KINNATE BIOPHARMA

A Clinical-Stage Precision Oncology Company

AACR 2023

Investor Presentation
April 17, 2023

Forward-Looking Statements

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.

These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, among other things: our ability to successfully complete our ongoing clinical trials and for those trials to produce positive results, our estimates of the number of patients who suffer from the diseases we are targeting and the number of patients that may enroll in our clinical trials; our ability to file INDs in the future; the commercializing of our product candidates, if approved; the timing of the initiation, progress and potential results of our ongoing and planned preclinical studies and clinical trials and our research programs; our ability to advance additional product candidates into, and successfully complete, preclinical studies and clinical trials with those additional product candidates; the timing or likelihood of regulatory filings and approvals; our product development and marketing strategy; the negative impacts of the COVID-19 pandemic; our ability and the potential to successfully manufacture and supply our product candidates for clinical trials and for commercial use, if approved; future strategic arrangements and/or collaborations and the potential benefits of such arrangements; our estimates regarding expenses, future revenue, capital requirements and needs for financing and our ability to obtain capital; the sufficiency of our existing cash and cash equivalents to fund our future operating expenses and capital expenditure requirements; our ability to retain the continued service of our key personnel and to identify, hire and retain additional qualified professionals; the implementation of our business model, strategic plans for our business and product candidates; the scope of protection we are able to establish and maintain for intellectual property rights, product candidates and our pipeline; our ability to contract with third-party suppliers and manufacturers and their ability to perform adequately; the pricing, coverage and reimbursement of our product candidates, if approved; and developments relating to our competitors and our industry, including competing product candidates and therapies, and changes to our preliminary and unaudited financial results upon completion of our financial statement closing procedures

These and other risks, uncertainties, assumptions and other factors are described in greater detail in our filings we have made and will make with the Securities and Exchange Commission, including, without limitation, under the heading "Risk Factors" in our Annual Report on Form 10-K for the fiscal year ended December 31, 2022. You may view our filings with the Securities Exchange Commission 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. You 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. 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 and differences.

Speakers

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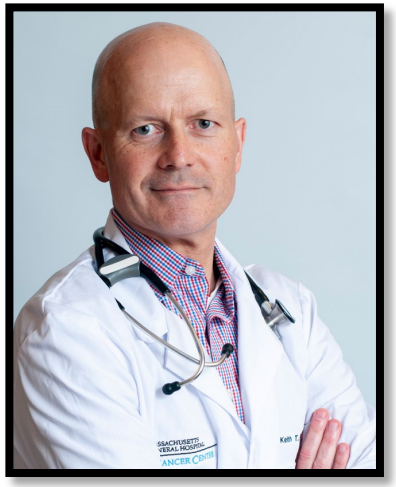

Keith Flaherty, MD
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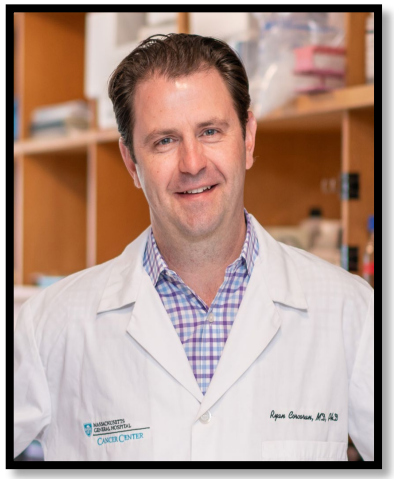

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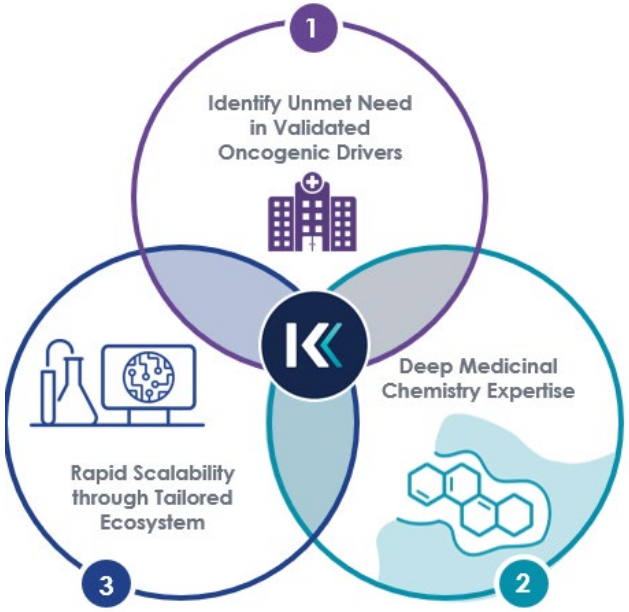
Agenda

- Opening Remarks
- Exarafenib Monotherapy Dose Escalation Data
- Preliminary Exarafenib + Binimetinib Combination Study Update
- Future Direction with RAF Monotherapy and Combination
- Early Pipeline
- Closing Remarks
- Q&A

Opening Remarks

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

DC = drug candidate nomination; IND=investigational new IND; FIH=first-in-human

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

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

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

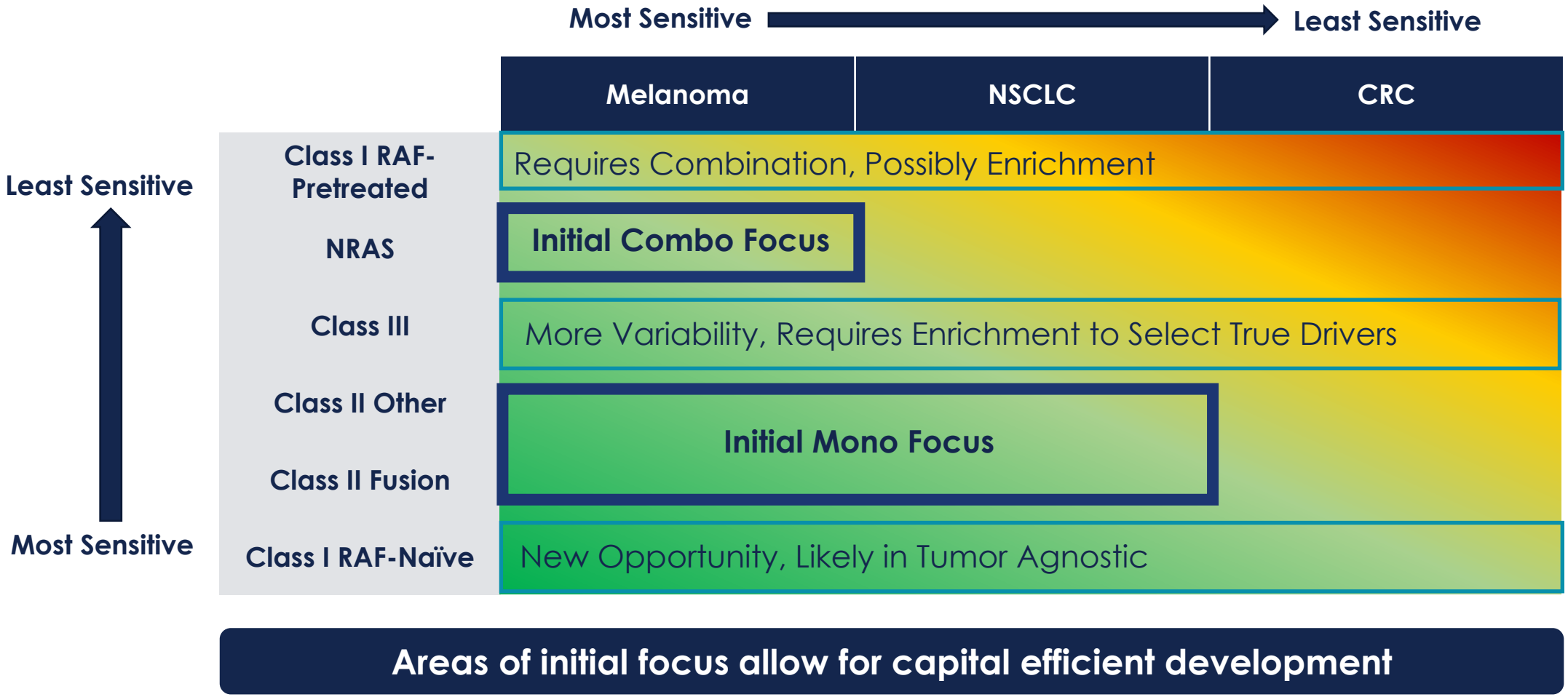
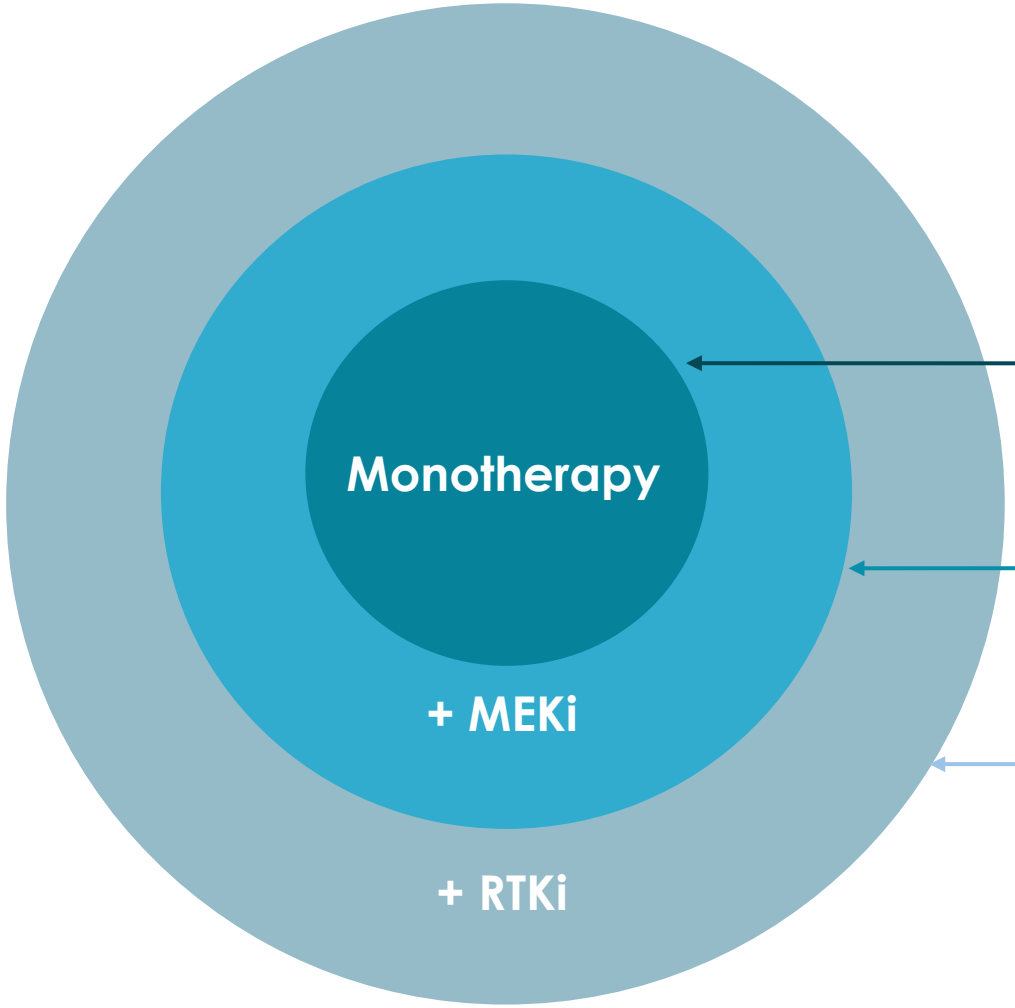


Illustration based on Kinnate preclinical data, prior clinical data from other pan-RAF inhibitors and publications. Note: other solid tumors beyond those in the illustration have potential opportunity with a pan-RAF but are not as well-characterized.

