ERASCA

On a Journey to Erase Cancer

Erasca Corporate Presentation

May 2026

Eric and his wife Margaret, inspiring our bold mission to erase cancer

Disclaimer: Forward Looking Statements & Market and Clinical Data

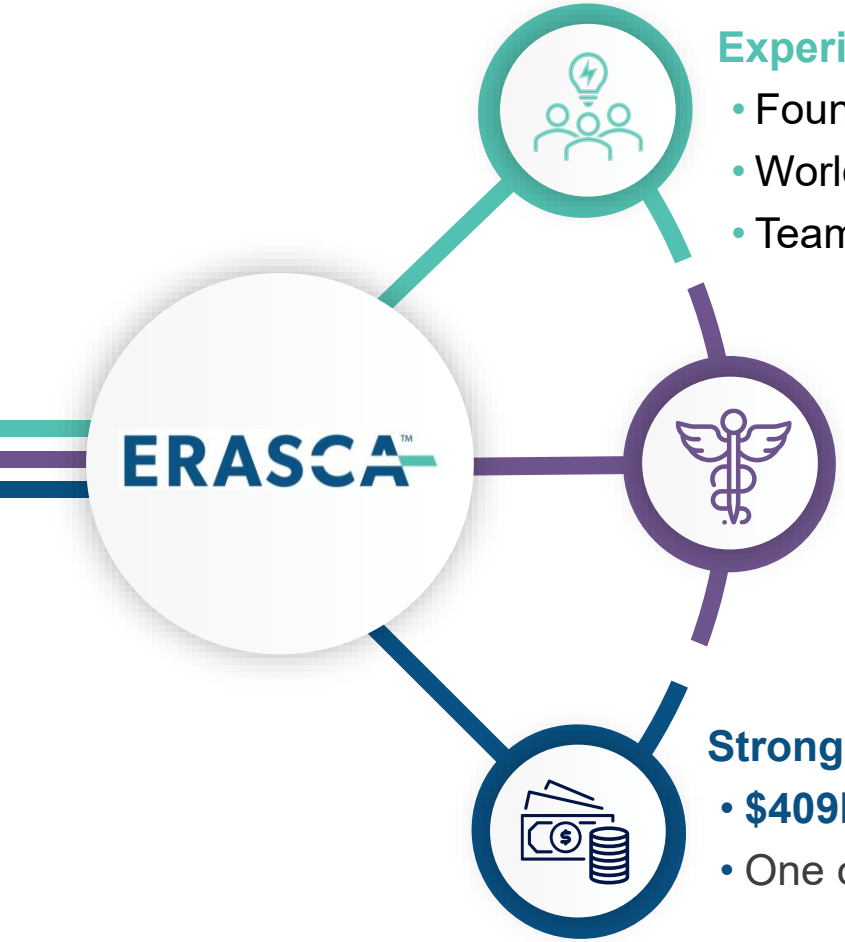
We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation are forward-looking statements, including statements regarding: our future results of operations and financial position, business strategy, research and development plans; the anticipated timing (including the timing of initiation and the timing of data readouts), costs, design and conduct of our ongoing and planned preclinical studies and clinical trials for our product candidates; the potential therapeutic benefits and potential patient population for each of our product candidates; the potential for ERAS-0015 to be best-in-class or serve as backbone therapy for future combination therapies; characterizations of the clinical profile of our product candidates and any comparisons against other products or product candidates in development; our intellectual property protection; and future results of anticipated product development efforts, including anticipated milestones. 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: the timing of our clinical data readouts, including for the AURORAS-1 and BOREALIS-1 trials may be delayed; our product candidates, including ERAS-0015 and ERAS-4001, may not demonstrate therapeutic benefits that we expect; this presentation includes clinical data generated by our third-party licensor, and such data are presented as received and have not been independently verified by us; topline and preliminary results of a clinical trial are not necessarily indicative of final results and one or more of the clinical outcomes may materially change as patient enrollment continues, following more comprehensive reviews of the data and as more patient data becomes available, including the risk that an unconfirmed partial response to treatment may not ultimately result in a confirmed partial response to treatment after follow-up evaluations; our observations, including those regarding the first dosage level at which a clinical response is detected and the efficacy and safety of ERAS-0015 compared to other products and product candidates (including our internal benchmark RMC-6236), are based on data generated within individual clinical trials, and comparisons of such clinical observations across different trials involve data from separate trials with distinct designs, patient populations, and methodologies, and therefore may not be directly comparable; any forward-looking statements regarding dose-response relationships reflect current expectations and/or assumptions are subject to risks and uncertainties that could cause actual results to differ materially; 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 two product candidates in clinical development and all of our other development efforts are in the preclinical stage; 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; 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; 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, 2025, 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.

Cross-Study Comparisons: The data presented for the CN and US trials in this presentation are based on separate studies and pooled and comparative data across such studies. Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from such data across separate studies as such pooling and comparative data is inherently limited and such data may not be directly comparable. The clinical data presented in this presentation comparing ERAS-0015 and other products and product candidates (including RMC-6236) are based on cross-study comparisons and are not based on any head-to-head clinical trials. Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable.

Our name is our mission: to erase cancer

Vision to one day erase cancer[1] in at least 100,000 patients annually as a leading global oncology company



Experienced leadership team and SAB with track record of serial successes

- Founded by **Jonathan Lim, MD & Kevan Shokat, PhD** around disruptive idea to target RAS
- World class scientific advisory board of **leading pioneers** in RAS/MAPK pathway
- Team with deep experience in efficient planning and execution of **global development**

Differentiated pipeline focused on shutting down the RAS/MAPK pathway

- **ERAS-0015 Pan-RAS molecular glue** with BIC potential in RASm solid tumors
- **ERAS-4001 Pan-KRASi** with FIC potential in KRASm solid tumors

Strong financial position with high quality investor base and industry visibility

- **$409M in cash, cash equivalents, and marketable securities[2]**; cash runway into **H2 2028**
- One of **Fierce Biotech's 2021 "Fierce 15"** most promising biotechnology companies

[1] Number of patients alive and free of cancer or free from cancer progression 2 yrs after starting an Erasca regimen, as measured by disease-free survival (adjuvant setting) and progression-free survival (metastatic setting)
[2] Unaudited, as of March 31, 2026
FIC: first-in-class; BIC: best-in-class; RASm: RAS mutated; KRASm: KRAS mutated

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SAB includes world's leading experts in the RAS/MAPK pathway

Kevan Shokat, PhD

Erasca co-founder. World expert in RAS who pioneered development of approaches to inhibit KRAS G12C (RAS-GDP) and active states of RAS (RAS-GTP)

UCSF University of California San Francisco

Michael Varney, PhD

Erasca Chair of R&D. World expert in structure-based drug design; former head of research at Agouron and former head of Genentech's Research and Early Development (gRED)

Genentech

René Bernards, PhD

World expert in functional cancer genetics and identifying new drug combinations based on genome-wide genetic approaches

Netherlands Cancer Institute Antoni van Leeuwenhoek

Stephen Blacklow MD, PhD

World expert in SHP2 who helped pioneer development of the first SHP2 inhibitor with Novartis

Harvard Medical School · Dana-Farber Cancer Institute

Karen Cichowski, PhD

World expert in RAS/MAPK pathway signaling and identifying novel combination therapies to shut it down

Harvard Medical School · Brigham and Women's Hospital

Ryan Corcoran, MD, PhD

World expert in ERK, having studied nearly every ERK inhibitor that has been or is being developed, as well as targeted therapies directed against KRAS, BRAF, and MEK mutations

Harvard Medical School · Massachusetts General Hospital Cancer Center

George Demetri, MD

World expert in targeted oncology therapies who pioneered the development of Gleevec®, which helped launched the precision oncology revolution

Harvard Medical School · Dana-Farber Cancer Institute

Piro Lito, MD, PhD

World expert in KRAS-targeted therapeutics and precision oncology, with a focus on resistance mechanisms to RAS inhibitors

Memorial Sloan-Kettering Cancer Center — The Best Cancer Care. Anywhere.

Pablo Rodriguez-Viciana, PhD

World expert in RAS/MAPK pathway with focus on the SHOC2 phosphatase complex as a unique regulatory node required for efficient pathway activation in the context of diseases such as cancer and RASopathies

UCL Cancer Institute

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Focused modality-agnostic RAS/MAPK pipeline

Program	Target	Modality	Indication	Discovery	IND-enabling	Phase 1	Phase 2	Phase 3	Worldwide Rights
ERAS-0015	RAS		RASm solid tumors	AURORAS-1					ERASCA
ERAS-4001	KRAS		KRASm solid tumors	BOREALIS-1					ERASCA
ERAS-12	EGFR D2/D3		EGFR & RAS/MAPK solid tumors						ERASCA

small molecule small molecule molecular glue large molecule

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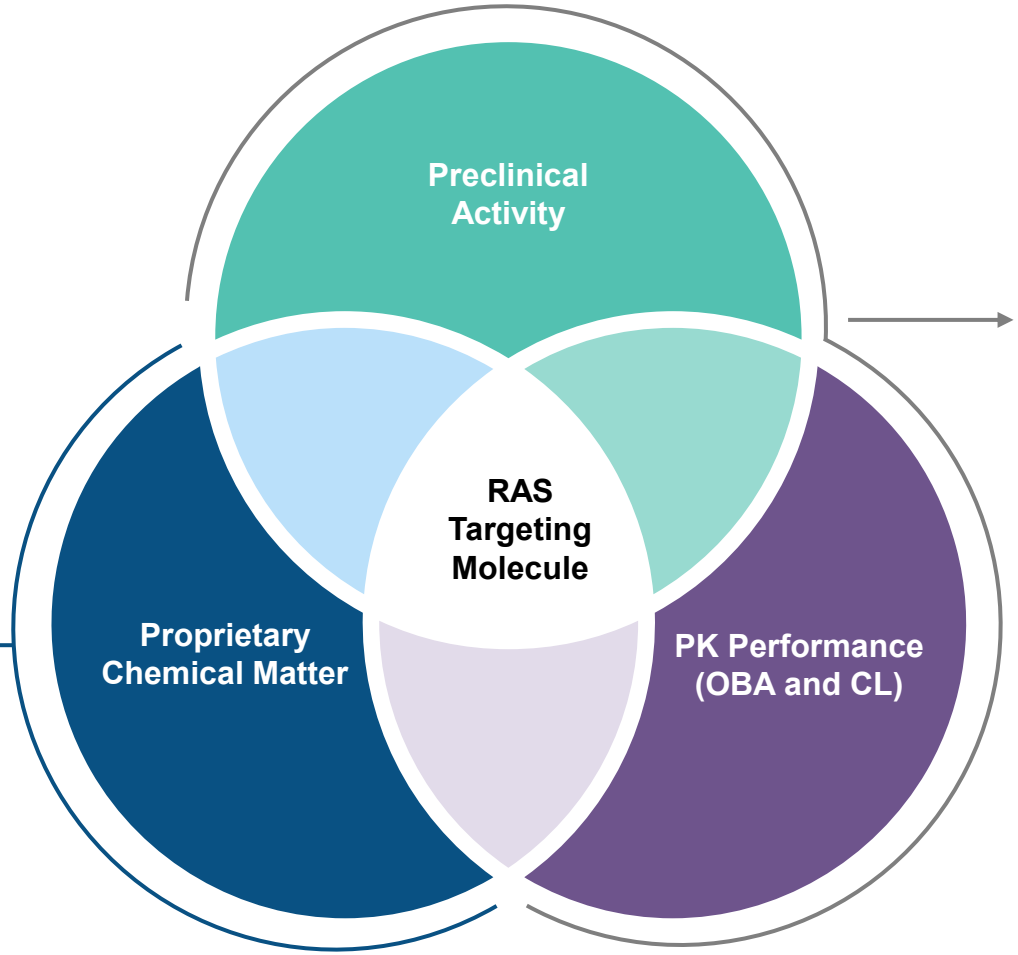
Ideal RAS targeting molecules integrate three key attributes

Bolsters ability to **maximize clinical and commercial value** in highly competitive market



- **Lower clinically active dose** could translate to:
  - Lower risk of solubility-limited absorption and exposure plateau observed with the most advanced pan-RAS MG in development
  - Better GI tolerability profile due to lower drug load
  - Improved therapeutic window for any potential off-target toxicities
- KRAS G12Ci class has demonstrated how higher potency can translate into **improved clinical activity**

MG = molecular glue; OBA: oral bioavailability; CL: clearance

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

	Preclinical Activity[1]	OBA[2]	IP[3]
ERAS-0015 Pan-RAS Molecular Glue	• In vitro: **0.2 – 13.8 nM IC50** in KRAS G12D/V/C/X, G13D, WT; activity in H/NRAS • In vivo: Tumor regression in KRAS G12D/V/R CDX models at low doses between **0.3 – 5 mpk PO QD**	• **38 – 48%** in small animal species • **17 – 22%** in large animal species	• IP exclusivity expected through **2043** • US patent covering composition of matter issued in Oct. 2025
ERAS-4001 Pan-KRAS Inhibitor	• In vitro: **0.7 – 10.8 nM IC50** in KRAS G12D/V/C and KRAS WT; **5.8 – 56 nM IC50** in KRAS G12X and G13D; no activity in H/NRAS • In vivo: Tumor regression in KRAS G12D/V CDX models at doses between **30 - 300 mpk PO BID**	• **Up to 27%** in small animal species • **16%** in large animal species	• IP exclusivity expected through **2043** • US patent covering composition of matter issued in Feb. 2026

Potential BIC **Pan-RAS MG** for RASm solid tumors, which showed ~5x – 10x greater antitumor activity and favorable ADME properties and PK performance in animal species (vs. most advanced pan-RAS MG in development)[4]

Potential FIC/BIC **Pan-KRAS** or **"KRAS-selective" SMi** that spared H/NRAS WT, predicted to provide a wider therapeutic window (vs. Pan-RAS MG) for KRASm solid tumors and address KRASwt activation to prevent resistance (vs. mutant-selective inhibitors)

