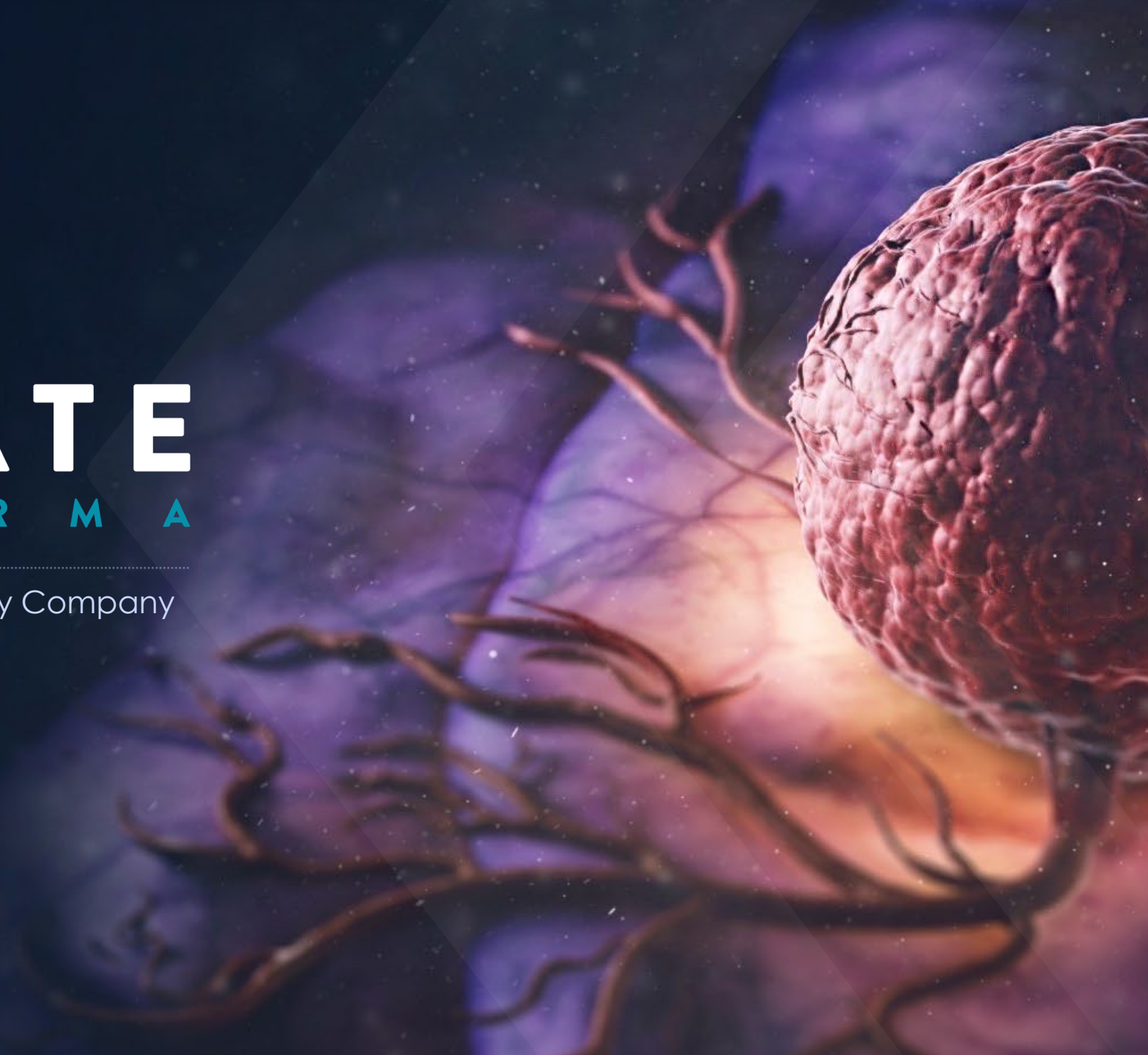
KINNATE BIOPHARMA

A Clinical-Stage Precision Oncology Company

Corporate Presentation
May 2023



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This presentation (including the accompanying oral presentation) contains forward-looking statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including statements that state or imply that the results of our preclinical studies or clinical trials to date are predictive of future clinical trial results, that certain of our approaches to drug development will be successful, faster than other approaches, or have the highest probability of success for us, that our estimates of the timing of future IND filings, data releases, and similar events will be achieved, that our pipeline programs will achieve best-in-class status or provide predicted benefits, that our cash resources will be sufficient to achieve certain objectives and that the markets for certain drugs will remain large and that we will be able to successfully address these markets, our expectations regarding our preliminary and unaudited financial results, statements regarding our future financial condition, results of operations, business strategy and plans, and objectives of management for future operations, as well as statements regarding industry trends, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potentially" "predict," "should," "will" or the negative of these terms or other similar expressions. We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs.

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These and other risks, uncertainties, assumptions and other factors are further described under the heading "Risk Factors" in our Quarterly Report on Form 10-Q for the quarter ended March 31, 2023, as well as in our subsequent filings we make with the Securities and Exchange Commission (SEC). You may view our filings with the SEC at their website (www.sec.gov). New risk factors emerge from time to time and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. Investors should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. Our forward-looking statements speak only as of the date of this presentation, and except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation. In addition, statements that "we believe" and similar statements reflect our beliefs and opinions on the relevant subject. These statements are based upon information available to us as of the date of this presentation, and while we believe such information forms a reasonable basis for such statements, such information may be limited or incomplete, and our statements should not be read to indicate that we have conducted an exhaustive inquiry into, or review of, all potentially available relevant information.

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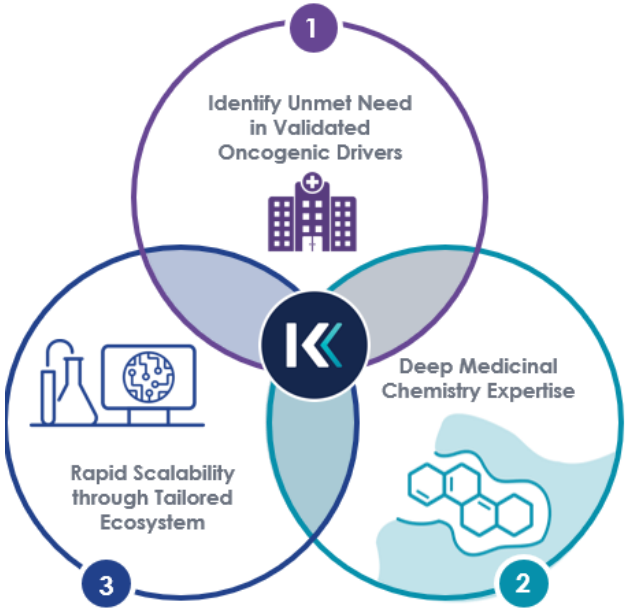
Certain data in this presentation are based on cross-study comparisons and are not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities or differences.

Highly Productive Discovery Engine Optimized for Speed, Probability of Success

Platform Focused on Design and Development of Wholly-Owned and Potential Best-in-Class Molecules



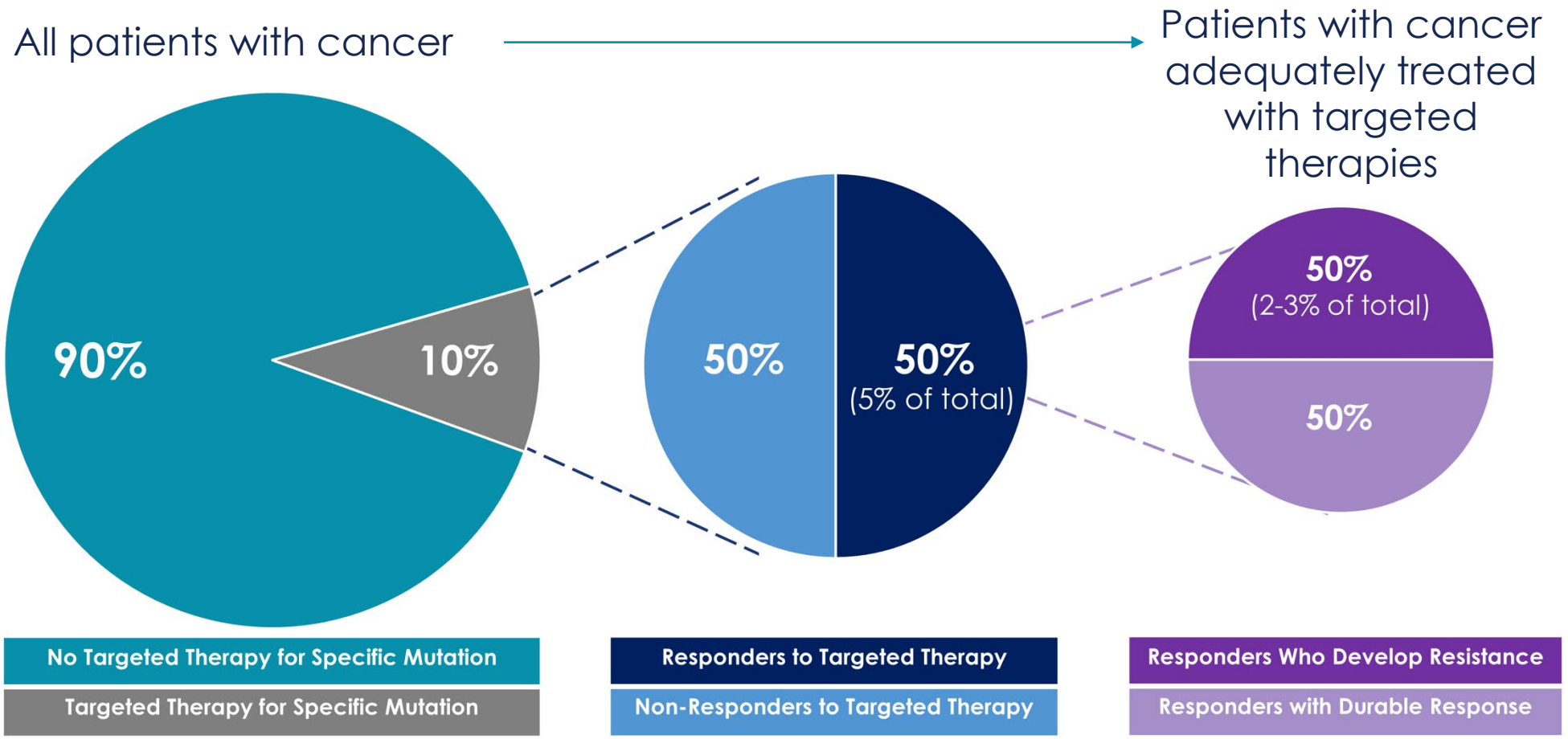
In Five Years Since Founding…

- Exarafenib – conceived, designed, DC/IND, delivered on FIH trial
- KIN-3248 – conceived, designed, DC/IND, now in clinic
- Third and fourth DCs generated from this engine
- Goal of one IND per year

Well-capitalized to invest in innovation:
$231.2 million cash on hand*

DC=drug candidate nomination; IND-investigational new IND; FIH=first-in-human
*Cash, cash equivalents and investments as of March 31, 2023

Expanding on the Promise of Targeted Therapies in Oncology



Source: Marquart JAMA ONOCLOGY 2018 https://pubmed.ncbi.nlm.nih.gov/29710180/; data available in USPIs for targeted cancer therapies approved in the U.S.

Multiple Ongoing Clinical, IND-Enabling and Discovery Programs

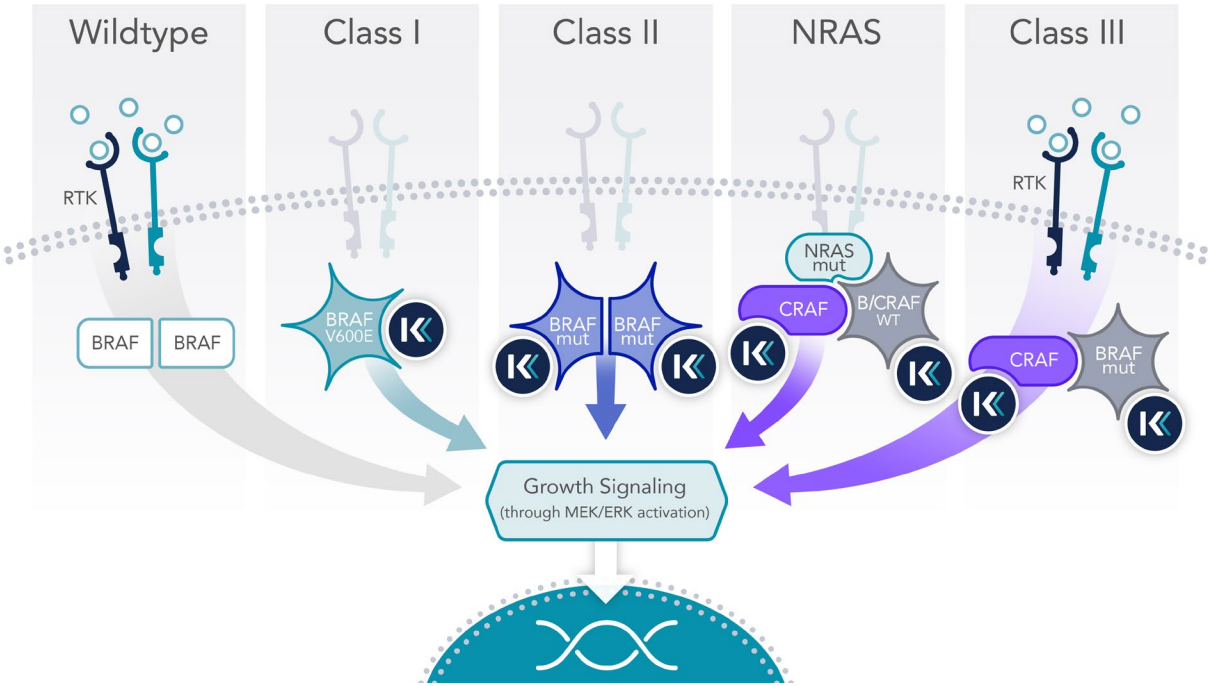
Target, Program	Study Name	Indications	Discovery	IND-Enabling	Phase 1a	Phase 1b	Phase 2/3	Anticipated Catalysts
Exarafenib RAF-Driven & Dependent	KN-8701	BRAF-Driven Advanced Adult Solid Tumors	Monotherapy					Dose Expansion Data in H1 2024
		Advanced NRAS Mutant Melanoma	Combination with Binimetinib					Expansion Dose Selection in H2 2023
KIN-3248 FGFR2/3-Driven	KN-4802	Naïve + Pre-treated FGFR2/3 Driven Advanced Adult Solid Tumors						Initial Clinical Data in H2 2023
KIN-7136 Brain Penetrant MEK		MAPK-Driven Advanced Adult Solid Tumors	Monotherapy & Combination with Exarafenib					Expect to Enter Clinic in H2 2023
KIN-8741 c-MET, Covers Acquired Resistance		c-Met-Driven Advanced Adult Solid Tumors						Expect to Enter Clinic in H1 2024
KIN-004 CDK12		Adult Solid Tumors						Exploring Strategic Alternatives
Multiple Undisclosed Leads in Research Stage, Goal of 1 IND a Year								

RAF Program

Exarafenib (KIN-2787)

Patients With Tumors Harboring BRAF or NRAS Represent a Broad Population With Significant Unmet Need[1,2]

- BRAF Class II/III alterations comprise more than half of oncogenic BRAF alterations[1,a]
  - Estimated to occur in 2.1% of solid tumors[2]
  - Most common in NSCLC, CRC, melanoma, and prostate cancer[2]
- BRAF Class II/III alterations are associated with poorer clinical outcomes than Class I[2]
  - Median OS was shorter for patients with NSCLC or melanoma with Class II/III vs Class I alterations[1]
- NRAS mutant melanoma is a RAF dependent cancer, representing ~20-25% of melanoma[3]
  - These patients also have poor clinical outcomes[4]
- No approved targeted therapies for tumors with BRAF Class II/III alterations and NRAS mutant melanoma[1,2]



	Class I	Class II	Class III
% of *BRAF*-pos[a]	45%	26%	28%

[a] Based on a real-world analysis of 5896 pts with metastatic/advanced cancers harboring *BRAF* alterations from a clinical databased of 160,000+ pts profiled by the Guardant360® assay. Multiple *BRAF* alterations were identified in 1.8% of pts (107/5896). [b] Median OS from metastatic diagnosis of NSCLC (n=938) or melanoma (n=333). 1. Severson P, et al. Poster presented at: 2022 AACR Annual Meeting; April 8–13; New Orleans, LA. Abstract 4122. 2. Severson P, et al. Poster presented at: ESMO Targeted Anticancer Therapies Congress 2022. March 7–8 [Virtual]. Abstract 40P. 3. Genomic data generated from AACR GENIE Project Data: Version 10.0-public Powering Precision Medicine Through An International Consortium. Cancer Discov 7(8): 818-831, 2017 (https://genie.cbioportal.org/); 4. Dummer et al., The Lancet, 2017.