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

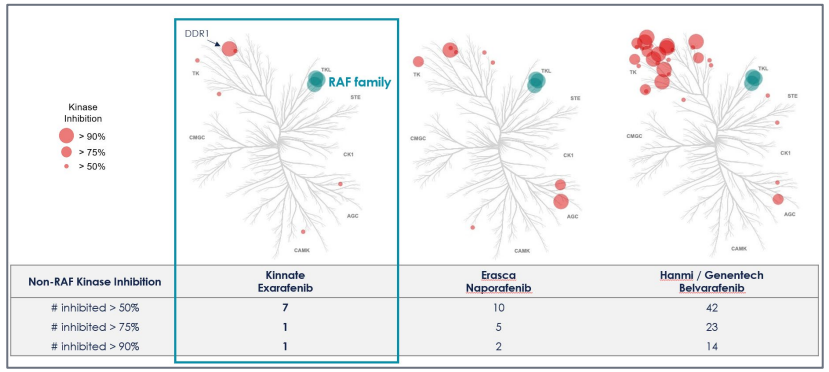
Combination with other RTKi (e.g., KRAS, EGFR) where RAF mutations co-occur (e.g., Class III or resistance mutations to primary drivers)

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

Substantial Preclinical Evidence of Differentiation

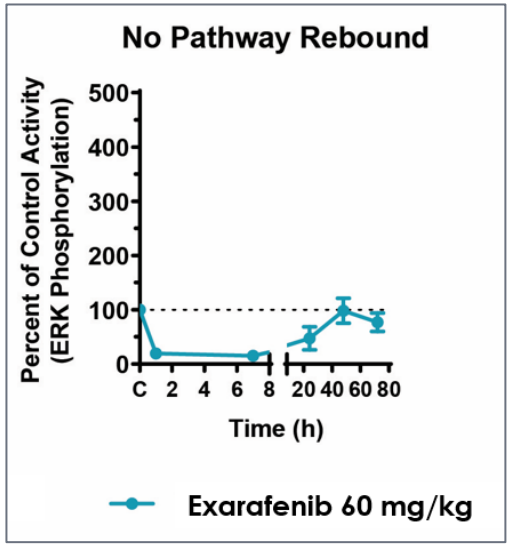
Exarafenib is Designed to Achieve the Optimal Product Profile Needed for BRAF-Driven Cancers

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	$BRAF^{Indel(V1AFTP)}$	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
	NCI-H358	NSCLC	$BRAF^{WT}$, $KRAS^{G12C}$	1	153	303	351
Wild Type (WT)	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

Based on Kinnate generated data

Phase 1 Exarafenib Monotherapy Dose Escalation Data

Exarafenib 300 mg bid is MTD and Expansion Dose

Expansion Cohorts of Phase 1 Trial Initiated Year End 2022

Enrolled Cohorts & Schema

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

Study Design

- 3+3 design, with accelerated titration (single patient cohorts) at DL1 & DL2
- DLT assessment period is 28 days (through 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

DLT = dose limiting toxicities

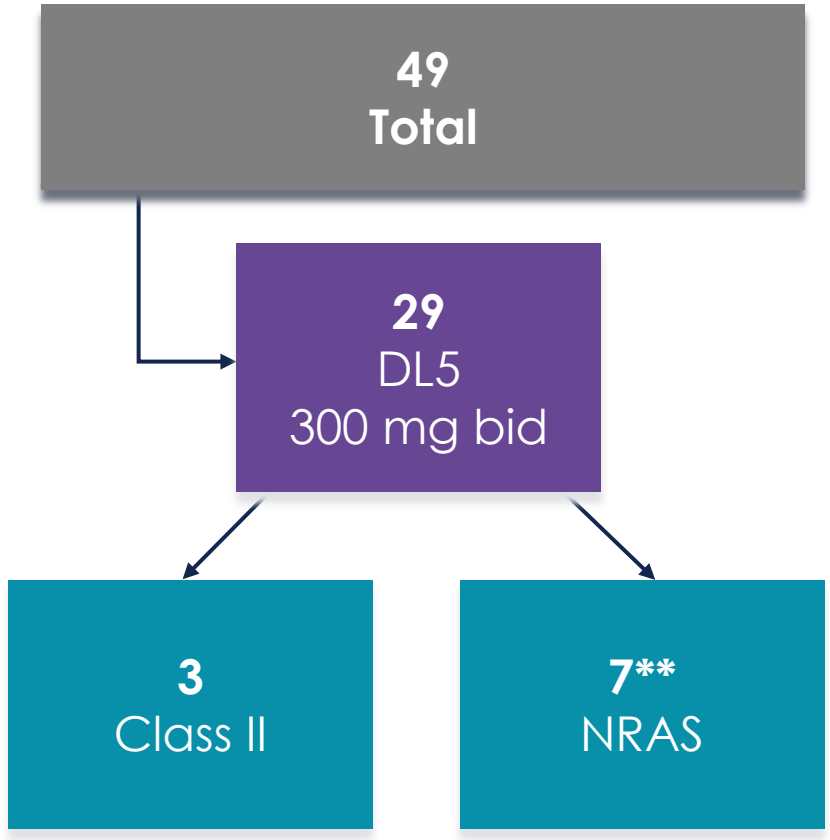
Enrollment Diversified Across Tumor Types & BRAF Alterations

Part A1: Enrollment (n) by Cancer Type, BRAF & NRAS Alteration

Cancer Type	Class I	Class II	Class III	NRAS	Total
NSCLC (Lung)	3	5	3		**11**
Melanoma	5	1	2	9	**17**
Colon & Rectal Carcinomas	11	1	8		**20**
Biliary (Cholangiocarcinoma & Ampullary Ca.)	1		3		**4**
Thyroid (Papillary)	3				**3**
Spindle Cell Sarcoma		1			**1**
Appendiceal Carcinoma			1		**1**
GIST (GI Stromal Tumor)	1				**1**
Breast Cancer			1		**1**
Skin Squamous Carcinoma (Apocrine)	1				**1**
TOTAL	**25**	**8**	**18**	**9**	**60**

Data cut date Feb 28, 2023; NSCLC = non-small cell lung cancer

Breakdown of Efficacy Evaluable Set*



Among 33 ctDNA positive cases evaluated to date, 29 cases (88%) had their primary BRAF or NRAS driver alteration confirmed on central Guardant OMNI testing

*Efficacy Evaluable Set includes all participants with documented BRAF (or melanoma with NRAS) genomic alterations (as specified for each study Part) who received at least 1 dose of exarafenib and have >= 1 measurable lesion at baseline for disease response assessment and at least 1 post-baseline efficacy assessment per RECIST Version 1.1 criteria. **Includes patient with NRAS and co-occurring BRAF Class III alterations. ctDNA=circulating tumor DNA

Baseline Demographics Indicate Heavily Pre-Treated Patients

Metric			All Patients (N=60)
Age (years)		**Mean (SD)**	61.6 (11.74)
		Median	63
		Min, Max	33, 84
Sex		**Male**	33 (55.0%)
		Female	27 (45.0%)
Ethnicity		**Hispanic or Latino**	2 (3.3%)
		Not Hispanic or Latino	49 (81.7%)
		Not Reported	9 (15.0%)
Race		**American Indian or Alaska Native**	0
		Asian	4 (6.7%)
		Black or African American	1 (1.7%)
		Native Hawaiian or Other Pacific Islander	0
		White	43 (71.7%)
		Other	4 (6.7%)
		Not Reported	8 (13.3%)
Adjudicated Prior Lines of Therapy		**Mean (SD)**	3.3 (1.98)
		Median	3
		Min, Max	1, 11
ECOG PS	**0**		20 (33.3%)
	1		38 (63.3%)
	2		2 (3.3%)

Data cut as of Feb 28, 2023
ECOG PS=Eastern Cooperative Oncology Group Performance Status

Safety Profile: Favorable Across Dose Levels & the MTD, 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

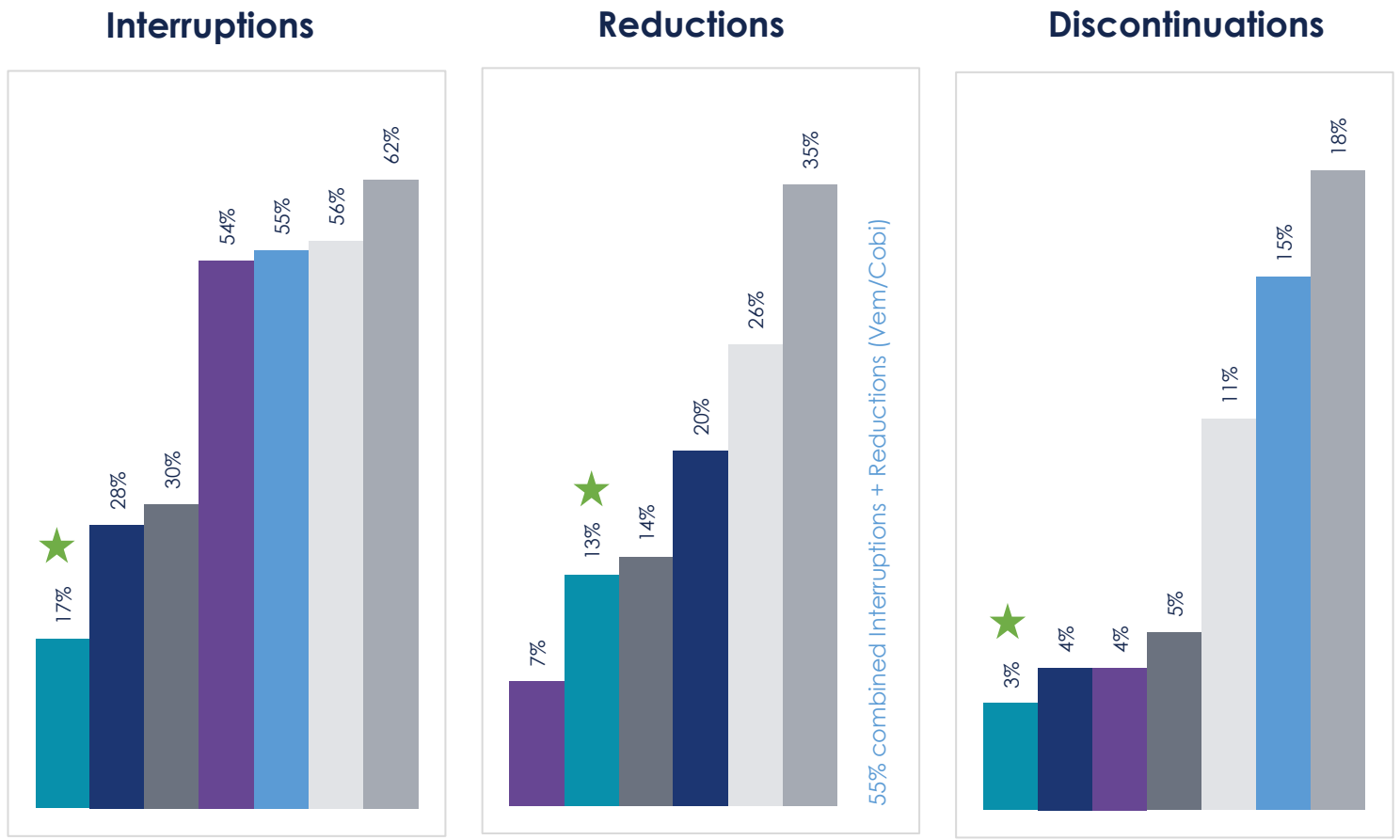
Exarafenib Achieved Substantial and Differentiated Dose Intensity

