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On a Journey to Erase Cancer

Erasca R&D Update

October 24, 2024

Eric N., naporafenib clinical trial participant and cancer survivor, and his wife Margaret

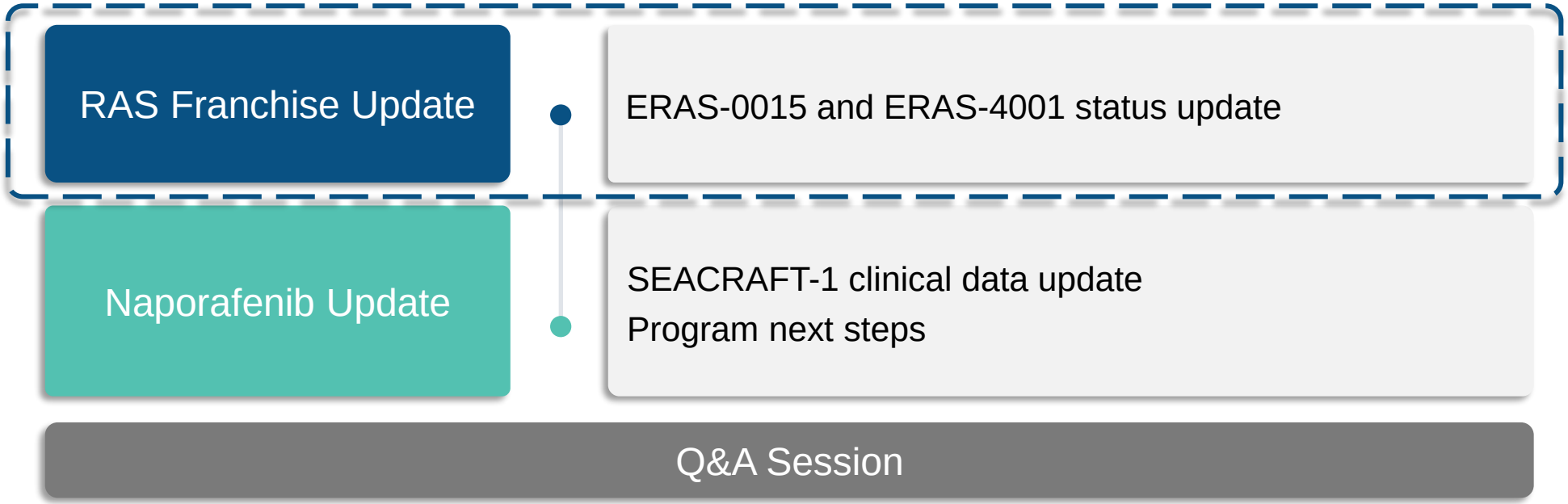
Disclaimer: Forward Looking Statements & Market Data

We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, research and development plans, the anticipated timing (including the timing of regulatory filings and the timing of clinical trial data readouts), costs, design and conduct of our ongoing and planned preclinical studies and clinical trials for our product candidates, the potential therapeutic benefits of our product candidates, the timing and likelihood of success of our plans and objectives, the impact of the deprioritization of certain programs, and future results of anticipated product development efforts, are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negative of these terms or other similar expressions. The inclusion of forward-looking statements should not be regarded as a representation by us that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: our approach to the discovery and development of product candidates based on our singular focus on shutting down the RAS/MAPK pathway, a novel and unproven approach; we only have one product candidate in clinical development and all of our other development efforts are in the preclinical or development stage; our assumptions about ERAS-0015's or ERAS-4001's development potential are based in part on the preclinical data generated by the licensors and we may observe materially and adversely different results as we conduct our planned studies; the analysis of pooled Phase 1 and Phase 2 naporafenib plus trametinib data covers two clinical trials with different designs and inclusion criteria, which cannot be directly compared, and therefore may not be a reliable indicator of ORR, mPFS, or mOS data; due to differences between trial designs and subject characteristics, comparing data across different trials may not be a reliable indicator of data; preliminary results of clinical trials are not necessarily indicative of final results and one or more of the clinical outcomes may materially change as patient enrollment continues, following more comprehensive reviews of the data and more patient data become available, including the risk that an uPR to treatment may not ultimately result in a cPR to treatment after follow-up evaluations; our SEACRAFT trials may not support the registration of naporafenib; later developments with the FDA or EU health authorities may be inconsistent with the feedback received to date regarding our development plans and trial designs; Fast Track Designation may not lead to a faster development or regulatory review or approval process, and does not increase the likelihood that our product candidates will receive marketing approval; our assumptions around which programs may have a higher probability of success may not be accurate, and we may expend our limited resources to pursue a particular product candidate and/or indication and fail to capitalize on product candidates or indications with greater development or commercial potential; potential delays in the commencement, enrollment, data readout, and completion of clinical trials and preclinical studies; our dependence on third parties in connection with manufacturing, research, and preclinical and clinical testing; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval, and/or commercialization, or may result in recalls or product liability claims; unfavorable results from preclinical studies or clinical trials; results from preclinical studies or early clinical trials not necessarily being predictive of future results; the inability to realize any benefits from our current licenses, acquisitions, or collaborations, and any future licenses, acquisitions, or collaborations, and our ability to fulfill our obligations under such arrangements; regulatory developments in the United States and foreign countries; our ability to obtain and maintain intellectual property protection for our product candidates and maintain our rights under intellectual property licenses; our ability to fund our operating plans with our current cash, cash equivalents, and marketable securities into the first half of 2027; and other risks described in our prior filings with the Securities and Exchange Commission (SEC), including under the heading “Risk Factors” in our annual report on Form 10-K for the year ended December 31, 2023, and any subsequent filings with the SEC. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.

Erasca Investor Presentation Agenda

RAS Franchise Update	ERAS-0015 and ERAS-4001 status update
Naporafenib Update	SEACRAFT-1 clinical data update Program next steps
Q&A Session	

Erasca Investor Presentation Agenda



ERAS-0015 and ERAS-4001 exhibit competitive profiles that exceed our TPP

ERAS-0015
Pan-RAS
Molecular Glue

Best-in-class potential for RASm solid tumors

- ~5x – 10x greater potency vs. pan-RAS MG in development
- Favorable ADME properties and PK performance in animals vs. pan-RAS MG in development
- Designed to address RASwt activation to prevent resistance vs. mutant-selective inhibitors

ERAS-4001
Pan-KRAS
Inhibitor

Potential first-in-class and best-in-class KRAS inhibitor

- Designed to spare H/NRAS WT
- Greater therapeutic window predicted vs. pan-RAS MG for KRASm solid tumors
- Designed to address KRASwt activation to prevent resistance vs. mutant-selective inhibitors

TPP = target product profile; RASm: RAS mutant; MG: molecular glue; ADME: absorption, distribution, metabolism, and excretion; PK: pharmacokinetic; WT: wild type; KRASm: KRAS mutant

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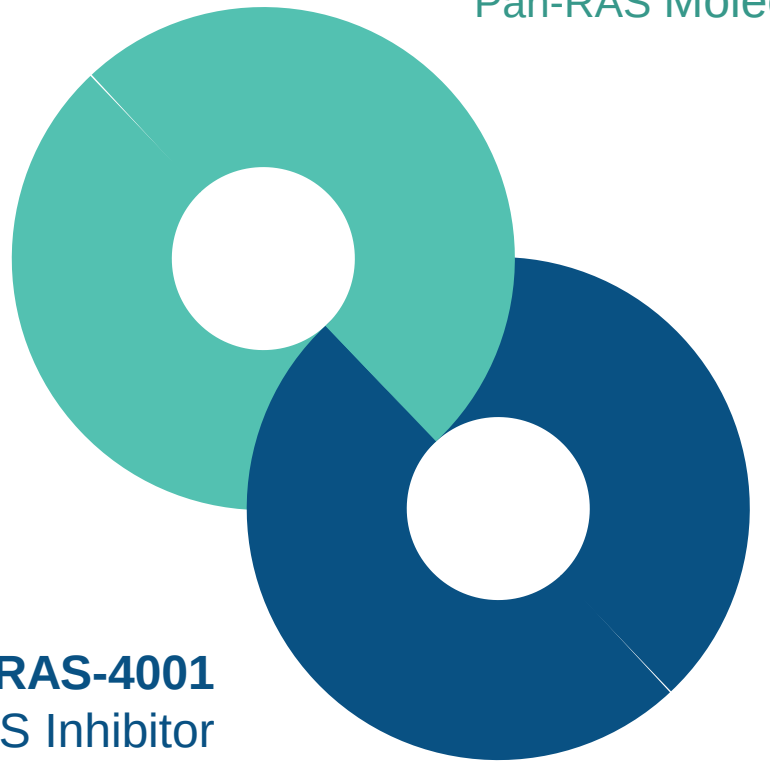
On track to file INDs for ERAS-0015 (H1 2025) and ERAS-4001 (Q1 2025)

ERAS-0015
Pan-RAS Molecular Glue



ERAS-4001
Pan-KRAS Inhibitor

Reproduced biological, biophysical, cellular, and in vivo data in house

Further characterized ERAS-0015's superior potency vs. RMC-6236

Completed in life portion of GLP toxicology studies for ERAS-0015; ERAS-4001 near completion