Exarafenib Achieved Monotherapy MTD With Best-in-Class Profile[1]

Substantial exposures achieved with monotherapy

- MTD at 300 mg bid
- ~19x free AUC vs. belvarafenib
- At least 5-fold coverage of the IC50 in Class II cell lines

Generally well-tolerated with only 3% (n=2) drug-related discontinuations

- 95% avg dose intensity at 300 mg bid
- Limited GI AEs, ~3% Grade 2+
- ~18% Grade 3+ drug- related AEs
- No evidence of paradoxical activation at therapeutically relevant exposures

Promising early efficacy, especially in priority BRAF Class II and NRAS subtypes

Overall

- 6 total PRs, including 5 RECIST confirmed
- 30% ORR in Class II & NRAS* (3 of 10) at 300 mg bid
- Deep response: avg 61% tumor reduction and 7 months DoT in responders

Class II

- 33% (1 of 3) ORR at 300 mg bid
- 71% (5 of 7) tumor reduction across doses
- 86% (6 of 7) DCR across doses

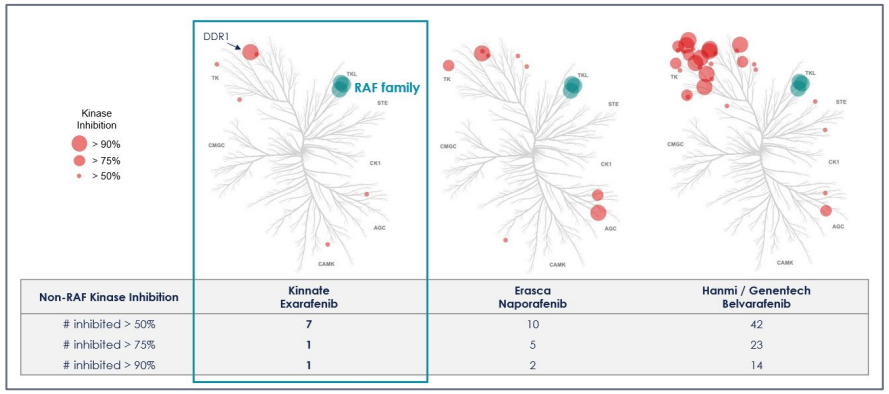
NRAS

- 29% (2 of 7) ORR at 300 mg bid*

[1]Data cut as of Feb 28, 2023. *Includes NRAS patient with co-occurring BRAF alteration
bid=two times daily; AUC=area under the curve; AE=adverse events; GI=gastrointestinal; PR=partial response; ORR=overall response rate; DCR=disease control rate; DoT=duration of therapy

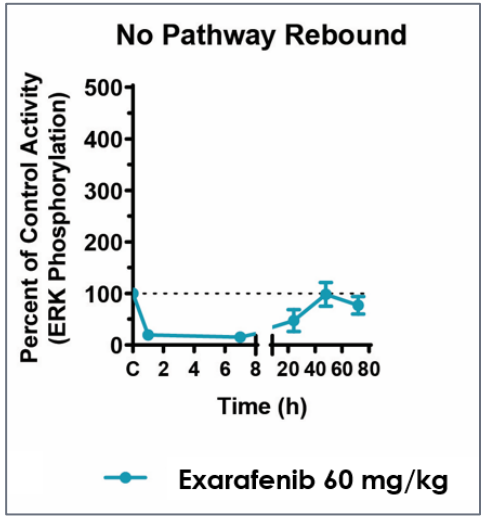
Unique Structural Attributes and Substantial Preclinical Evidence Support the Case for Exarafenib's Best-in-Class Profile

Selective drug



Non-RAF Kinase Inhibition	Kinnate Exarafenib	Erasca Naporafenib	Hanmi / Genentech Belvarafenib
# inhibited > 50%	7	10	42
# inhibited > 75%	1	5	23
# inhibited > 90%	1	2	14

Equipotent inhibition



Broad alteration coverage

BRAF Status	Tumor Cell Line	Lineage	MAPK Pathway Alteration(s)	pERK Inhibition EC50 (nM)			
				Pfizer Binimetinib	Erasca Naporafenib	Hanmi / Genentech Belvarafenib	Kinnate Exarafenib
Class I	A-375	Melanoma	$BRAF^{V600E}$	7	171	67	67
	Colo800	Melanoma	$BRAF^{V600E}$	6	242	108	112
Class II	BxPC-3	Pancreatic	$BRAF^{Indel(VTAFTP)}$	3	32	42	51
	OV-90	Ovarian	$BRAF^{Indel(NVTAP)}$	4	24	22	26
	NCI-H2405	NSCLC	$BRAF^{Indel(LNVTAP)}$	6	5	8	10
Class III	WM3629	Melanoma	$BRAF^{D594G}$, $NRAS^{G12D}$	5	6	4	9
	CAL-12T	NSCLC	$BRAF^{G466V}$	3	19	41	18
Wild Type (WT)	NCI-H358	NSCLC	$BRAF^{WT}$, $KRAS^{G12C}$	1	153	303	351
	CHL-1	Melanoma	$BRAF^{WT}$, $NRAS^{WT}$	5	291	443	580
	BJ	Normal fibroblast	Wild type	31	4686	2923	7963

Kinase	Exarafenib IC50 (nM)
CRAF	0.573
$BRAF^{V600E}$	1.53
ARAF	2.41
BRAF	3.46

Superior drug properties

	Erasca Naporafenib	Hanmi / Genentech Belvarafenib	Kinnate Exarafenib
Human Plasma Free Fraction (%)	<1	<1	7
Aqueous Solubility (uM):			
pH = 2	50	266	312
pH = 4.5	7	0.4	196
pH = 7.4	6	0.1	29

Exarafenib is designed to achieve the optimal product profile needed for BRAF-driven cancers

Based on Kinnate generated data. Certain data on this slide are based on cross-study comparisons and are not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities or differences.

Exarafenib Clinical Data Provides Initial Validation of Predicted Sensitivity to BRAF Inhibition

Alteration and Tumor Types Continue to Inform How Sensitive Patient Will Be to Pan-RAF Therapy

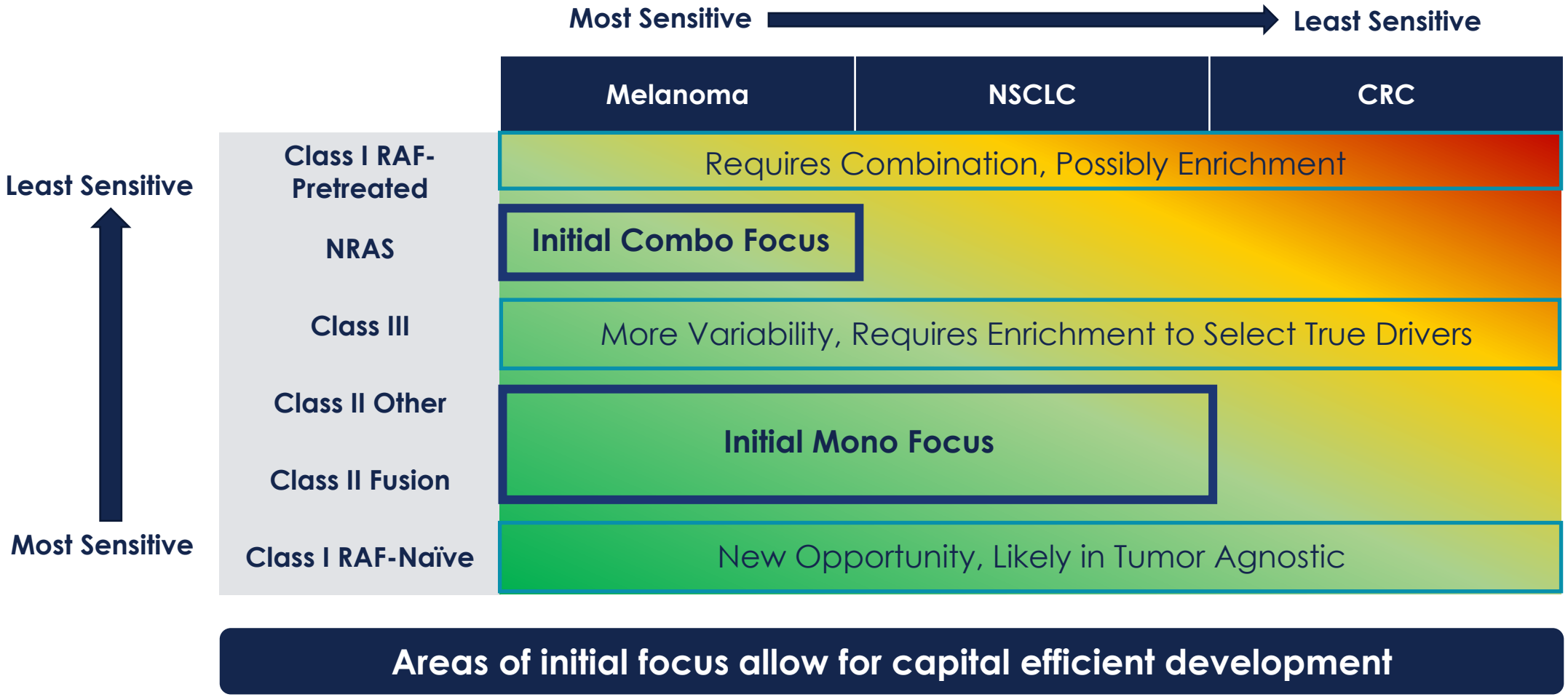
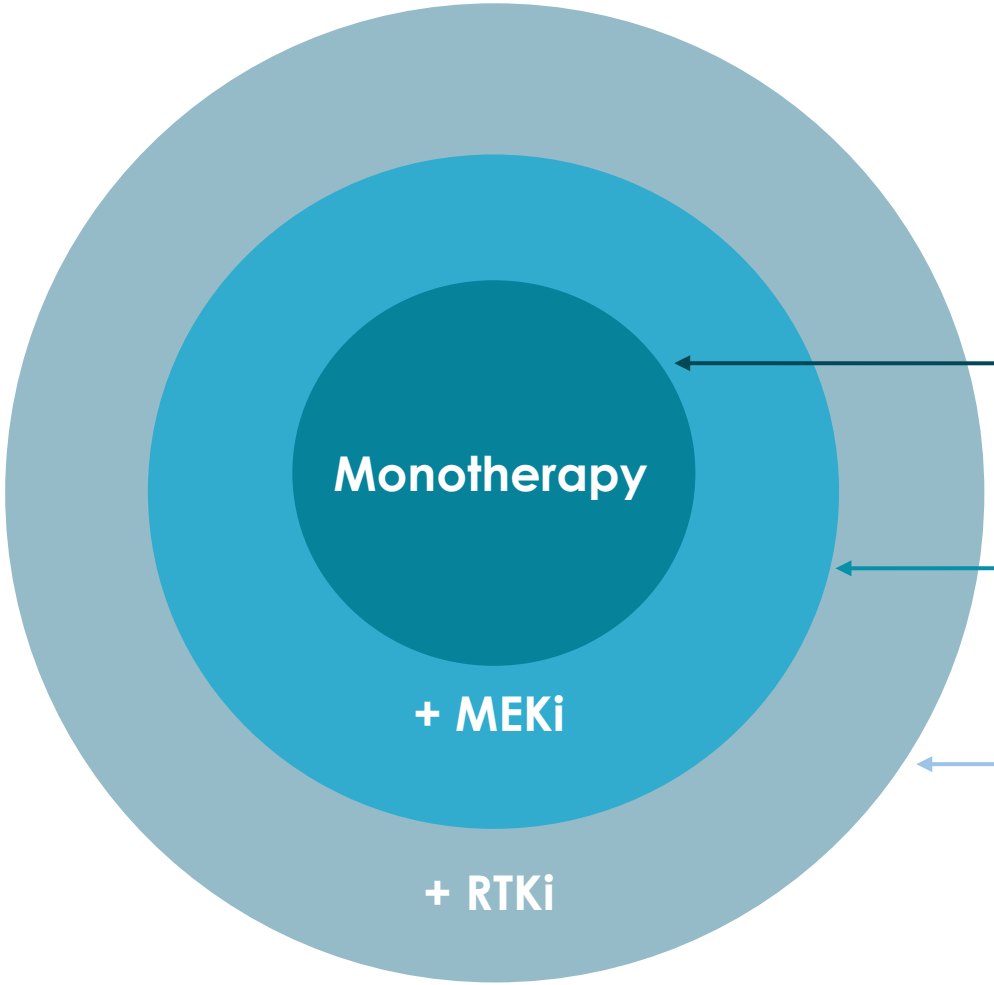


Illustration based on Kinnate preclinical data, prior clinical data from other pan-RAF inhibitors and publications. Note: other solid tumors beyond those in the illustration have potential opportunity with a pan-RAF but are not as well-characterized.

Well-tolerated Monotherapy Makes Promising Combination Partner



Monotherapy option may be best for sensitive populations like Class II and Class I RAF naïve patients

Combination with MEKi to further inhibit MAPK pathway to increase responses and duration in more difficult patient populations e.g., NRAS melanoma and Class I pre-treated

Combination with other RTKi (e.g., KRAS, EGFR) where RAF mutations co-occur (e.g., Class III or resistance mutations to primary drivers)

With compelling PK/PD, responses and especially a favorable safety/tolerability profile, exarafenib can serve as backbone for other combinations

Exarafenib Demonstrated a Favorable Safety and Tolerability Profile Across Dose Levels & 300 mg bid

- At therapeutically relevant exposures, no cutaneous (skin) evidence of paradoxical activation
- Only 2 DLTs observed, both at highest dose level (400 mg bid), Gr. 3 acneiform rash and Gr. 3 macular rash
- TRAEs at any Grade occurred in 73.3% (n=44) of patients, with 18.3% (n=11) of TRAEs reported as Gr. 3+
- Most common TRAEs of any Grade were skin related (21.7%; n=13), with 5% (n=3) of patients having skin events that were Gr. 3+
- Gr. 2 GI TRAEs occurred in 3.3% (n=2) of patients with no Gr. 3 GI TRAEs observed
- Reversible, asymptomatic increased ALT/AST TRAEs were reported at Gr. 3 (n=4; 6.7%) and Gr. 4 (n=1; 1.7%)
- At 300 mg bid (n=29), Gr. 1 (n=7) and Gr. 2 (n=8) TRAEs occurred in 48.3% of patients, with 28% (n=8) of TRAEs reported as Gr. 3+
- Overall relative mean dose intensity was 97% and was 95% in patients treated at 300 mg bid

Tolerability profile led to only 3% (n=2/60) of patients to discontinue therapy due to TRAEs

Safety Analysis population: n = 60 participants across all Dose Levels and n = 29 participants at 300 mg bid (DL5). TRAE=Treatment Related Adverse Events.. AEs are reported by maximum toxicity grade, observed at a 5% or greater frequency across all 60 patients treated in 6 dose levels. Relative dose intensity refers to the total actual dose received divided by total planned dose for the period patient received the assigned dose. Data cut off: Feb. 28, 2023.

Exarafenib Safety Profile Across Dose Levels and 300 mg bid

	All Dose Levels (n=60)					Dose Level 5 (300 mg bid) (n=29)				
SOC, Preferred Term	**Gr. 1**	**Gr. 2**	**Gr. 3**	**Gr. 4**	**Any Grade**	**Gr. 1**	**Gr. 2**	**Gr. 3**	**Gr. 4**	**Any Grade**
Any Exarafenib-related AE (TRAEs)	15 (25.0%)	18 (30.0%)	9 (15.0%)	2 (3.3%)	**44 (73.3%)**	7 (24.1%)	8 (27.6%)	6 (20.7%)	2 (6.9%)	**23 (79.3%)**
Gastrointestinal disorders										
Nausea	9 (15.0%)	1 (1.7%)			**10 (16.7%)**	3 (10.3%)	1 (3.4%)			**4 (13.8%)**
Oral pain	3 (5.0%)				**3 (5.0%)**	1 (3.4%)				**1 (3.4%)**
Vomiting	4 (6.7%)	1 (1.7%)			**5 (8.3%)**	2 (6.9%)	1 (3.4%)			**3 (10.3%)**
General disorders & admin. site conditions										
Asthenia	4 (6.7%)	1 (1.7%)			**5 (8.3%)**	3 (10.3%)	1 (3.4%)			**4 (13.8%)**
Fatigue	5 (8.3%)	3 (5.0%)			**8 (13.3%)**	4 (13.8%)	1 (3.4%)			**5 (17.2%)**
Investigations										
Alanine aminotransferase (ALT) increased	2 (3.3%)	3 (5.0%)	4 (6.7%)	1 (1.7%)	**10 (16.7%)**	1 (3.4%)	3 (10.3%)	3 (10.3%)	1 (3.4%)	**8 (27.6%)**
Aspartate aminotransferase (AST) increased	4 (6.7%)	3 (5.0%)	4 (6.7%)		**11 (18.3%)**	2 (6.9%)	3 (10.3%)	3 (10.3%)		**8 (27.6%)**
Blood alkaline phosphatase increased	3 (5.0%)				**3 (5.0%)**	1 (3.4%)				**1 (3.4%)**
Blood bilirubin increased	2 (3.3%)	1 (1.7%)			**3 (5.0%)**	1 (3.4%)	1 (3.4%)			**2 (6.9%)**
Blood creatine phosphokinase increased	4 (6.7%)				**4 (6.7%)**	2 (6.9%)				**2 (6.9%)**
Metabolism and nutrition disorders										
Decreased appetite	5 (8.3%)	1 (1.7%)			**6 (10.0%)**	2 (6.9%)	1 (3.4%)			**3 (10.3%)**
Musculoskeletal & connective tissue disorders										
Myalgia	3 (5.0%)	1 (1.7%)			**4 (6.7%)**	3 (10.3%)				**3 (10.3%)**
Nervous system disorders										
Dizziness	3 (5.0%)				**3 (5.0%)**	2 (6.9%)				**2 (6.9%)**
Skin & subcutaneous tissue disorders										
Dermatitis acneiform	8 (13.3%)	3 (5.0%)	1 (1.7%)	1 (1.7%)	**13 (21.7%)**	5 (17.2%)	2 (6.9%)		1 (3.4%)	**8 (27.6%)**
Pruritus	7 (11.7%)	2 (3.3%)			**9 (15.0%)**	4 (13.8%)	2 (6.9%)			**6 (20.7%)**
Rash (any) *	12 (20.0%)	5 (8.3%)	1 (1.7%)		**18 (30.0%)**	6 (20.7%)	3 (10.3%)			**9 (31.0%)**

Safety Analysis population, n = 60 participants across all Dose Levels. Relatedness to exarafenib reported per Investigator assessment. Exarafenib-related or Treatment Related AEs (TRAEs) occurring in ≥ 5% (≥ 3 pts) in Safety Analysis population are annotated by SOC, Preferred Term, Maximum toxicity grade. * Rash (any) includes any of the following Preferred Terms: 'rash'; 'rash macular'; 'rash popular'; 'rash maculopapular'; 'rash morbilliform'

Data cut off: Feb. 28, 2023

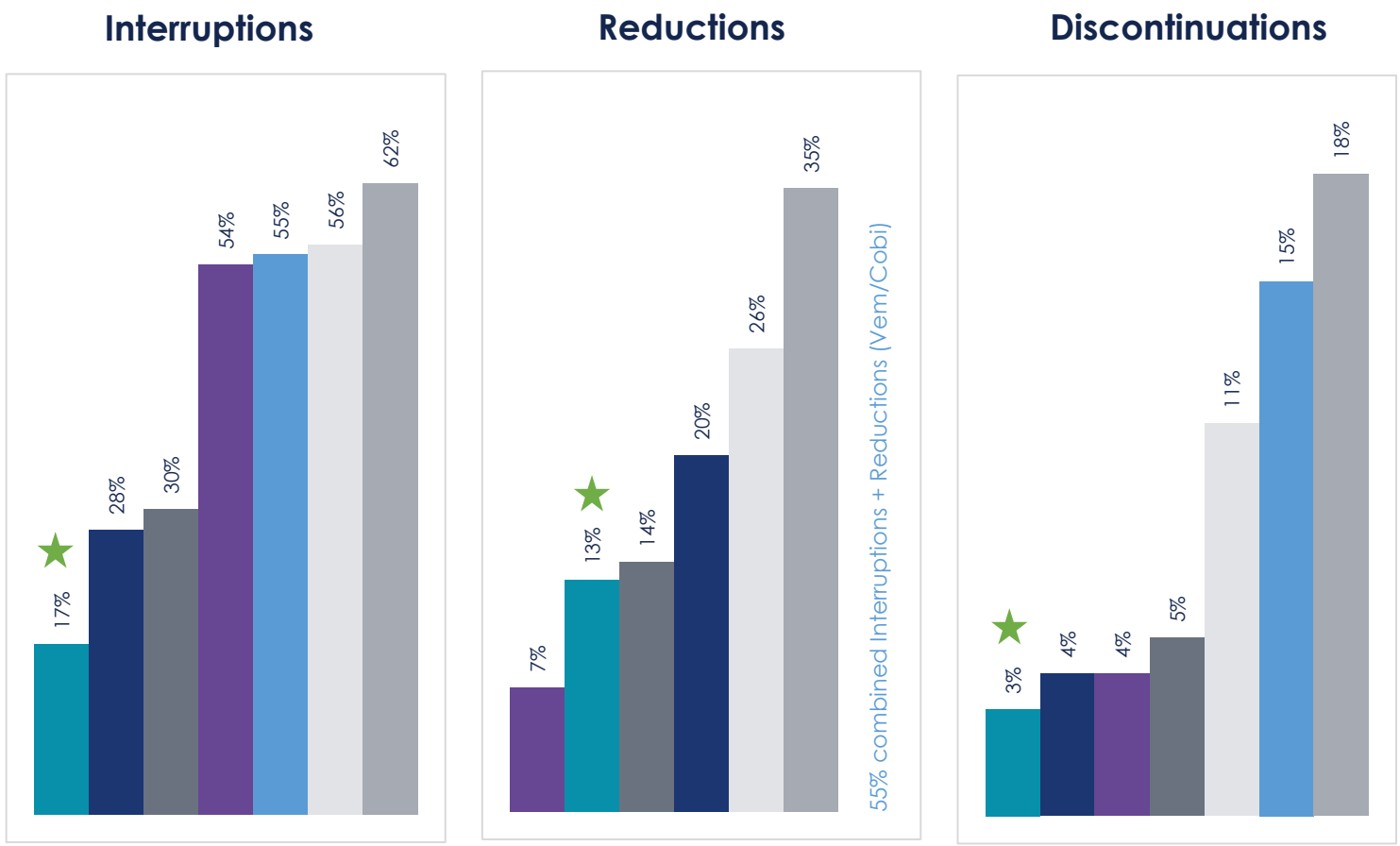
Exarafenib Achieved Substantial and Differentiated Dose Intensity

Tolerability Profile Led to Only 2 Patients Discontinuing Treatment Due To Drug-Related Toxicity

In all exarafenib treated patients (n=60), the relative mean dose intensity* was **97%** and in patients treated at 300 mg bid (n=29), it was **95%**. The median for both patient sets was 100%.