TPP: target product profile; OBA: oral bioavailability; IP: intellectual property; FIC: first-in-class; BIC: best-in-class; WT: wildtype; SMi: small molecule inhibitor; MG: molecular glue; [1] in vitro potency assessed by CTG 2D and 3D-cell proliferation assay IC50s; [2] OBA assessed by %F; [3] IP includes composition of matter, methods of use, and methods of making licensed compounds; date is absent any patent term adjustments or extensions, [4] These data were generated in head-to-head in vitro assay and/or in vivo experiments

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

ERAS-0015
Pan-RAS
Molecular Glue

Potential best-in-class Pan-RAS molecular glue

- ~5x – 10x greater antitumor activity vs. most advanced pan-RAS MG in development[1]
- Favorable ADME properties and PK performance in animals vs. most advanced pan-RAS MG in development[1]
- 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 Pan-KRAS inhibitor

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

[1] These data were generated in head-to-head in vitro assay and/or in vivo experiments
TPP = target product profile; MG: molecular glue; ADME: absorption, distribution, metabolism, and excretion; PK: pharmacokinetic; WT: wild type; KRASm: KRAS mutant

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ERAS-0015: Potential best-in-class pan-RAS molecular glue

ERAS-0015
Pan-RAS
Molecular Glue

ERAS-0015 has potential to become preferred RAS-targeting agent as monotherapy and a backbone for combo therapy

Preclinical Differentiation[1]

In Vivo Activity	Comparable anti-tumor activity at 1/10th-1/5th of RMC-6236 dose in multiple mouse models
PK Properties	• Higher oral bioavailability (improved %F) • Lower CL, longer $T_{1/2}$ • Preferential tumor distribution with longer residence time
Cellular Potency	Improved potency across RAS-altered cell lines
CYPA Binding	Improved binding affinity (8-21x)

[1]These data were generated in head-to-head assay and in vivo experiments
CYPA: cyclophilin A

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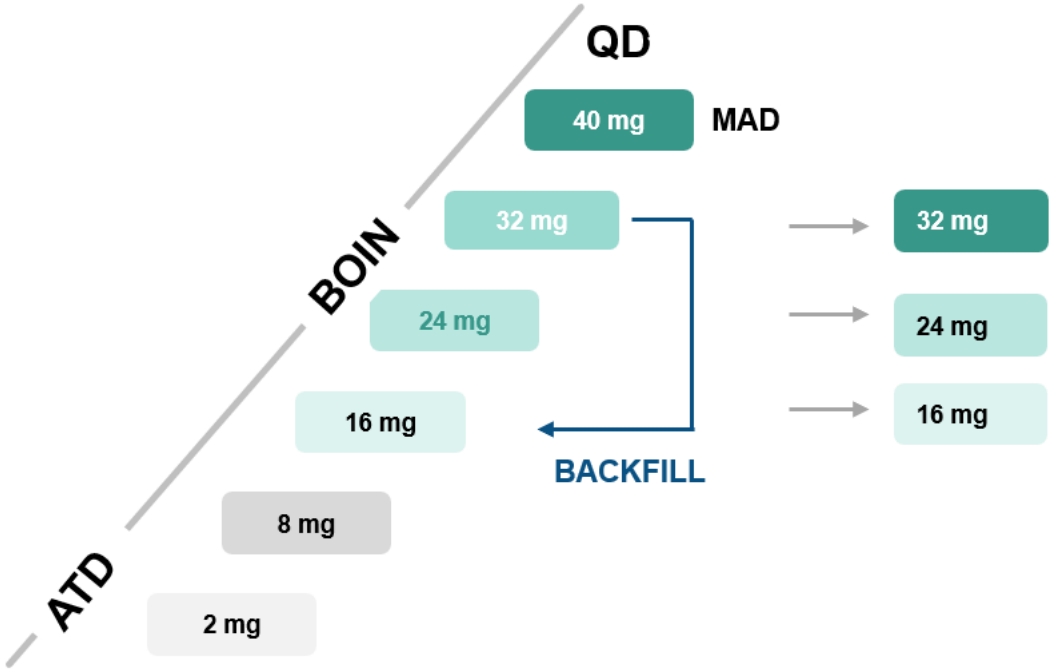
CN and US Trials: Similar trial designs support generalizability between them

China Trial (JYP0015M101) FPD to DCO ~10.5 months[1]

Phase Ia: Dose Escalation[3] **Phase Ib: Dose Expansion**



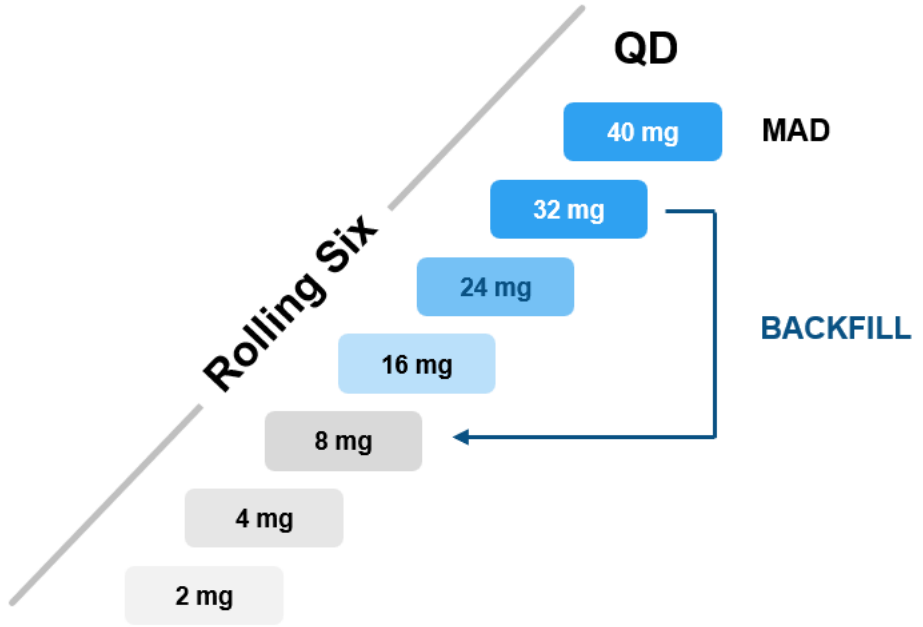
Primary Endpoints: DLTs, Safety assessments

Secondary Endpoints: PK parameters, ORR, DCR, TTR, DOR, PFS, OS

Key Eligibility Criteria: all RASm solid tumors; exposed to SOC; ECOG 0-1; measurable disease; RASi naive

US Trial (AURORAS-1) FPD to DCO ~9.5 months[2]

Part 1: Dose Escalation[3]



Primary Endpoints: DLTs, Safety assessments, PK parameters

Secondary Endpoints: ORR, DCR, TTR, DOR, PFS, OS

Key Eligibility Criteria: PDAC, KRAS G12X/G13X NSCLC, KRAS G12X/G13X select GI tumors; exposed to SOC; ECOG 0-1; measurable disease; RASi naive

[1]DCO as of Feb 2026; [2]DCO as of Apr 2026; [3]Dose escalation is completed; doses >40 mg QD will not be evaluated. FPD = First Patient Dosed; DCO = Data cutoff; ATD= Accelerated Titration Design; BOIN= Bayesian Optimal Interval; DCR=disease control rate; DLT=dose limiting toxicity; DOR=duration of response; MAD=maximum administered dose; NSCLC=non-small-cell lung cancer; ORR=objective response rate; OS=overall survival; PDAC=pancreatic adenocarcinoma; PK=pharmacokinetics; PFS=progression free survival; QD=once daily; RASi=RAS inhibitor; SOC=standard of care; TTR=time to response; Note: see Disclaimer slide regarding Cross-Study Comparisons

CN and US Trials: Baseline characteristics (PDAC and NSCLC)

CN Trial (JYP0015M101)	RASm PDAC (2, 8, 16, 24, 32, 40 mg)	RASm NSCLC[1] (16, 24, 32 mg)
	N=78	N=42
Median Age (range)	64.5 years (35 – 79)	65.0 years (36 - 75)
Female	36% (28/78)	36% (15/42)
ECOG PS		
0	9% (7/78)	2% (1/42)
1	91% (71/78)	98% (41/42)
Smoking Status		
Current	NA	5% (2/42)
Past	NA	48% (20/42)
Never	NA	48% (20/42)
Number of prior anti-cancer therapies, median (range)	1 (0-6)	1 (0-6)
Select prior anti-cancer therapies/regimens		
Gemcitabine + nab-paclitaxel	NA[4]	
FOLFIRINOX	NA[4]	
Checkpoint inhibitor		81% (34/42)
Platinum-based chemotherapy		88% (37/42)

US Trial (AURORAS-1)[2]	PDAC (2, 4, 8, 16, 24, 32, 40 mg)	KRAS G12X/G13X NSCLC[3] (8, 16, 24, 32, 40 mg)
	N=42	N=22
Median Age (range)	67.0 years (41 - 84)	67.0 years (45 - 78)
Female	29% (12/42)	59% (13/22)
ECOG PS		
0	24% (10/42)	27% (6/22)
1	76% (32/42)	73% (16/22)
Smoking Status		
Current	NA	9% (2/22)
Past	NA	59% (13/22)
Never	NA	32% (7/22)
Number of prior anti-cancer therapies in the metastatic setting, median (range)	2 (1-4)	2 (0-5)
Select prior anti-cancer therapies/regimens		
Gemcitabine + nab-paclitaxel	62% (26/42)	
FOLFIRINOX	81% (34/42)	
Checkpoint inhibitor		91% (20/22)
Platinum-based chemotherapy		96% (21/22)

CN Trial DCO: Feb 2026 / US Trial DCO: Apr 2026

[1] 2 and 8 mg cohorts did not enroll patients with NSCLC

[2] Safety analysis set: all patients with PDAC or NSCLC that received at least one dose of ERAS-0015

[3] 2 and 4 mg cohorts did not enroll patients with NSCLC

[4] Most common prior regimens were gemcitabine-based or 5-FU-based

PDAC: pancreatic ductal carcinoma; NSCLC: non-small cell lung cancer; NA=not available

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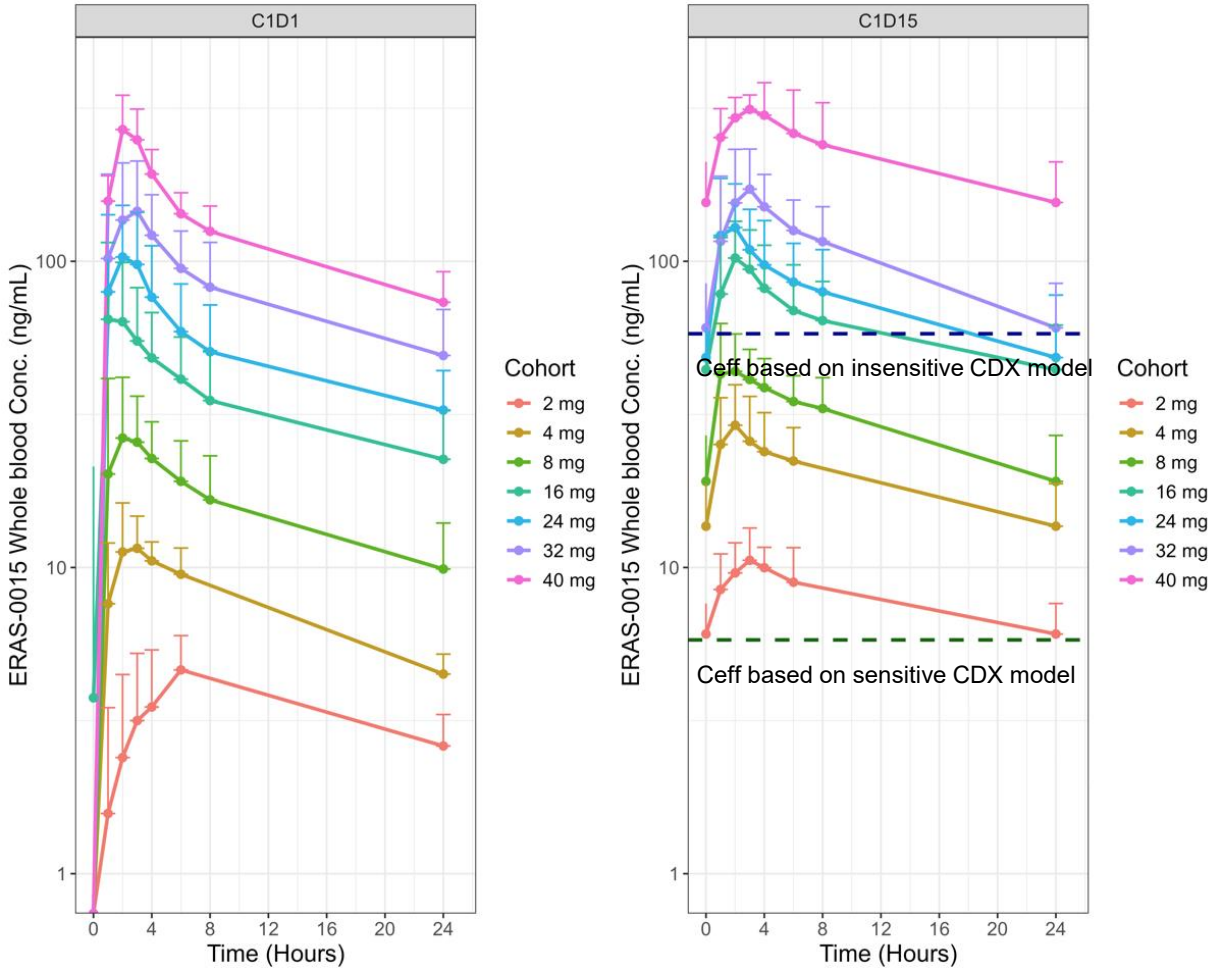
US Trial: Dose-dependent increase in PK exposure up to 40 mg MAD with no exposure plateau observed; PAD defined based on steady-state exposures that exceeded target threshold