Advancing CMC per plan

CMC: chemistry, manufacturing and controls

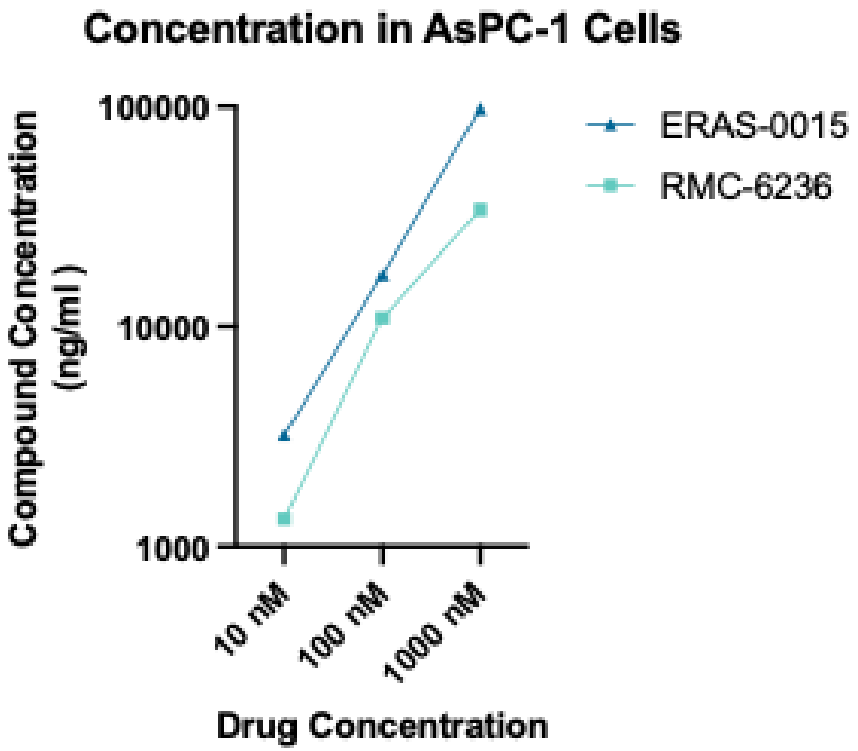
ERAS-0015's higher CYPA binding affinity may be a differentiator from RMC-6236; new data reinforces potential best-in-class profile

	Assay	Study conducted by	ERAS-0015 (nM)	RMC-6236 (nM)	Fold difference: ERAS-0015/ RMC-6236
Licensor data	SPR K_D	JOYO 嘉越 PHARMA	45.2	194	**4.3**
Erasca data	SPR K_D	ERASCA™	4.5	92	**20.5**
	ITC K_D	ERASCA™	5.3	44.1	**8.3**

Stronger binding to cyclophilin A (CYPA) may enable more potent RAS inhibition

SPR: surface plasmon resonance; K_D: equilibrium dissociation constant; ITC: isothermal titration calorimetry

New ERAS-0015 data in KRAS G12D PDAC cells demonstrated higher tumor cell concentrations vs. RMC-6236 in vitro, potentially driven by higher CYPA binding affinity

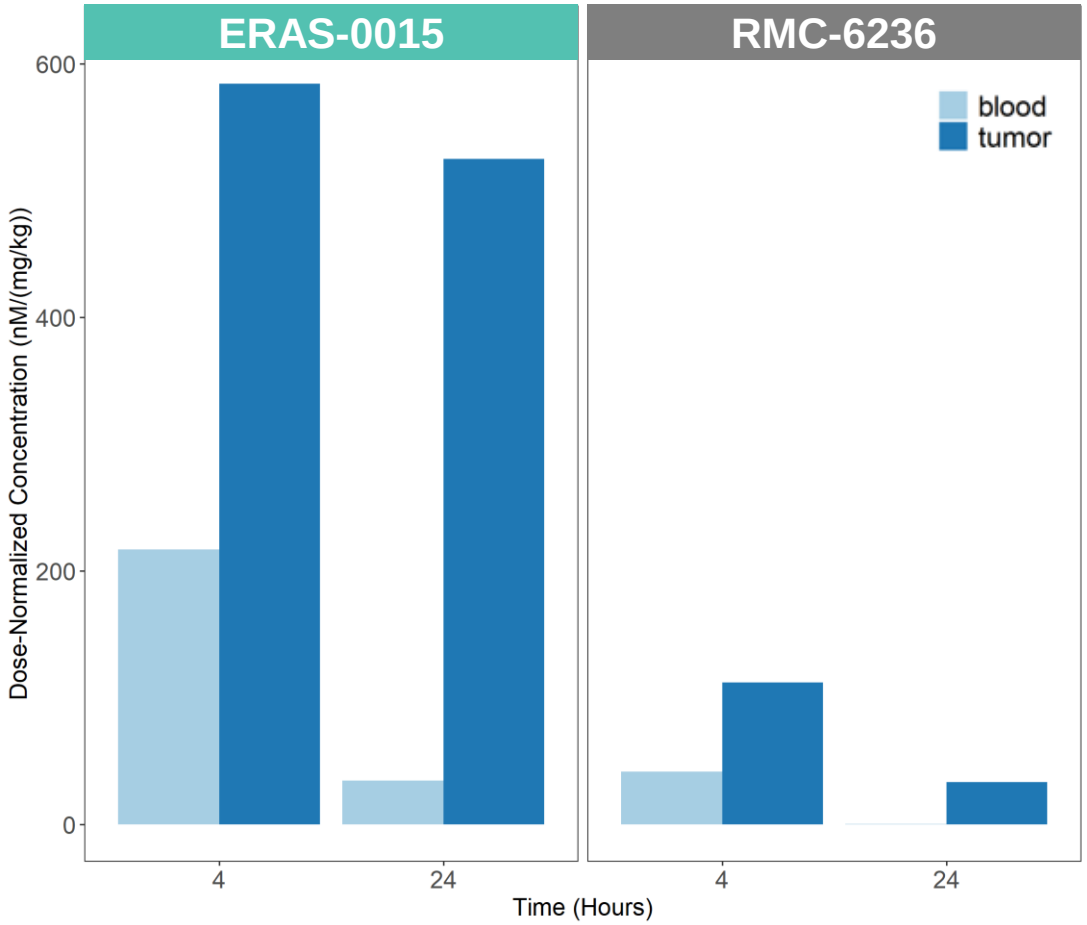


	10 nM Treatment		100 nM Treatment		1000 nM Treatment	
Compound	**Cellular (ng/ml)**	**Ratio to RMC-6236**	**Cellular (ng/ml)**	**Ratio to RMC-6236**	**Cellular (ng/ml)**	**Ratio to RMC-6236**
ERAS-0015	3,215	**2.4x**	17,200	**1.6x**	98,000	**2.9x**
RMC-6236	1,335	1x	10,950	1x	33,900	1x

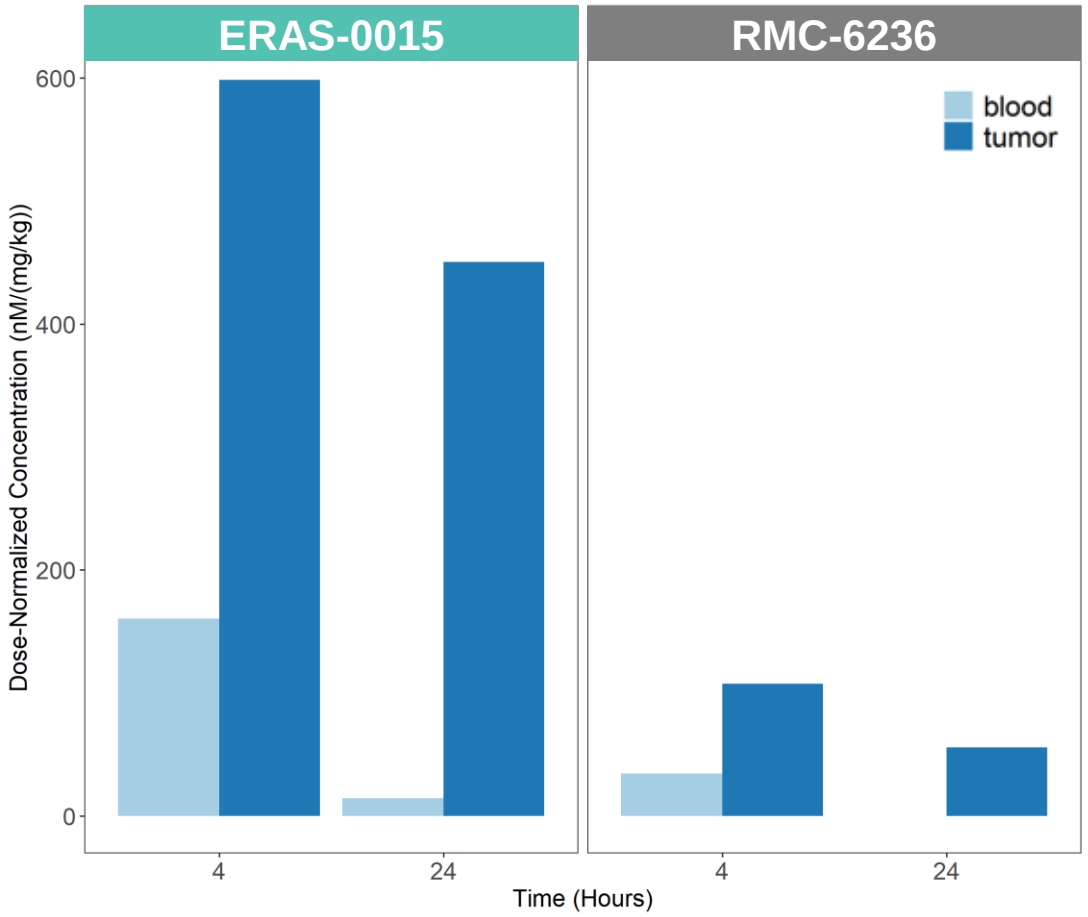
Compound concentration gradient in the media was linear across 3 doses for all compounds (data not shown)
PDAC: pancreatic ductal adenocarcinoma; CYPA: cyclophilin A

ERAS-0015 demonstrated preferential tumor distribution and longer residence time vs. RMC-6236 in vivo