RAF Drug-Related Tolerability Landscape Assessment

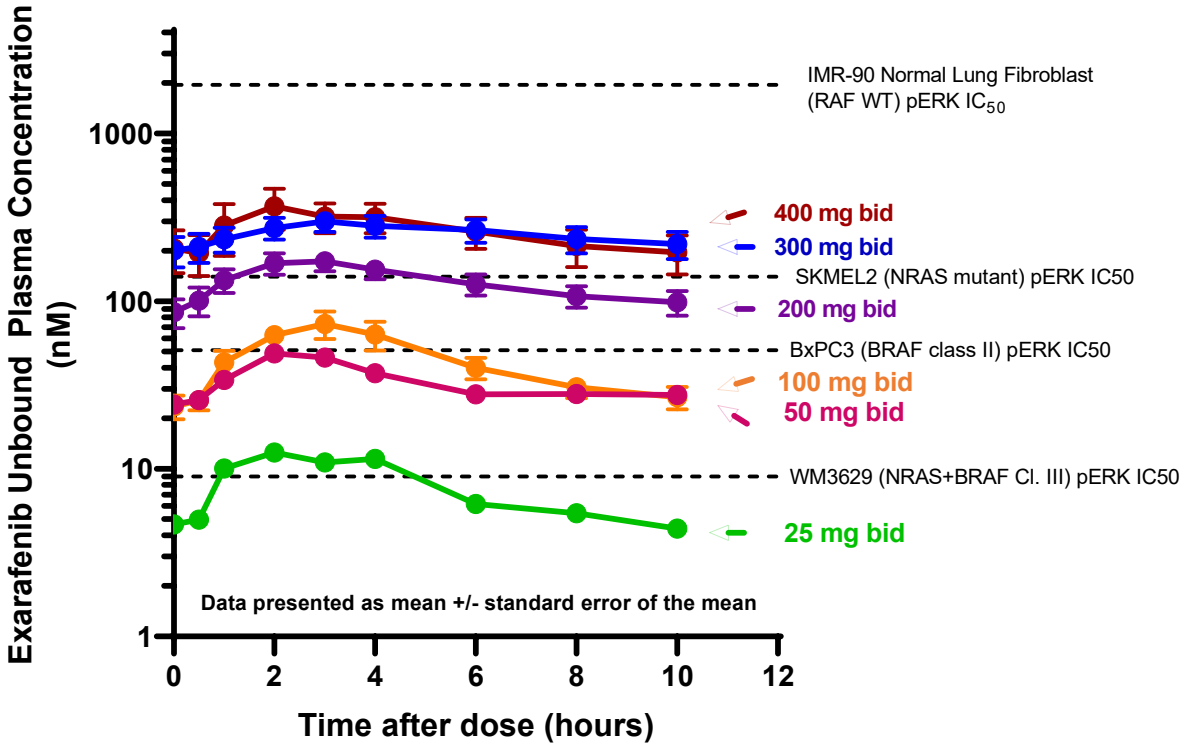
- Exarafenib
- FORE-8394 - Solid Tumors
- Belvarafenib - Solid Tumors
- Vemurafenib + Cobimetinib – Melanoma
- Encorafenib + Binimetinib – Melanoma
- Dabrafenib + Trametinib – NSCLC
- Dabrafenib + Trametinib – Met. Melanoma



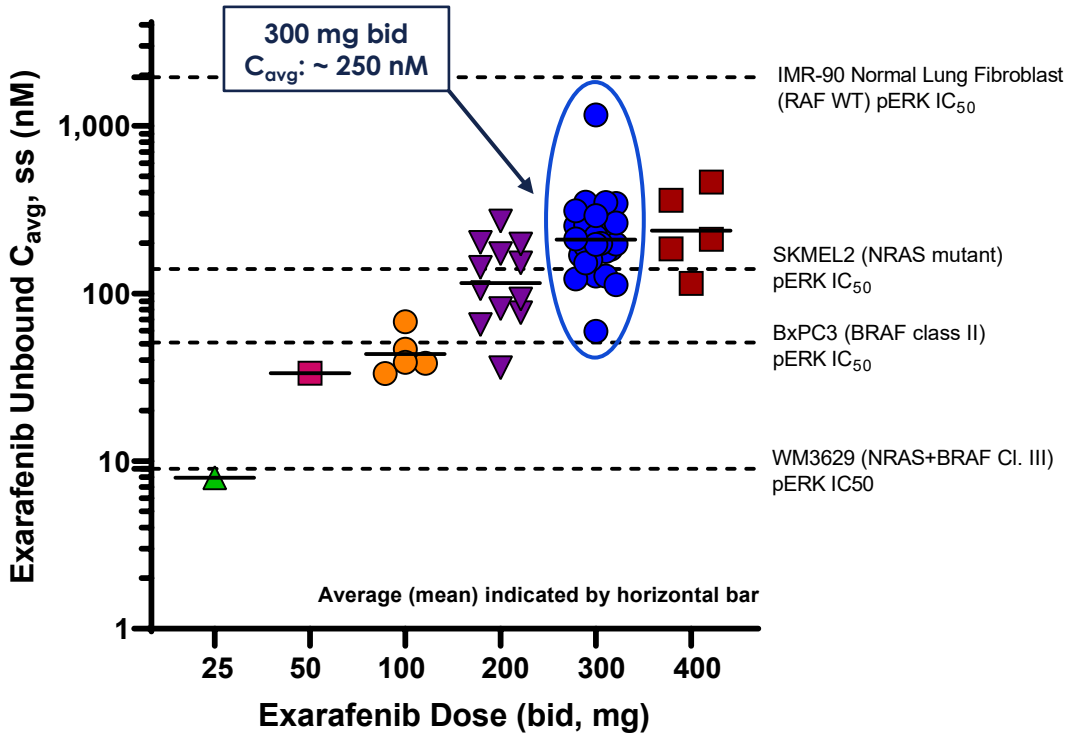
*Relative dose intensity refers to the total actual dose received divided by total planned dose for the period patient received the assigned dose. Exarafenib data cut as of Feb 28, 2023. USPI used as source for Dabrafenib, Encorafenib, Vemurafenib – Accessed 27 March 2023. Cobimetinib USPI for vemurafenib + cobimetinib data. Belvarafenib data from Yen et al, Nature 2021; FORE-8394 data from Sherman, ESMO 2022 Poster. Note: Belvarafenib/FORE-8394 data represent Treatment Emergent Adverse Events leading to Interruptions, Reductions, Discontinuations; relationship to study drug not specified.

Favorable Pharmacokinetics: Exarafenib Delivered Dose-Dependent & Steady State Exposures

Unbound Exarafenib Exposures at Steady State (nM; all Dose Levels)



Unbound Average Exarafenib Exposures At Steady State (nM; all Dose Levels)



- 8-hour half life
- Unbound exarafenib exposures and C_{avg} concentrations increase dose proportionately
- Steady state exarafenib exposures are achieved at Cycle 1 Day 15
- At 300 mg bid, unbound C_{avg} concentrations exceed in vitro pERK IC_{50} values across all BRAF/NRAS mutant cell lines - including 5-fold higher relative to a representative BRAF Class II cell line

PK Dataset: March 16, 2023

Pan-RAF Inhibitor Clinical PK Comparison

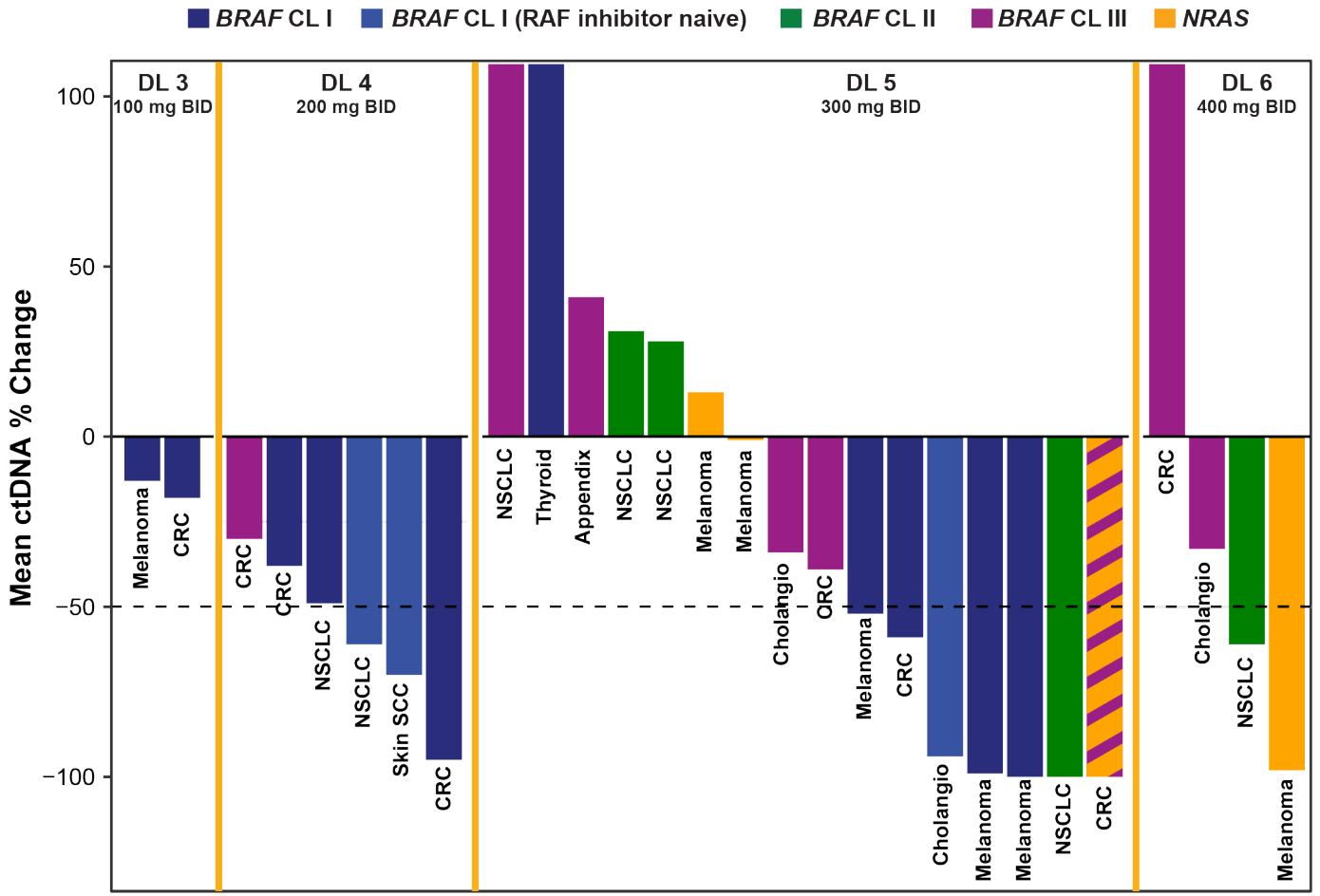
Exarafenib is Achieving Monotherapy Exposures Not Seen with Other Agents

RAF Inhibitor	Naporafenib	Belvarafenib	Exarafenib
Company	ERASCA	Genentech A Member of the Roche Group / Hanmi	KINNATE BIOPHARMA
Dose	600 mg bid	450 mg bid	300 mg bid (DL5)
Human Fraction Unbound (%)	<1	0.11	6.8
Clinical Total $AUC_{0\text{-}24,ss}$ (ng*h/mL)	79,000[1]	127,000[2]	38,000[3]
Clinical Free $AUC_{0\text{-}24,ss}$ (ng*h/mL)	< 790	140	2,600

Based on Kinnate generated data for Human Fraction Unbound and approximate published clinical PK data for Total AUCs for Naporafenib & Belvarafenib; Exarafenib Clinical Total AUC from KN-8701 trial. Certain data on this slide are based on cross-study comparisons and are not based on any head-to-head clinical trials. Cross-study comparisons are inherently limited and may suggest misleading similarities or differences. (1)Janku et al., Phase 1 Study of LXH254 in Patients With Advanced Solid Tumors Harboring MAPK Pathway Alterations. Poster 2586. ASCO Annual Meeting (2018),(2) Yen et al, Nature 2021, (3) 2x 0–10-hour AUC.

Significant Decrease in Mean ctDNA Levels Spanned Multiple Cancer Types and BRAF/NRAS Alterations

71% (20/28) of MR Evaluable Patients Show Decreases in Mean ctDNA Levels at C2D1 or C3D1*

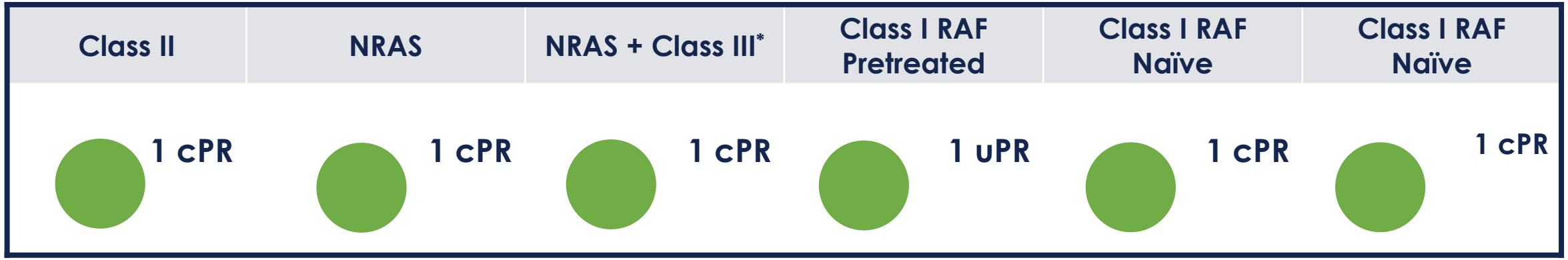


*Analysis: March 17, 2023; 28 patients evaluable for molecular response (MR)
MR is defined as a ≥ 50% average reduction in a pre-specified panel of tumor associated mutations at C2D1 or C3D1 compared to baseline (C1D1 or screening) samples.

Breadth of Responses Across Alteration and Tumor Types[1]

33% ORR in Class II & 29% ORR in NRAS at 300 mg bid; 33% ORR (2 of 6) in Class 1 Naïve at 200 mg bid+

Responses Across All Enrolled Alteration Types...



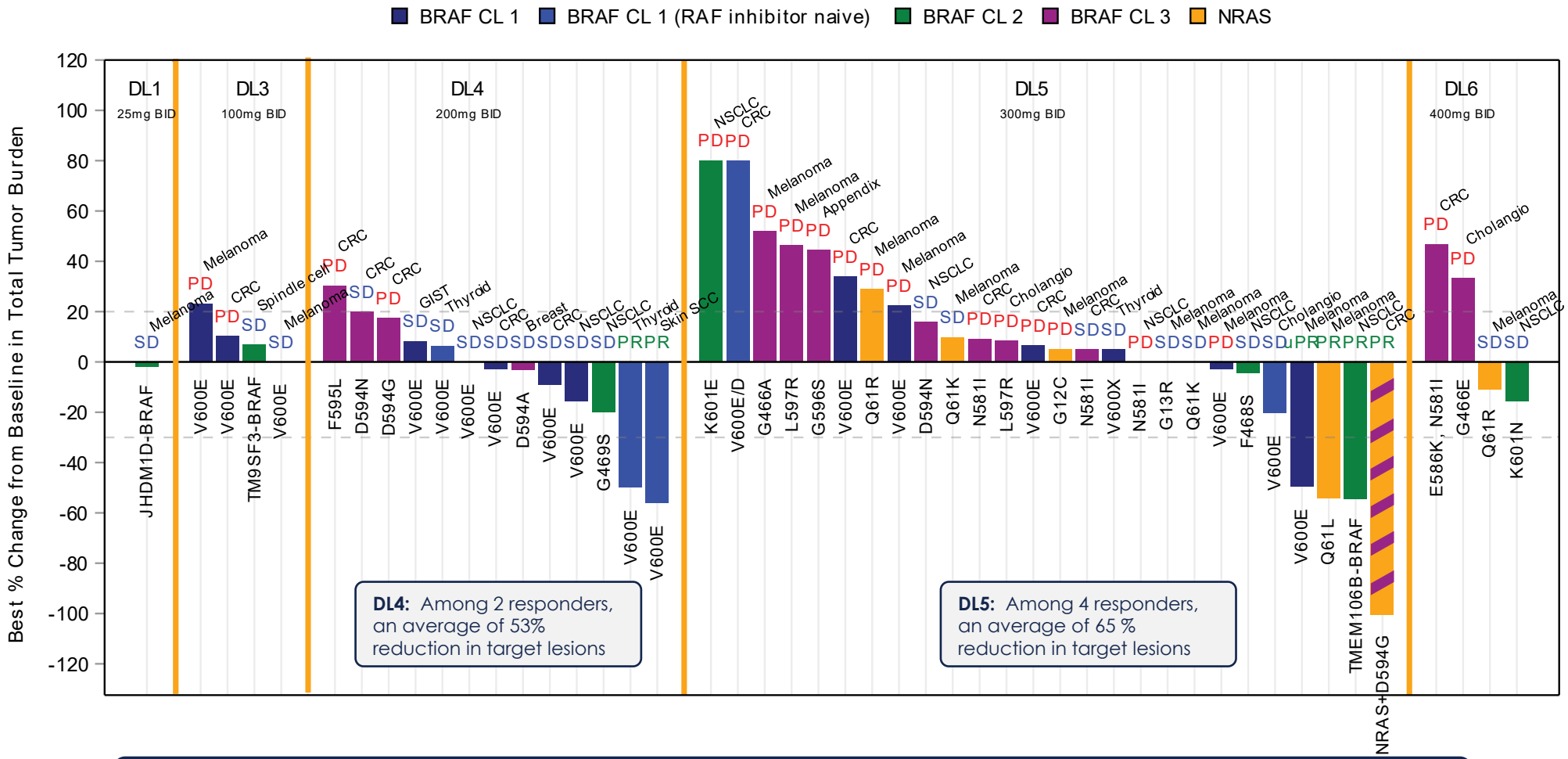
...And, Across Broad Range of Tumor Types

- NSCLC
- Melanoma x2
- CRC
- PTC
- SCC

[1]Data cut as of Feb 28, 2023 * Includes patient with NRAS co-occurring with a BRAF Class III alteration
ORR = overall response rate; uPR = unconfirmed partial response; cPR = confirmed partial response; NSCLC = non-small cell lung cancer; PTC = papillary thyroid cancer, CRC = colorectal cancer; SCC = squamous cell carcinoma