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

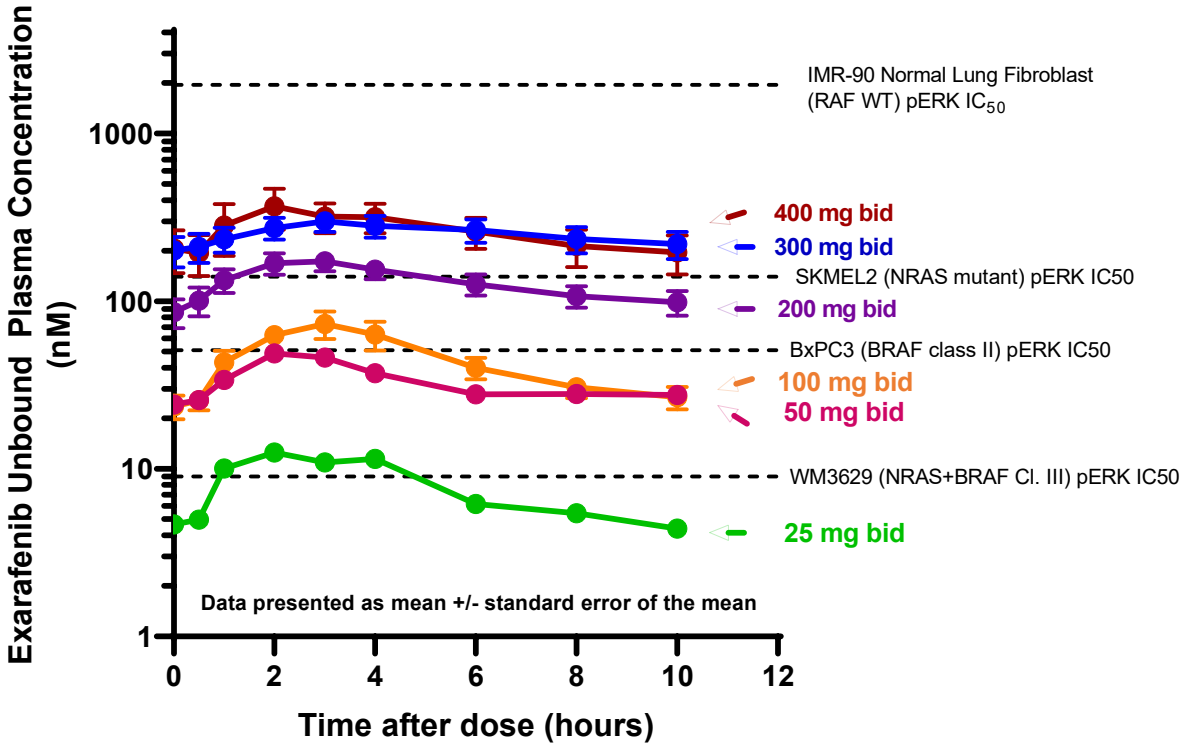
- Exarafenib
- FORE-8394 - Solid Tumors
- Belvarafenib - Solid Tumors
- Vemurafenib + Cobimetinib – Melanoma
- Encorafenib + Binimetinib – Melanoma
- Dabrafenib + Trametinib – NSCLC
- Dabrafenib + Trametinib – Met. Melanoma



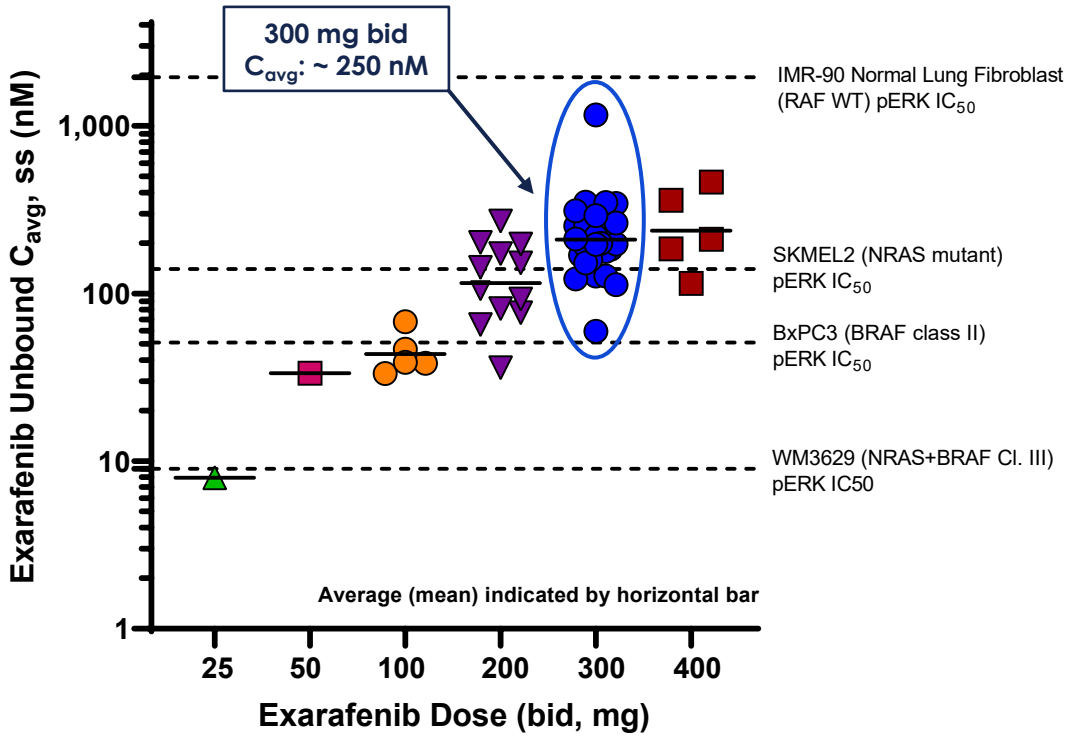
*Relative dose intensity refers to the total actual dose received divided by total planned dose for the period patient received the assigned dose. Exarafenib data cut as of Feb 28, 2023.
USPI used as source for Dabrafenib, Encorafenib, Vemurafenib – Accessed 27 March 2023. Cobimetinib USPI for vemurafenib + 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

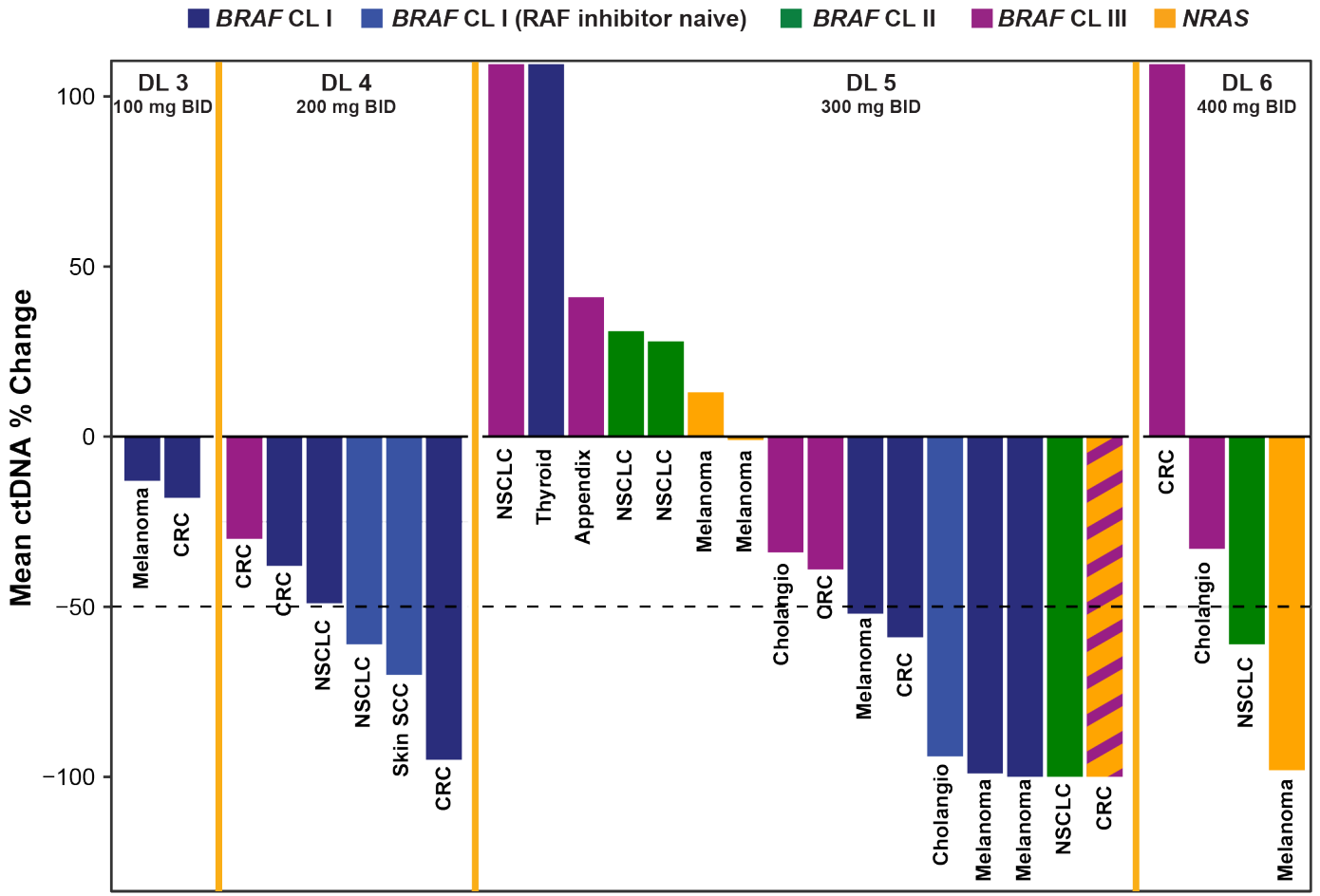
Exarafenib is Achieving Monotherapy Exposures Not Seen with Other Agents

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...

Class II	NRAS	NRAS + Class III*	Class I RAF Pretreated	Class I RAF Naïve	Class I RAF Naïve
1 cPR	1 cPR	1 cPR	1 uPR	1 cPR	1 cPR

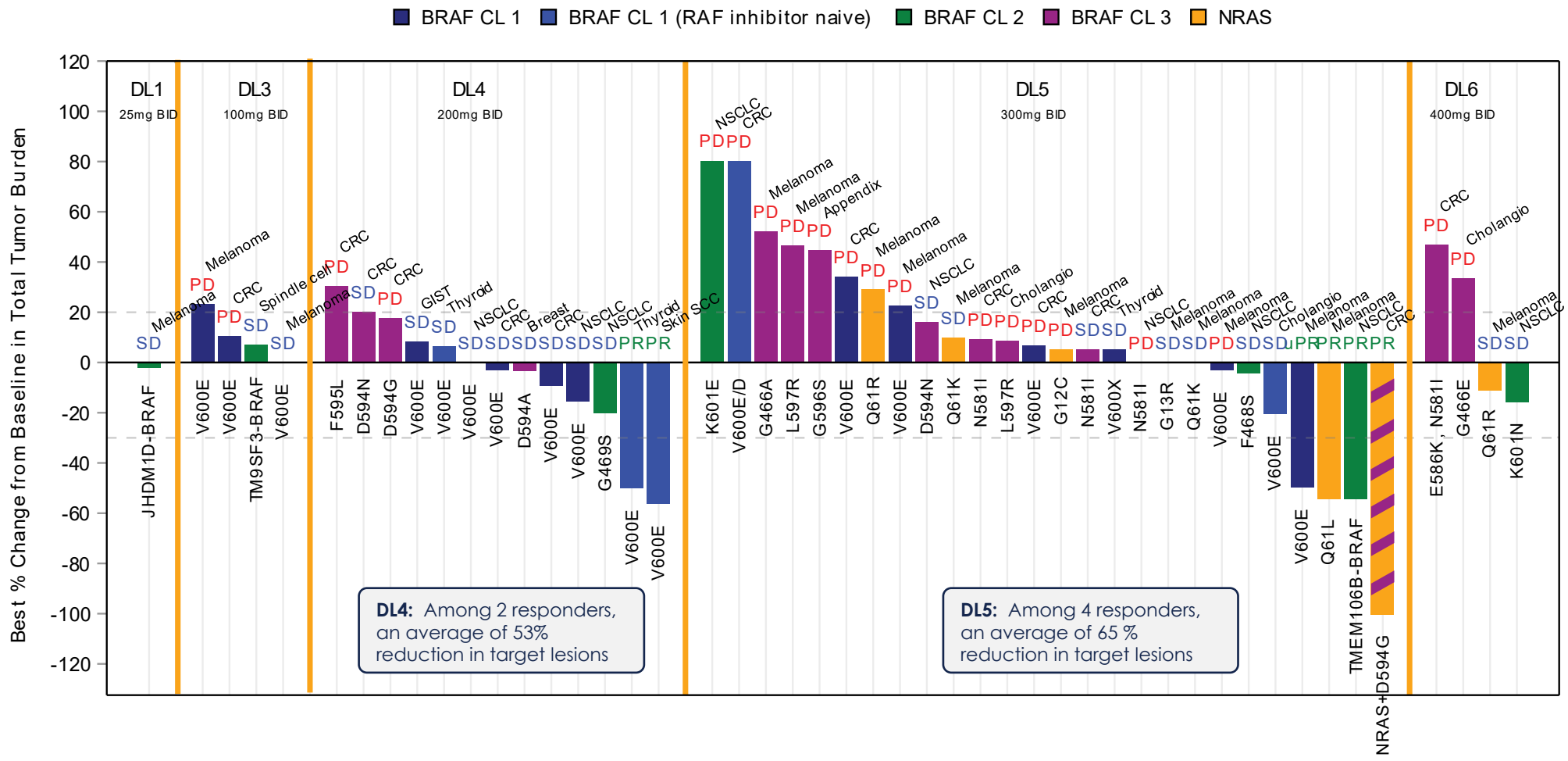
...And, Across Broad Range of Tumor Types