Tumor PK Distribution Assessment in the KRAS G12D PDAC CDX Model, PK-59



Tumor PK Distribution Assessment in the KRAS G12R PDAC CDX Model, PSN-1



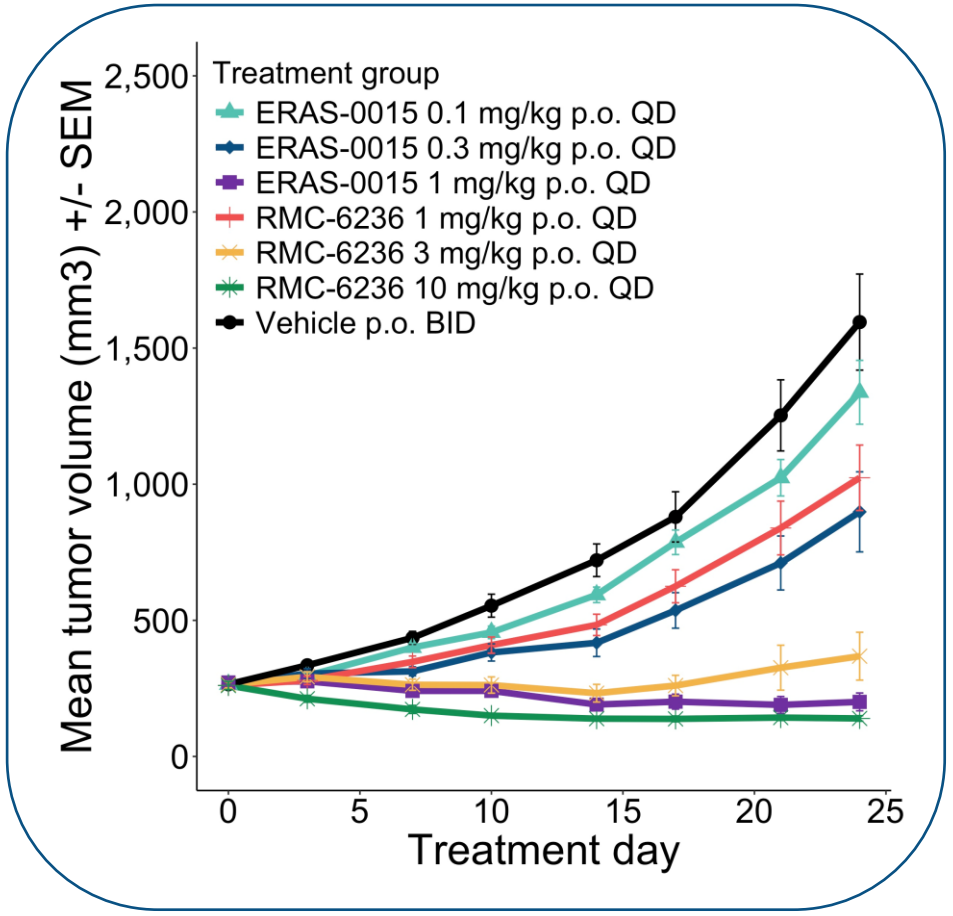
PDAC: pancreatic ductal adenocarcinoma; CDX: cell-line derived xenograft

ERAS-0015 showed ~8x-10x higher potency than RMC-6236 across multiple in vivo models

Model and TGI Measurement Day	ERAS-0015 Dose (mg/kg QD)	ERAS-0015 TGI	RMC-6236 Dose (mg/kg QD)	RMC-6236 TGI	Potency Ratio
PK-59 Day 23 (KRAS G12D PDAC)	0.3	106%	3	105%	**~10**
PSN-1 Day 14 (KRAS G12R PDAC)	1	97%	10	95%	**~10**
SW620 Day 26 (KRAS G12V CRC)	3	102%	25	103%	**~8**
NCI-H727 Day 22 (KRAS G12V NSCLC)	1	104%	10	104%	**~10**

TGI: tumor growth inhibition; PDAC: pancreatic ductal adenocarcinoma; CRC: colorectal cancer; NSCLC: non-small cell lung cancer; QD: once a day

Reproduced in vivo activity of ERAS-0015 in NCI-H727 NSCLC CDX model demonstrating comparable TGI at 1/10th the dose of RMC-6236



ERAS-0015 was well tolerated at all doses

Compound	Dose (mg/kg QD)	TGI on Day 25
ERAS-0015	1	105%
RMC-6236	10	109%

TGI: tumor growth inhibition; NSCLC: non-small cell lung cancer; CDX: cell-line derived xenograft; p.o.: orally administered; QD: once a day; BID: twice a day

ERAS-4001 showed potent activity against both GTP- and GDP-bound KRAS

Assay Class	Assay	Target	ERAS-4001 IC50 (nM)
Biochemical Functional	RAS-RAF Binding Assay (RBD)	RBD KRAS G12D GDP	1.6
		RBD KRAS G12D GMPPNP*	6.8

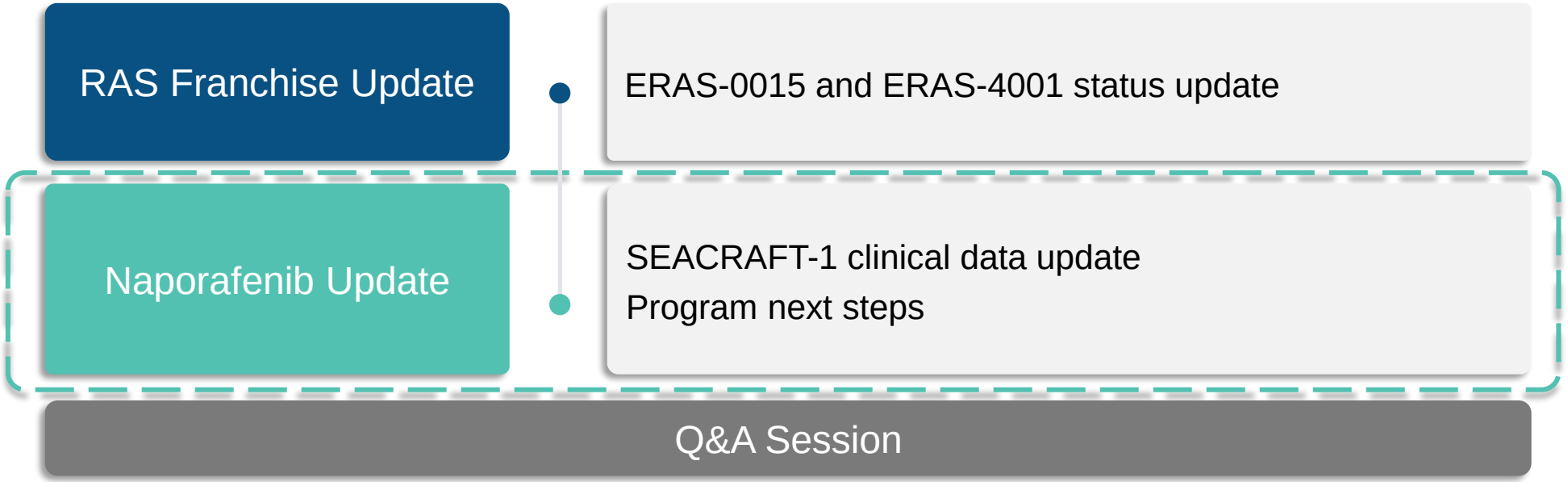
* GMPPNP is a nonhydrolyzable GTP analogue intended to approximate GTP-bound KRAS

ERAS-4001 showed tumor regression in multiple KRAS G12X in vivo models

Model	Study conducted by	Sensitivity to pan-KRAS inhibitors	ERAS-4001 Dose (mg/kg BID)	TGI (%)
PK-59 (KRAS G12D PDAC CDX)	WuXi AppTec	sensitive	30	101
			100	107
HPAC (KRAS G12D PDAC CDX)	ERASCA	sensitive	50	114
LU-01-1381 (KRAS G12D NSCLC PDX)	WuXi AppTec	sensitive	30	108
RKN (KRAS G12V Ovarian CDX)	WuXi AppTec	sensitive	30	117
NCI-H727 (KRAS G12V NSCLC CDX)	WuXi AppTec	insensitive	30	30
			100	77
			300	101
NCI-H727 (KRAS G12V NSCLC CDX)	ERASCA	insensitive	30	41
			100	76
			300	97

CDX: cell-line derived xenograft; PDAC: pancreatic ductal adenocarcinoma; NSCLC: non-small cell lung cancer

Erasca Investor Presentation Agenda



Two-pronged naporafenib development approach in patients with RAS/MAPK-driven tumors

SEACRAFT-1 (SC-1): RAS Q61X Solid Tumors

- RAS Q61X mutation commonly found in melanoma, lung, thyroid, and GI malignancies
- High unmet need with limited targeted therapy options
- Preliminary Phase 1b data for naporafenib + trametinib in Q4 2024

SEACRAFT-2 (SC-2): NRASm Melanoma

- Potential for approval in US and Europe based on high unmet need and regulatory alignment
- Compelling Ph 1 and 2 POC data generated
- Phase 3 Stage 1 data for naporafenib + trametinib anticipated in 2025

GI: gastrointestinal; NRASm = NRAS mutant; POC = proof-of-concept

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Naporafenib development scenarios based on SEACRAFT-1 data