Tumor Regressions Observed Across Dose Levels

Average of 65% Reduction in Target Lesions Among 4 Responders at 300 mg bid

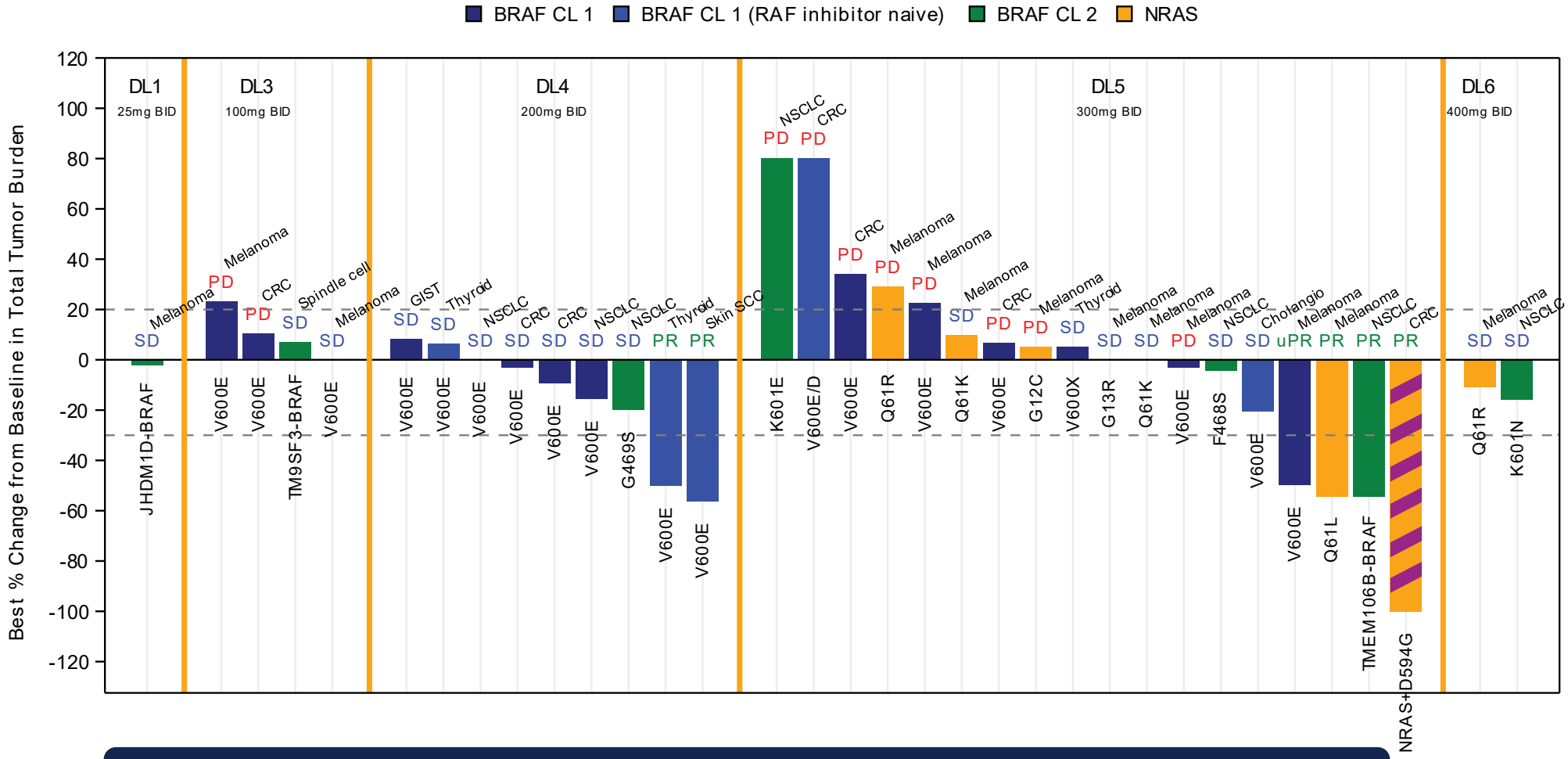


33% ORR in Class II & 29% ORR in NRAS at 300 mg bid

Efficacy Evaluable Population: 1 Pt (DL1, 25 mg bid) is not included as pt. had baseline measurable lesion, but had PD based upon appearance of a new lesion prior to post-baseline assessment – hence no percentage change could be calculated. Pts are analyzed by Dose Level at which they achieved or confirmed their best response. Data cut as of Feb 28, 2023.

Tumor Regressions in Patients with BRAF Class II, NRAS Alterations

Meaningful Activity in Class II and NRAS Supports Initial Focus in These Priority Segments

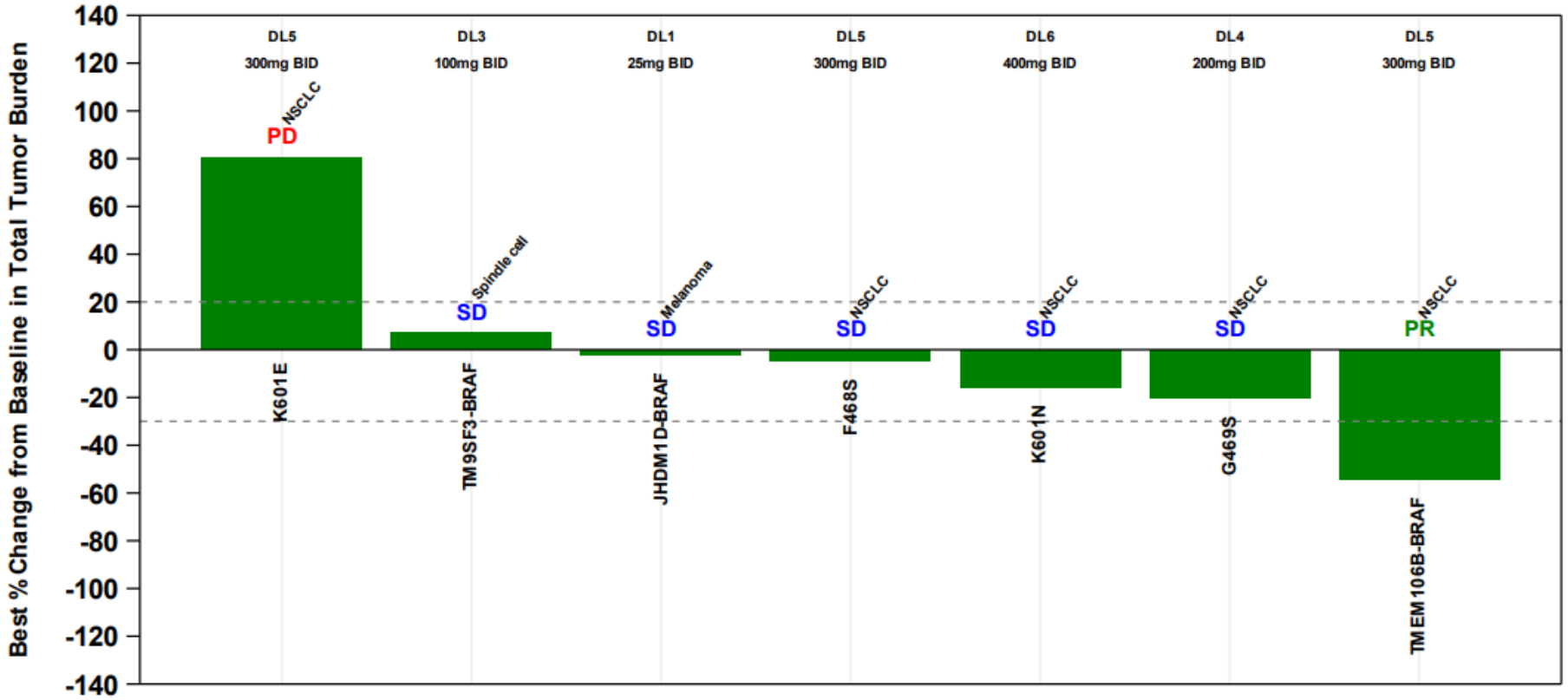


33% ORR in Class II & 29% ORR in NRAS at 300 mg bid

Efficacy Evaluable Population: 1 Pt (DL1, 25 mg bid) is not included as pt. had baseline measurable lesion, but had PD based upon appearance of a new lesion prior to post-baseline assessment – hence no percentage change could be calculated. Pts are analyzed by Dose Level at which they achieved or confirmed their best response. Data cut as of Feb 28, 2023.

Tumor Regressions in Patients with BRAF Class II

Meaningful Activity in Class II Supports Initial Focus in Priority Segment

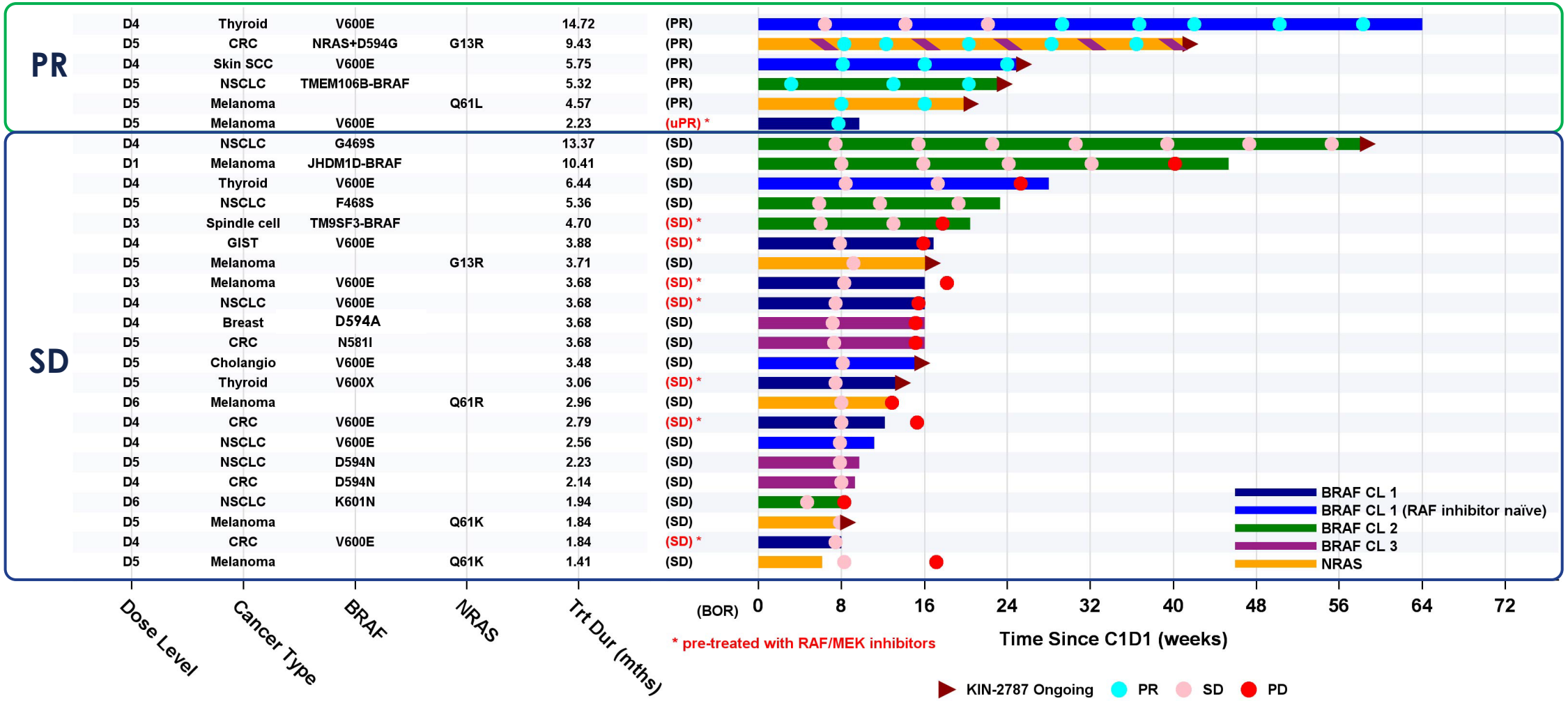


33% (1 of 3) ORR at 300 mg bid with 86% (6 of 7) DCR across doses

Efficacy Evaluable Population: 1 Pt (DL1, 25 mg bid) is not included as pt. had baseline measurable lesion, but had PD based upon appearance of a new lesion prior to post-baseline assessment – hence no percentage change could be calculated. Pts are analyzed by Dose Level at which they achieved or confirmed their best response. Data cut as of Feb 28, 2023.

Sustained Duration with Follow Up Ongoing

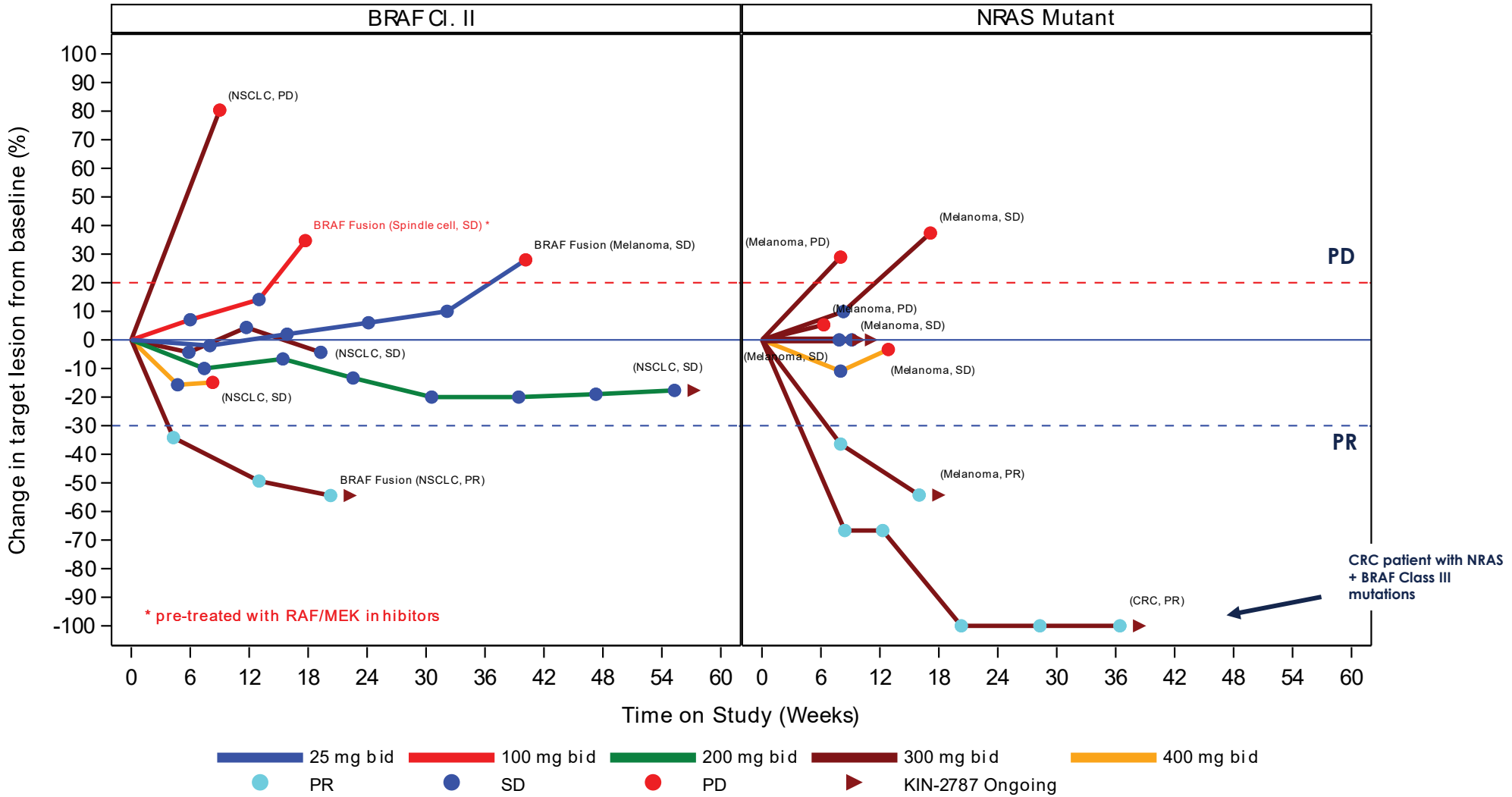
Responders Stayed on Treatment for an Average of 7 Months; mDOR Not Reached



Data cut as of Feb 28, 2023

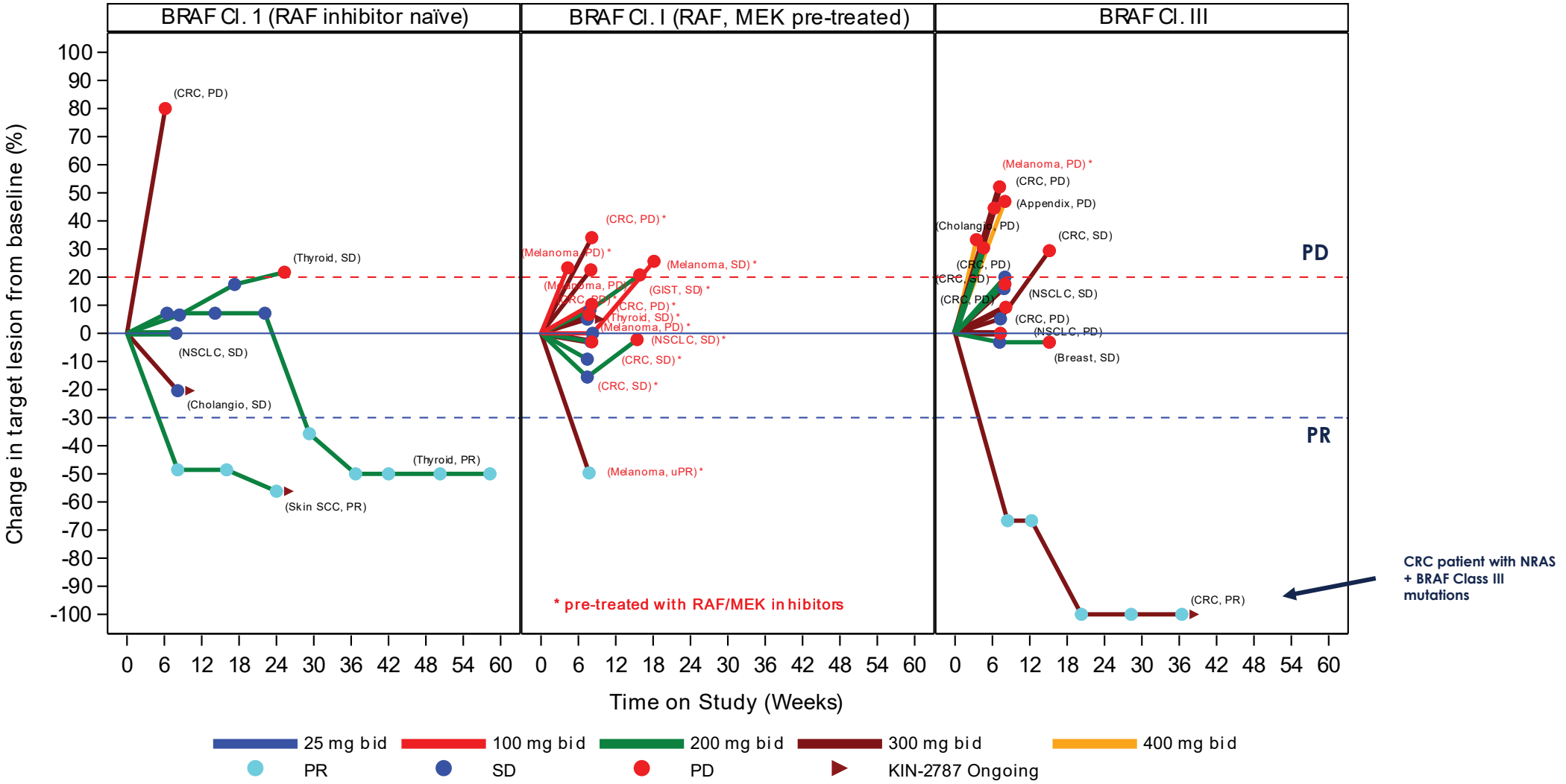
Compelling Data Supports Priority Focus in Class II and NRAS

Exarafenib Monotherapy Induced Rapid Response in These Patient Subtypes



Data cut as of Feb 28, 2023

Potential New Opportunity in BRAF Class 1 Naïve, Enrichment Needed in BRAF Class III and Class 1 Pretreated Patient Subtypes