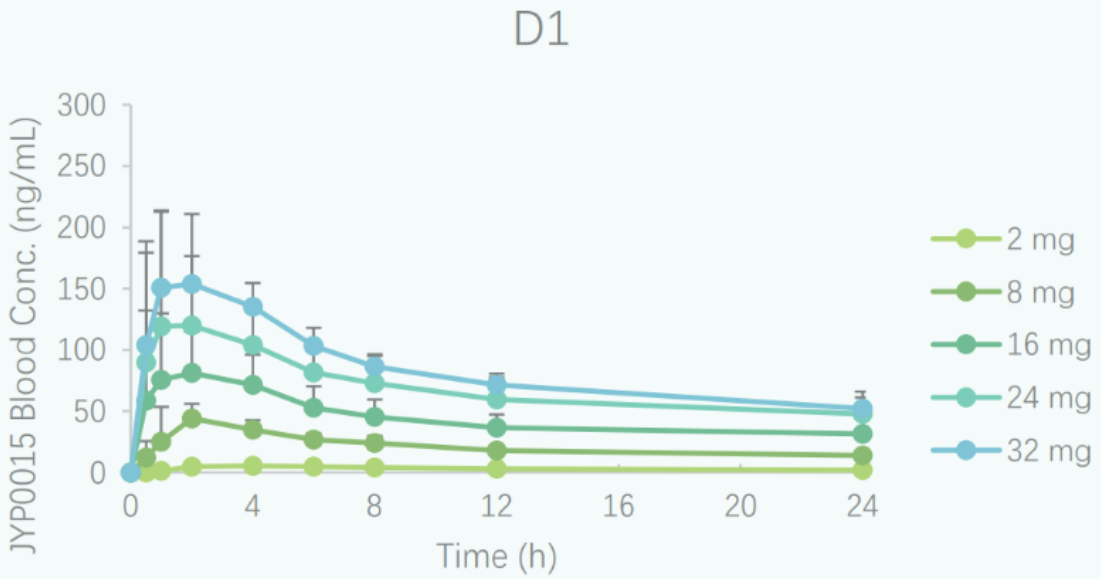


- **PK exposure** increased with dose with low-to-moderate PK variability
- **Pharmacologically active dose (PAD)** range: 16-32 mg QD
  - Mean steady-state average exposure ($C_{avg, ss}$) at ≥16 mg exceeded target exposure threshold (based on insensitive NCI-H727 CDX model)
- **Recommended doses for expansion (RDEs)**: 24 and 32 mg QD based on totality of the clinical data
- Ph 1 monotherapy data in today's presentation to be highlighted at PAD (safety and NSCLC, PDAC efficacy) and RDEs (NSCLC, PDAC efficacy)

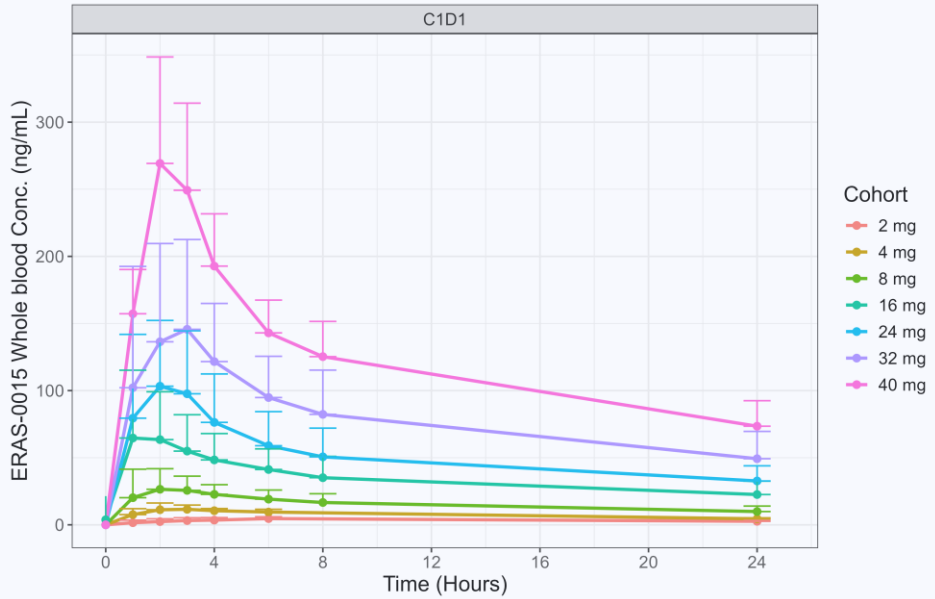
AURORAS-1 PK data are as of April 2, 2026; data points in the figures are presented as Mean + SD
Note: MAD: maximum administered dose; $C_{avg,ss}$: average concentration at steady-state

Single-dose PK data between CN and US trials have been largely comparable

CN Trial (JYP0015M101)[1]



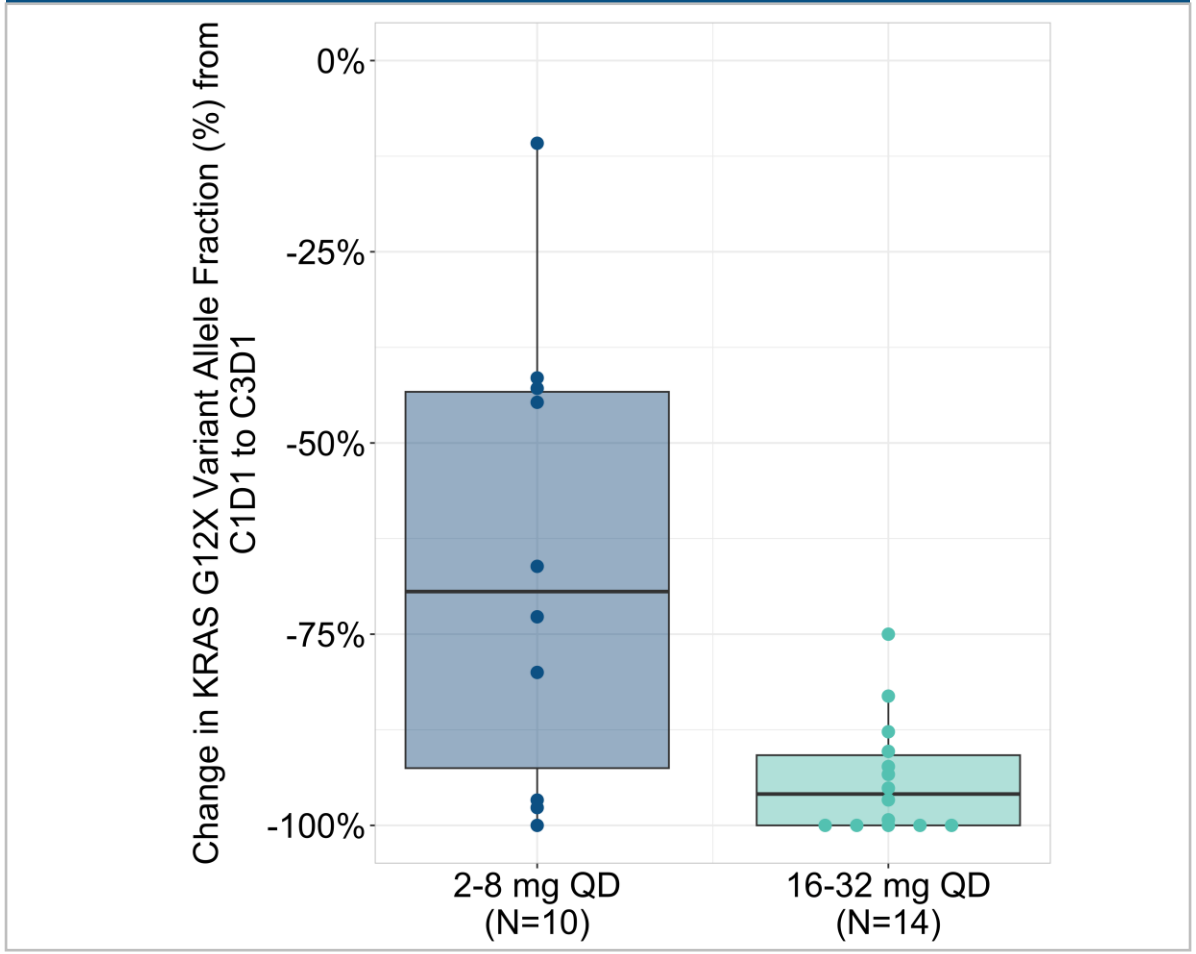
US Trial (AURORAS-1)[2]



[1]Data generated by Joyo Pharma 05Jan2026
[2]Data generated by Erasca 02April2026
Note: see Disclaimer slide regarding Cross-Study Comparisons

US Trial PD: Greater KRAS G12X ctDNA reduction at PAD doses (16-32 mg) vs. below PAD (2-8 mg)

Change in solid tumor ctDNA KRAS G12X VAF from C1D1 to C3D1: Below PAD (2-8 mg) vs at PAD range (16-32 mg)



100% of patients (14/14) at PAD showed at least **75% reduction of KRAS G12X variant allele fraction**, including 5 out of 14 patients showing 100% reduction

ctDNA = circulating tumor DNA; PD = pharmacodynamics; RASm = RAS mutant; PAD = pharmacologically active dose; VAF = variant allele fraction

Predicted sensitivity of Big 3 tumor types to pan-RAS inhibition

	NSCLC	PDAC	CRC
Relative anticipated sensitivity in the clinic to pan-RAS inhibition	**High**	**Med**	**Low**
Pan-RAS response dynamics	Faster, deeper responses	Up to 50% of patients may exhibit delayed RECIST responses (detectable after ≥2 scans, i.e., ~14 weeks)	Primary and adaptive resistance, often mediated by EGFR, is a key issue for pan-RAS monotherapy
Potential ERAS-0015 dose to achieve optimal anti-tumor activity	Monotherapy at PAD (16-32 mg)	Monotherapy at RDE (24-32 mg)	Combo. with anti-EGFR at PAD

Note: PAD=pharmacologically-active dose; RDE=recommended dose for expansion

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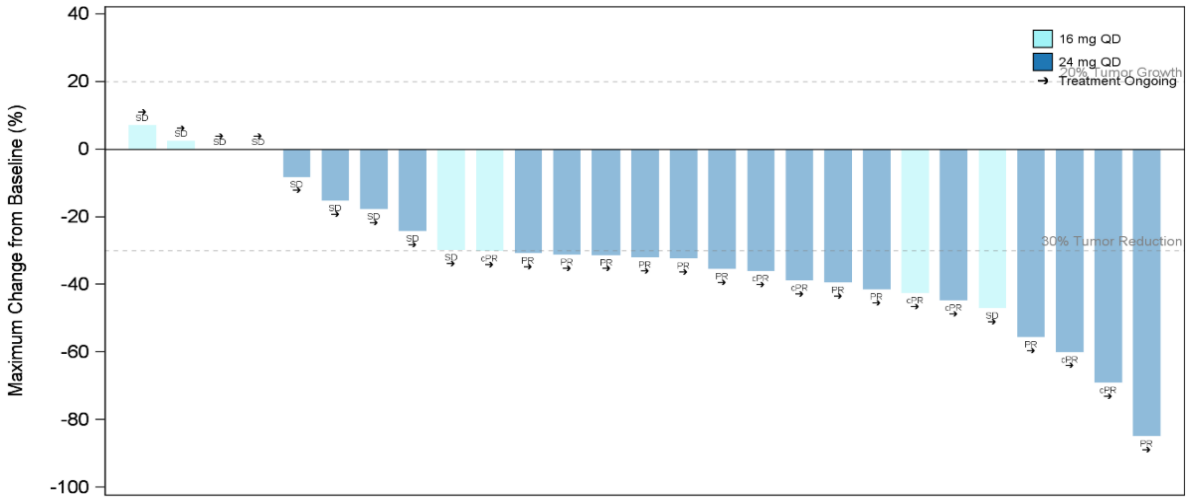
CN Trial: Encouraging preliminary efficacy signal observed for ERAS-0015 in 2L+ KRAS G12X NSCLC

Within pharmacologically active dose range

2L+ KRAS G12X NSCLC	16 mg QD (N=6)	24 mg QD1 (N=21)	ALL (N=27)
CR, n (%)	0	0	0
PR, n (%)	2 (33)	15 (71)	17 (63)
SD, n (%)	4 (67)	6 (29)	10 (37)
PD, n (%)	0	0	0
uORR, n (%)	**2 (33)**	**15 (71)**	**17 (63)**
DCR, n (%)	6 (100)	21 (100)	27 (100)



	2L+ KRAS G12X NSCLC	Post-ICI/platinum (2/3L) KRAS G12X
Dose	16-24 mg QD[1]	16-24 mg QD[1]
uORR	**17 (63)[2]**	**8 (73)**
N	27	11