SEACRAFT-1 (SC-1):
RAS Q61X Solid Tumors

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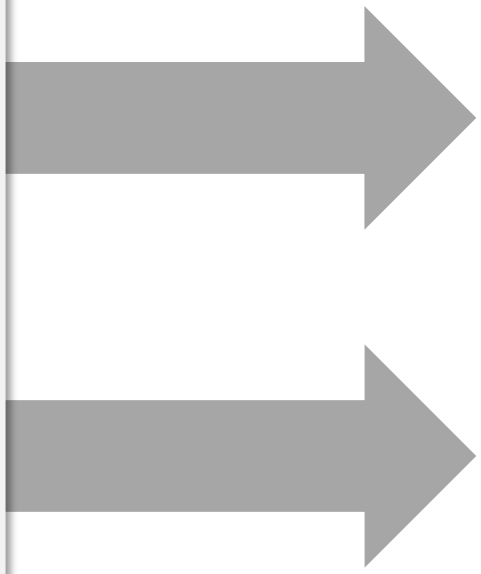

Tissue-agnostic indication

Tissue-specific indication(s)

GI: gastrointestinal; POC: proof-of-concept

Naporafenib development scenarios based on SEACRAFT-1 data

SEACRAFT-1 (SC-1):
RAS Q61X Solid Tumors

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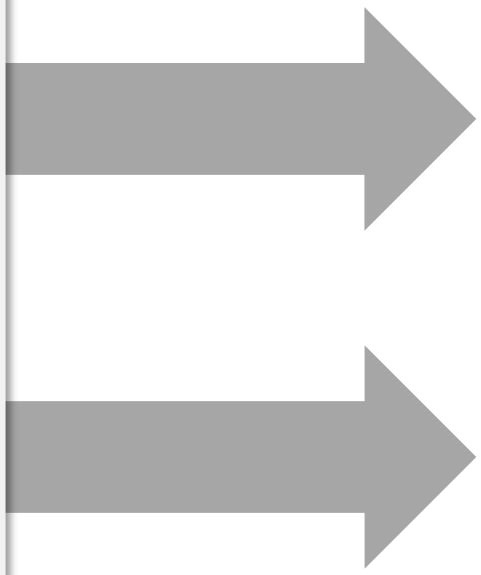


Tissue-agnostic indication

Tissue-specific indication(s)

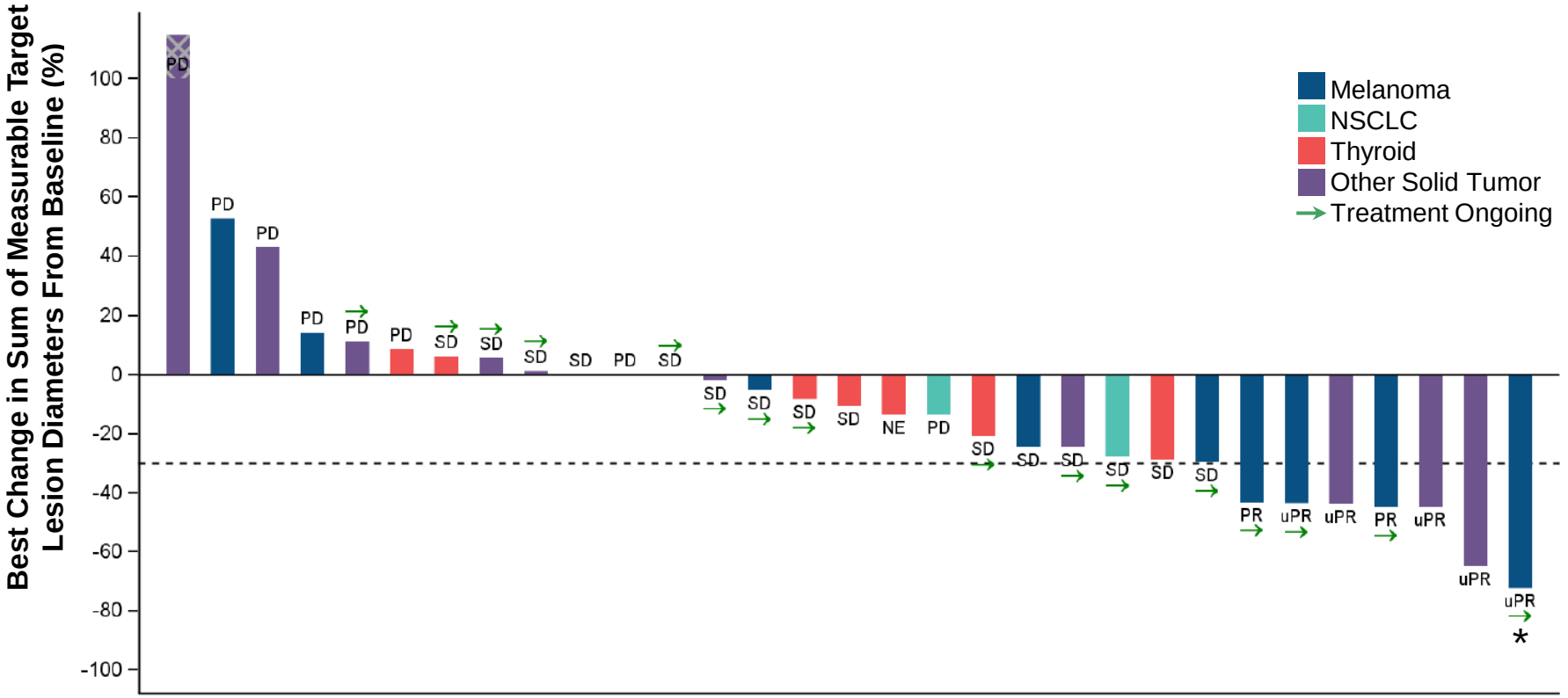
GI: gastrointestinal; POC: proof-of-concept

High hurdle for pursuing a tissue-agnostic indication

Based on encouraging preclinical and early phase clinical data, SEACRAFT-1 was designed to explore whether pursuing a RAS Q61X tissue-agnostic indication was feasible

Limited efficacy observed across multiple non-melanoma tumor types in SEACRAFT-1 does not support pursuing tissue-agnostic indication for naporafenib + trametinib

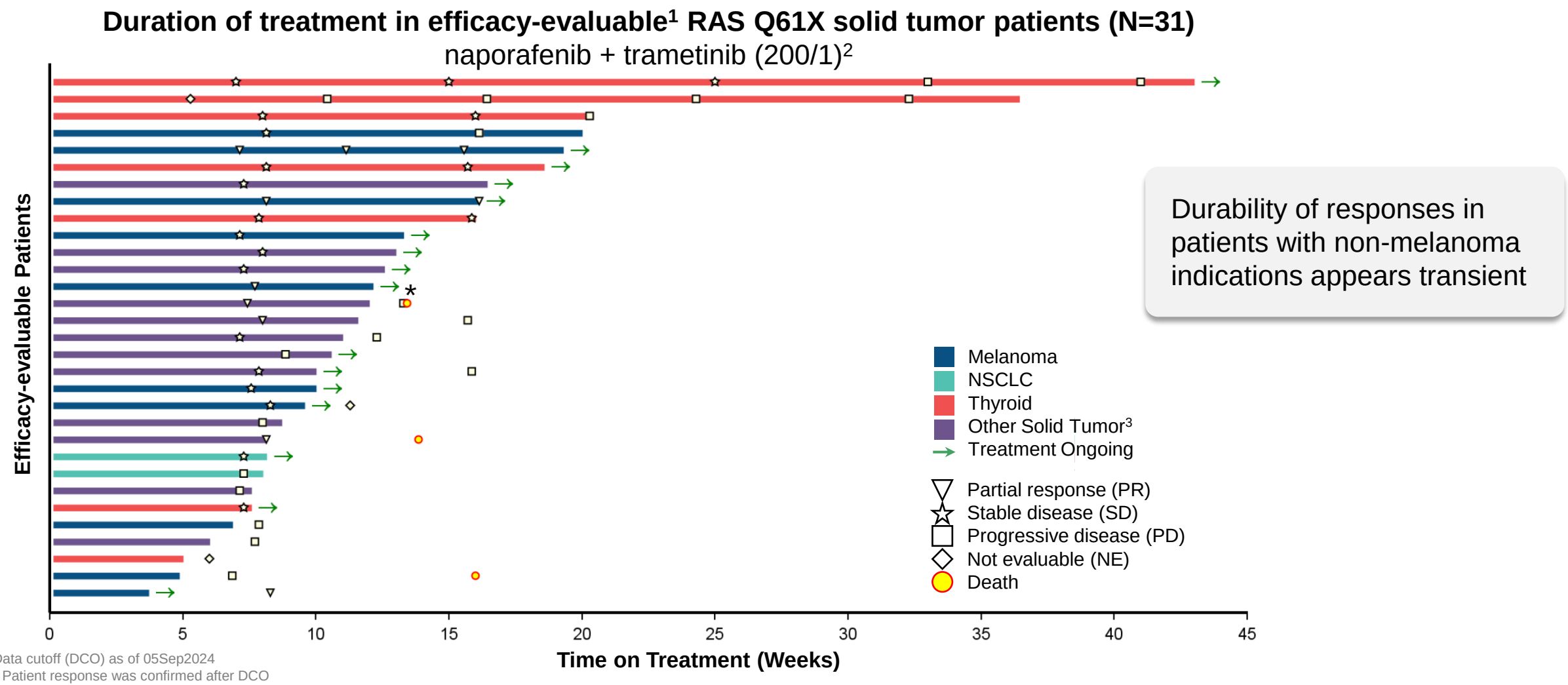
Efficacy-evaluable[1] RAS Q61X solid tumor patients (N=31)
naporafenib + trametinib (200/1)[2]



- **23%** (7/31) response rate (3 confirmed PRs, 4 uPRs[3])
- **71%** (22/31) disease control rate[4]
- Best response observed in CRC (n=10) and PDAC (n=11) was SD (data not shown)