Data cut as of Feb 28, 2023

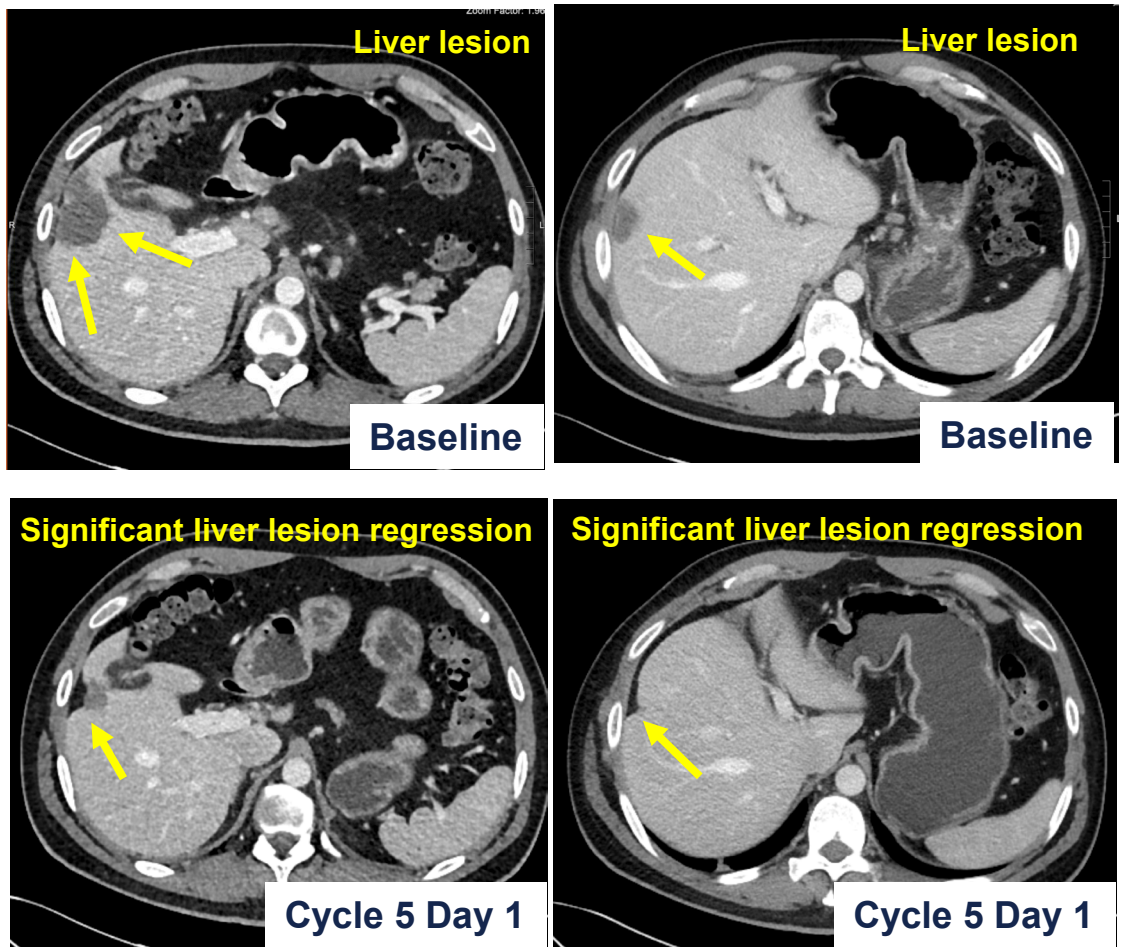
Confirmed Partial Response in Patient with BRAF Class II Lung Cancer Highlights Impressive Activity in Our Core Population

Lung Cancer
BRAF Class II Fusion
DL6 (400 mg bid) → DL5 (300 mg bid)
Patient received Pembrolizumab, Pemetrexed, Cisplatin, Cabozantinib

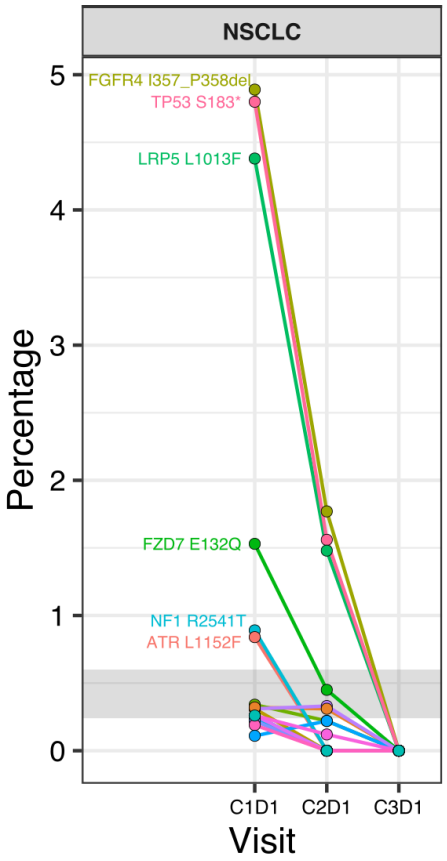
- PR on 1st scan with rapid response (**-34%** on 2 target lesions)
- Drug interruption (rash) & pt resumed exarafenib at DL5 (300 mg bid)
- Confirmed PR on 2nd & 3rd scans (**-54%** reduction of target lesions), significant reductions in non-target lesions
- Complete molecular response by C3D1
- Pt remains on exarafenib for ~ 5 months

Exarafenib had monotherapy activity in a key cancer and molecular subtype (Lung, Class II)

Liver Lesions



ctDNA Analysis



BRAF Fusion not detected at baseline on ctDNA assay

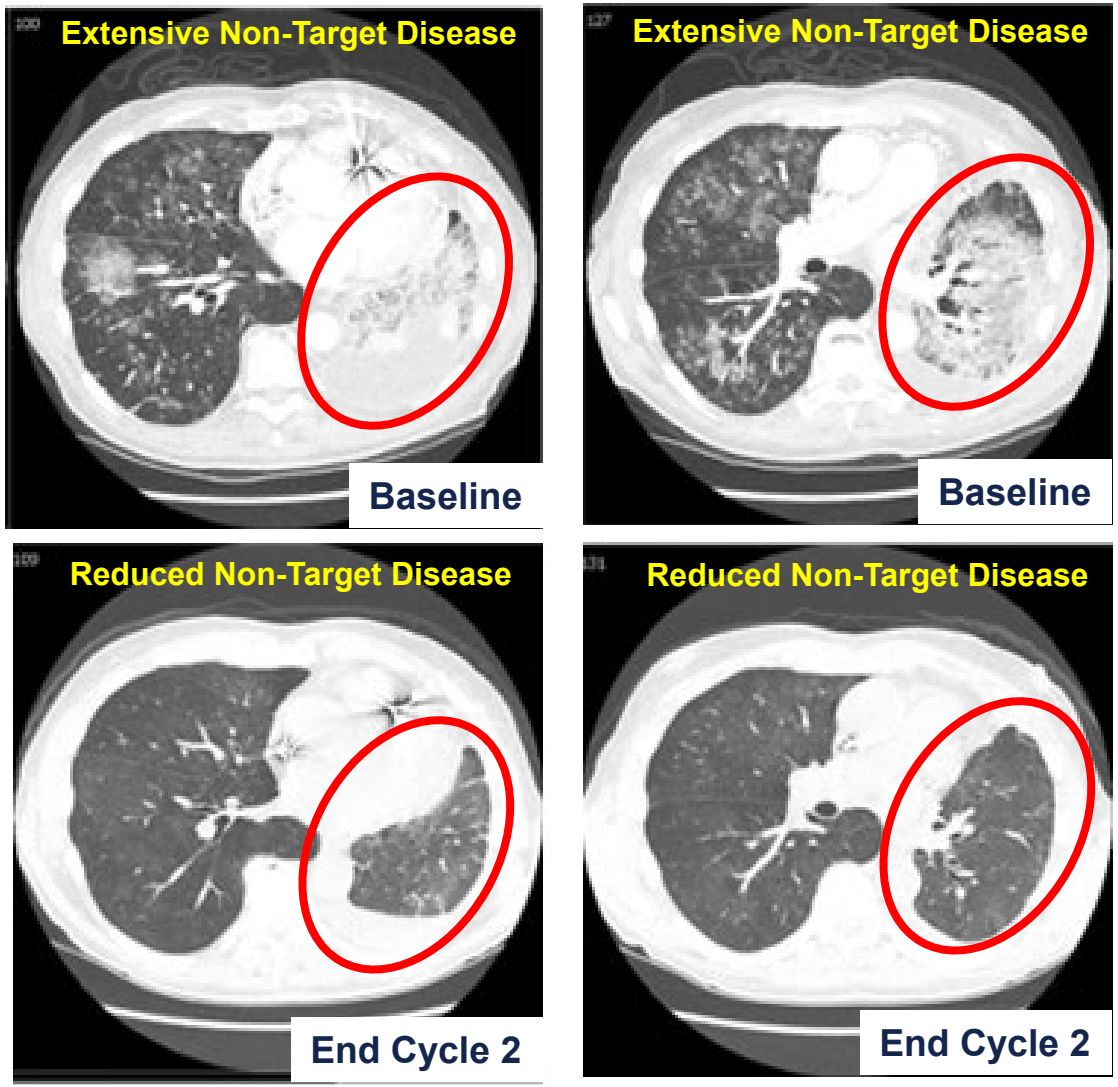
Substantial Clinical Benefit in Patient with BRAF Class II Lung Cancer; Patient Remains on Therapy for 13+ Months

Lung Cancer
BRAF G469S SNV (Class II)
DL4 (200 mg bid)
Patient received Pembrolizumab, Pemetrexed, Carboplatin

- Pt weaned off supp. oxygen in 2 weeks
- Significant reductions of non-target disease
- Prolonged Stable Disease (**-20%** on Target lesions) on 7 successive scans

Exarafenib had substantial & prolonged tumor control in a key cancer and molecular subtype (Lung, Class II)

Pulmonary Disease



Data cut as of Feb 28, 2023

Confirmed Partial Response in Patient with NRAS Co-Occurring with BRAF Class III Colorectal Cancer; Deep Molecular Response

NRAS (G13R) with BRAF Class III SNV (D594G) Colorectal Cancer

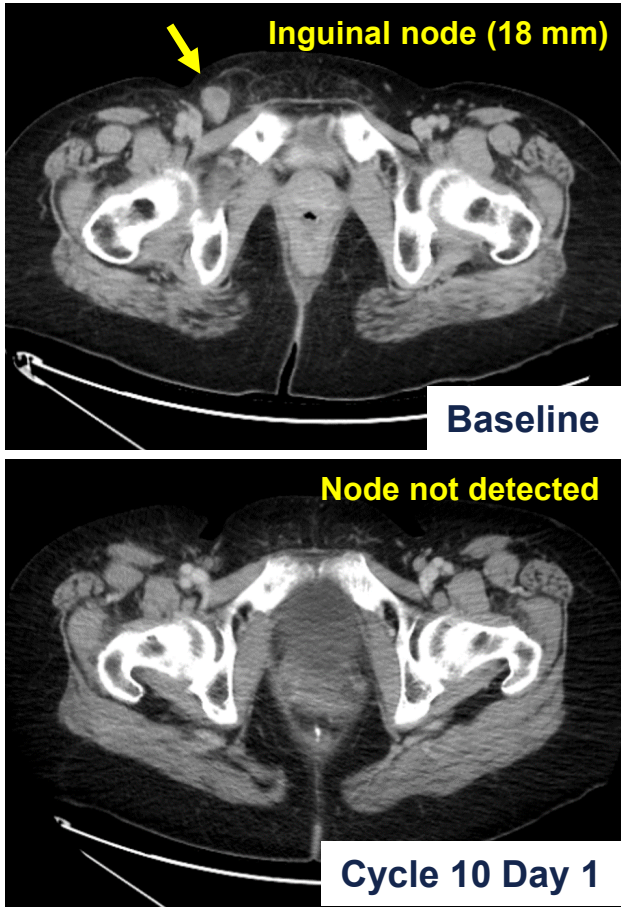
DL5 (300 mg bid)

Refractory to multiple prior lines of Oxaliplatin, Capecitabine, Irinotecan

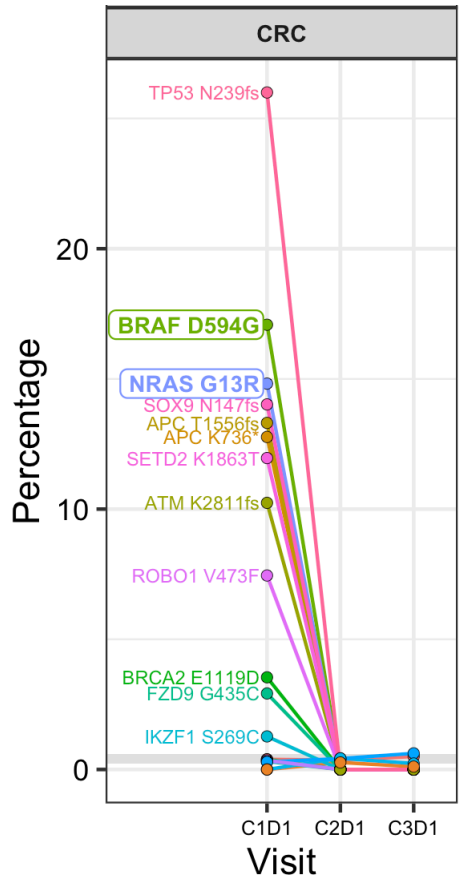
- Rectal bleeding resolved after 2 weeks
- PR on first scan with deep response **(-67%)**
- PR confirmed on 4 more assessments
- CR on target lesion **(-100%)**
- Significant reduction in non-target pulmonary disease
- Complete molecular response by 4 weeks
- Pt remains on exarafenib for 10+ months

Exarafenib had monotherapy activity even in the most challenging patient subtype, CRC

Pelvic Lesion



ctDNA Analysis



Data cut as of Feb 28, 2023

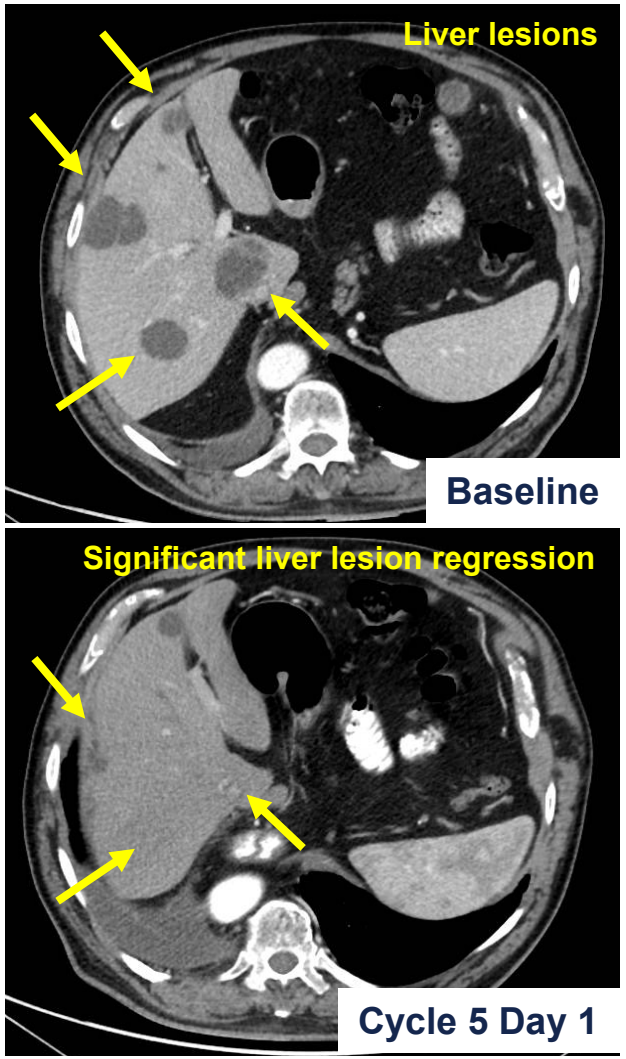
Deep, Confirmed Partial Response in Patient with NRAS Mutant Melanoma

Melanoma
NRAS (Q61L)
DL5 (300 mg bid)
Refractory to Nivolumab, Cisplatin, Doxorubicin

- Extensive abdominal & thoracic disease
- PR on first scan with deep response **(-36%)** and confirmed on 2nd scan **(-54%)**
- Target pulmonary lesion now undetectable
- Pt remains on exarafenib for ~ 5 months

Exarafenib demonstrated monotherapy activity even in a challenging cancer and molecular subtype, NRAS mutant melanoma

Liver Lesions



Data cut as of Feb 28, 2023

Exarafenib + Binimetinib Phase 1 Dosing Schema & Study Design

Enrollment Continues at Exarafenib 200 mg bid + Binimetinib 15 mg bid

Enrolled Cohorts & Schema

Participants receive combination dose twice daily (bid) with continuous dosing on 28-day cycles

Cohort 1
100 mg bid exarafenib + 45 mg bid binimetinib

Cohort 2
100 mg bid exarafenib + 15 mg bid binimetinib

Cohort 3
200 mg bid exarafenib + 15 mg bid binimetinib

- Objectives: Evaluate safety, PK & PD; establish MTD/RP2D; assess preliminary anti-tumor activity
- Population: Adults with advanced, unresectable or metastatic solid tumors with BRAF alterations, including NRAS mutant melanoma

3+3 Study Design

- DLT assessment period is 28 days (thru end of Cycle 1)
- Tumor assessments (per RECIST v1.1) occur every 2 cycles (8 weeks)
- Protocol permits (1) 'Backfill' enrollment of additional participants at the highest previously cleared dose level and (2) Intra-patient dose escalation up to previously cleared dose levels

Early, Compelling Findings with Combination Supports Strategy

2 of 7 Efficacy Evaluable Patients Achieved RECIST PRs

- 12 treated patients, primarily with NRAS mutant melanoma
- 7 patients remain on combination therapy, including all responders
- Safety evaluation ongoing

NRAS Mutant Melanoma

1uPR in;
41% Reduction

NRAS Mutant Melanoma

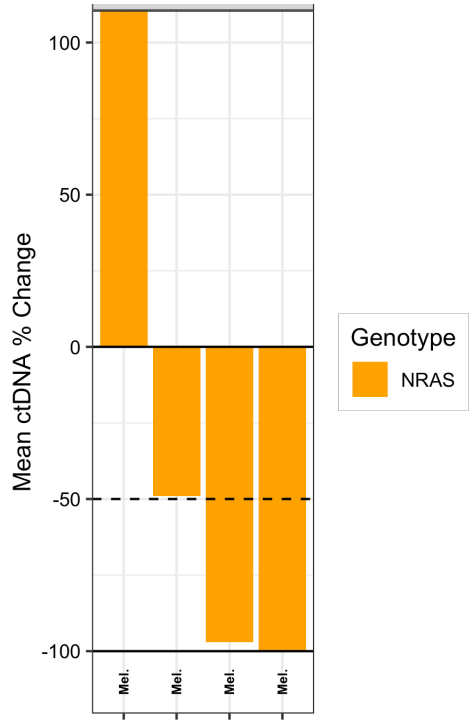
25% reduction in target lesions

BRAF Class II Fusion

1cPR
41% Reduction

CA 19.9 dropped from 540 to <20 (normal) in 8 weeks

ctDNA: Positive Molecular Responses



- Priority development of combination in patients with NRAS mutant melanoma
- RAFi pre-treated patients with BRAF Class I-driven cancers also expected to be enrolled

Expansion Dose Selection Expected in H2 2023

Data as of March 31, 2023

Data-Informed Strategy Optimized for Probability of Success

Monotherapy Dose Expansion Ongoing

Cohort 1: NSCLC
BRAF Class II

Cohort 2: Melanoma
BRAF Class II

Cohort 3: Other Solid Tumors
BRAF Class II

Class II Lung and Melanoma are Priority Cohorts (enroll ~55 patients)