63%

uORR for ERAS-0015

in 2L+ KRAS G12X NSCLC at the lower PAD doses of 16 and 24 mg

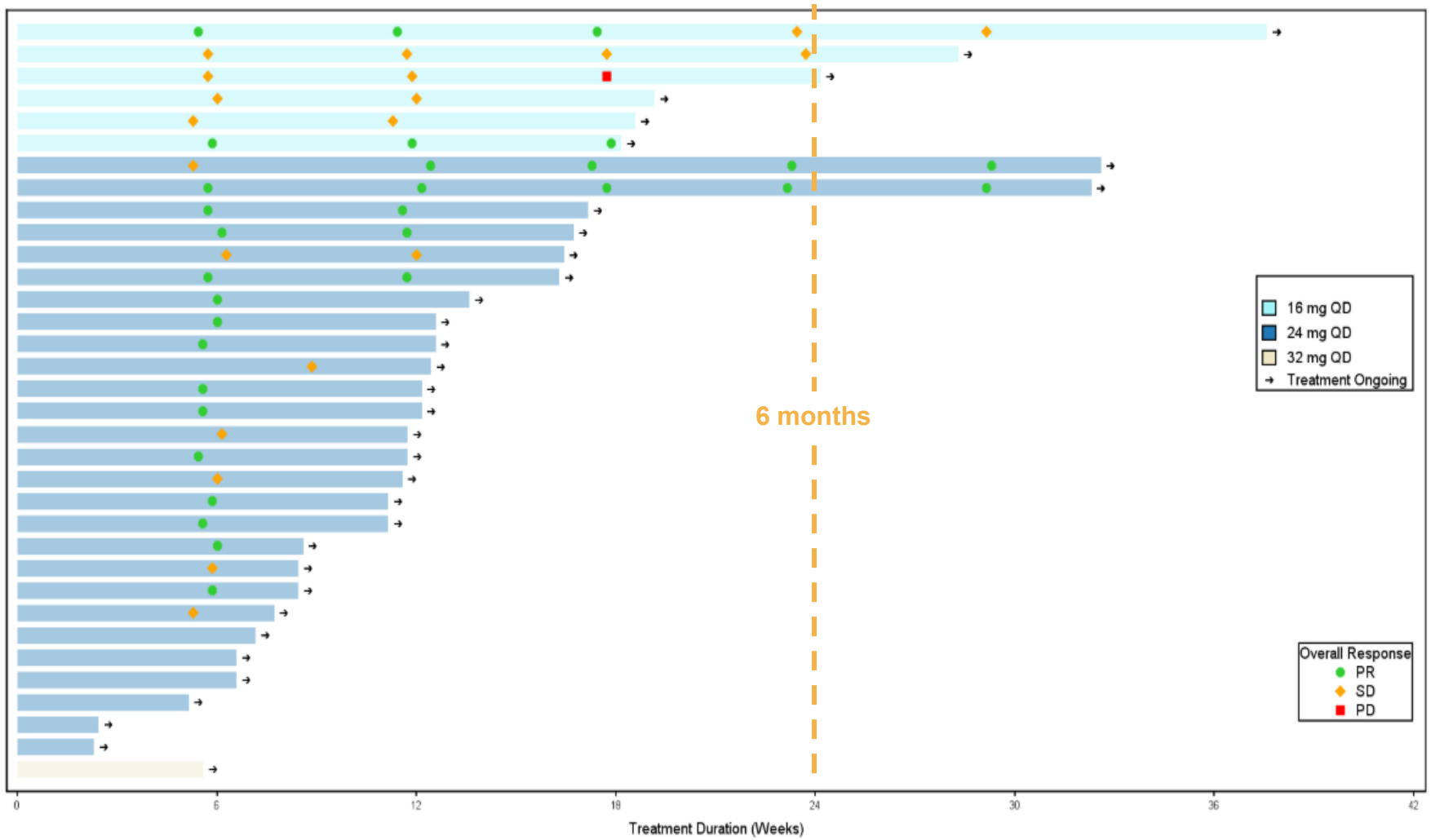
DCO Feb 2026

[1] No patients with NSCLC were enrolled at 32 mg and included in the efficacy evaluable population as of the DCO; Efficacy evaluable analysis set: patients with at least one post-dose tumor assessment

[2] 7 ongoing cPRs and 10 ongoing PRs out of 27 patients with KRAS G12X mutations

DCR: disease control rate; NA: not available; NSCLC: non-small-cell lung cancer; uORR: objective response rate (confirmed and unconfirmed responses); PAD: pharmacologically active dose range; PD: progressive disease; cPR: confirmed partial response; PR: unconfirmed partial response; SD: stable disease;

CN trial: Most patients with 2L+ KRAS G12X NSCLC remain on treatment suggesting potentially favorable safety and tolerability[1]



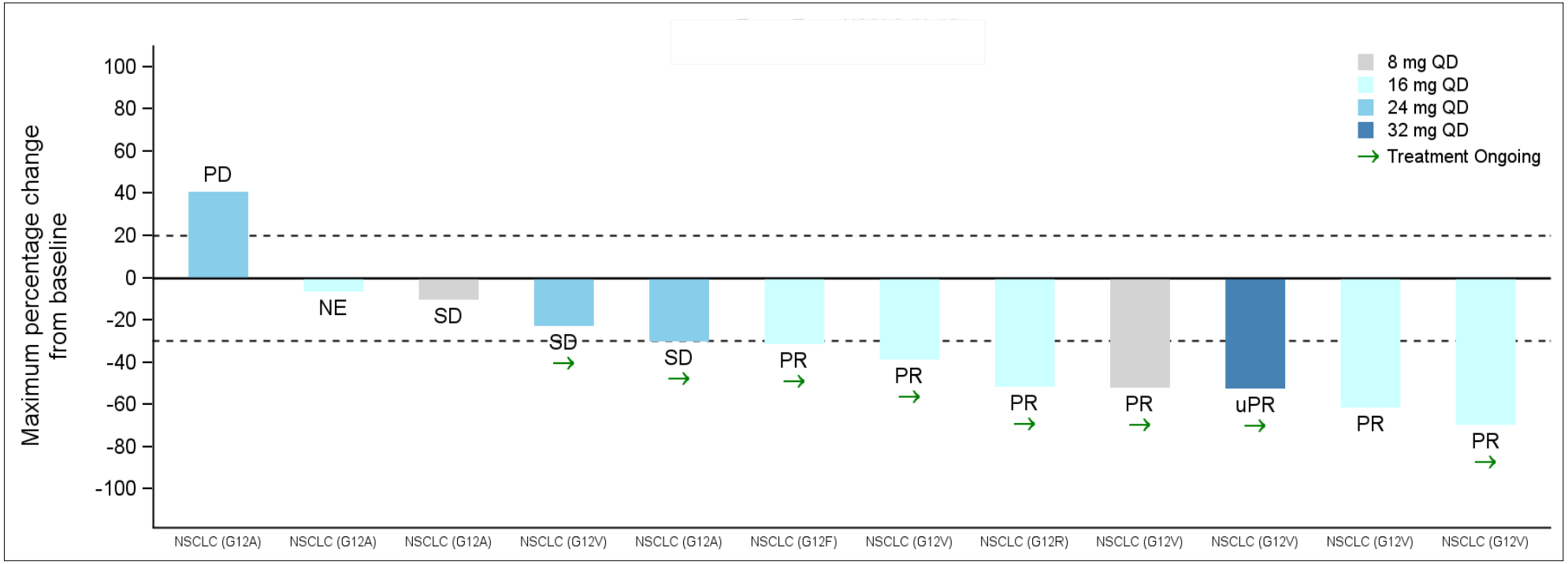
100% of responders – including all patients with uPRs – remain on treatment

Reinforces the safety, tolerability and durability of response of ERAS-0015

DCO Feb 2026

[1] Full analysis set: all patients who received at least one dose of ERAS-0015
PR: partial response; PD: progressive disease; QD: once daily; SD: stable disease;

US trial: Encouraging preliminary ERAS-0015 efficacy data in patients with 2L+ KRAS G12X NSCLC consistent with data from CN trial



Responses observed throughout the dose range indicate sensitivity of NSCLC to pan-RAS inhibition

60% uORR observed at the PAD

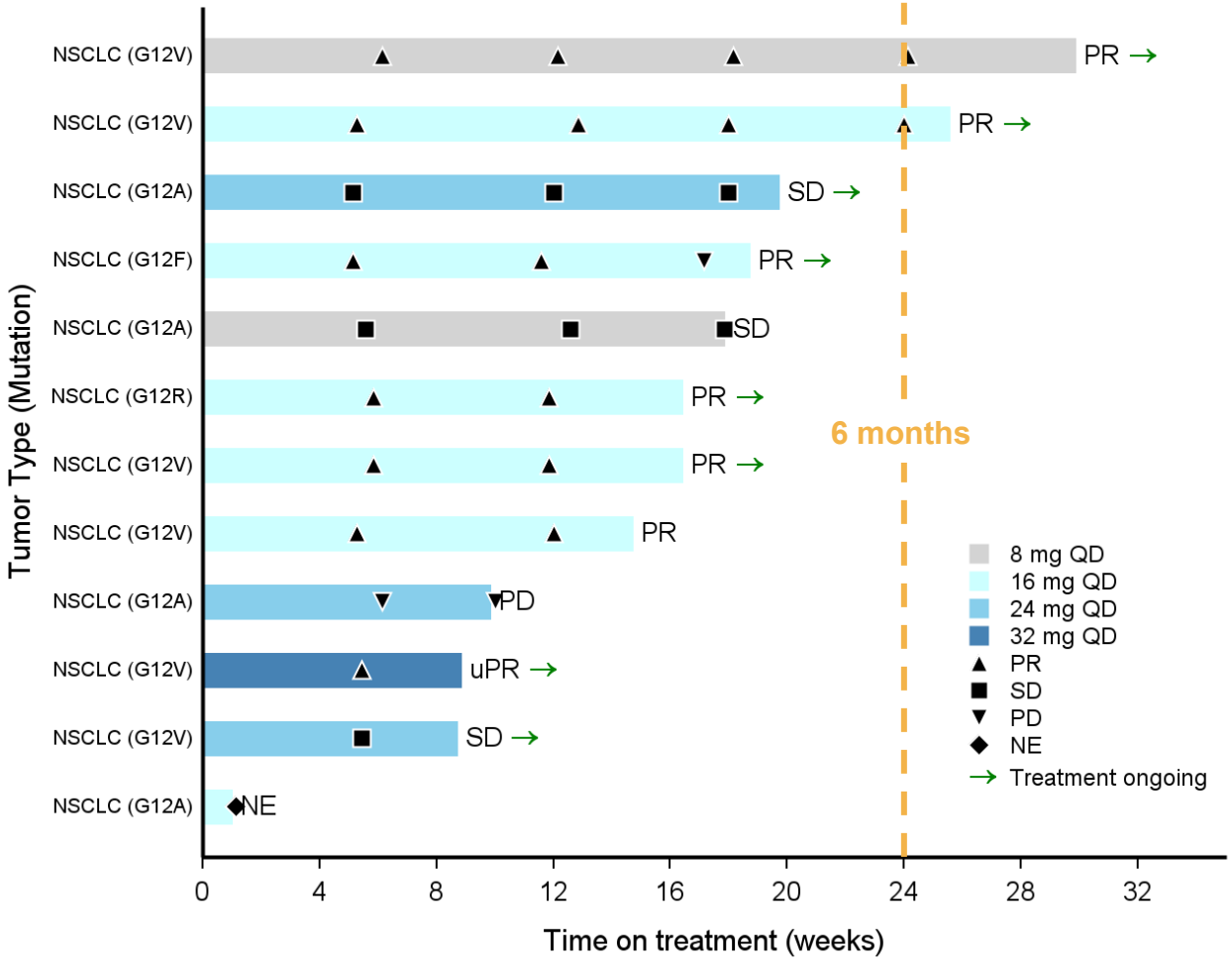
71% uORR observed in post-ICI/platinum (2/3L) KRAS G12X

ERAS-0015 2L+ KRAS G12X NSCLC	8 mg QD N=2	16 mg QD N=6	24 mg QD N=3	32 mg QD N=1	40 mg QD N=0	Total N=12	Post-ICI/platinum (2/3L) KRAS G12X N=7
uORR[1], n (%)	1 (50)	5 (83)	0	1 (100)	NA	7 (58)	5 (71)
DCR, n (%)	2 (100)	5 (83)	2 (67)	1 (100)	NA	10 (83)	6 (86)

DCO Apr 2026

1 uORR includes confirmed and unconfirmed responses; DCO 4Apr2026; Efficacy evaluable analysis set: all participants in the safety analysis set who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date; DCR=disease control rate; NA=not applicable; NSCLC=non-small-cell lung cancer; ORR=objective response rate; PAD=pharmacologically active dose; uPR=unconfirmed PR; PD=progressive disease; PR=partial response; QD=once daily; SD=stable disease; Note: see Disclaimer slide regarding Cross-Study Comparisons

US trial: Most patients with 2L+ KRAS G12X NSCLC remain on treatment as of DCO



6 out of 7 responders—including all patients with uPRs—remain on treatment

Reinforces the safety, tolerability and potential durability of response of ERAS-0015

DCO Apr2026; NSCLC=non-small-cell lung cancer; PD=progressive disease; PR=partial response; QD=once daily; SD=stable disease
Efficacy evaluable analysis set: all participants in the safety analysis set who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date.
Response at the end of the bar represents the best objective response based on investigator assessment denoted as CR/PR for confirmed CR/PR or uCR/uPR for unconfirmed CR/PR.

ERAS-0015: Preliminary NSCLC data suggest differentiated preclinical properties could potentially drive improved clinical activity, consistent with KRAS G12Ci precedent

First-in-class molecules

37%

Sotorasib ORR[1]
in 2L+ KRAS G12C NSCLC,
960 mg QD, N=124

43%

Adagrasib ORR[2]
in 2L+ KRAS G12C NSCLC,
600 mg BID, N=112

38%

RMC-6236 ORR[4]
in Post-ICI/plat (2/3L)
KRAS G12X NSCLC,
120-220 mg QD, N=40

Potentially best-in-class molecules

KRAS G12Ci's →

59%

Divarasib ORR[3]
in 2L+ KRAS G12C NSCLC,
400 mg QD, N=44

Pan-RAS MG's →

62%

(95% CI: 45%, 78%)
ERAS-0015 uORR
in 2L+ KRAS G12X NSCLC,
16-32 mg QD, N=37

75%

(95% CI: 48%, 93%)
in Post-ICI/plat (2/3L)
KRAS G12X NSCLC,
16-32 mg QD, N=16

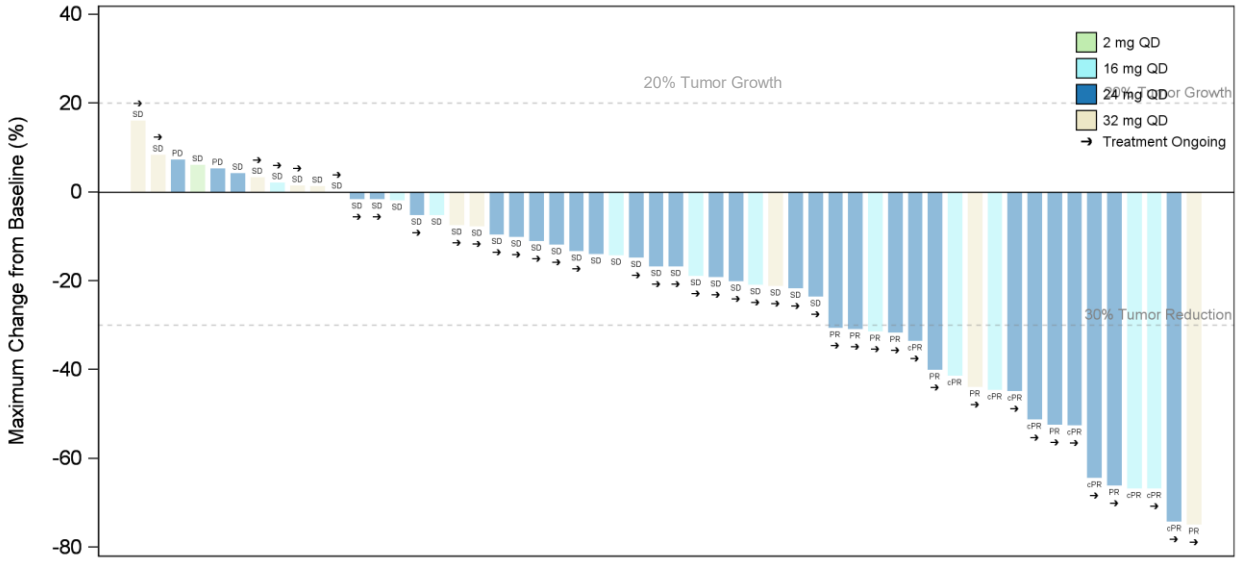
[1]Skoulidis F et al, NEJM 2021; exploratory subgroup analyses from phase 2 CodeBreak 100 trial showed 39.6% ORR in 2L only subgroup (N=53) and 35.2% ORR in 3L+ subgroup (N=71); [2]Jänne P et al, NEJM 2022; [3]Sacher A at al, JCO 2025; [4]Punekar et al, JTO 2025; N=40 selected from 73 patients (120-220mg) with at least 14 weeks prior to data cutoff date (to allow 2 potential scans), RAS G12X NSCLC, 2-3L post-IO and platinum, no prior docetaxel
NSCLC: non-small-cell lung cancer; ORR: overall response rate; QD: once daily; BID: twice daily; ICI: immune checkpoint inhibitor; plat: platinum
Note: Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons

ERASCA

CN trial: Encouraging preliminary efficacy data for ERAS-0015 in 2L+ KRAS G12X PDAC

Within pharmacologically active dose range (PAD)

ERAS-0015 2L+ KRAS G12X PDAC[1]	2 mg QD (N=1)	16 mg QD (N=11)	24 mg QD (N=32)	32 mg QD (N=10)	ALL (N=54)
CR, n (%)	0	0	0	0	0
PR, n (%)	0	5 (45)	12 (38)	2 (20)	19 (35)
SD, n (%)	1 (100)	6 (55)	18 (56)	8 (80)	33 (61)
PD, n (%)	0	0	2 (6)	0	2 (4)
uORR, n (%)	0	**5 (45)**	**12 (38)**	**2 (20)**	**19 (35)**
DCR, n (%)	1 (100)	11 (100)	30 (94)	10 (100)	52 (96)



	ERAS-0015	
	2L+ KRAS G12X PDAC	2L KRAS G12X PDAC
Dose	16-32 mg QD (PAD)	16-32 mg QD (PAD)
uORR, n (%)	**19 (36)[2]**	**11 (41%)[3]**
N	53	27

36%

uORR for ERAS-0015

in 2L+ KRAS G12X PDAC at the PAD

DCO Feb 2026

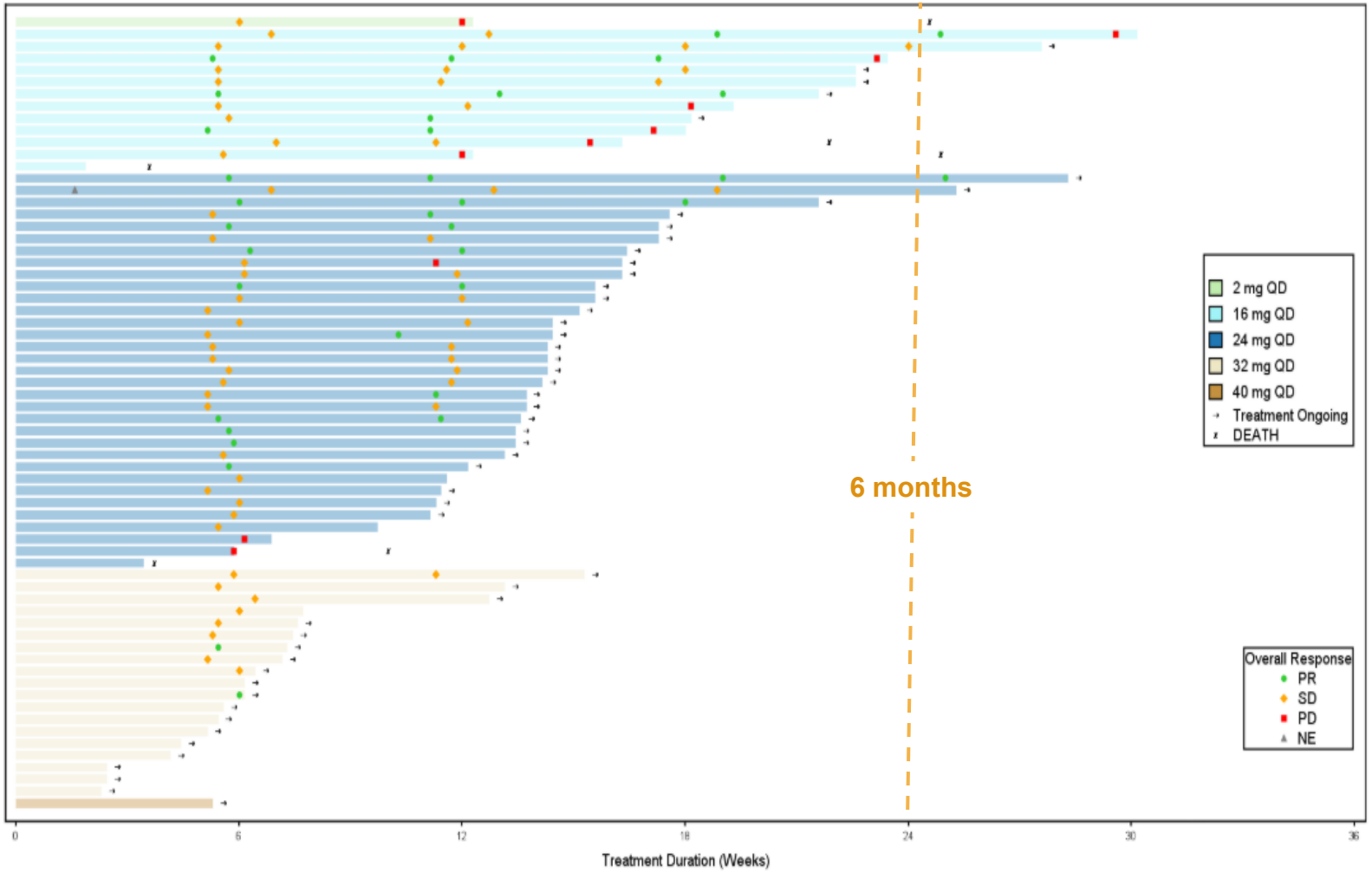
[1] Efficacy evaluable analysis set: Patients with at least one post-dose tumor assessment

[2] 7 ongoing cPRs, 3 cPRs that have discontinued treatment, and 9 ongoing uPRs out of 53 patients with KRAS G12X mutations

3 4 cPRs and 7 ongoing uPRs out of 27 patients with KRAS G12X mutation

DCR: disease control rate; uORR: objective response rate (confirmed and unconfirmed responses); PAD: pharmacologically active dose range; PD: progressive disease; PDAC: pancreatic adenocarcinoma; cPR: confirmed partial response; PR: unconfirmed partial response; SD: stable disease;

CN trial: Most patients with 2L+ KRAS G12X PDAC remain on treatment suggesting potentially favorable safety and tolerability[1]



84% (16/19) of responders – including all uPRs at 24-32 mg – remain on treatment

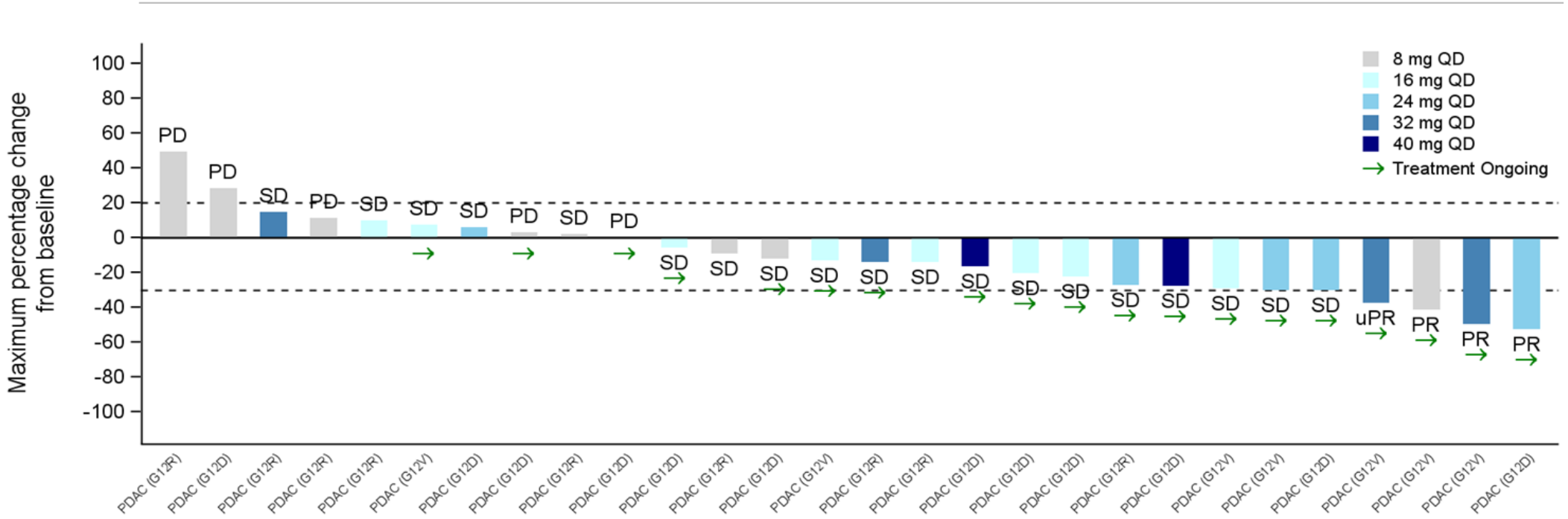
Reinforces the safety, tolerability and durability of response of ERAS-0015

DCO Feb 2026

[1] Full analysis set: all patients who received at least one dose of ERAS-0015

NE: not evaluable; PR: partial response; PD: progressive disease; QD: once daily; SD: stable disease;

US trial: Encouraging preliminary ERAS-0015 efficacy data in patients with 2L+ KRAS G12X PDAC consistent with data from CN trial



Preliminary data support the hypothesis that PDAC may be less sensitive than NSCLC, with high DCRs but limited responses at lower doses…

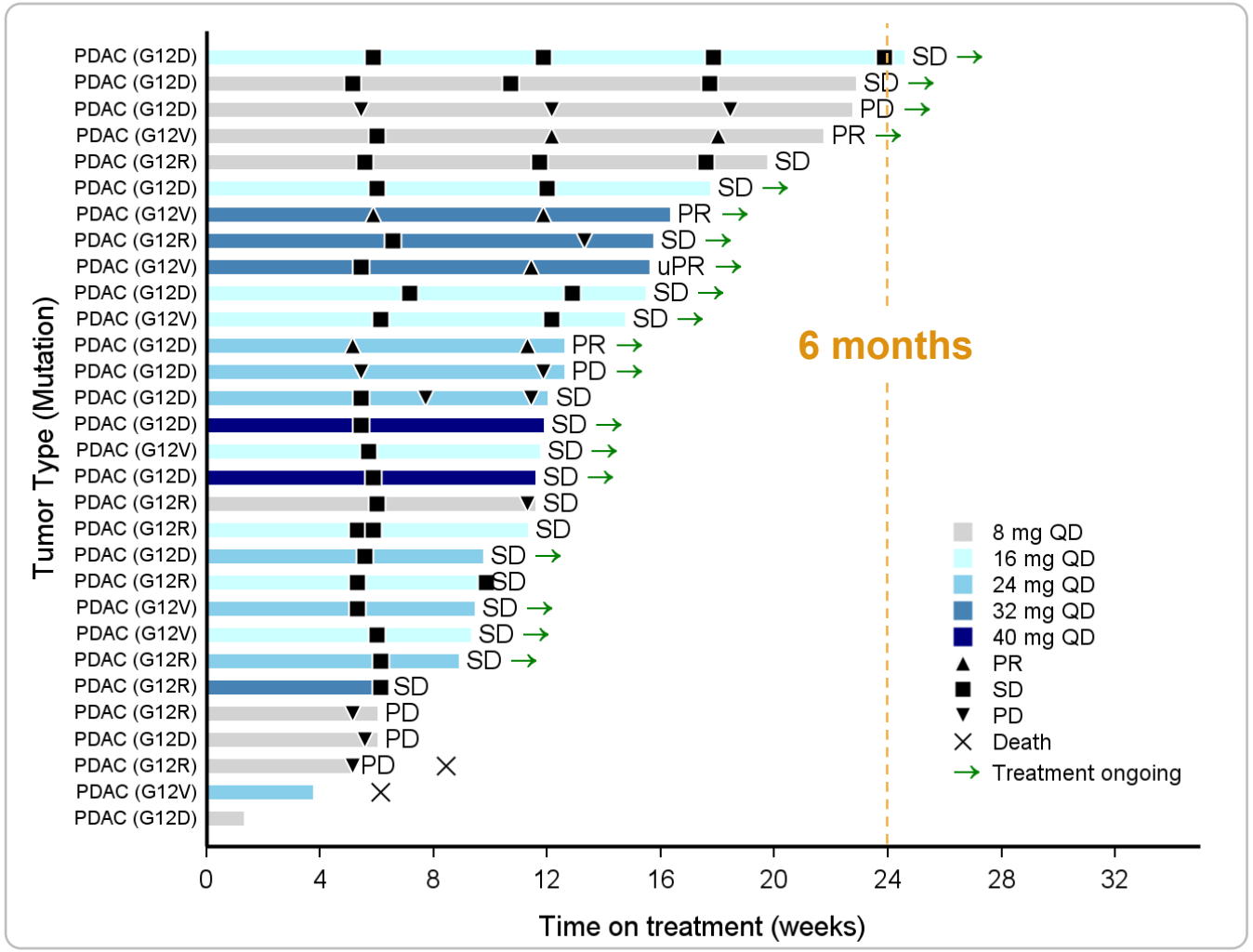
ERAS-0015 2L+ KRAS G12X PDAC[1]	8 mg QD N=9	16 mg QD N=8	24 mg QD N=7	32 mg QD N=4	40 mg QD N=2	Total N=30
uORR[1] , n (%)	1 (11%)	0	1 (14%)	2 (50%)	0	4 (13%)
DCR, n (%)	4 (44%)	8 (100%)	5 (71%)	4 (100%)	2 (100%)	23 (77%)