Data cutoff (DCO) as of 05Sep2024
* Patient response was confirmed after DCO
[1] Defined as patients who received at least one dose of study drug, had measurable disease at baseline per RECIST, and had at least one post-baseline response assessment; CRC and PDAC patients excluded
[2] naporafenib 200 mg BID + trametinib 1 mg QD (BID: twice a day; QD: once a day)
[3] Melanoma patient with uPR continuing study treatment with next scan pending; Patients with other solid tumors will remain unconfirmed
[4] Disease control rate (DCR) = CR + PR + SD; uPR is included
PR: partial response; uPR: unconfirmed partial response; PD: progressive disease: SD: stable disease; NE: not evaluable; NSCLC: non-small cell lung cancer; CRC: colorectal cancer; PDAC: pancreatic ductal adenocarcinoma

Durability of response in SEACRAFT-1 has been limited in non-melanoma patients

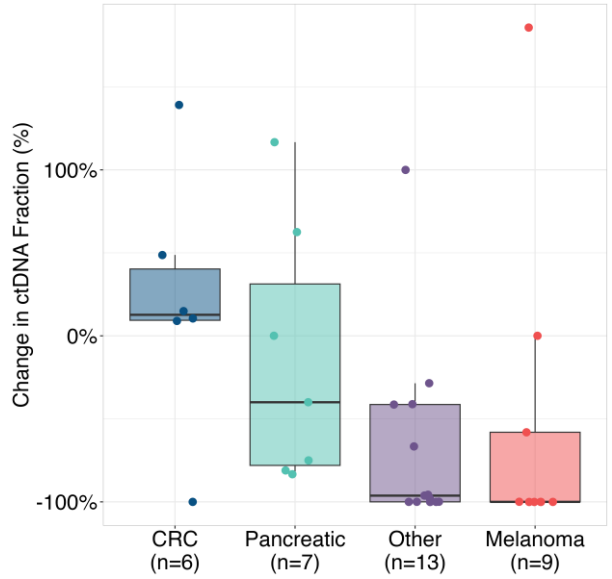


Durability of responses in patients with non-melanoma indications appears transient

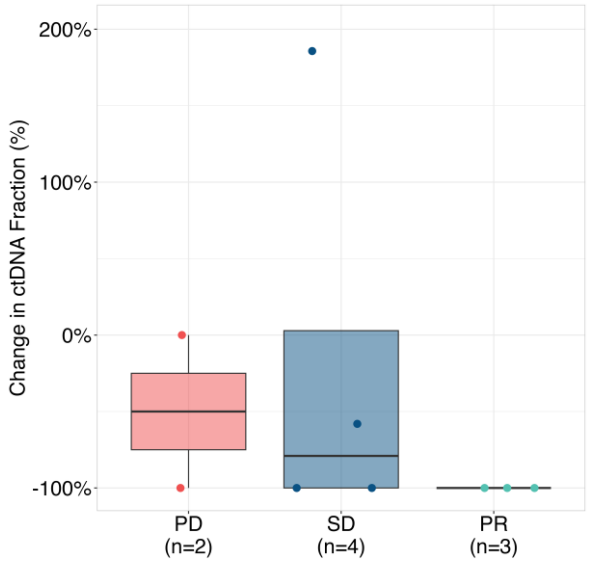
Data cutoff (DCO) as of 05Sep2024
* Patient response was confirmed after DCO
[1] Defined as patients who received at least one dose of study drug, had measurable disease at baseline per RECIST, and had at least one post-baseline response assessment; CRC and PDAC patients excluded
[2] naporafenib 200 mg BID + trametinib 1 mg QD (BID: twice a day; QD: once a day)
[3] Includes 11 tissue types, each for which fewer than four patients' data were available
NSCLC: non-small cell lung cancer

Decrease in ctDNA correlates with RECIST response, highlighting responsiveness of melanoma vs. other tumor types

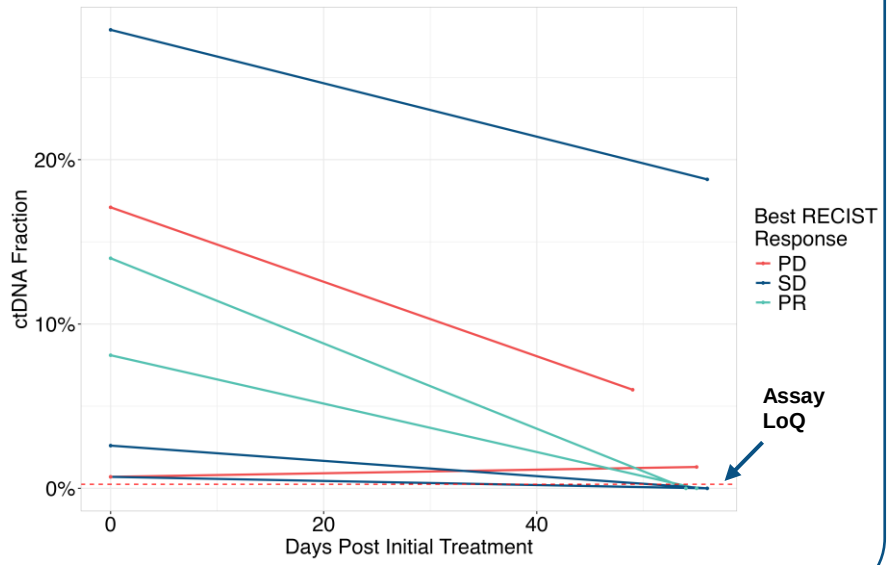
Change in ctDNA across tumor types



Change in ctDNA in melanoma by best response



Patient-specific ctDNA change in NRAS melanoma



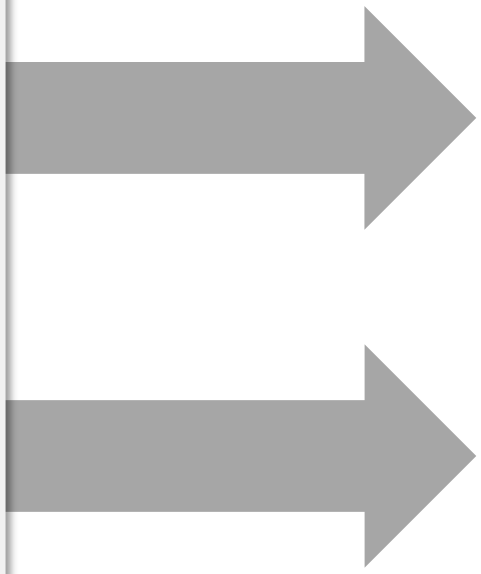
- Across all tumor types, ctDNA was undetectable at the majority of timepoints where tumor shrinkage was observed by imaging
- Greatest decreases in ctDNA at C2D1 were observed in melanoma relative to other solid tumors
- In NRAS Q61X melanoma patients, PRs and SDs correlated with undetectable ctDNA at tumor imaging timepoints

Note: 1 of 10 efficacy evaluable patients not evaluated for ctDNA
ctDNA: circulating tumor DNA; LoQ: limit of quantification; PD: progressive disease; PR: partial response; SD: stable disease

Naporafenib development scenarios based on SEACRAFT-1 data

SEACRAFT-1 (SC-1):
RAS Q61X Solid Tumors

- RAS Q61X mutation commonly found in melanoma, lung, thyroid, and GI malignancies
- High unmet need with limited targeted therapy options
- Preliminary Phase 1b data for naporafenib + trametinib in Q4 2024

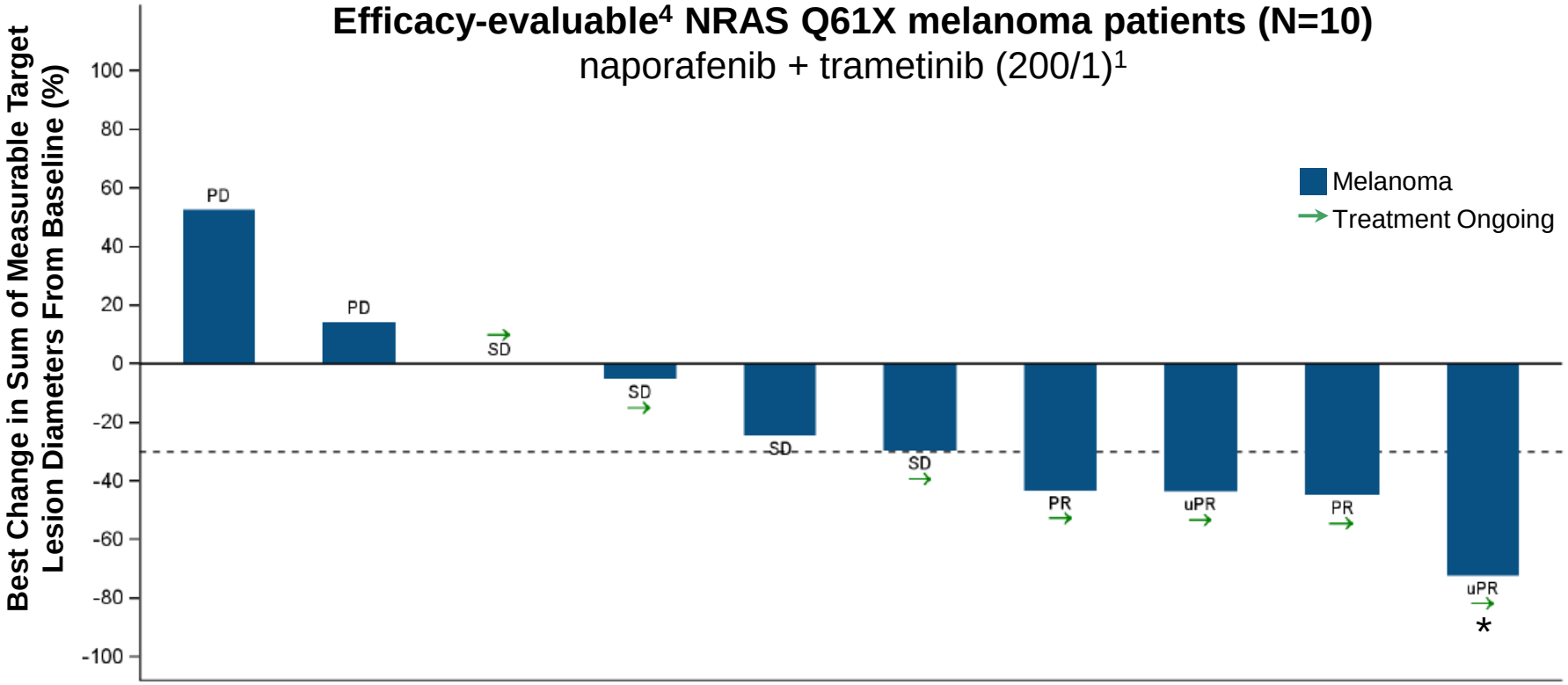


Tissue-agnostic indication

Tissue-specific indication(s)