Cohort 4: BRAF Class III Melanoma

Cohort 5: BRAF Class III NSCLC

Cohort 6: BRAF Class III Other Solid Tumors

Recruitment Deemphasized, Pending Enrichment Strategy

Cohort 7: BRAF Class I (RAFi naive) Solid Tumors

Long-term Opportunity (enroll ~15 patients)

Exarafenib + Binimetinib Combination Dose Escalation Ongoing

NRAS Mutant Melanoma

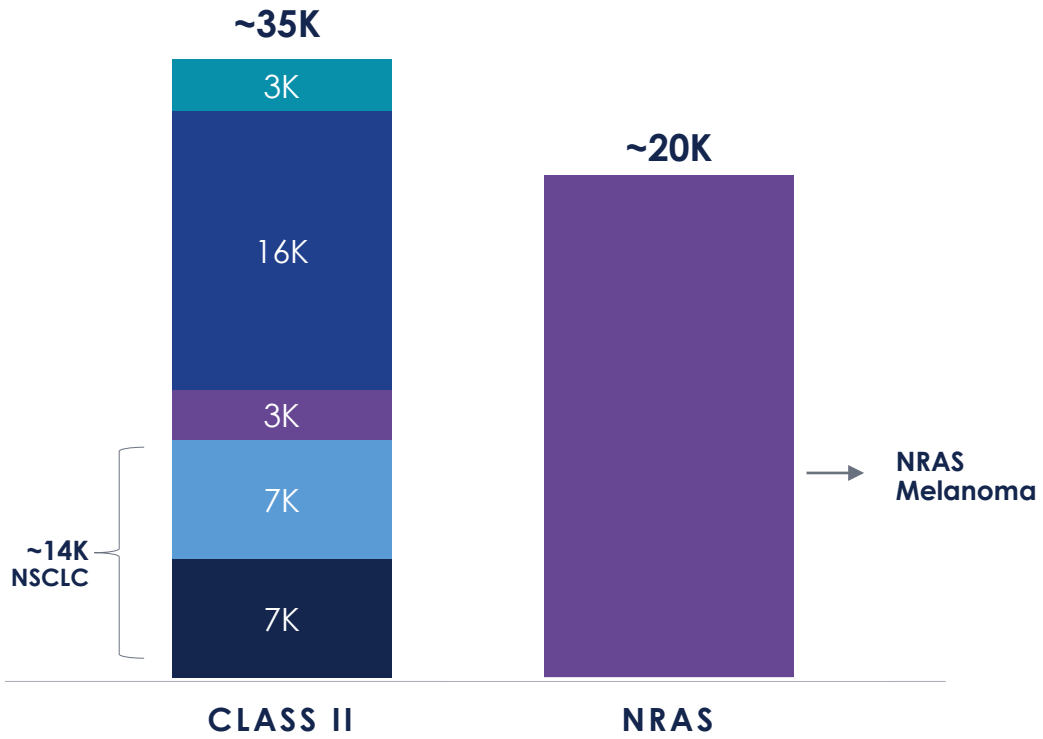
Class I Pre-Treated

Strategy to be Refined, May Require Enrichment

~55K Patients Potentially Addressable with Class II & NRAS Melanoma

Potential for Additional Opportunities in Other Patient Subtypes

Patients with No Approved Targeted Therapy Available Today



Additional Opportunities

Class III: ~47K patients	→	Requires enrichment, monotherapy or in combination with other RTK inhibitors
$2.5bn[1] Class I naïve market	→	Monotherapy, tumor agnostic priority
$1bn Class 1 resistance	→	Combination, *~40 – 60%*[2,3] patients eligible
NRAS-driven cancers: ~28K patients (excludes melanoma, CRC)	→	Future combination

Notes: Kinnate calculations of prevalence; reflects approximate prevalence in U.S., EU4, UK, Japan (unless otherwise noted). Class I resistance includes BRAF KD Duplication and BRAF Splice Variants (dimer-based). Class III includes undefined oncogenes. NRAS mutations includes Q61, G12, G13. Assumes unresectable or advanced metastatic. "Other" tumor types with BRAF Class II alterations and "NRAS-driven cancers" include Anaplastic Thyroid, Bladder, Breast, Cholangiocarcinoma, Endometrial, Esophagogastric, Other Thyroid, Ovarian, Pancreatic, Prostate. (1) 2022 sales of approved MEK/RAF products (Dabrafenib, Vemurafenib, Encorafenib, Trametinib, Cobimetinib, Binimetinib) based on company financial reports except Vemurafenib based on Wall Street consensus estimates. (2) D.B. Johnson, et al., Acquired BRAF inhibitor resistance, Eur. J. Cancer 51 (18) (2015) 2792-2799. (3) K. Kemper, et al., Phenotype switching: tumor cell plasticity as a resistance mechanism and target for therapy, Cancer Res. 74 (21) (2014) 5937-5941.

FGFR2/3 Program

KIN-3248

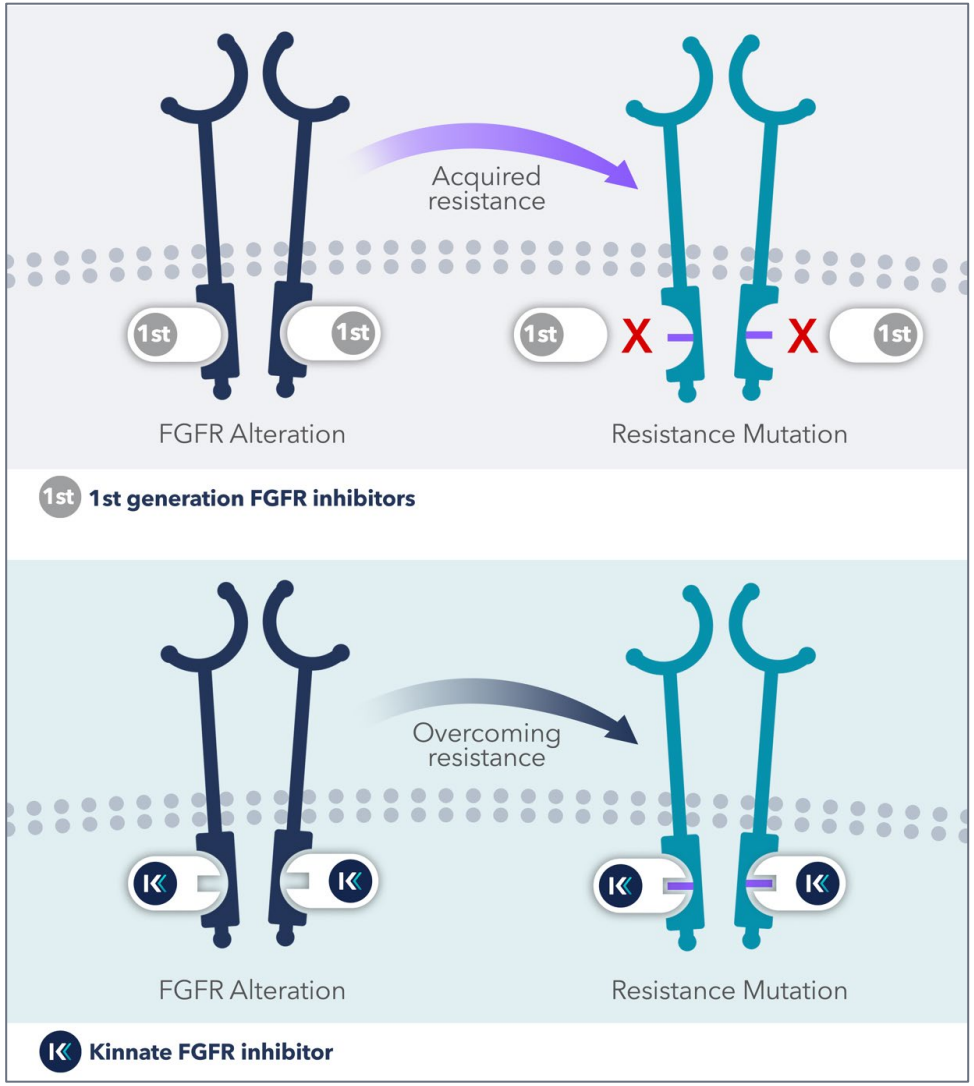
Kinnate FGFR 2/3 Inhibitor Program

KIN-3248 is a Potentially Potent, Highly Selective, Covalent FGFR Inhibitor

Acquired resistance limits clinical benefit of approved and in-development FGFR inhibitors

KIN-3248 Designed to Target

- FGFR2 & FGFR3 driver alterations in ICC & UC, and other tumor types
- Known & predicted 'on-target' FGFR2 & FGFR3 kinase domain mutations that confer clinical resistance (e.g., gatekeeper & molecular brake)
- FGFR1, R2 & R3 isoforms, thereby reducing opportunities for bypass resistance



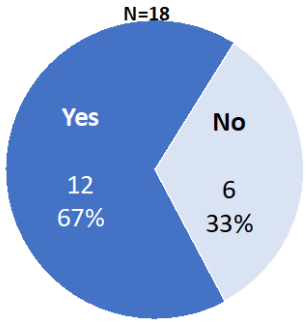
FGFR, fibroblast growth factor receptor; UC, urothelial carcinoma; ICC, intrahepatic cholangiocarcinoma

Meaningful On-Target Acquired Resistance to FGFR Inhibitors

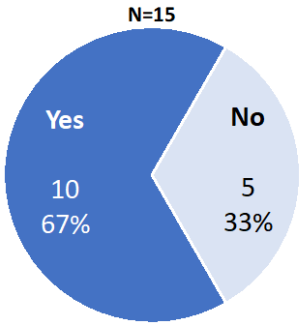
67% of FGFRi-Treated ICC Patients Developed FGFR2 Kinase Domain Mutations at Progression

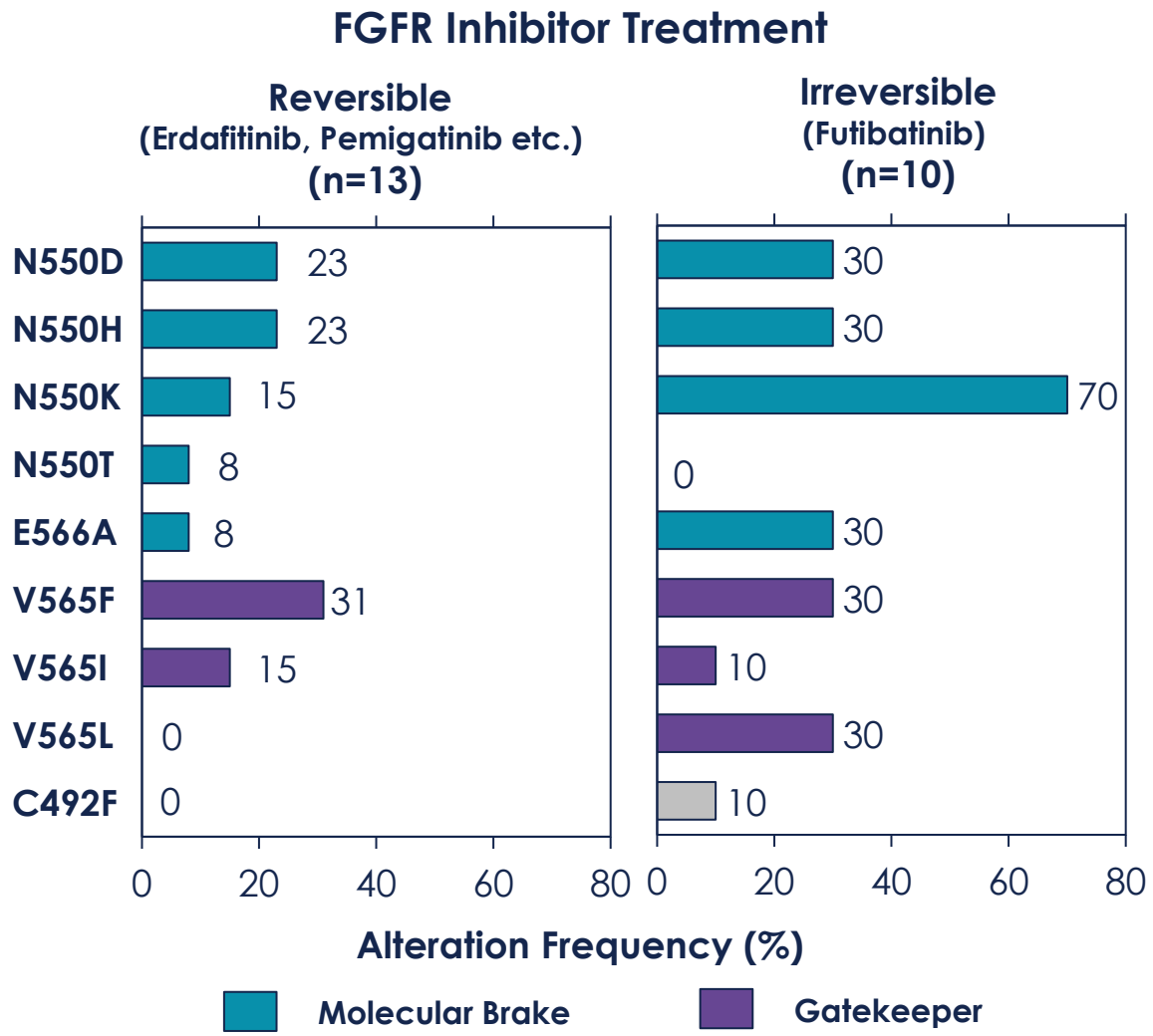
FGFR2 Kinase Domain Mutation

Reversible FGFRi (Erdafitinib, Pemigatinib, etc)



Irreversible FGFRi (Futibatinib)

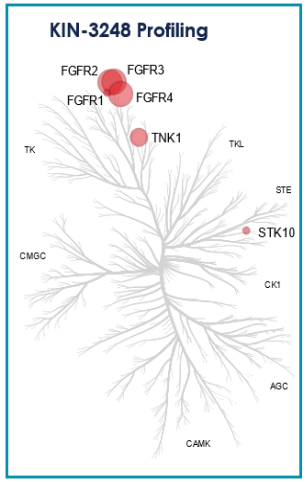


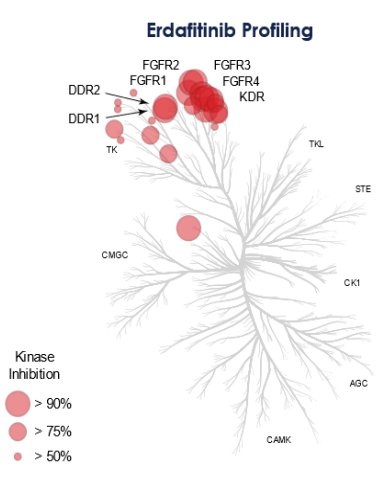


Adapted from: Goyal et al., Landscape of Acquired Resistance to Selective FGFR Inhibitors in FGFR2 Fusion or Rearrangement+ Cholangiocarcinoma. EORTC-NCI-AACR Symposium (Oct 2020). Analysis includes Reversible FGFR inhibitor treated patients (n=13) and Irreversible FGFR inhibitor treated patients (n=10; all patients received futibatinib)

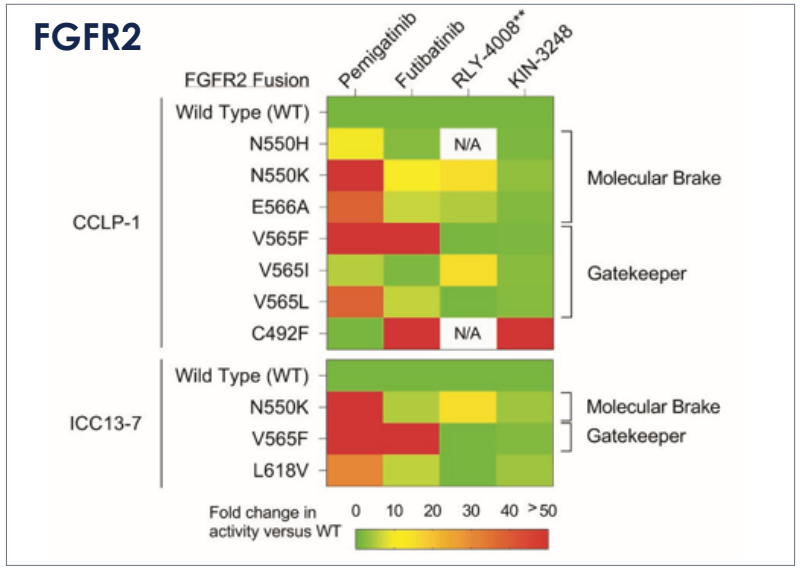
Preclinical Evidence Demonstrates KIN-3248 Directly Targets FGFR2/3 Driver Alterations and Acquired Resistance Mechanisms

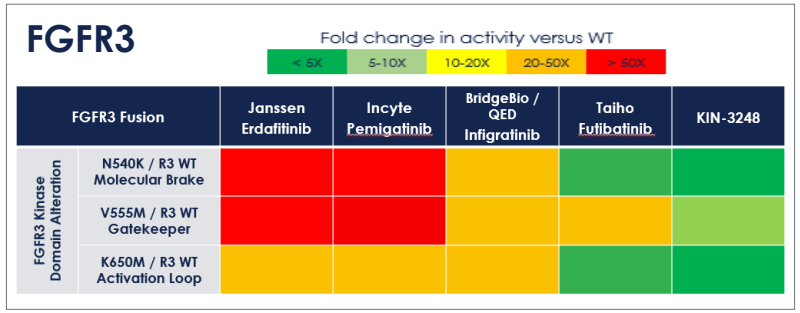
KIN-3248 Displays A Selective & Differentiated Kinase Profile



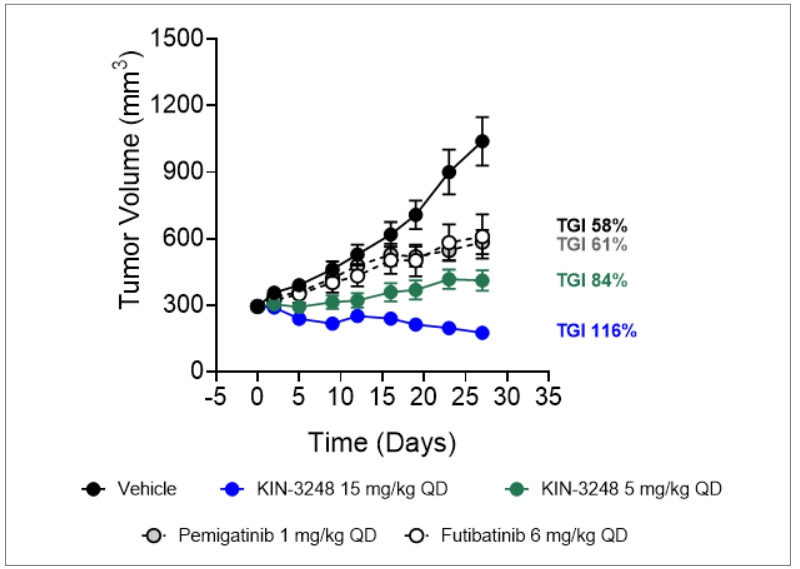


KIN-3248 Inhibits Growth of FGFR2/3 Fusion+ Cells Harboring Secondary Resistance Mutations[1]