- 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

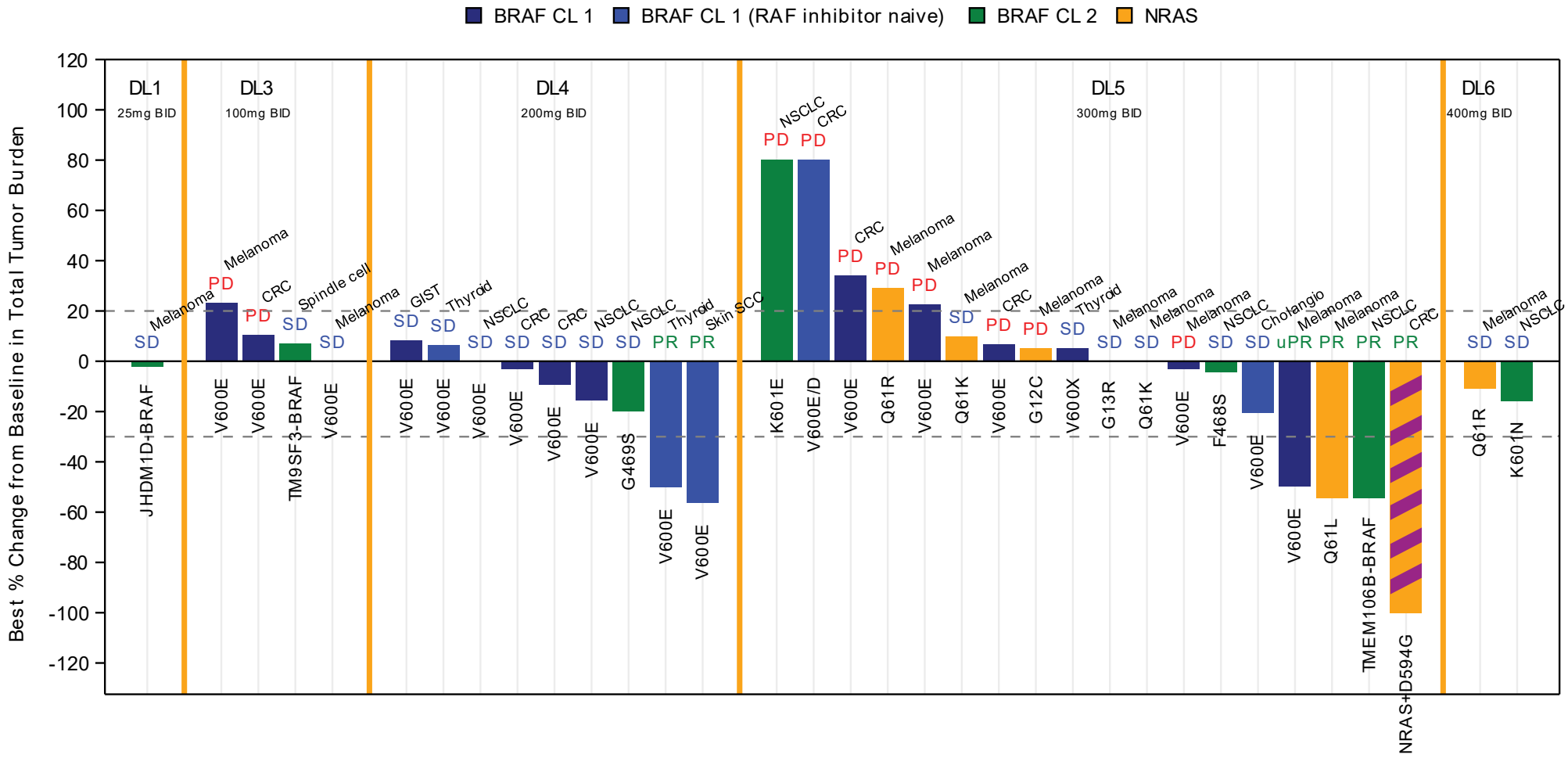


33% ORR in Class II & 29% ORR in NRAS at 300 mg bid

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



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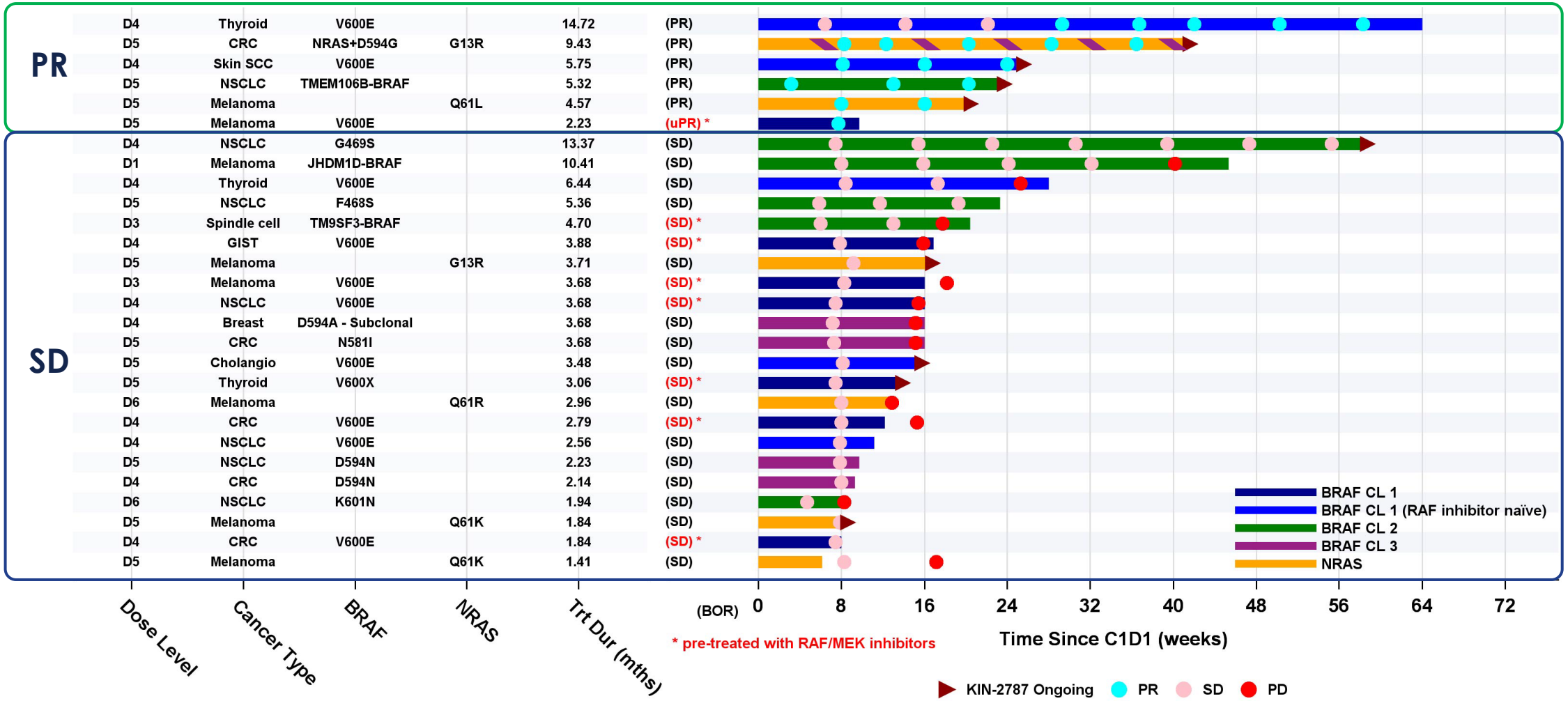
Sustained Duration with Follow Up Ongoing

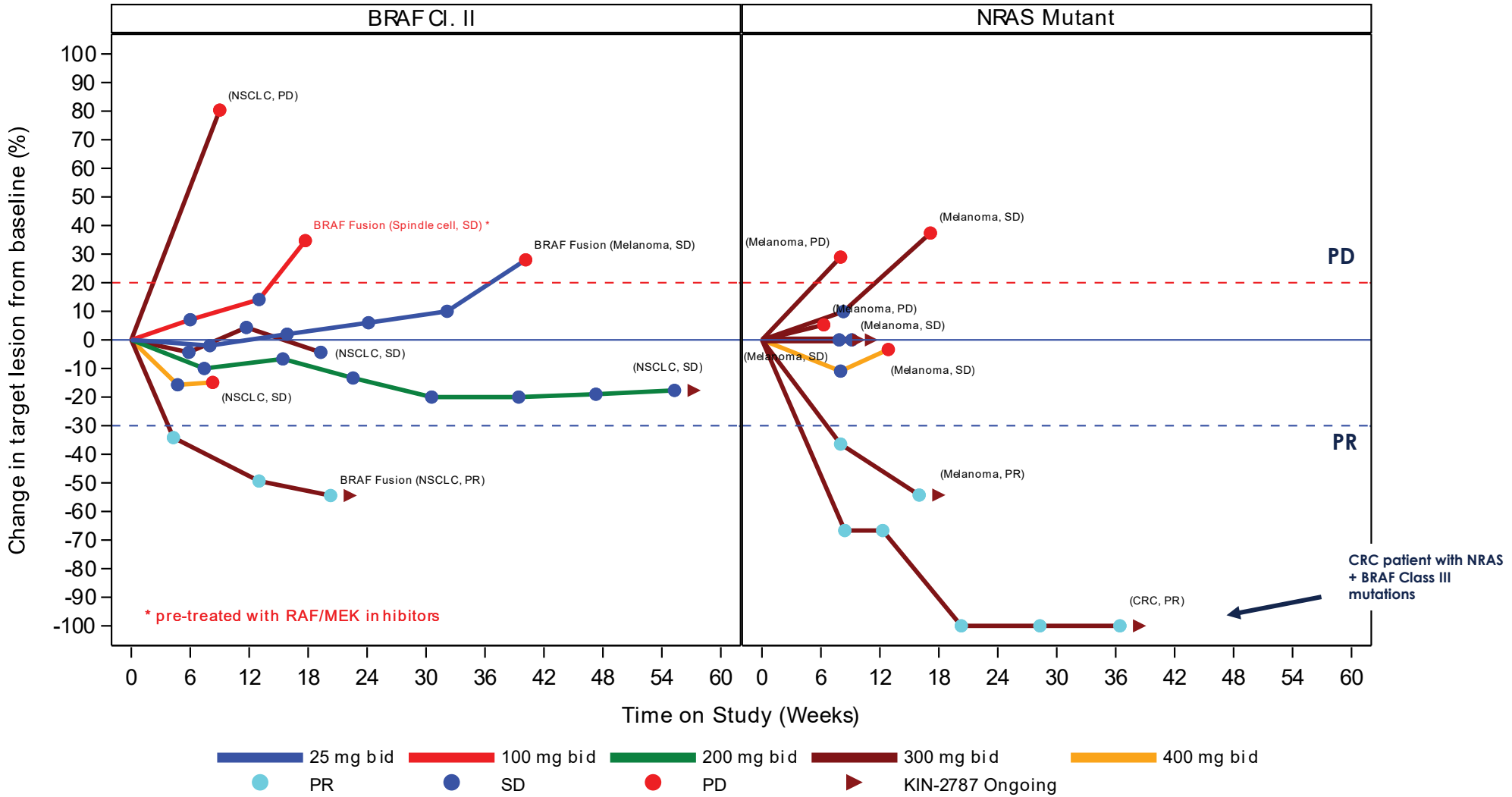
Responders Stayed on Treatment for an Average of 7 Months; mDOR Not Reached