GI: gastrointestinal; POC: proof-of-concept

Positive preliminary efficacy observed in SEACRAFT-1 melanoma cohort bolsters rationale for pursuing tissue-specific NRASm melanoma indication in SEACRAFT-2



40% (4/10) response rate (3 confirmed PRs, 1 uPR[2])

80% (8/10) disease control rate[3]

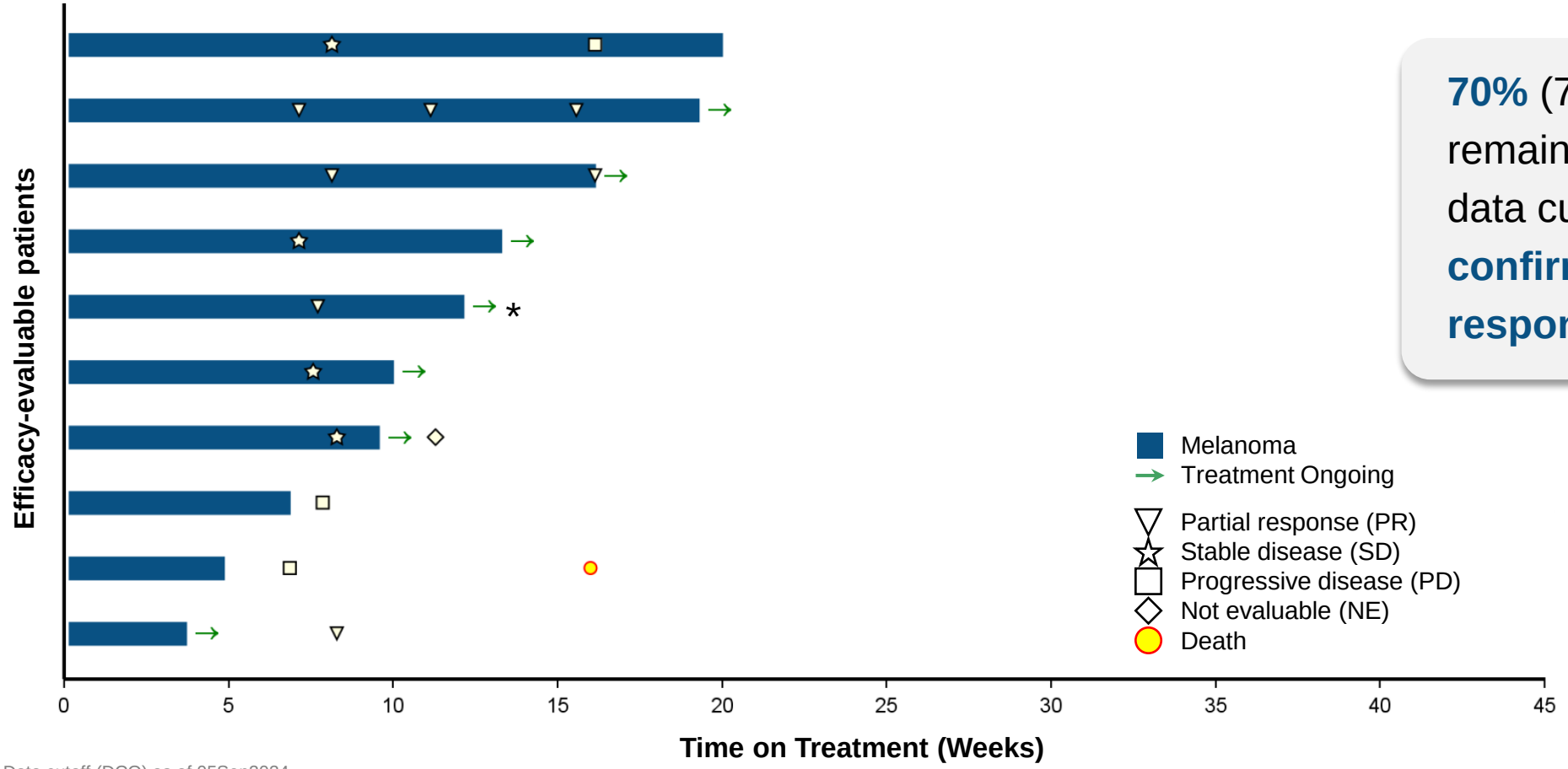
Response observed in patient with mucosal melanoma, a population that had not been enrolled in previous studies

Data cutoff (DCO) as of 05Sep2024
* Patient response was confirmed after DCO
[1] naporafenib 200 mg BID + trametinib 1 mg QD (BID: twice a day; QD: once a day)
[2] Melanoma patient with uPR continuing study treatment with next scan pending
[3] Disease control rate (DCR) = CR + PR + SD; uPR is included
[4] Defined as patients who received at least one dose of study drug, had measurable disease at baseline per RECIST, and had at least one post-baseline response assessment
NRASm: NRAS mutant; PR: partial response; uPR: unconfirmed partial response; PD: progressive disease: SD: stable disease

Early but encouraging durability observed in SEACRAFT-1 melanoma cohort

Duration of treatment in efficacy-evaluable[2] NRAS Q61X melanoma patients (N=10)

naporafenib + trametinib (200/1)[1]



70% (7/10) of patients remained on treatment as of data cutoff, including **all confirmed and unconfirmed responders**

Data cutoff (DCO) as of 05Sep2024

* Patient response was confirmed after DCO

[1] naporafenib 200 mg BID + trametinib 1 mg QD (BID: twice a day; QD: once a day)

[2] Defined as patients who received at least one dose of study drug, had measurable disease at baseline per RECIST, and had at least one post-baseline response assessment

Napo + tram generally well tolerated in SEACRAFT-1, primarily Grade 1/2 TRAEs

Treatment-related Adverse Events Reported in ≥ 10% of All Patients

(arranged by descending frequency in the All Grades column)

	Naporafenib + Trametinib (200/1)[1] (N = 52)	
Preferred Term	**All Grades, n (%)**	**Grade ≥ 3, n (%)**
Rash	21 (40.4)	3 (5.8)
Dermatitis acneiform	11 (21.2)	1 (1.9)
Constipation	9 (17.3)	0
Diarrhea	8 (15.4)	2 (3.8)
Stomatitis	8 (15.4)	2 (3.8)
Fatigue	8 (15.4)	1 (1.9)
Rash maculo-popular	7 (13.5)	2 (3.8)
Nausea	7 (13.5)	1 (1.9)
AST increased	7 (13.5)	1 (1.9)
Pruritis	6 (11.5)	0

Data cutoff (DCO) as of 03Sep2024

[1] naporafenib 200 mg BID + trametinib 1 mg QD (BID: twice a day; QD: once a day)

TRAE: treatment-related adverse events; AST: aspartate aminotransferase

Mandatory primary rash prophylaxis substantially decreased incidence and severity of dermatologic toxicities

Dermatologic Treatment-emergent Adverse Events

naporafenib 200 mg BID + trametinib 1 mg QD

	Phase 1*	Phase 2*	SEACRAFT-1
	N = 54	N = 30	N = 52
Dermatologic[1] toxicities, n (%)	49 (90.7)	26 (86.7)	38 (73.1)
Grade ≥3 dermatologic toxicities	20 (37.0)	11 (36.7)	6 (11.5)
Dermatitis acneiform, n (%)	17 (31.5)	9 (30.0)	11 (21.2)
Grade ≥3 dermatitis acneiform	4 (7.4)	1 (3.3)	1 (1.9)
Rash, n (%)	23 (42.6)	11 (36.7)	22 (42.3)
Grade ≥3 rash	9 (16.7)	4 (13.3)	3 (5.8)

*Novartis introduced primary prophylaxis into both trials by amendment late in the enrollment period for each study
1 "Dermatologic" refers to a group of adverse events describing a variety of dermatologic toxicities
Data cutoff dates: SEACRAFT-1 03Sep2024; Ph 1 (CLXH254X2102) 04Aug2022; Ph 2 (CLXH254C12201) 30Dec2022
BID: twice a day; QD: once a day

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Mandatory primary rash prophylaxis led to a clinically significant decrease in drug discontinuations due to AEs and improved relative dose intensity

Discontinuations due to adverse events
naporafenib 200 mg BID + trametinib 1 mg QD

Naporafenib / trametinib	Phase 1*	Phase 2*	SEACRAFT-1
	N = 54	N = 30	N = 52
Permanent discon., n (%)	10 (18.5)	6 (20.0)	5 (9.6)
Permanent discon. due to skin tox TEAE, n	5/10	5/6	0/5

Relative Dose Intensity (RDI)
naporafenib 200 mg BID + trametinib 1 mg QD

Naporafenib / trametinib	Phase 1*	Phase 2*	SEACRAFT-1
	N = 54	N = 30	N = 51[1]
Median RDI, %	66.3 / 59.2	57.5 / 62.4	98.5 / 100

