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	~1.8K	CPI + Chemo	Only ~27% of patients respond; Increased PFS (Durva+Gem/Cis: 7.2mo[2])
2nd Line	~1.1K	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?

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