Data cut as of Feb 28, 2023

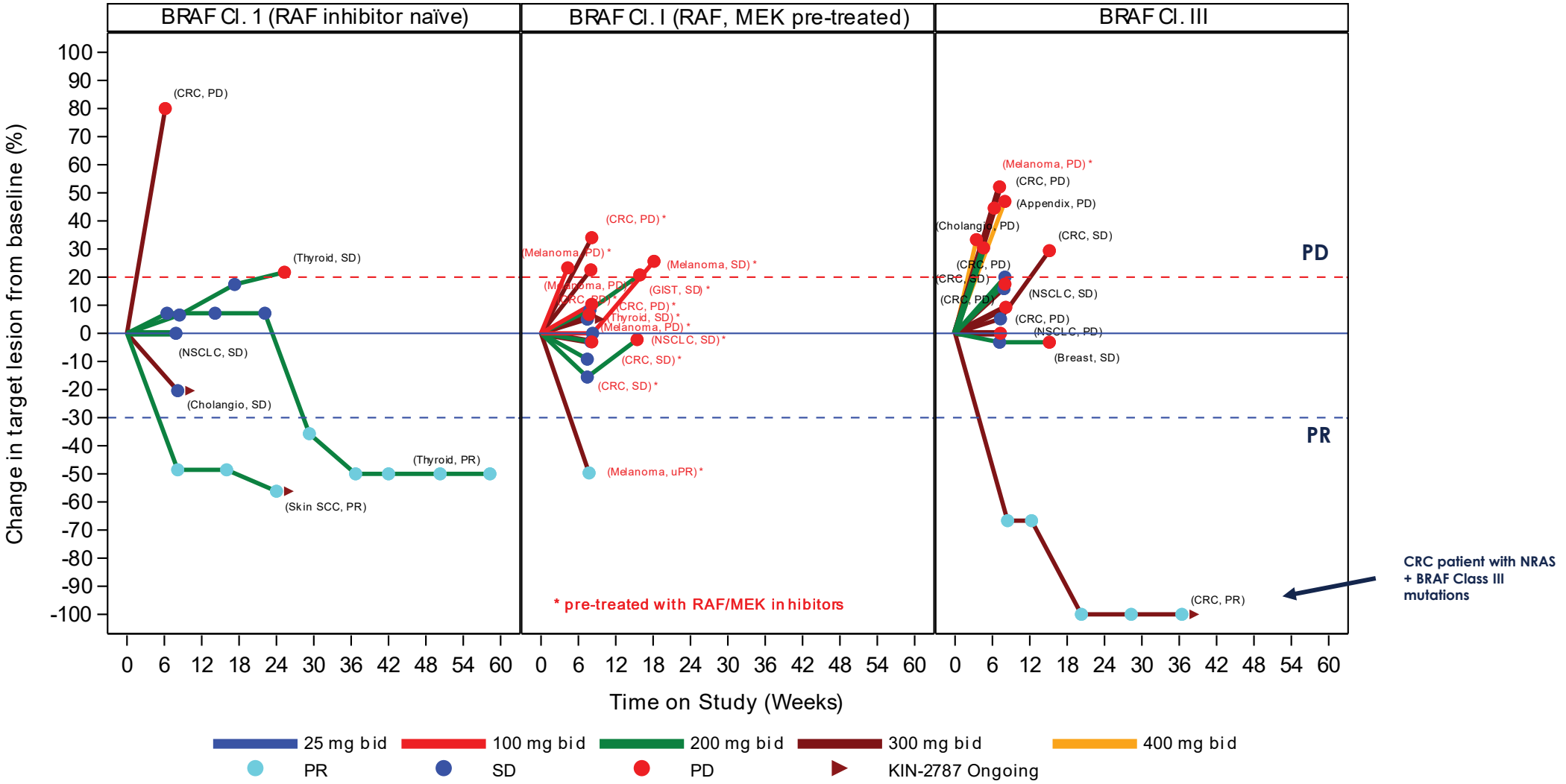
Compelling Data Supports Priority Focus in Class II and NRAS

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

Patient Case Studies: Exarafenib Monotherapy

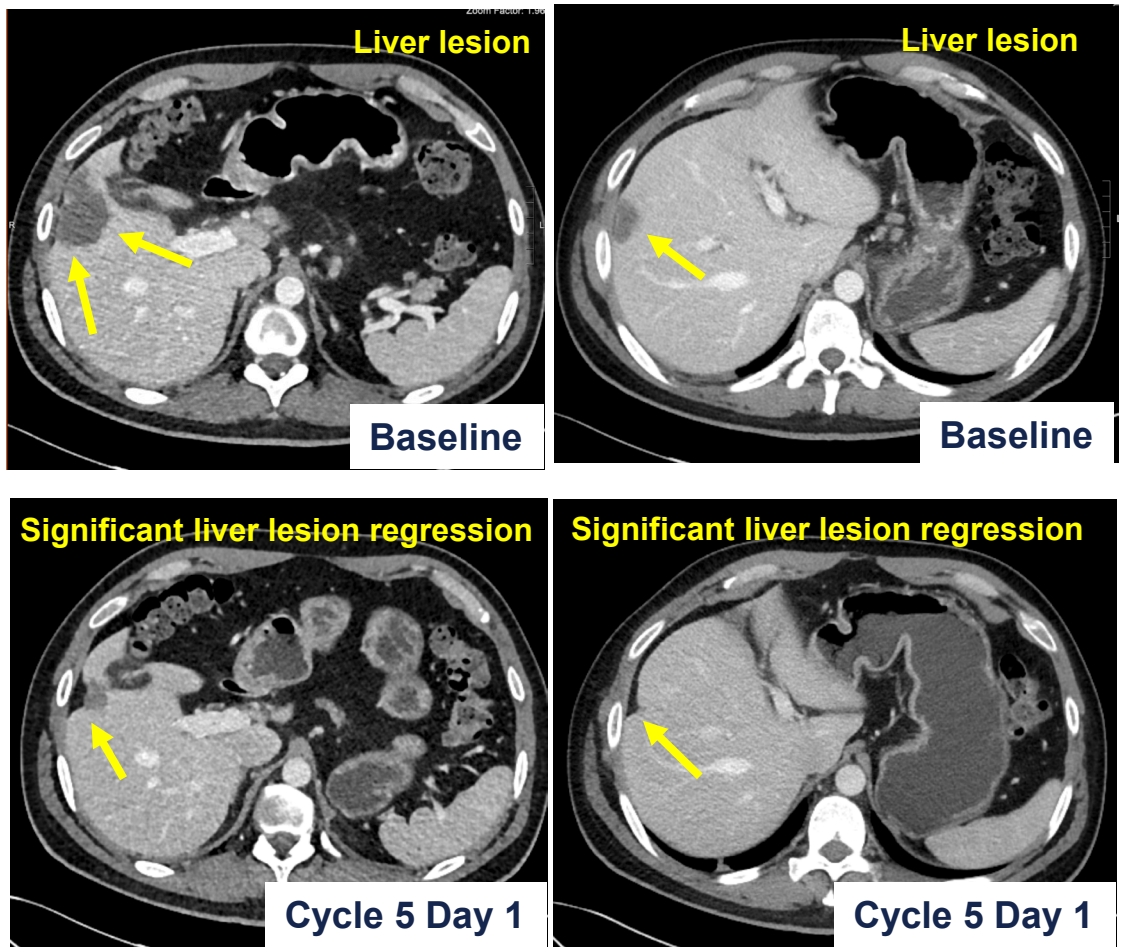
Confirmed Partial Response in Patient with BRAF Class II Lung Cancer Highlights Impressive Activity in Our Core Population

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

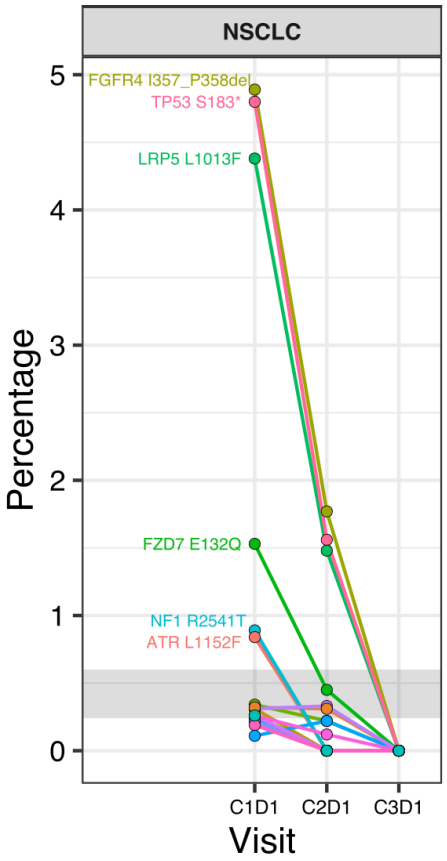
- PR on 1st scan with rapid response (**-34%** on 2 target lesions)
- Drug interruption (rash) & pt resumed exarafenib at DL5 (300 mg bid)
- Confirmed PR on 2nd & 3rd scans (**-54%** reduction of target lesions), significant reductions in non-target lesions
- Complete molecular response by C3D1
- Pt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