…ERAS-0015 showed increased efficacy with longer minimum follow-up and earlier line of treatment

ERAS-0015 KRAS G12X PDAC uORR		ERAS-0015 dose			
Line of treatment (LOT)	Weeks after first dose prior to DCO	≥8 mg	PAD 16 - 32 mg	RDEs 24 - 32 mg	32 mg
2L	14	2/9=22%	1/5=20%	1/1=100%	1/1=100%
	8	3/12=25%	2/8=25%	2/4=50%	1/2=50%
3L+	14	1/10=10%	1/5=20%	1/3=33%	1/2=50%
	8	1/18=6%	1/11=9%	1/7=14%	1/2=50%

[1]uORR includes confirmed and unconfirmed responses; DCO 4Apr2026; Efficacy evaluable analysis set: all participants in the safety analysis set who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date; DCR: disease control rate; NA: not available; NSCLC: non-small cell lung cancer; ORR: objective response rate; PAD: pharmacologically active dose range; uPR: unconfirmed PR; PR: partial response; PDAC: pancreatic ductal adenocarcinoma; PD: progressive disease; QD: once daily; SD: stable disease; Note: see Disclaimer slide regarding Cross-Study Comparisons.

US trial: Time on treatment for patients with 2L+ KRAS G12X PDAC consistent with data from CN trial



All patients with either confirmed or unconfirmed responses remain on treatment

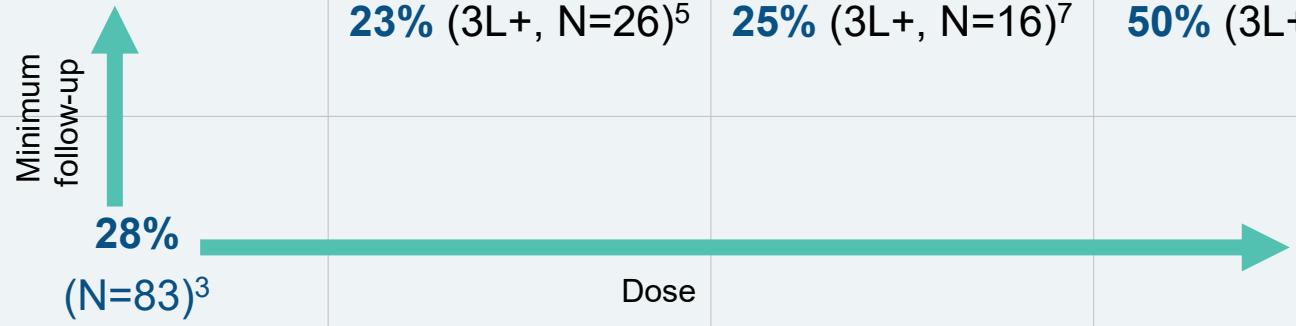
Reinforces the safety, tolerability and durability of response of ERAS-0015

DCO Apr2026; PDAC=pancreatic adenocarcinoma; PD=progressive disease; PR=partial response; QD=once daily; SD=stable disease
Efficacy evaluable analysis set: all participants in the safety analysis set who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date.
Response at the end of the bar represents the best objective response based on investigator assessment denoted as CR/PR for confirmed CR/PR or uCR/uPR for unconfirmed CR/PR.
Note: see Disclaimer slide regarding Cross-Study Comparisons.

ERAS-0015 showed increased response rates in 2L+ KRAS G12X PDAC with longer minimum follow-up and earlier line of treatment

ERAS-0015 uORR by dose, minimum follow-up, and line of treatment in 2L+ KRAS G12X PDAC

Time of Assessment	≥8 mg	16 – 32 mg PAD	24 – 32 mg RDE	32 mg only
$uORR_{14wk}$[1] (received first dose at least 14 weeks prior to DCO)		**40%** (2L, N=20)[4] **23%** (3L+, N=26)[5]	**42%** (2L, N=12)[6] **25%** (3L+, N=16)[7]	**50%** (2L, N=2)[8] **50%** (3L+, N=2)[9]
$uORR_{8wk}$[2] (received first dose at least 8 weeks prior to DCO)	**28%** (N=83)[3]			



[1]$uORR_{14wk}$=Objective Response Rate (confirmed and unconfirmed responses) for patients who received first dose of ERAS-0015 at least 14 weeks prior to data extract date (US and CN trial); [2]$uORR_{8wk}$=Objective Response Rate (confirmed and unconfirmed responses) for patients with KRAS G12X (US) or RASm (CN) PDAC who received first dose of ERAS-0015 at least 8 weeks prior to data extract date (US trial) or at least one ERAS-0015 post-dose tumor assessment (CN trial); [3]US: 3 cPR, 1 uPR, N=30; CN: 10 cPR, 9 uPR, N=53; [4]US: 1 uPR, N=5, CN: 4 cPR, 3 uPR, N=15; [5]US: 1 cPR, N=5, CN: 5 cPR, N=21; [6]US: 1 uPR, N=1, CN: 2 cPR, 2 uPR, N=11; [7]US: 1 cPR, N=3, CN: 3 cPR, N=13; [8]US: 1 uPR, N=1, CN: 0 c/uPR, N=1; [9]US: 1 cPR, N=2, CN: 0 c/uPR, N=0
Note: see Disclaimer slide regarding Cross-Study Comparisons.

Anti-EGFR treatment has demonstrated limited monotherapy efficacy in 2L+ KRASm CRC but substantially improved activity in combination with KRAS G12Ci

	Cetuximab monotherapy[1]	Panitumumab monotherapy[2]	Panitumumab + 960mg QD sotorasib[3]
ORR	1.2%	0%	26%
PFS	1.8 months	~7.4 weeks	5.6 months
OS	4.5 months	ND	NR

"**ERBITUX** is not indicated for treatment of RAS-mutant colorectal cancer"[4]

- Cetuximab Package Insert (2021)

"**Vectibix** is not indicated for the treatment of patients with RAS-mutant CRC unless used in combination with **sotorasib** in KRAS G12C-mutated mCRC"[5]

- Panitumumab Package Insert (2025)

[1]Kareptis et al. NEJM 2008; [2]Amado et al. JCO 2008; [3] Fakih et al. NEJM 2023; [4] ERBITUX (cetuximab) US Package insert. Lilly 2021; [5] Vectibix (panitumumab) US Package insert, Amgen 2025
Note: ND=not determined
Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons

ERASCA

ERAS-0015 shows promising clinical potential to combine with SOC in CRC

ERAS-0015 + panitumumab[1]

- Initiated ERAS-0015 (at 16 mg QD) plus anti-EGFR mAb (at approved dose) combo cohort in Q1 2026
- Tolerated thus far with no DLTs observed to date (N=3)
- uPR in first efficacy-evaluable patient at initial scan

2 patients have cleared DLT window

Toxicity manageable to date with AEs consistent with the known profile of panitumumab (per investigator feedback)

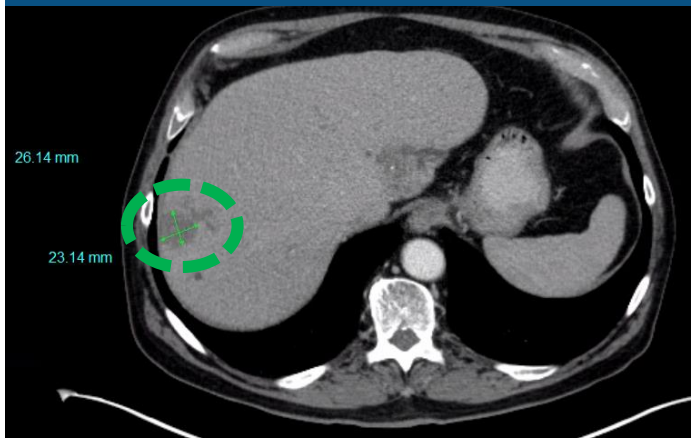
[1] As of 31Mar2026
DLT: dose-limiting toxicity

ERASCA

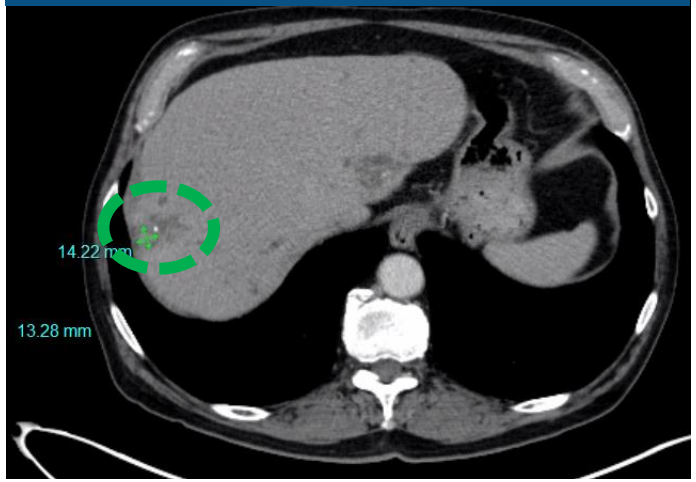
AURORAS-1 combination case study in KRAS G12D CRC: Ongoing uPR in 77-year-old male treated with ERAS-0015 16 mg + panitumumab

Diagnosis	Stage IV MSS CRC; KRAS G12D
Prior Therapy	FOLFIRINOX (Jan-June 21) 5FU+Bev (Aug 21-Dec 24) Radiation of liver lesions (Nov 23) Experimental Treatment (Jan-Dec 25)
ERAS-0015 / Panitumumab Treatment (4-wk cycle)	**Cycle 1:** ERAS-0015 (16 mg QD) + panitumumab (6mg/kg Q14D) **Cycle 3:** Restaging CT (-34% per RECIST; ***ongoing unconfirmed partial response***) Patient continues on treatment[1]
Treatment-related adverse events	Rash Acneiform (Gr 2) Paronychia (Gr 1) Pruritus (Gr 1) Mucositis (Gr 1)

Baseline



Cycle 3



uPR = unconfirmed partial response; CRC: colorectal cancer; MSS: microsatellite stable; 5-FU: 5-fluorouracil; Bev: bevacizumab; QD: every day; Q14D: every 14 days

[1]As of 31Mar2026

ERASCA

US trial: ERAS-0015 was generally well-tolerated with mostly low-grade TRAEs and no discontinuations

Summary of TRAEs occurring in ≥10% of patients					
Patients with RASm NSCLC and PDAC Treated at PAD (16-32mg) ERAS-0015 (N=43)					
TRAEs, n (%)	**Grade 1**	**Grade 2**	**Grade 3[1]**	**Grade 4**	**All Grades**
Rash[2]	20 (47)	8 (19)	1 (2)	0	29 (67)
Diarrhea	12 (28)	1 (2)	0	0	13 (30)
Stomatitis	6 (14)	1 (2)	0	0	7 (16)
Nausea	4 (9)	1 (2)	0	0	5 (12)
TRAEs leading to dose interruptions					5 (12)
TRAEs leading to dose reductions					3 (7)
TRAEs leading to dose discontinuations					0

[1] One Grade 3 TRAE of pneumonitis progressed to Grade 5 after withdrawal of supportive care per patient decision. The patient was a 66 year-old male with heavily pretreated metastatic pancreatic adenocarcinoma who received 24 mg of ERAS-0015. The patient had pulmonary metastases, a history of right lung cryoablation and no history of lung radiation. The patient presented to the ER approximately a month after starting ERAS-0015 with Grade 3 pneumonitis that was treated aggressively with immediate discontinuation of ERAS-0015, high dose steroids and infliximab. The patient requested withdrawal of supportive care and ultimately died of the event.

[2] Rash events are identified using following preferred term rash pustular, rash papular, rash maculo-papular, rash macular, rash, erythema and dermatitis acneiform (uncoded terms rash acneiform and rash, are also included).