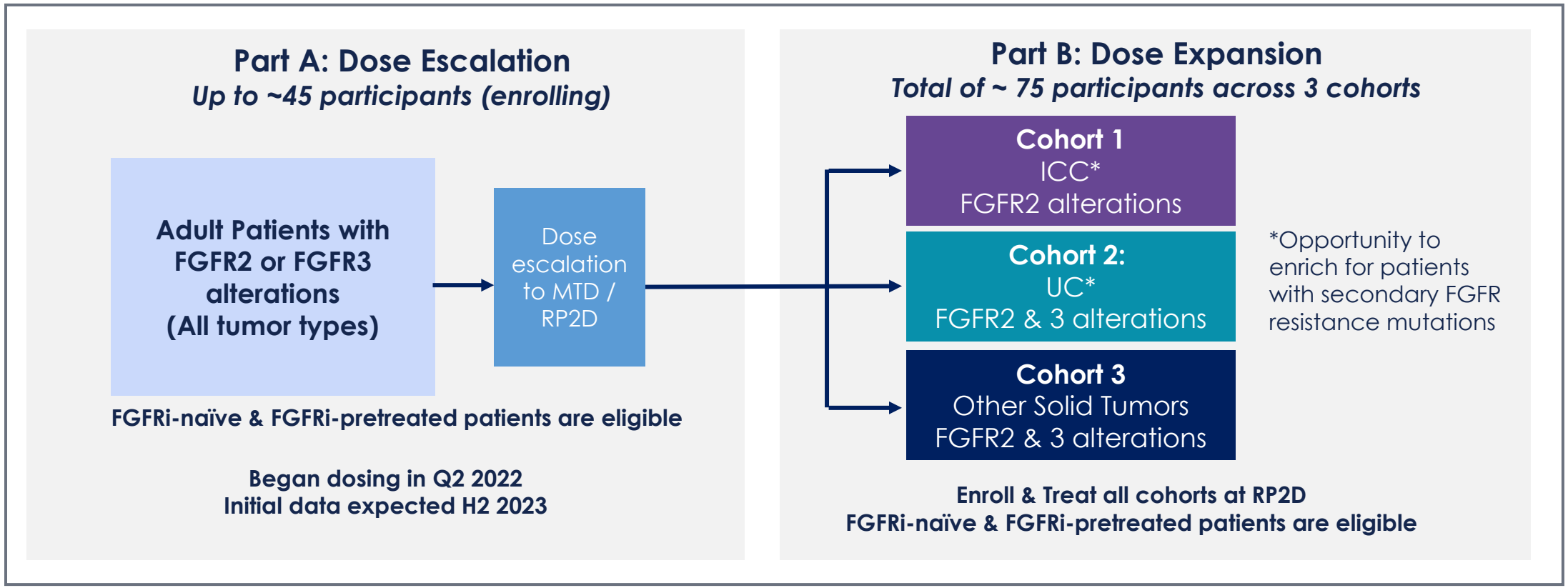
KIN-3248 Demonstrated Tumor Reductions Against Secondary, Acquired FGFR2 Gatekeeper Resistance Mutation *In Vivo*[2]



(1) Analysis includes Kinnate-generated data for clonal competition & cellular sensitivity experiments, apart from data for RLY-4008's profile that was abstracted from Relay's S1 public SEC filing (https://www.sec.gov/Archives/edgar/data/0001812364/000119312520192936/d904779ds1a.htm#rom904779_12). (2) Tumor growth inhibition (TGI) was calculated as follows: TGI = (1 – (TVf-TVi)treated / (TVf-TVi)control)) x 100%, where TVf is the final tumor volume and TVi is the initial tumor volume.

KIN-3248 Development Plan: Ongoing Phase 1 Trial

Initial Dose Escalation Data Expected H2 2023

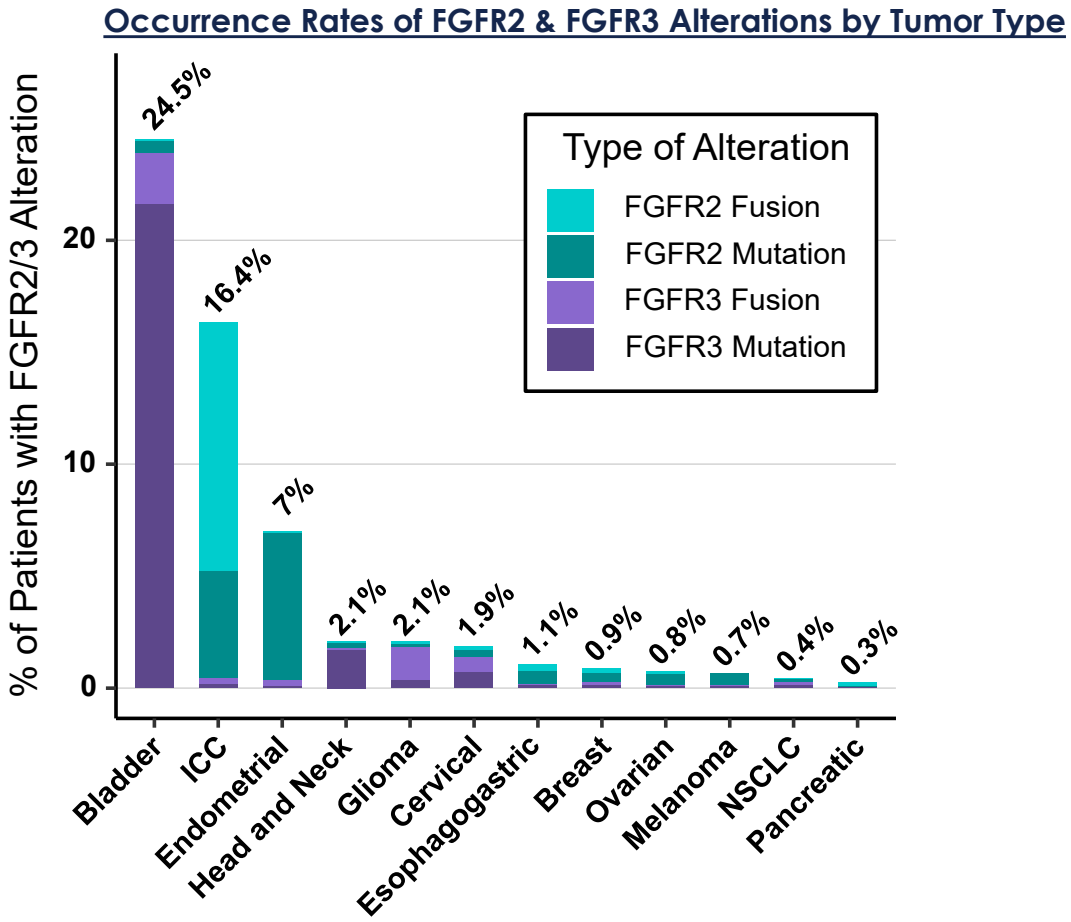


- Phase 1 trial objectives: Evaluate Safety, PK & PD; Establish MTD/RP2D; Assess preliminary anti-tumor activity (NCT05242822)
- Population: Adults with advanced or metastatic solid tumors
- FGFR2 & FGFR3 gene alterations previously detected in tissue-based or blood-based genomic testing

FGFR, fibroblast growth factor receptor; MTD, maximum tolerated dose; RP2D, recommended phase 2 dose; PK, pharmacokinetics; PD, pharmacodynamics: UC, urothelial carcinoma; ICC, intrahepatic cholangiocarcinoma

FGFR Inhibitor Market Opportunity

Global Sales of FGFR Inhibitors are Expected to Reach >$2B by 2028[1]



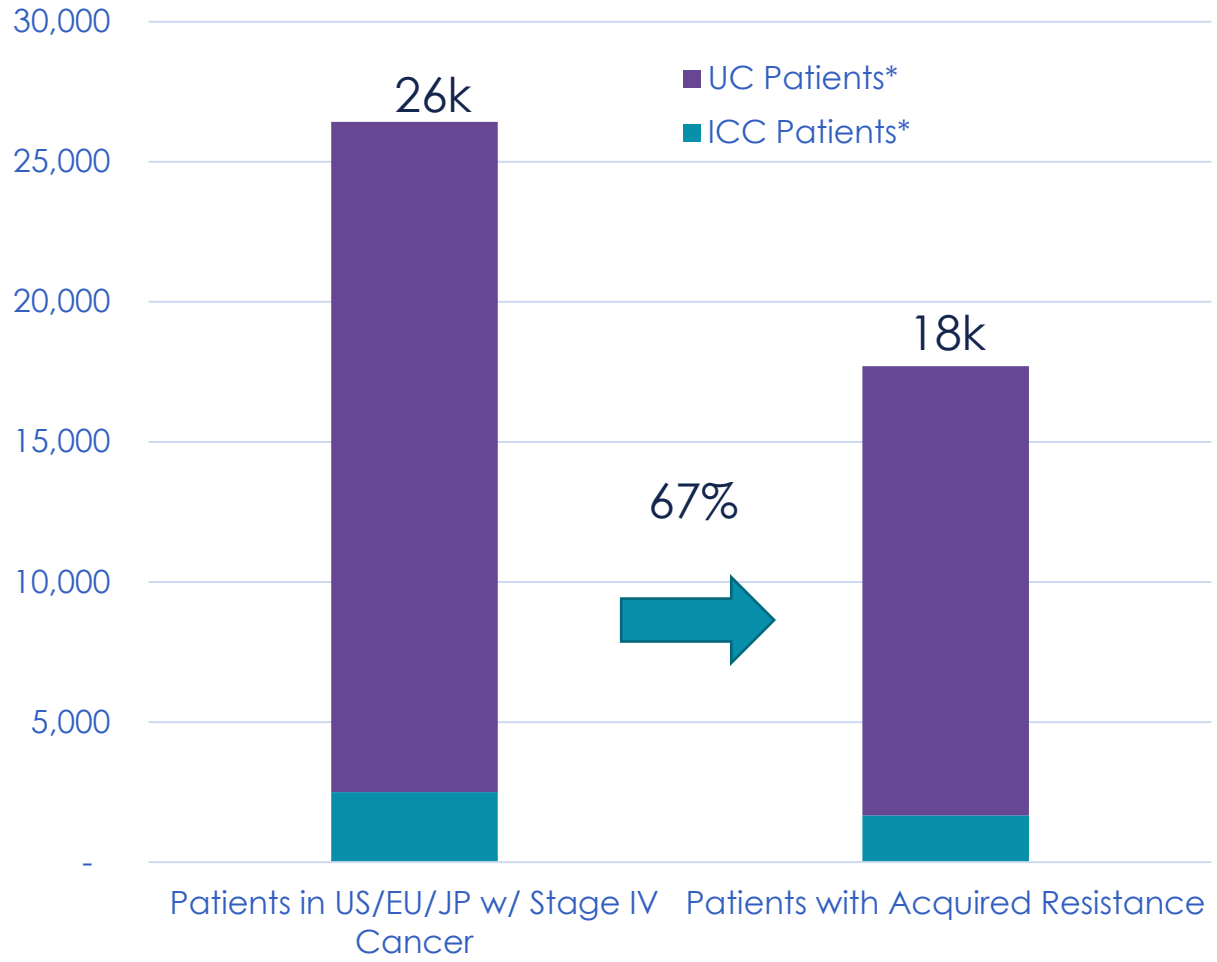
KIN-3248 is designed to target FGFR2/3 alterations

- FGFR2/3 alterations include fusions, mutations (indels and SNVs) and other rearrangements, which are likely oncogenic drivers of tumor
- FGFR2/3 amplifications are often not the primary drivers of solid tumors
- FGFR alterations are most common in UC and ICC, our primary focus
- FGFR alternations also found in other tumor types e.g., endometrial, breast, etc.

Data generated from AACR GENIE Project Data: Version 10.0-public Powering Precision Medicine Through An International Consortium. Cancer Discov 7(8): 818-831, 2017 (https://genie.cbioportal.org/). Analysis includes mutations that are annotated as at least *Likely Oncogenic* in oncokb.org and rearrangements including fusions, intergenic and intragenic events. Unknown frame fusions were included, but out-of-frame fusions were not included. Source: Evaluate Pharma; includes analyst estimates of approved and development candidates, including erdafitinib, pemigatinib, futibatinib and RLY-4008. UC, urothelial carcinoma; ICC, intrahepatic cholangiocarcinoma

FGFR Inhibitor Market Opportunity – UC & ICC Patients

US, EU and Japan Patients with Active Disease



*Reflects FGFR2 or FGFR3 Alterations

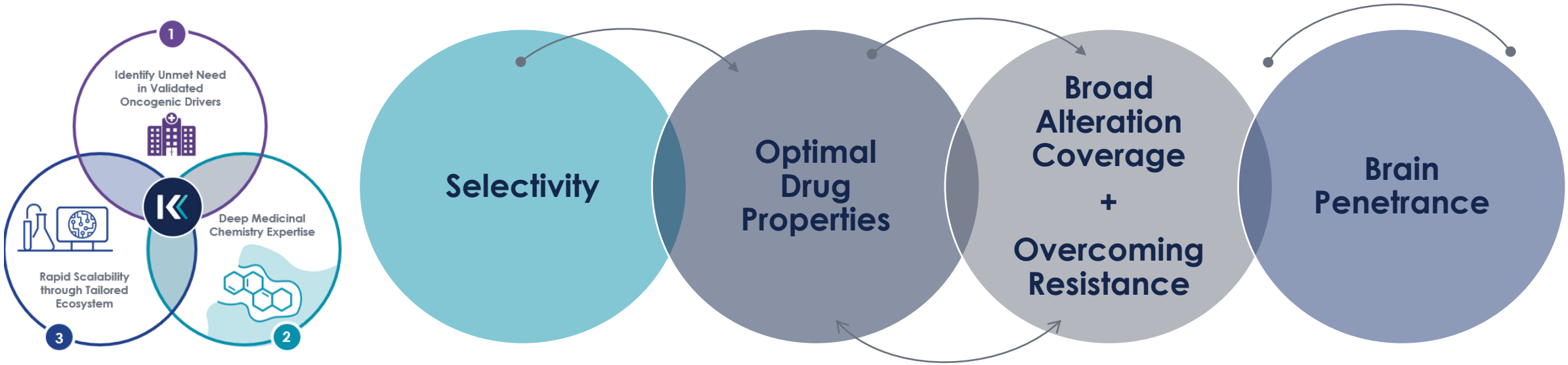
Growth Opportunities

- FGFR alterations have been found in other tumor types (e.g., breast)
- FGFR amplifications
- NGS technologies identify additional patients with FGFR alterations
- Geographic expansion (e.g., China)

Kinnate calculations based on Kantar data (stage IV metastatic UC) and data generated from AACR GENIE Project Data: Version 10.0-public Powering Precision Medicine Through An International Consortium. Cancer Discov 7(8): 818-831, 2017 (https://genie.cbioportal.org/); Adapted from: Goyal et al., Landscape of Acquired Resistance to Selective FGFR Inhibitors in FGFR2 Fusion or Rearrangement+ Cholangiocarcinoma. EORTC-NCI-AACR Symposium (October 2020).

Kinnate Discovery Engine

Deep Expertise in Chemistry and Structure-based Drug Design Drives Pipeline of Highly Selective Compounds



MEK Inhibitor: KIN-7136

KIN-7136: Potential Best-in-Class Brain Penetrant MEK Inhibitor

Expected to Enter the Clinic in H2 2023

2022 sales of the 3 approved MEK products were ~$1.1bn[1]

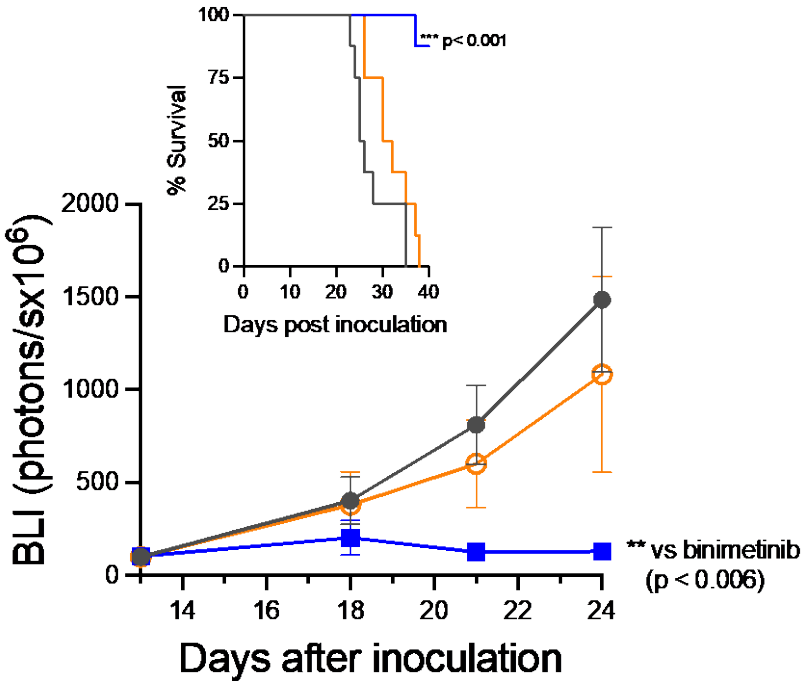
- ✓ **Brain penetrant**
- ✓ **Highly selective, dual mechanism**
- ✓ **Quality drug-like properties**
- ✓ **Potentially paradigm-breaking simplicity of structure**

(1) MEK inhibitors: Mekinist, Cotellic, Mektovi . Source: Company financial reports unless otherwise noted; Mekinist sales assumed to be 50% of Tafinlar + Mekinist sales (reported jointly). Mektovi sales only include U.S. and Japan.