Data cut as of Feb 28, 2023

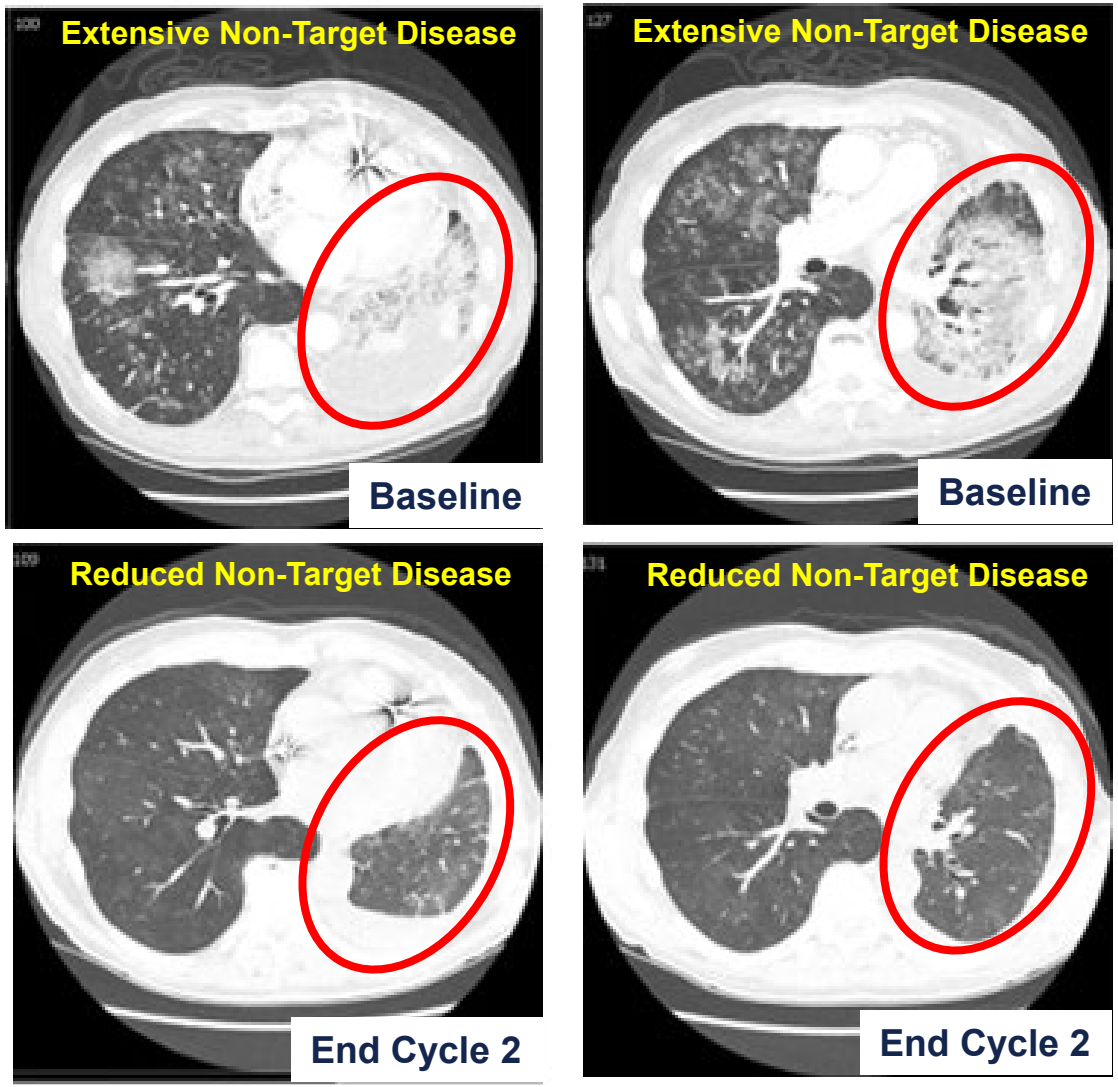
Substantial Clinical Benefit in Patient with BRAF Class II Lung Cancer; Patient Remains on Therapy for 13+ Months

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

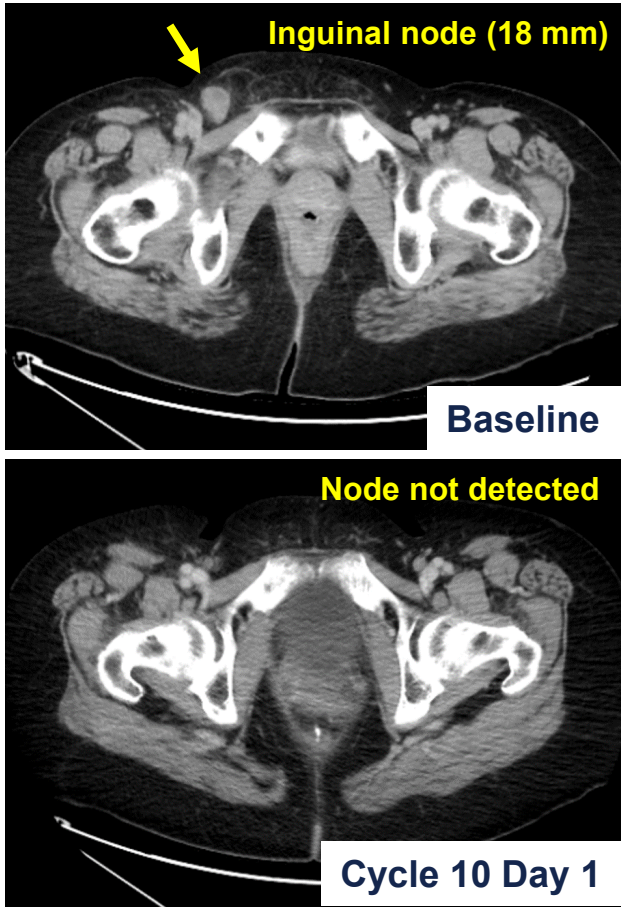
DL5 (300 mg bid)

Refractory to multiple prior lines of Oxaliplatin, Capecitabine, Irinotecan

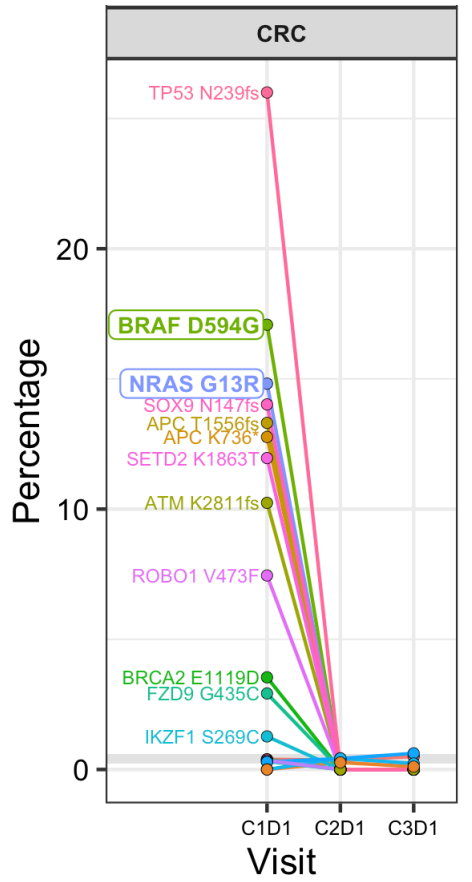
- Rectal bleeding resolved after 2 weeks
- PR on first scan with deep response **(-67%)**
- PR confirmed on 4 more assessments
- CR on target lesion **(-100%)**
- Significant reduction in non-target pulmonary disease
- Complete molecular response by 4 weeks
- Pt remains on exarafenib for 10+ months

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

Pelvic Lesion



ctDNA Analysis



Data cut as of Feb 28, 2023

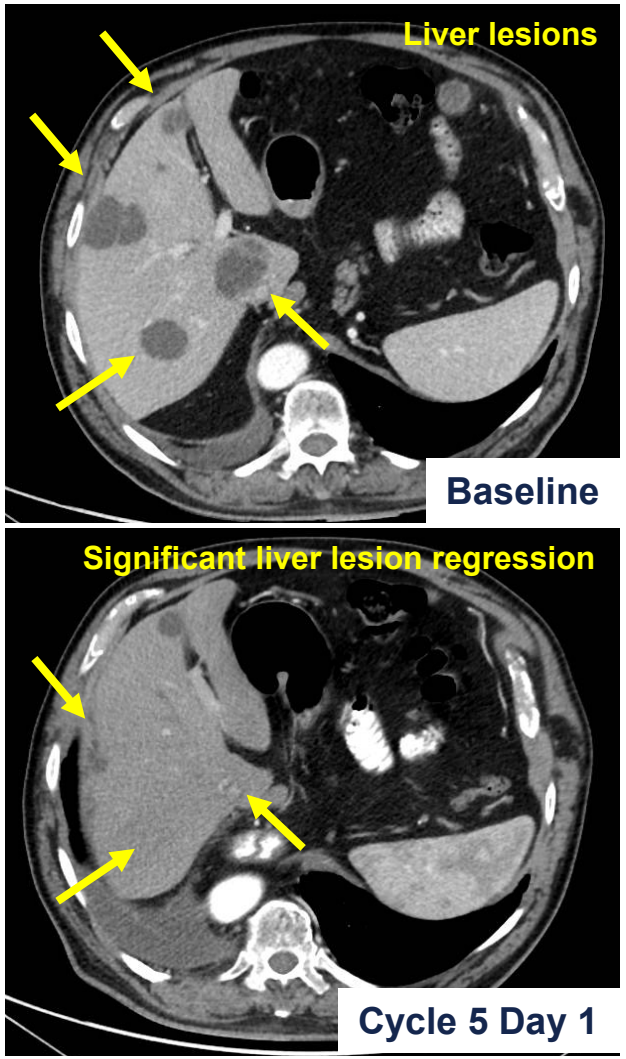
Deep, Confirmed Partial Response in Patient with NRAS Mutant Melanoma

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

Preliminary Exarafenib + Binimetinib Combination Study Update

Exarafenib + Binimetinib Phase 1 Dosing Schema & Study Design

Enrollment Continues at Exarafenib 200 mg bid + Binimetinib15 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 profile evaluation ongoing

NRAS Mutant Melanoma

1uPR in;
41% Reduction

NRAS Mutant Melanoma

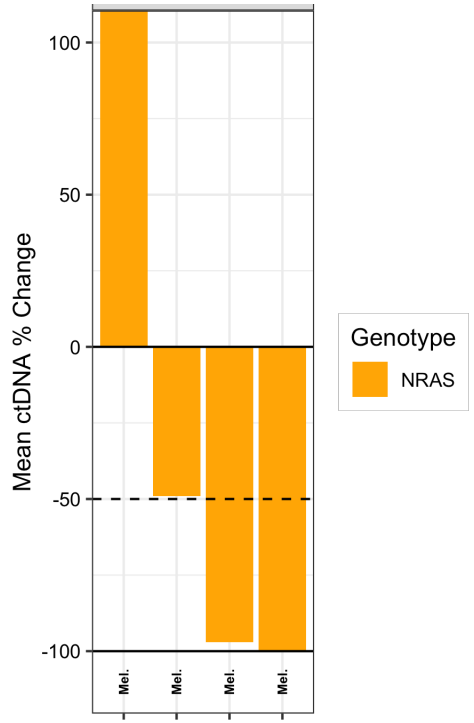
25% reduction in target lesions

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

Monotherapy Dose Expansion & Combination Dose Escalation Strategy

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

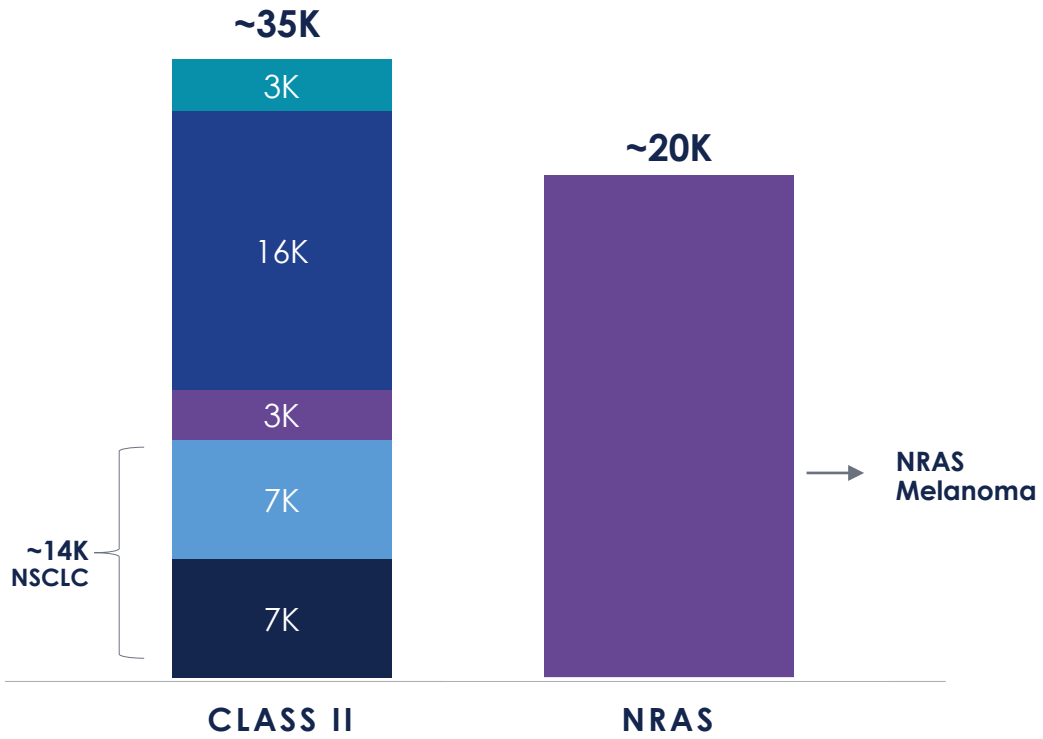
Strategy to be Refined, May Require Enrichment