Note: TRAEs = treatment-related adverse events; DCO Apr2026

ERASCA

Valuable investigator insights on safety and tolerability

- Close engagement with ERAS-0015 investigators helps optimize our development approach
- To date, feedback on ERAS-0015 has been highly encouraging:[1]

"There's Grade 2 rash that's tolerable and intolerable. I get the sense at least from my patients that the ***majority [of patients] that have Grade 2 rash is tolerable****. That means that they're* ***not desiring to come off study or dose reduce, it's not getting super infected, and it's not painful****."*

"I have ***some patients who they just prefer to live with the Grade 2 rash*** *as opposed to do all the antibiotics and the topicals because it honestly doesn't bother them that much."*

"I think we would all agree that this looks so far to be ***less of a Grade 2 than your competitors****, which is* ***incredibly important and that's a big differentiator****."*

[1] Reflects opinions of select investigators; quotes are anecdotal and do not constitute comparative clinical evidence

Rapidly advancing ERAS-0015, a potential best-in-class next-generation pan-RAS molecular glue with emerging clinical differentiation

Rapid Execution and Strong Momentum

- High investigator and patient engagement
- Completed dose escalation in <1 year from FPD
- Identified two monotherapy RDEs
- Initiated monotherapy expansion and combination dose escalation cohorts ahead of guidance

Encouraging Clinical Profile

- Compared favorably to benchmark across key efficacy and safety/tolerability attributes
- Well-behaved, linear PK with no evidence of exposure plateau
- Combinable with anti-EGFR with initial signs of anti-tumor activity

Potential to be preferred RAS-targeting agent

- Emerging profile supports potential as leading monotherapy
- Early data suggest ERAS-0015 could potentially become preferred RAS-targeting backbone for combination therapy
- Accelerating clinical development efforts based on high unmet need and investigator enthusiasm

We are deeply grateful to the patients, their families, and the investigators and study teams, for their participation in these studies

FPD: first patient dosed; RDE: recommended dose for expansion; PK: pharmacokinetics

ERASCA

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

ERAS-0015
Pan-RAS
Molecular Glue

Potential best-in-class Pan-RAS molecular glue

- ~5x – 10x greater antitumor activity vs. most advanced pan-RAS MG in development[1]
- Favorable ADME properties and PK performance in animals vs. most advanced pan-RAS MG in development[1]
- 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 Pan-KRAS inhibitor

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

[1] These data were generated in head-to-head assay and/or in vivo experiments
TPP = target product profile; MG: molecular glue; ADME: absorption, distribution, metabolism, and excretion; PK: pharmacokinetic; WT: wild type; KRASm: KRAS mutant

ERAS-4001 selectively bound KRAS with high affinities, long residence times

SPR-based kinetic biophysical binding characterization of ERAS-4001

Target	KD (nM)	$t_{1/2}$ (s)
KRAS G12D	0.0006	273,079
KRAS G12V	0.0069	30,159
KRAS G12C	0.016	7,724
KRAS WT	0.058	3,409
HRAS WT	117	18.1
NRAS WT	2,660	1.2

SPR = surface plasmon resonance

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

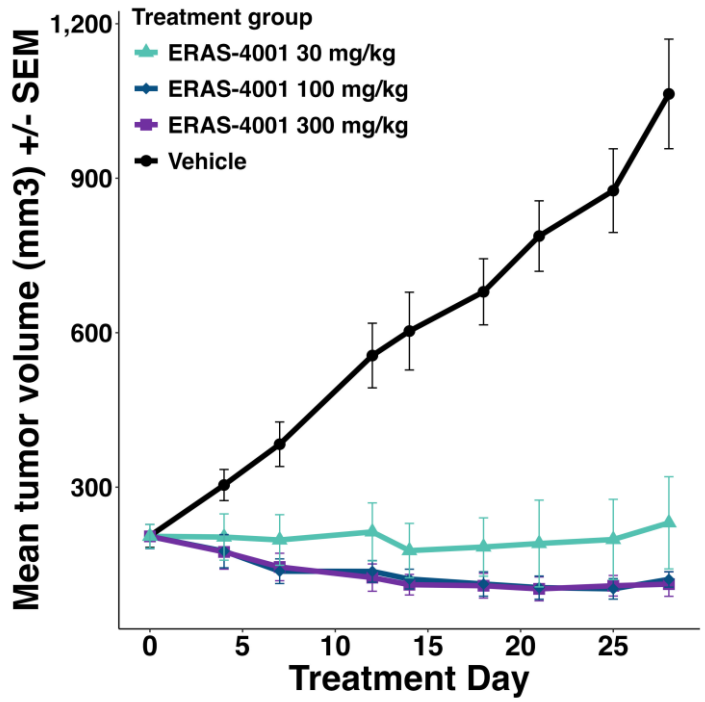
ERAS-4001 potently and selectively inhibited cell growth in KRAS G12X, G13D and WT cell lines

KRAS Mutation	Tumor type	Cell line	ERAS-4001 cell growth inhibition (nM)
KRAS G12D	Pancreatic	AsPC-1	**1.8**
	Pancreatic	Panc 04.03	**1.9**
	Pancreatic	HPAC	**1.0**
	Pancreatic	PK-59	**2.6**
KRAS G12V	Lung	NCI-H727	**3.5**
	Lung	NCI-H441	**0.7**
	Ovary	RKN	**2.3**
	Colorectal	SW620	**9.1**
KRAS G12C	Lung	LU99	**2.7**
	Pancreatic	MIA PaCa-2	**1.1**
	Lung	NCI-H2030	**4.5**
KRAS G12A	Multiple Myeloma	RPMI-8226	**6.5**
	Lung	NCI-H1573	**37.7**
KRAS G13D	Colorectal	LoVo	**5.8**
	Colorectal	HCT-116	**56**
KRAS WT	Lung	NCI-H1975	**10.8**
	Stomach	MKN-1	**3.6**
KRAS Independent	Melanoma	A375	**>2,000**
	Lung	NCI-H226	**3,497**

ERASCA

ERAS-4001 achieved tumor regression in a KRAS G12D PDAC CDX model at doses at or above 100 mg/kg BID

KRAS G12D CDX Panc 04.03
TGI curves from 28 day repeat dose study



TGI, PD (pERK) and PK ($AUC_{0\text{-last}}$) Summary

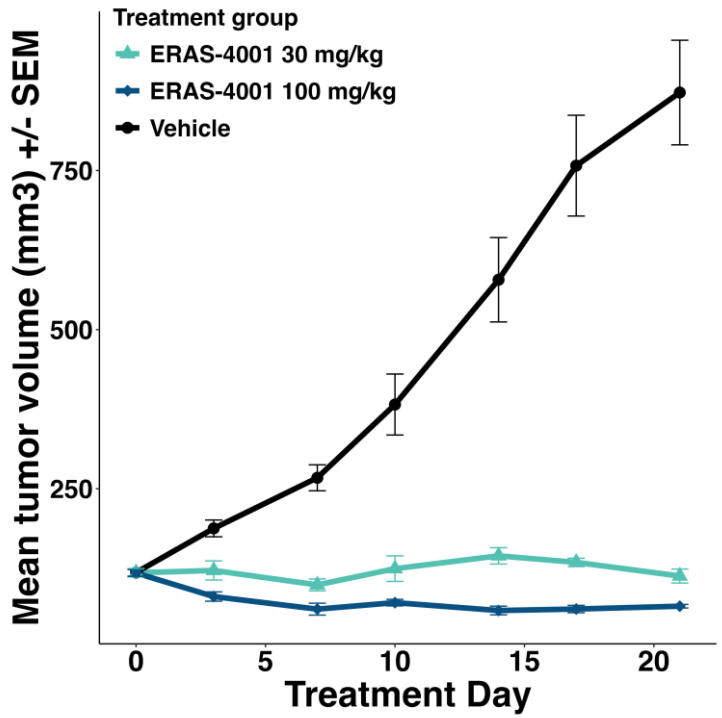
Therapy	Dose	TGI on Day 28	pERK Inhibition at 8 hr	$AUC_{0\text{-last}}$ (nmol/L·h)
ERAS-4001	30 mg/kg	97%	17%	1,547
	100 mg/kg	110%	64%	5,153
	300 mg/kg	111%	80%	12,971

- ERAS-4001 was well tolerated at doses up to 300 mg/kg BID for 28 days (i.e., no dose reductions or holidays; no body weight loss or significant health observations)

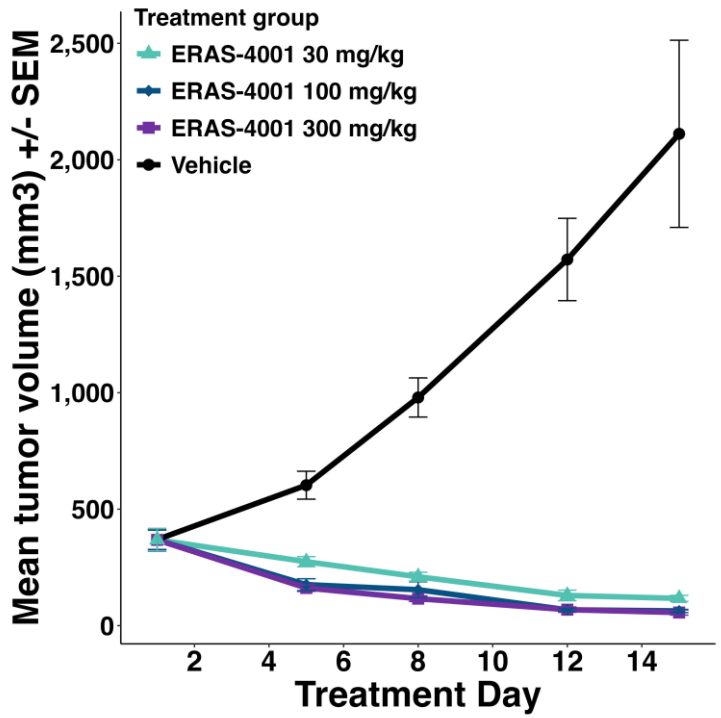
ERAS-4001 dosed orally twice daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

ERAS-4001 achieved tumor regressions in sensitive KRAS G12D and G12V CDX models at doses as low as 30 mg/kg BID

TGI in KRAS G12D PDAC CDX PK-59



TGI in KRAS G12V Ovarian CDX RKN

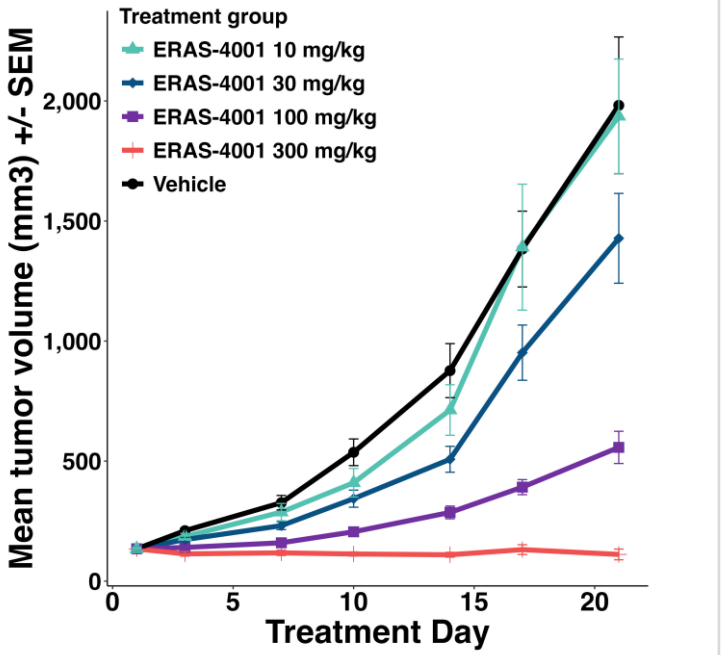


- ERAS-4001 was well tolerated in both studies at doses up to 300 mg/kg BID (i.e., no dose reductions or holidays; no body weight loss or significant health observations)

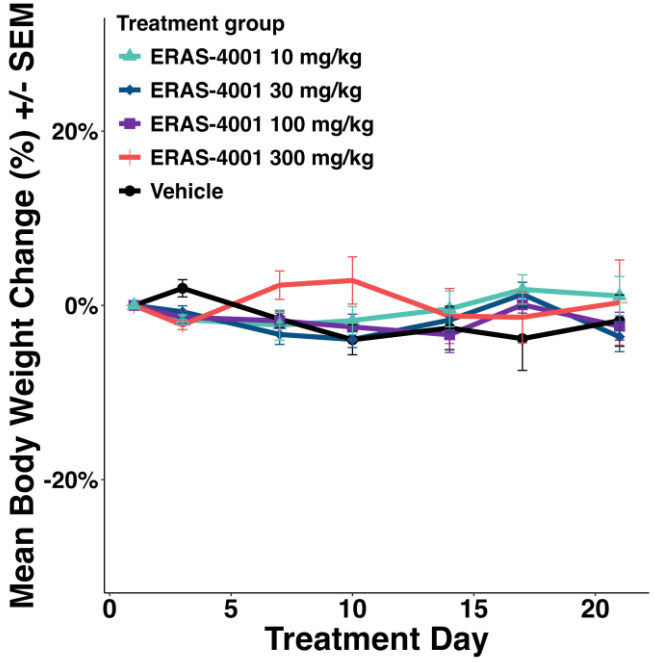
ERAS-4001 dosed orally twice daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

ERAS-4001 achieved tumor regression in a pan-KRASi insensitive KRAS G12V NSCLC CDX model

TGI in KRAS G12V NCI-H727



% Body Weight Change



TGI Summary

Therapy	Dose	TGI on Day 21
ERAS-4001	10 mg/kg	3%
	30 mg/kg	30%
	100 mg/kg	77%
	300 mg/kg	101%

- ERAS-4001 was well tolerated at doses ranging from 10 mg/kg p.o. BID to 100 mg/kg p.o. BID (i.e., no dose holidays or mortality)
- ERAS-4001 at 300 mg/kg p.o. BID showed borderline tolerability with 4 out of 6 mice receiving continuous treatment, one mouse receiving a dose holiday due to body weight loss on days 16-21, and one mouse death on day 13
- Observed borderline tolerability may be model and/or study specific; ERAS-4001 at 300 mg/kg p.o. BID was well tolerated in the Panc 04.03 CDX TGI study (no dose holidays or mortality)

ERAS-4001 dosed orally twice daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

ERAS-4001 showed promising PK in mouse, rat, and dog

	PK Parameter	Mouse	Rat	Dog
IV	Dose (mpk)	1.7	2	2.1
	C_0 (nM)	1,722	1,083	1,669
	$T_{1/2}$ (h)	1.9	3	5.8
	V_d (L/kg)	5.16	10.1	14.1
	CL (mL/kg/min)	45.5	70.9	53.1
	$AUC_{0\text{-last}}$ (nM·h)	938	615	827
Oral	Dose (mpk)	30.3	30.9	15.3
	C_{max} (nM)	2,090	584	323
	T_{max} (h)	1.5	4	0.5
	$T_{1/2}$ (h)	1.5	2.3	5.4
	$AUC_{0\text{-last}}$ (nM·h)	4,498	2,562	962
	Bioavailability (F %)	27	27	16

Anticipated key milestones in 2026-2027

Program *Mechanism*	**Trial Name** *Indication*	**Anticipated Milestones**
ERAS-0015 *Pan-RAS molecular glue*	**AURORAS-1** *RASm solid tumors*	• **2H26:** - Initiate monotherapy expansion cohorts - Initiate combination dose escalation cohorts • **1H27:** - Monotherapy expansion data - Combination dose escalation data
ERAS-4001 *Pan-KRAS inhibitor*	**BOREALIS-1** *KRASm solid tumors*	• **2H26:** Preliminary Ph 1 monotherapy data • **2027:** - Initiate monotherapy expansion cohorts - Initiate combination dose escalation cohorts

Compelling investment thesis

EXPERIENCED TEAM WITH TRACK RECORD OF SERIAL SUCCESSES

Seasoned drug developers who have advanced multiple programs from discovery to IND to global approvals

WORLD-CLASS SCIENTIFIC ADVISORY BOARD

Includes leading pioneers in the RAS/MAPK pathway (Shokat, UCSF; Lito, MSKCC; Rodriguez-Viciana, UCL; Cichowski, HMS; Blacklow, HMS; Corcoran, MGH), precision oncology (Demetri, DFCI; Bernards, NKI), and biopharma (Varney, Genentech)

PROMISING PIPELINE TARGETS LARGE, UNDERSERVED MARKETS ACROSS MULTIPLE TUMOR TYPES

Potential to address unmet needs in millions of patients diagnosed annually with RAS/MAPK solid tumors

CLINICAL ADVANCEMENT OF INDUSTRY LEADING RAS-TARGETING FRANCHISE

Potential best-in-class/first-in-class RAS programs comprising ERAS-0015 pan-RAS molecular glue and ERAS-4001 pan-KRAS small molecule inhibitor

MULTIPLE POTENTIAL NEAR-TERM AND LONG-TERM VALUE DRIVERS

Focused clinical development plan with near-term clinical readouts

ERASCA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THANK YOU!