KIN-7136 Inhibits Intracranial Tumor Growth

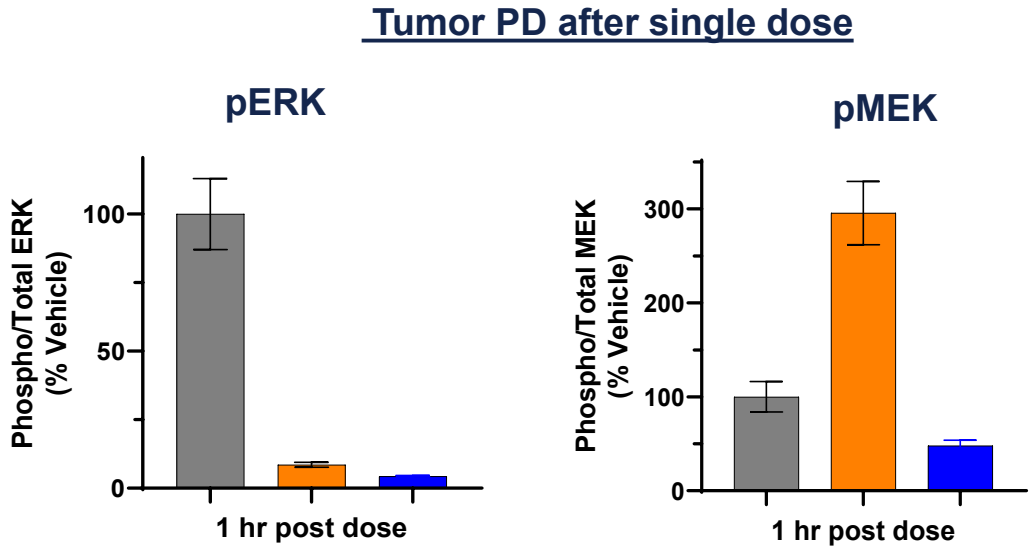
Dual Function Inhibits MEK Activity (pERK) and Suppresses Activation (pMEK) in BRAF Class I Melanoma

$BRAF^{V600E}$ Intracranial Tumor



KIN-7136 Brain Penetration
Rat $K_{p,uu}$ = 0.5 to 0.9

$BRAF^{V600E}$ Subcutaneous Tumor PD



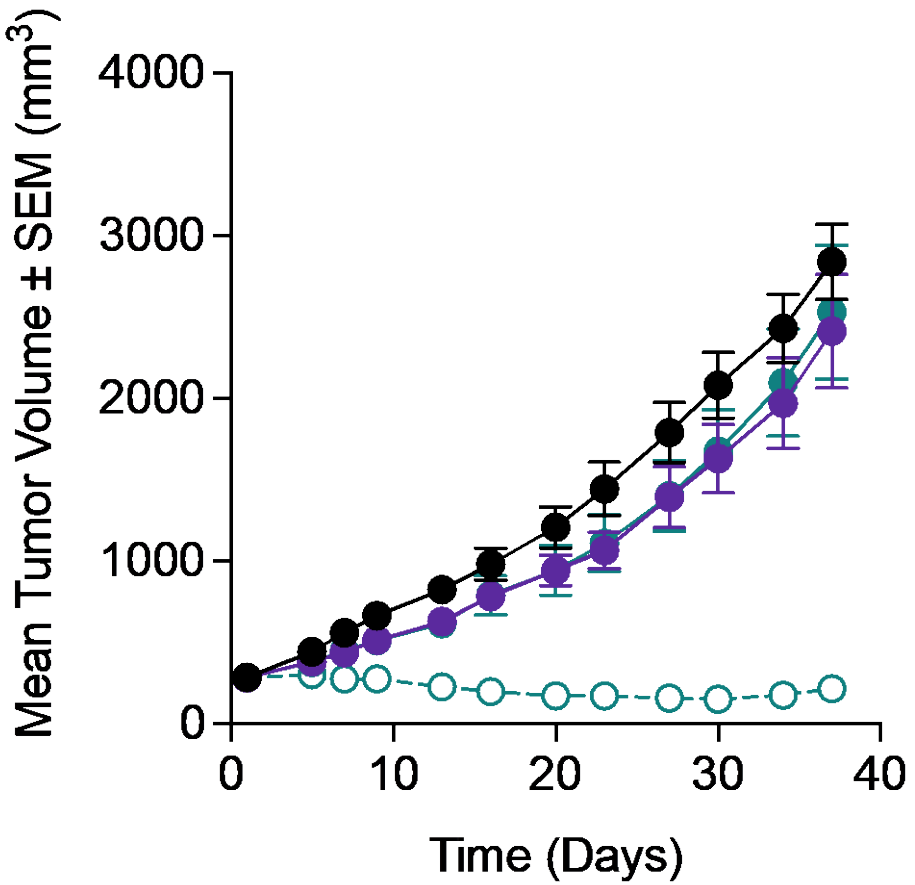
KIN-7136 suppresses upstream priming (pMEK) of MAPK pathway vs binimetinib

- Vehicle
- KIN-7136 10 mg/kg PO BID
- Binimetinib 3 mg/kg PO BID

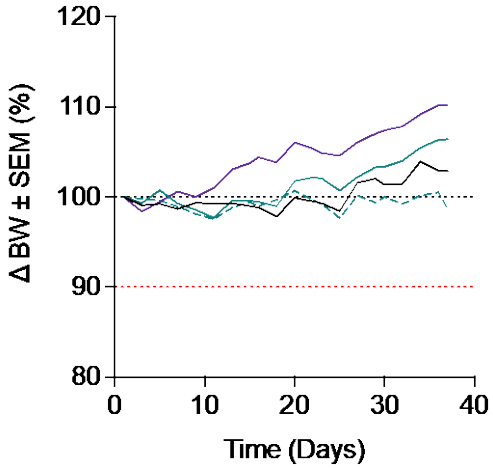
Study doses are exposure-matched to clinical exposure of binimetinib

KIN-7136 Provides Combination Benefit with Exarafenib Preclinically

Activity in NRAS^{Q61K} melanoma



Change in Body Weight



Well-tolerated in all treatment groups

- Vehicle
- KIN-7136 (10 mg/kg BID)
- Exarafenib (10 mg/kg BID)
- KIN-7136 (10 mg/kg BID) + Exarafenib (10 mg/kg BID)

MEK Combination Development Strategy Built for the Long-Term

Combination Development with Binimetinib

KN-8701: Exarafenib + Binimetinib Dose Escalation (ongoing) → **Exarafenib + Binimetinib Dose Expansion**

Combination Development with KIN-7136

Exarafenib + KIN-7136 Dose Escalation

If positive PoC with binimetinib, KIN-7136 may be effective as a long-term combination strategy

- 2 parallel cohorts – KIN-7136 monotherapy and combination with exarafenib
- Per FDA feedback, planned initiation of combination dose escalation after one cohort in monotherapy cleared
- Broad patient population to rapidly achieve MTD/RP2D – including BRAF, NRAS, KRAS and NF1 alterations

c-MET Inhibitor: KIN-8741

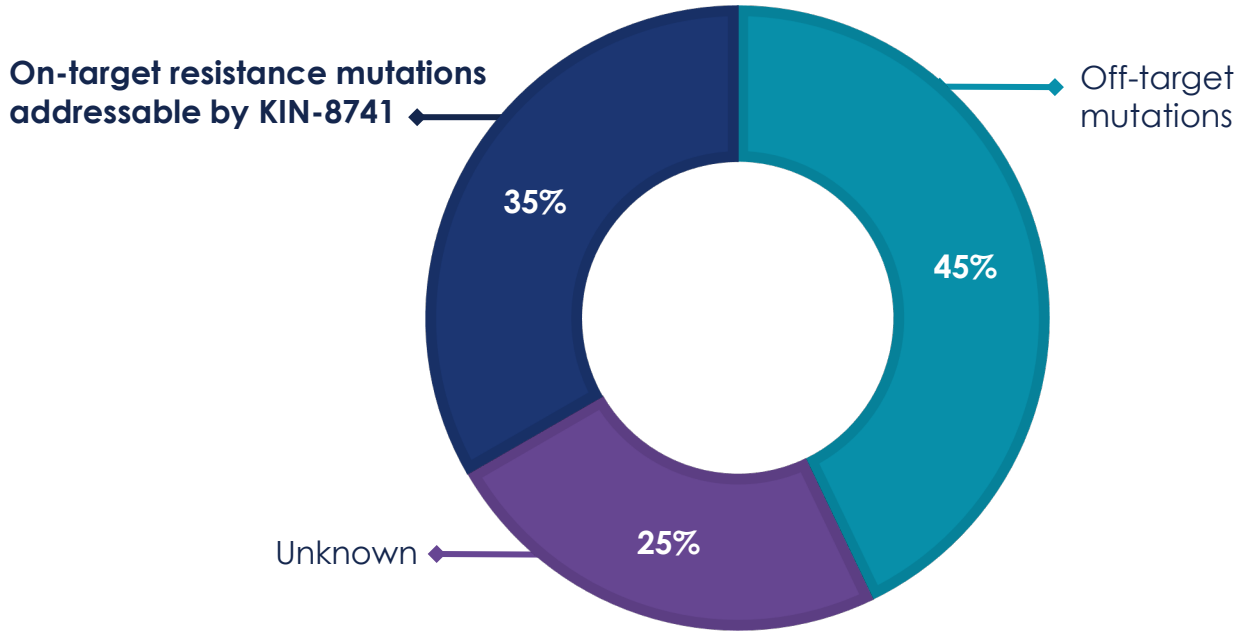
KIN-8741 Targets c-MET and Acquired Resistance Mechanisms

c-MET ~$2B Potential Market[1] Represents Significant Commercial Opportunity for KIN-8741

KIN-8741 is designed to be a best-in-class c-METi for NSCLC with MET Δ exon 14 alterations and secondary resistance mutations

- 3-4% of NSCLC patients present with actionable MET Δ exon 14 alterations
- 35% develop on target resistance mutations with approved Type 1 MET inhibitors

Distribution of Mechanisms of Resistance to c-MET Inhibitors[2]



Acquired resistance limits clinical benefit of approved and in-development c-MET inhibitors

(1) Based on Global Data Wall Street consensus estimates for Tabrecta, Orpathys, Tepmetko, telisotuzumab vedotin, and elzovantinib for 2028E; (2) Recondo et al. Molecular Mechanisms of Acquired Resistance to MET Tyrosine Kinase Inhibitors in Patients with MET Exon 14–Mutant NSCLC. Clin Cancer Res 2020; 26:2615–25.

KIN-8741 Has Potential Best-in-Class Profile and Properties

Has Unique Binding Mode Unlike Prior Type I/II Inhibitors; Expected to Enter the Clinic in H1 2024

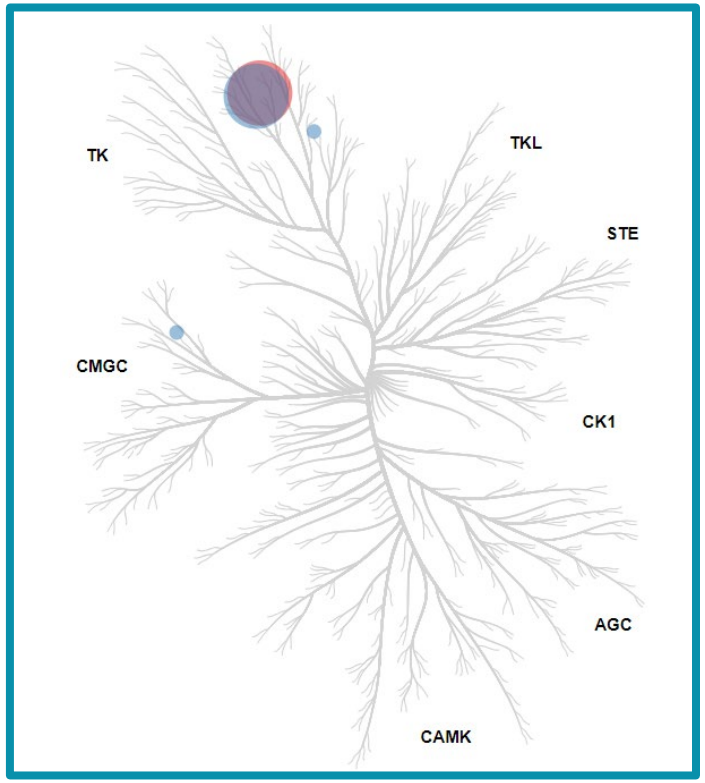
	KIN-8741	Type I approved inhibitors[1]	Type II inhibitors[2]
Highly selective	✓	✓	X
Covers acquired resistance mutations	✓	X	✓
Quality drug-like properties	✓	✓	X

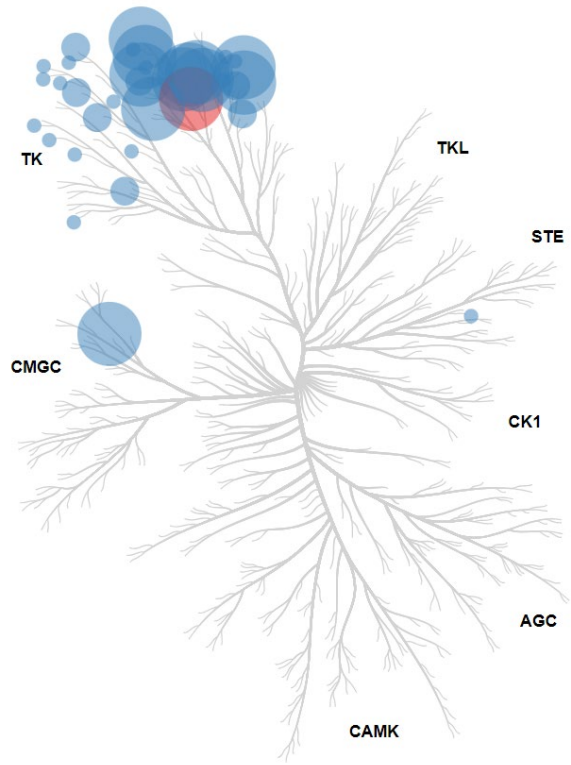
(1) Type I: Capmatinib, Savolitinib & Tepotinib
(2) Type II: Cabozantinib, Merestinib & Glesatinib

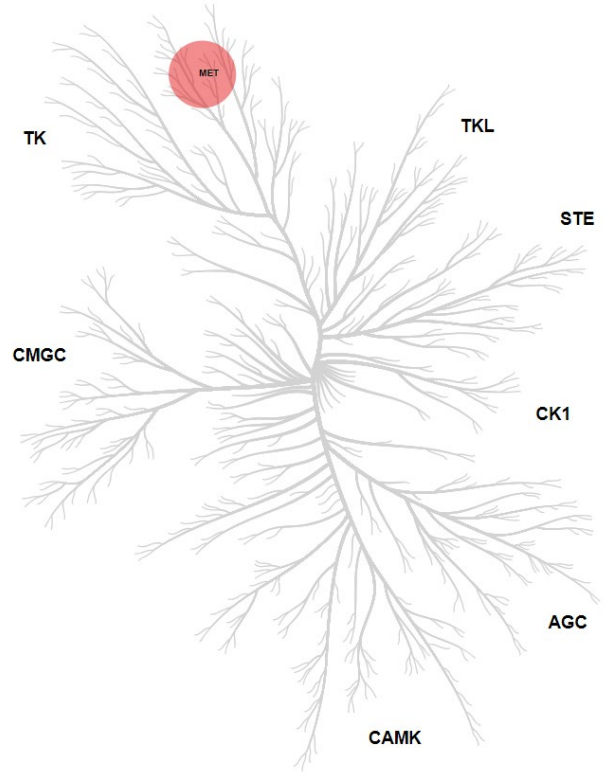
KIN-8741 Has Been Shown to be Highly Selective

Node Color
- Target
- Off-Target

Node Size
- 51-75
- 76-90
- 90-100







Kinase Inhibition	KIN-8741	Cabozantinib (Type II)	Capmatinib (Type I)
Non-MET # inhibited > 90%	1 (RON)	6	0
Non-MET # inhibited > 75%	1 (RON)	12	0

- Kinome wide enzyme inhibition profiling at 100 nM across > 695 kinases at Reaction Bio (including wild type, atypical, mutant)
- Only wild type kinases pictured in kinome trees

KIN-8741 Demonstrated Broad Mutation Coverage *In Vitro*

Inhibition of kinase activity (number of inhibited kinases at 100nM)

Biochemical Inhibition	KIN-8741	Cabozantinib (Type II)	Capmatinib (Type I)
#MET and MET mutants > 90%	26/28	4/28	2/28

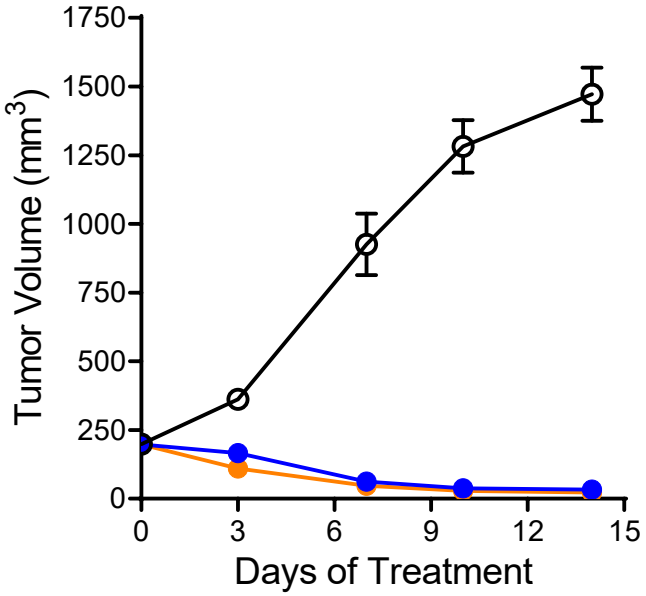
Cellular viability profiling in engineered TPR-MET Ba/F3 cells

Type	Compound	WT	D1228X/Y1230X					Other KD mutations		
			D1228N	D1228H	Y1230H	Y1230C	Y1230S	H1094Y	L1195V	F1200I
I	Capmatinib	2.9	10000	10000	10000	10000	10000	0.3	35	26.4
	Tepotinib	11.1	3290	3040	3410	2210	2810	0.6	123	122
II	Cabozantinib	53.3	190	368	44.9	39.4	35.6	53.2	1030	4480
	Merestinib	22.4	138	129	25.9	23.2	18	7.8	203	837
KIN-8741		**14.8**	**5.1**	**3.3**	**9.5**	**12.7**	**9**	**7.3**	**52.6**	**20.1**

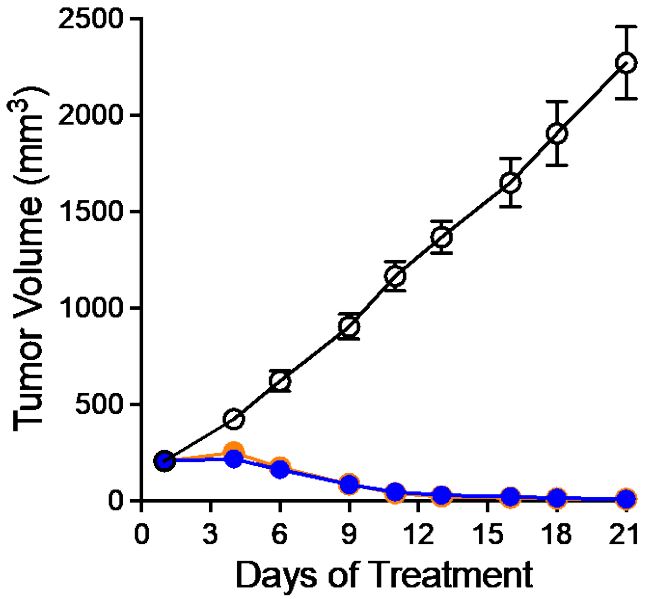
EC_{50} (nM)	<30	<100	<300	<1000	>1000

Antitumor Activity Achieved Across c-MET Mutation Models

Activating Alterations in NSCLC

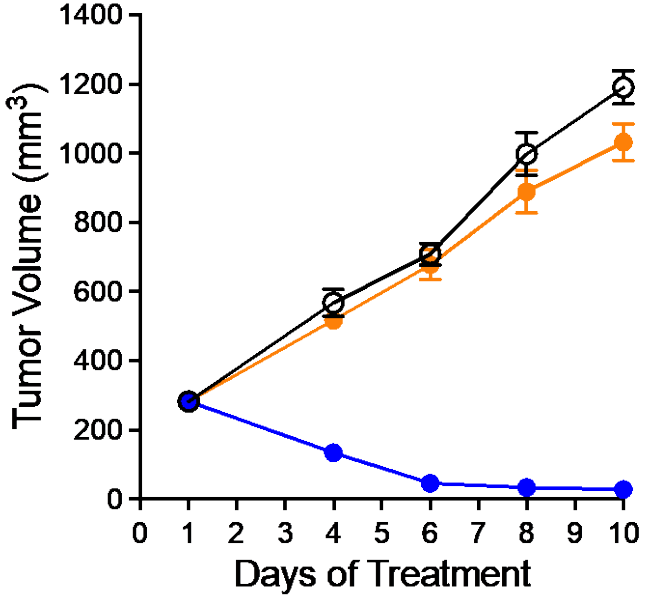


c-MET$^{\Delta ex14}$



c-MET Amp

Acquired Resistance to 1st Gen



c-MET-D1228N

Safety Profile

- No body weight changes in efficacy studies across doses or models
- Dose Range Finding Studies In-life Observations (2 weeks)
  - Rat: Tolerated up to 300 mg/kg (>30-fold the AUC that results in TGI_{80})
  - Dog: Tolerated up to 75 mg/kg (>30-fold the AUC that results in TGI_{80})

Veh
KIN-8741 60mg/kg BID
Capmatinib 10mg/kg BID*

*Dose exposure matched to approved human dose

Key Company Facts

Offices: San Francisco and San Diego, California

Founded: 2018

NASDAQ listing: KNTE

Financials: $231.2 million of cash*

Employees: 94

Pronunciation: KY-nate / kaɪˈneɪt /

Our mission is to inspire hope for those battling cancer by expanding on the promise of targeted therapies.

**Cash, cash equivalents and investments as of March 31, 2023
**Full-time employees as of March 31, 2023