RAF Market Opportunity

~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



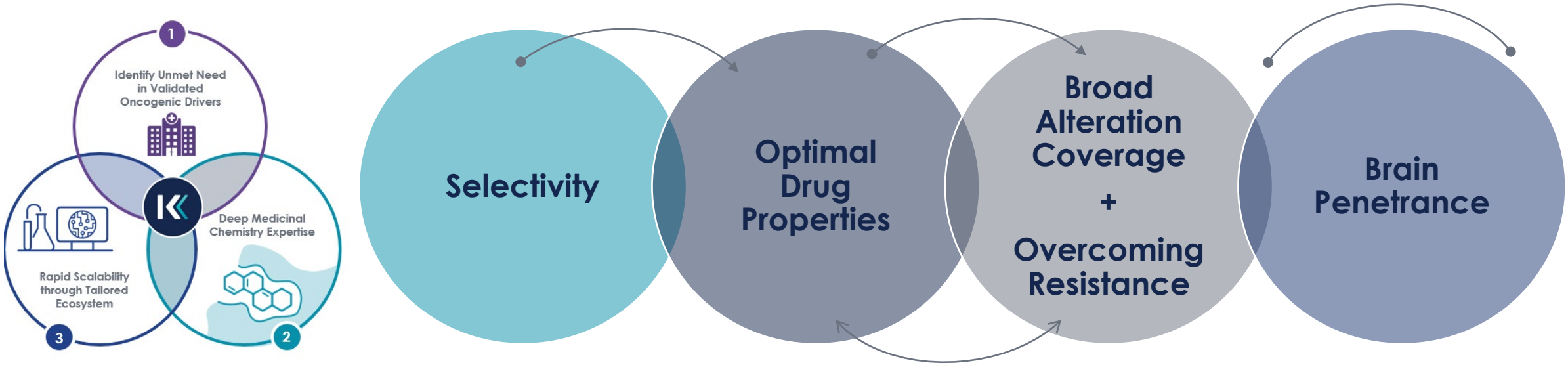
Additional Opportunities

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.

Early Pipeline

Deep Expertise in Medicinal 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]

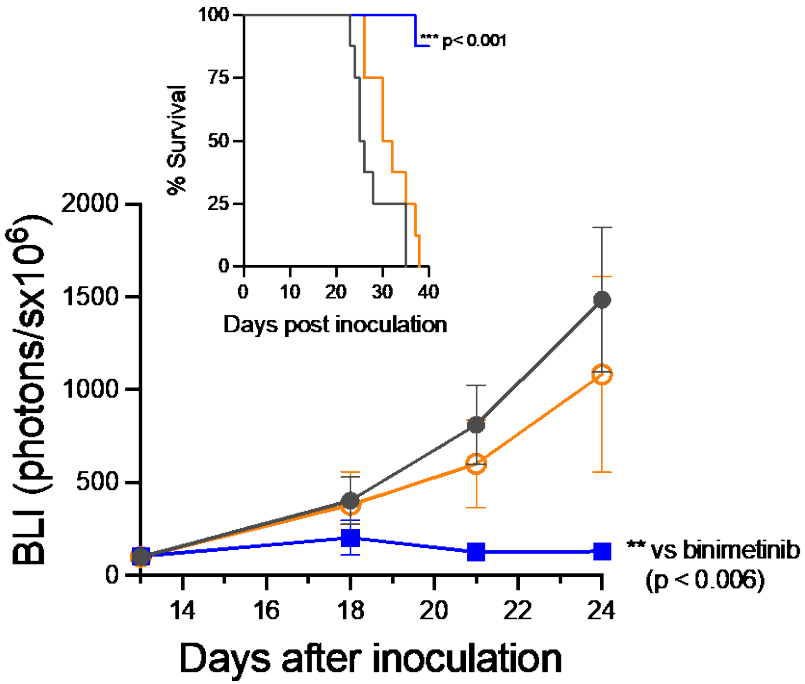
- ✓ **Brain penetrant**
- ✓ **Highly selective, dual mechanism**
- ✓ **Quality drug-like properties**
- ✓ **Paradigm-breaking simplicity of structure**

(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

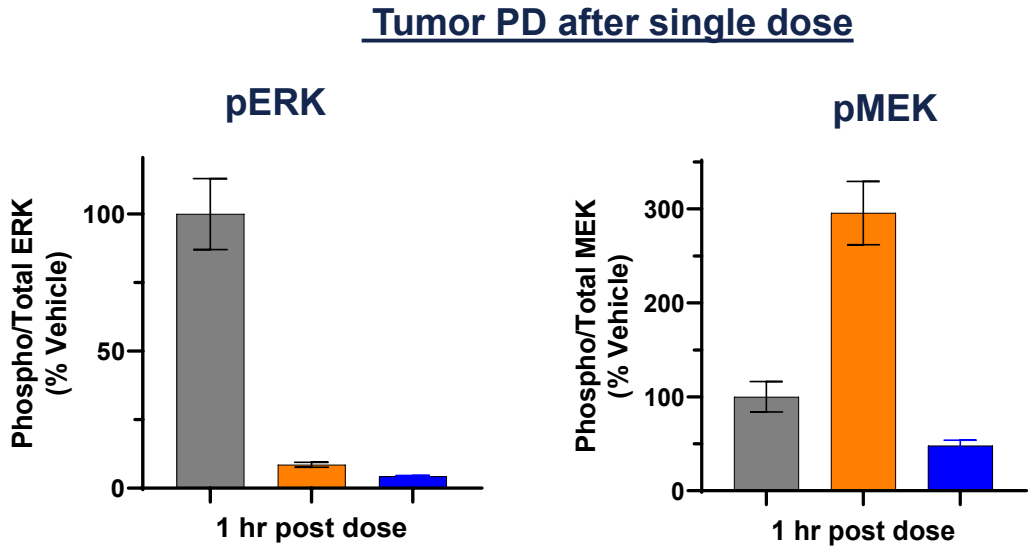
Dual Function Inhibits MEK Activity (pERK) and Suppresses Activation (pMEK) in BRAF Class I Melanoma

$BRAF^{V600E}$ Intracranial Tumor



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

$BRAF^{V600E}$ Subcutaneous Tumor PD



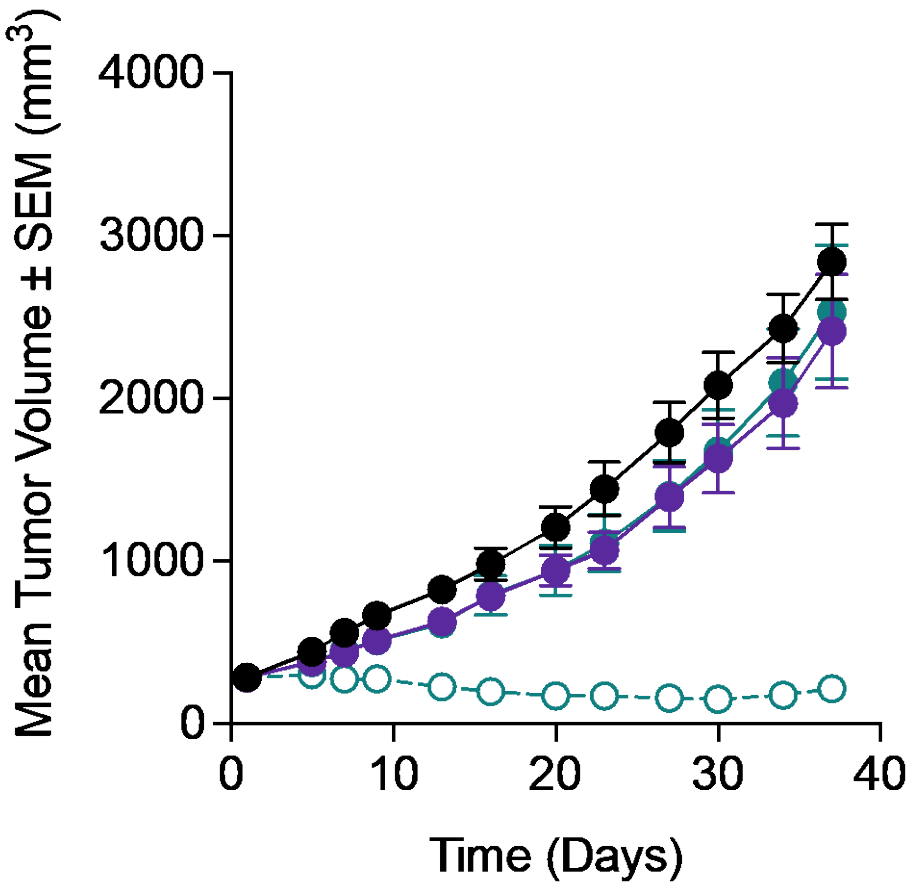
KIN-7136 suppresses upstream priming (pMEK) of MAPK pathway vs binimetinib

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

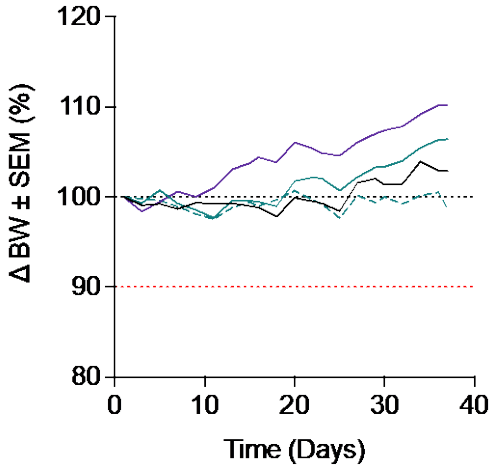
Study doses are exposure-matched to clinical exposure of binimetinib

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

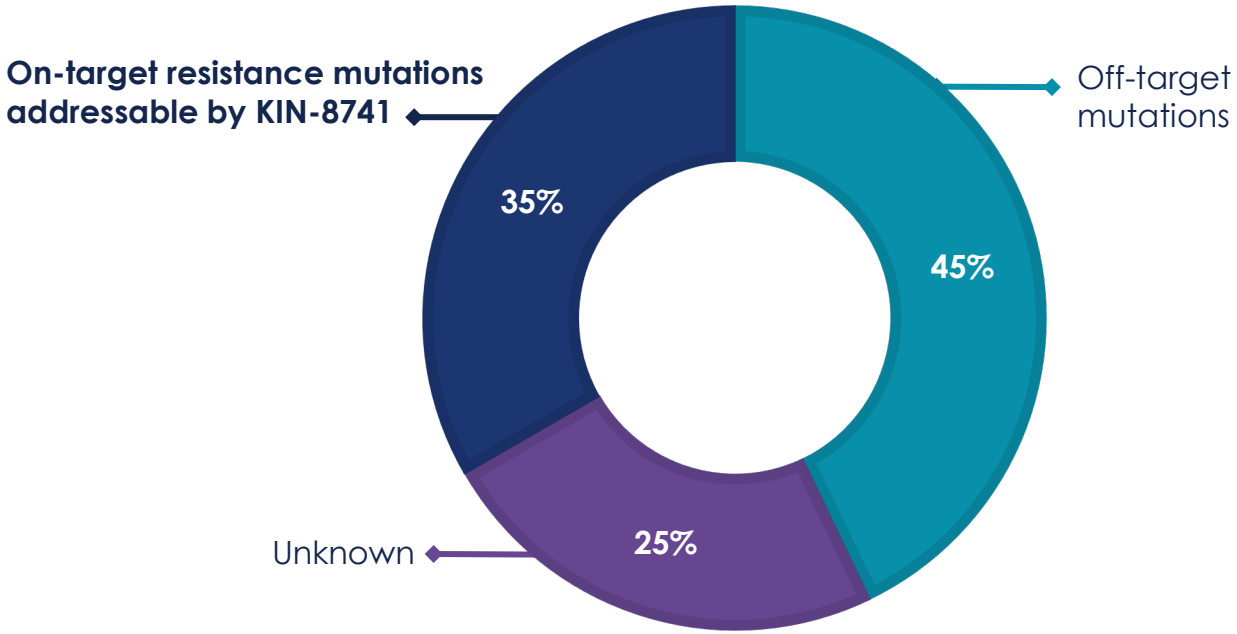
KIN-8741 Targets c-MET and Acquired Resistance Mechanisms