*Novartis introduced primary prophylaxis into both trials by amendment late in the enrollment period for each study
Data cutoff dates: SEACRAFT-1 03Sep2024; Ph 1 (CLXH254X2102) 04Aug2022; Ph 2 (CLXH254C12201) 30Dec2022
[1] Dosing data pending for one patient
AE: adverse event; discon: discontinuation; BID: twice a day; QD: once a day

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Consistent, meaningful efficacy for napo + tram in NRASm melanoma

	MEKi		SOC	Pooled Ph 1 and Ph 2[4]	
	Binimetinib[1]	Trametinib[2]	Chemo[3]	Naporafenib + Trametinib	
	45mg	2mg	1g/m² IV	200mg+1mg	400mg+0.5mg
	N=269	N=33	N=133	N=39	N=32
ORR %	15%	15%	7%	31%	22%
mPFS months	2.8	~*2.8**	1.5	**5.1**	**4.9**
mOS months	~10-11 months (Benchmark #1: *NEMO Study*)			**~13 months**	**~14 months**
	~7 months *(Benchmark #2: Chart Review)*				
	~7 months *(Benchmark #3: C12201 BRAFm Patients[5])*				

Benchmarks most like SEACRAFT-2 patient population

Prelim. **SEACRAFT-1 response rate of 40%** in NRASm melanoma further supports potential in SC-2

Potential win on both SEACRAFT-2 primary endpoints (PFS and OS)

1 Dummer et al 2017; binimetinib is administered BID
2 Pooled analysis from the following publications: Falchook et al, 2012; Pigne et al, 2023; Salzmann et al, 2022; trametinib is administered QD
3 Dacarbazine is the approved chemotherapy in this indication
4 Ph 1 = CLXH254X2102 with DCO 4 Aug 2022; Ph 2 = CLXH254C12201 with DCO 30 Dec 2022
5 BRAF/MEK inhibitor-resistant BRAFm melanoma patients in Novartis's Phase 2 trial
*Assumes trametinib efficacy is similar to published binimetinib efficacy results
SOC: standard of care; mPFS: median progression free survival; mOS: median overall survival; SC-1: SEACRAFT-1; SC-2: SEACRAFT-2; NRASm: NRAS mutant
The pooled phase 1 and phase 2 napo + tram data covers two clinical trials with different designs and inclusion criteria, which cannot be directly compared, and therefore may not be a reliable indicator of efficacy data
Due to differences between trial designs and subject characteristics, comparing data across different trials may not be a reliable indicator of data

ERASCA

Data bolster strong potential for naporafenib and RAS-targeting franchise

Conviction on SEACRAFT-2 Phase 3

Promising SC-1 melanoma data support development in SC-2 tissue-specific path

Improved tolerability observed following mandatory primary rash prophylaxis (e.g., TRAEs, discontinuations, RDI)

High investigator enthusiasm; site activation ahead of schedule

Conviction on RAS-targeting franchise

In-life portion of tox studies tracking as anticipated: ERAS-0015 completed, ERAS-4001 near completion

Reproduced favorable activity and generated new supporting data

Nonclinical data continue to bolster potential differentiation in multiple models

SC-1: SEACRAFT-1; SC-2: SEACRAFT-2; TRAEs: treatment-related adverse events; RDI: relative dose intensity

Deep modality-agnostic RAS/MAPK pathway-focused pipeline

Program	Target	Modality	Indication	Discovery	IND-enabling	Phase 1	Phase 2	Phase 3	Worldwide Rights
Naporafenib	BRAF/CRAF		RAS/MAPK solid tumors			SEACRAFT-1			ERASCA
			NRASm melanoma				SEACRAFT-2		ERASCA
ERAS-0015	RAS		RASm solid tumors			AURORAS-1 (planned)			ERASCA JOYO[1]
ERAS-4001	KRAS		KRASm solid tumors			BOREALIS-1 (planned)			ERASCA
ERAS-12	EGFR D2/D3		EGFR & RAS/MAPK solid tumors						ERASCA

small molecule large molecule

Note: Pipeline also includes ERAS-801 brain-penetrant EGFR inhibitor for EGFR-altered GBM (for which we are concluding a Phase 1 trial and exploring advancement via investigator-sponsored trials), ERAS-007 ERK1/2 inhibitor, and ERAS-601 SHP2 inhibitor. ERAS-007 and ERAS-601 are being assessed in preclinical studies as potential combination partners with other programs in our pipeline for RAS/MAPK pathway inhibition. Via Erasca Ventures, we made an equity investment into Affini-T Therapeutics, which is developing TCR T-cell therapies against KRAS G12V, KRAS G12D, and KRAS G12C.

[1] Licensor Joyo Pharmatech, Ltd., retains rights to People's Republic of China, Hong Kong and Macau, subject to Erasca's option to convert our territory to worldwide

Anticipated key milestones and clinical trial readouts

Program *Mechanism*	**Trial Name** *Indication* *(Combo partner if applicable)*	**Anticipated Milestone**
Naporafenib *Pan-RAF inhibitor*	**SEACRAFT-2** *NRASm Melanoma* *(+ trametinib)*	• **2025:** Ph 3 stage 1 randomized dose optimization data[1]
ERAS-0015 *Pan-RAS molecular glue*	**AURORAS-1** *RASm solid tumors*	• **H1 2025:** IND filing[2] • **2026:** Ph 1 monotherapy data[3]
ERAS-4001 *Pan-KRAS inhibitor*	**BOREALIS-1** *KRASm solid tumors*	• **Q1 2025:** IND filing • **2026:** Ph 1 monotherapy data[3]

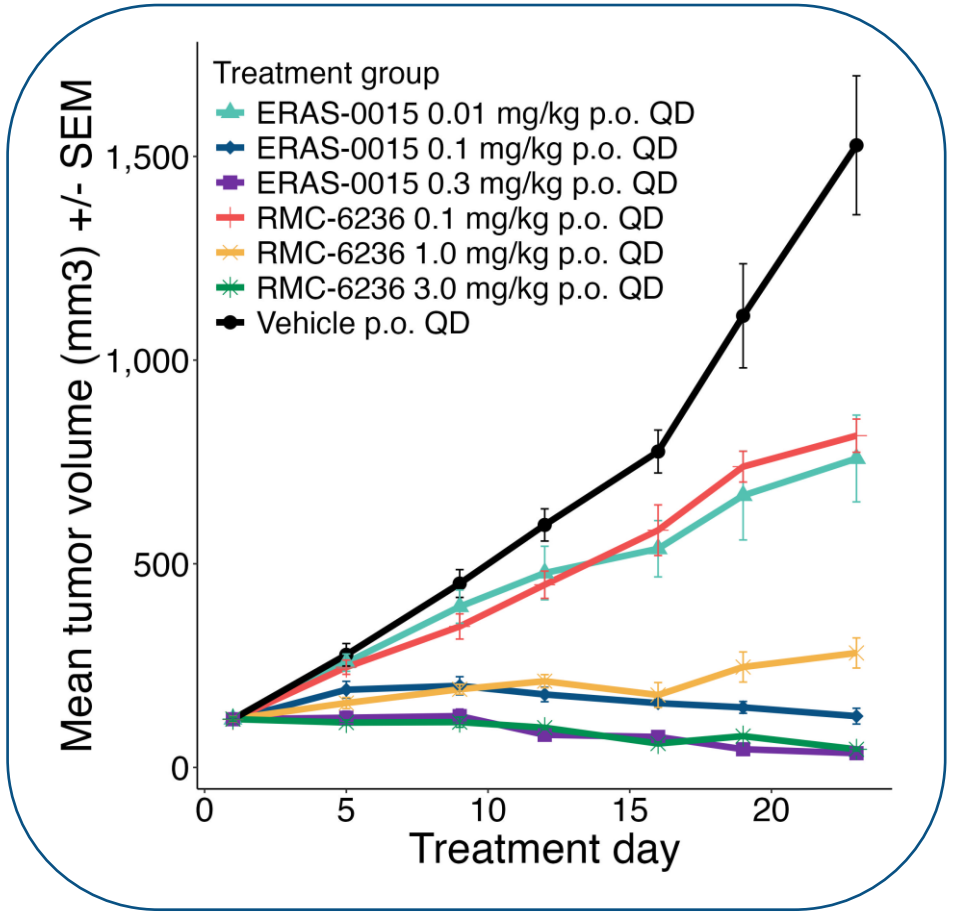
$460M in cash, cash equivalents, and marketable securities[4]; cash runway into **H1 2027**

[1] Data to include safety, pharmacokinetics (PK), and efficacy at relevant dose(s) in relevant population(s) of interest
[2] Timing of IND is subject to adjustment pending detailed program planning, driven predominantly by CMC timelines
[3] Subject to change pending detailed program planning, but assuming target US IND filing timing is achieved, data to include safety, PK, and efficacy at relevant dose(s) in relevant population(s) of interest
[4] Unaudited, as of June 30, 2024

ERASCA


ERASCA
THANK YOU!