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

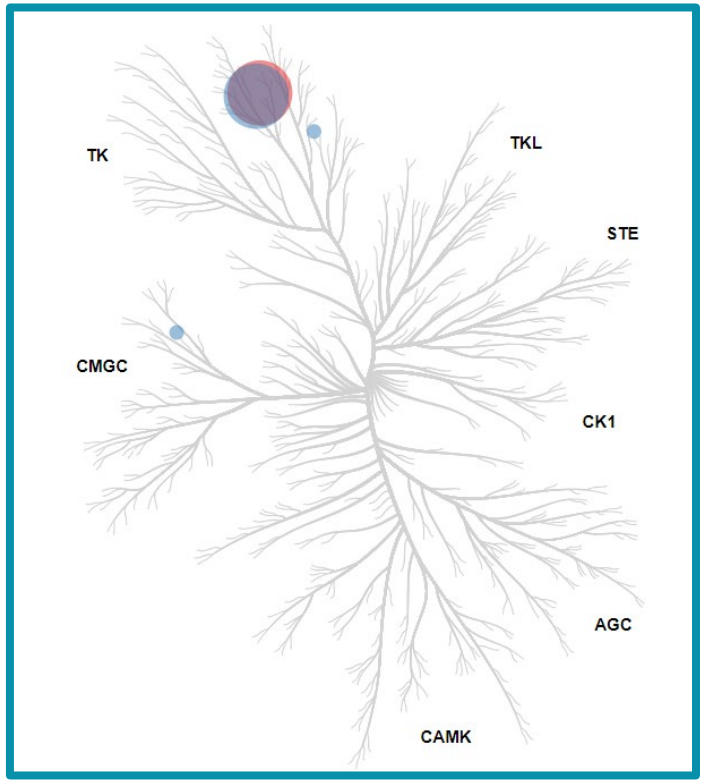
KIN-8741 is Highly Selective

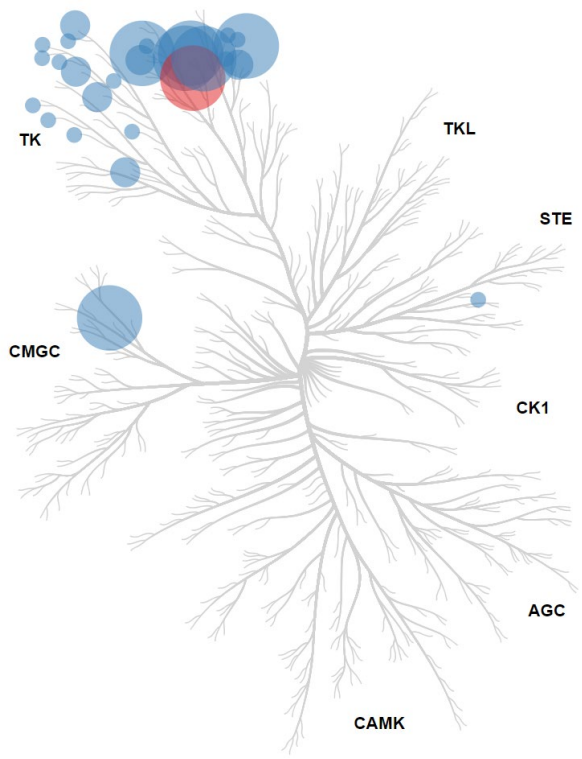
Node Color

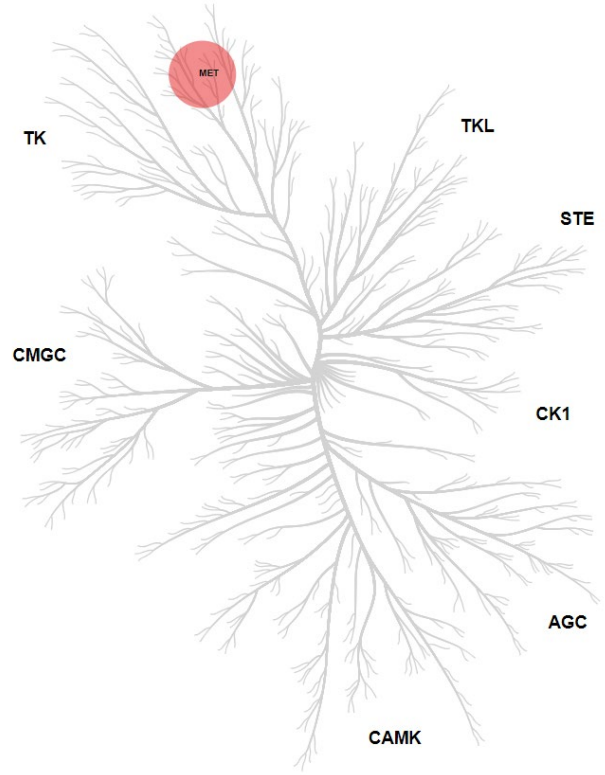
- Target
- Off-Target

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 trees are 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

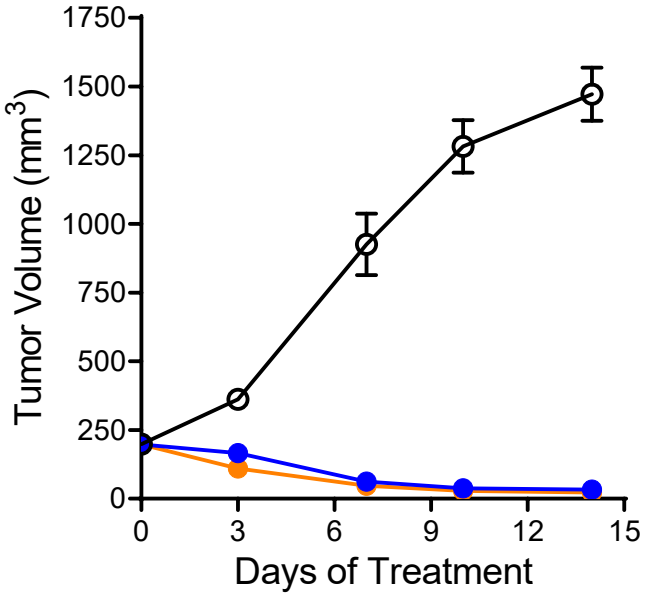
Cellular viability profiling in engineered TPR-MET Ba/F3 cells

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**

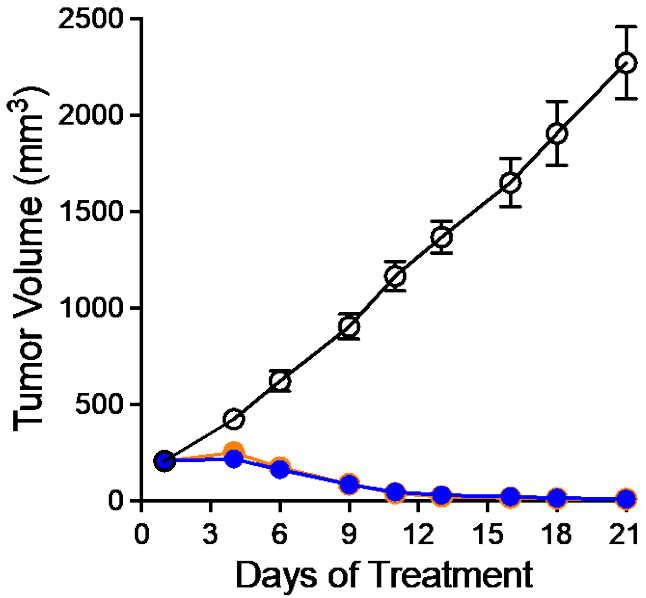
EC_{50} (nM)	<30	<100	<300	<1000	>1000

Antitumor Activity Achieved Across c-MET Mutation Models

Activating Alterations in NSCLC

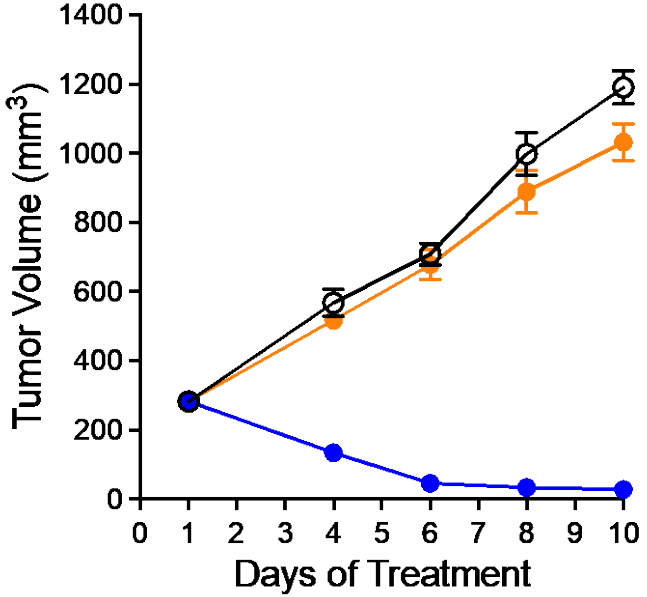


c-MET$^{\Delta ex14}$



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

Closing Remarks

Multiple Ongoing Clinical, IND-Enabling and Discovery Programs

~$231MM Cash On Hand[1]; Prioritization Allows Anticipated Cash Runway Into Early 2025

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								

[1]Cash, cash equivalents and investments as of March 31, 2023 (preliminary, estimated and unaudited financial results)

Please stand by as we transition to the Q&A portion of our presentation

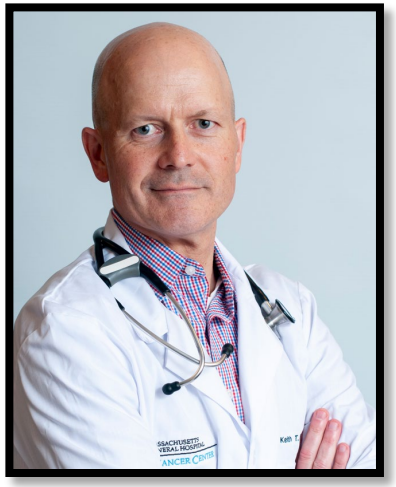
Speakers

Nima Farzan
CEO

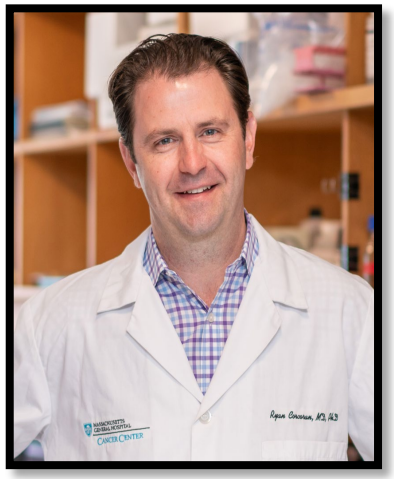
Richard Williams, MBBS, PhD
CMO

Rob Kania, PhD
SVP, Drug Discovery

Neha Krishnamohan
CFO



Keith Flaherty, MD
Harvard Medical School



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Thank you to the patients, clinical trial investigators and operations staff who participate in our research programs

Our mission

is to inspire hope

for those battling cancer by
expanding on the promise

of targeted therapies.