ERAS-0015: Comparable TGI at 1/10th the dose of RMC-6236; achieved tumor regression in the KRAS G12D PDAC CDX model PK-59

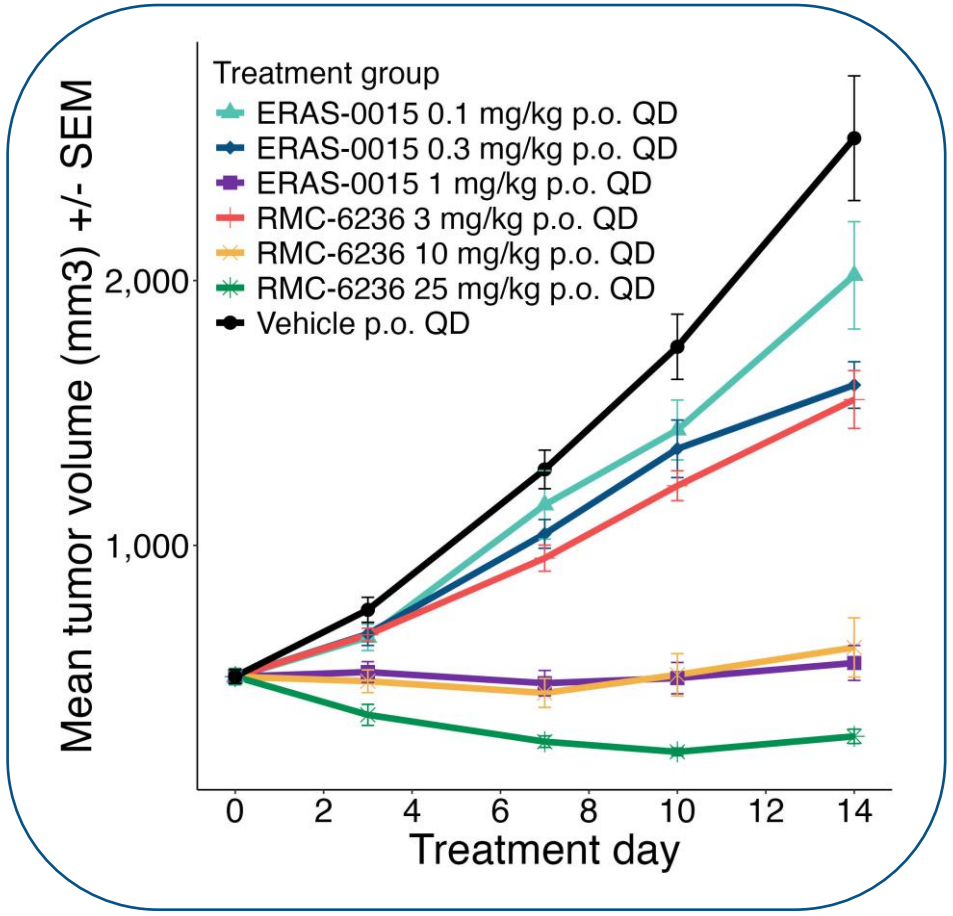


ERAS-0015 was well tolerated at all doses

Compound	Dose (mg/kg QD)	TGI on Day 23
ERAS-0015	0.3	106%
RMC-6236	3	105%

TGI: tumor growth inhibition; PDAC: pancreatic ductal adenocarcinoma; CDX: cell-line derived xenograft; p.o.: orally administered; QD: once a day

ERAS-0015: Comparable TGI at 1/10th the dose of RMC-6236; achieved tumor regression in the KRAS G12R PDAC CDX model PSN-1

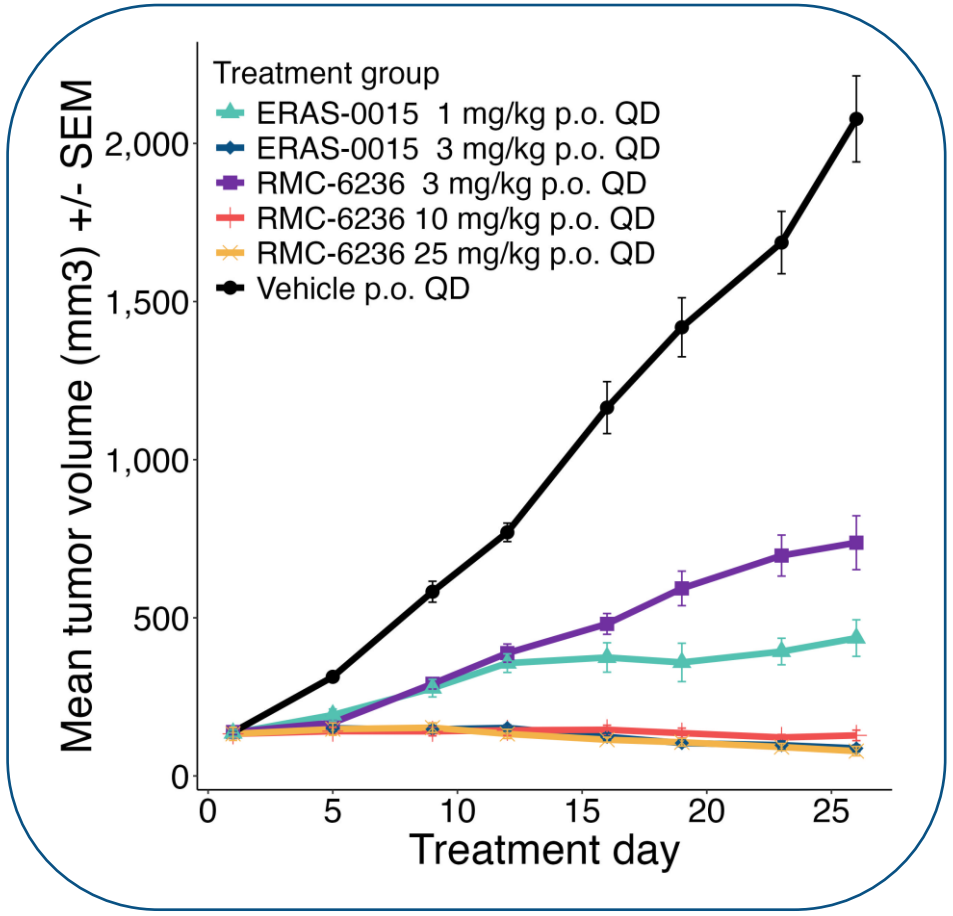


Compound	Dose (mg/kg QD)	TGI on Day 14
ERAS-0015	1	97%
RMC-6236	10	95%

ERAS-0015 was well tolerated at all doses

TGI: tumor growth inhibition; PDAC: pancreatic ductal adenocarcinoma; CDX: cell-line derived xenograft; p.o.: orally administered; QD: once a day

ERAS-0015: Comparable TGI at ~1/8th the dose of RMC-6236; achieved tumor regression in the KRAS G12V CRC CDX model SW620

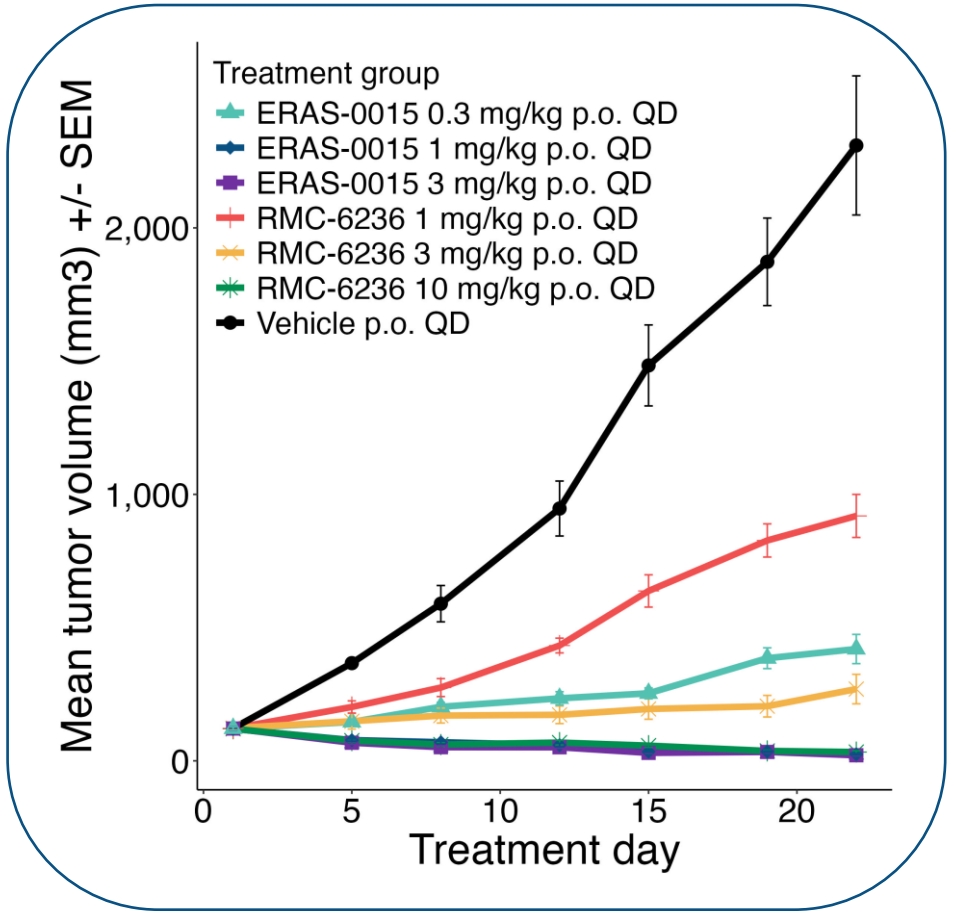


ERAS-0015 was well tolerated at all doses

Compound	Dose (mg/kg QD)	TGI on Day 26
ERAS-0015	3	102%
RMC-6236	25	103%

TGI: tumor growth inhibition; CRC: colorectal cancer; CDX: cell-line derived xenograft; p.o.: orally administered; QD: once a day

ERAS-0015: Comparable TGI at 1/10th the dose of RMC-6236; achieved tumor regression in the KRAS G12V NSCLC CDX model NCI-H727



ERAS-0015 was well tolerated at all doses

Compound	Dose (mg/kg QD)	TGI on Day 22
ERAS-0015	1	104%
RMC-6236	10	104%

TGI: tumor growth inhibition; NSCLC: non-small cell lung cancer; CDX: cell-line derived xenograft; p.o.: orally administered; QD: once a day