RAS targeting landscape drives importance of identifying development candidates with first-in-class or best-in-class potential

Pan-RAS	Adlai Nortye AN9025	GENFLEET THERAPEUTICS GF-276	Roche RO7673396	ERASCA **ERAS-0015**	REVOLUTION MEDICINES RMC-6236
Pan-KRAS	KESTREL THERAPEUTICS KST-6051	AMGEN AMG-410	AstraZeneca JAB-23E73	ERASCA **ERAS-4001**	
		BBOT BBO-11818	Pfizer PF-07934040 (G12X)	Alterome THERAPEUTICS ALTA-3263	
Mutant Selective[1]	Lilly LY3962673 (G12D)	Quanta THERAPEUTICS INC. QTX3034 (KRAS G12D)	Incyte INCB161734 (G12D)	REVOLUTION MEDICINES RMC-9805 (G12D)	
	Roche RG6620/GDC-7035 (G12D)	Quanta THERAPEUTICS INC. QTX3054 (KRAS G12V)	VERASTEM ONCOLOGY VS-7375 (G12D)	Boehringer Ingelheim BI 3706674 (wt amp, G12V)	
	ARVINAS ARV-806 (G12D degrader; IV)	Tyligand TSN1611 (G12D)	astellas ASP3082 (G12D degrader; IV)	REVOLUTION MEDICINES RMC-5127 (G12V)	

Note: Select clinical-stage coopetitors shown based on public disclosures; list is not intended to be exhaustive; updated as of May 2026

[1] Mutant selective beyond KRAS G12C inhibitors

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ERAS-0015's higher CYPA binding affinity may be a differentiator from RMC-6236, demonstrating potential best-in-class profile

Assay	ERAS-0015 (nM)	RMC-6236 (nM)	Binding affinity difference: ERAS-0015/ RMC-6236
SPR K_D	4.5	92	**21x**
ITC K_D	5.3	44.1	**8x**

8-21x higher binding affinity to cyclophilin A (CYPA) may enable more potent RAS inhibition

These data were generated in head-to-head in vitro assay and/or in vivo experiments
SPR: surface plasmon resonance; K_D: equilibrium dissociation constant; ITC: isothermal titration calorimetry
These data were generated in head-to-head in vitro assay and/or in vivo experiments

ERAS-0015 demonstrated significantly more potent inhibition of cell growth across KRAS mutant cell lines vs. RMC-6236

Mutation	Tumor type	Cell line	ERAS-0015 cell growth inhibition (nM)	RMC-6236 cell growth inhibition (nM)	ERAS-0015:RMC-6236 Fold Potency
KRAS G12C	NSCLC	H358 (adagrasib-resistant)	0.8	3.6	**4.5x**
	NSCLC	LU99	1.4	5.4	**3.9x**
KRAS G12D	NSCLC	A-427	13.3	59.2	**4.5x**
	CRC	SW620	0.2	1.3	**6.5x**
	CRC	GP2d	0.9	4.6	**5.1x**
	PDAC	AsPc-1	2.0	26.7	**13.4x**
	PDAC	HPAC	4.8	15.5	**3.2x**
	PDAC	PK-59	10.7	10.7	**1x**
	PDAC	KP-4	5.0	19.7	**3.9x**
	PDAC	Panc 04.03	5.7	26.4	**4.6x**
KRAS G12V	Lung Cancer	NCI-H727	0.4	1.7	**4.3x**
	Lung Cancer	NCI-H441	1.4	16.7	**11.9x**
	CRC	SW480	0.8	6.8	**8.5x**
	PDAC	CAPAN-1	2.5	7.1	**2.8x**
	Ovarian leiomyosarcoma	RKN	0.7	1.6	**2.3x**
KRAS G12R	PDAC	PSN-1	5.3	17.1	**3.2x**
KRAS G12S	NSCLC	A-549	4.1	38.3	**9.3x**
KRAS Q61R	PDAC	Panc 02.13	7.4	44.3	**6x**
KRAS G13D	CRC	LoVo	2.8	1.5	**0.5x**
	CRC	HCT-116	5.5	26.2	**4.8x**
KRAS WT Amplified	Gastric	MKN-1	13.8	55.8	**4x**
EGFR L858R / T790M	NSCLC	H1975	6.5	11.4	**1.8x**
MET amplified	NSCLC	EBC-1	4.4	16.9	**3.8x**
BRAF V600E	Melanoma	A375	>6,000	>6,000	**N/A**

Sub-nM to nM potency against multiple KRAS wildtype, KRAS mutant, and RTK altered cell lines

RTK: receptor tyrosine kinase
These data were generated in head-to-head in vitro assay and/or in vivo experiments.

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ERAS-0015 demonstrated comparable antitumor activity to RMC-6236 at 1/10th to 1/5th of the dose in multiple in vivo mouse models

Model	KRAS Mutation	Tumor Origin	Dose achieving tumor regression[1]	
			ERAS-0015	RMC-6236
PK-59 CDX	G12D	PDAC	**0.3 mg/kg**	3 mg/kg
NCI-H727 CDX	G12V	NSCLC	**1 mg/kg**	10 mg/kg
SW620 CDX	G12V	CRC	**3 mg/kg**	25 mg/kg
PSN-1 CDX	G12R	PDAC	**5 mg/kg**	25 mg/kg
CT26 Syngeneic	G12D	CRC	**3 mg/kg**	NA[2]

[1] Achieving >100% tumor growth inhibition (TGI)
[2] Not achieved at any dose tested
These data were generated in head-to-head in vitro assay and/or in vivo experiments.

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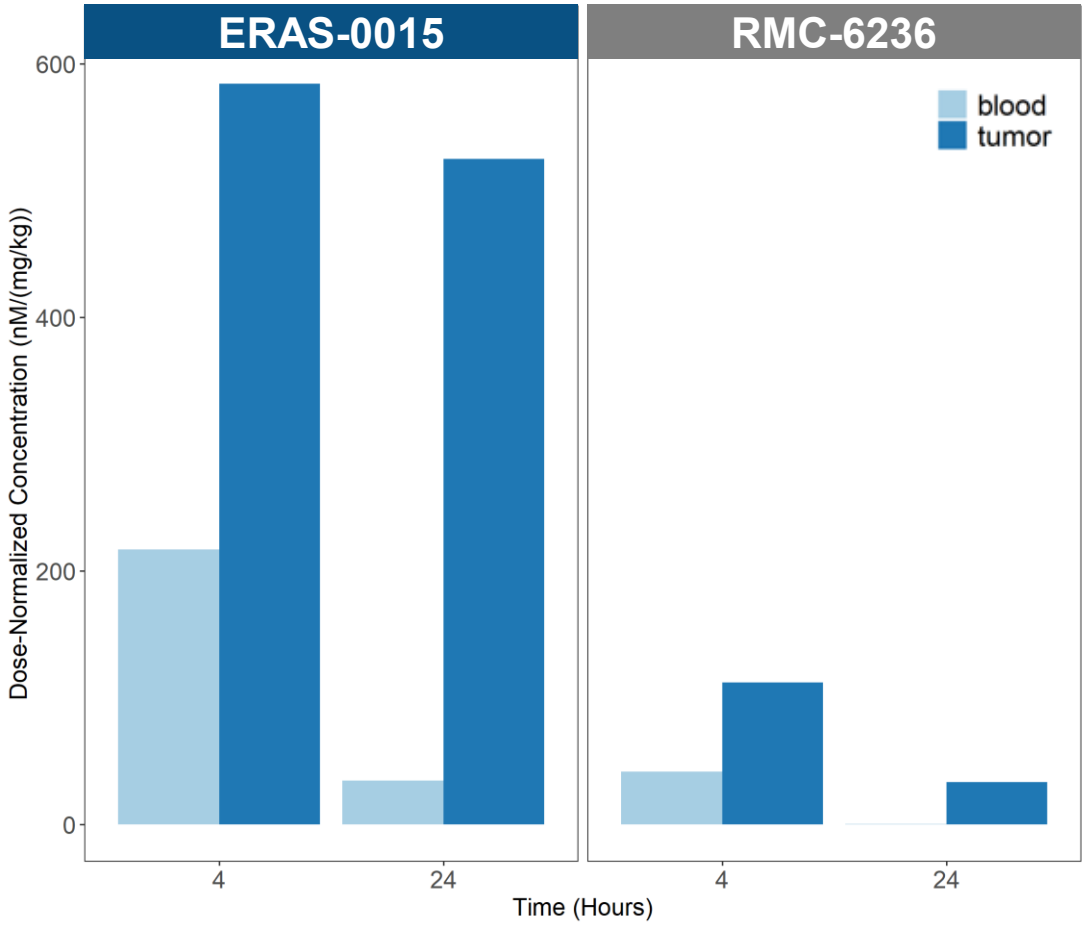
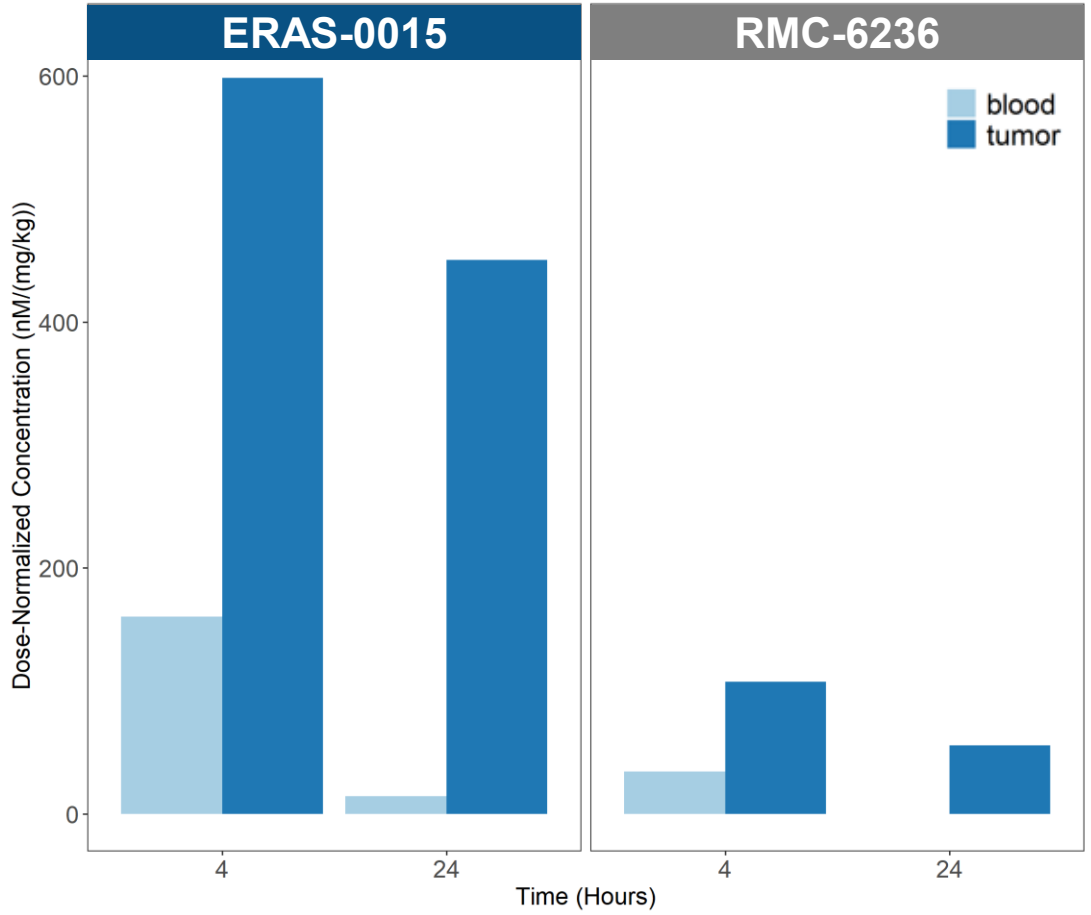
ERAS-0015 demonstrated preferential tumor distribution and longer residence time vs. RMC-6236 in vivo

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
These data were generated in head-to-head in vitro assay and/or in vivo experiments

ERAS-0015 showed promising PK in mouse, rat, dog, and monkey

		Mouse		Rat		Dog		Monkey	
		ERAS-0015	RMC-6236	ERAS-0015	RMC-6236	ERAS-0015	RMC-6236	ERAS-0015	RMC-6236
IV	Dose (mpk)	**1**	1	**1**	1	**1**	1	**1**	No Data
	$T_{1/2}$ (h)	**5.0**	1.7	**5.7**	1.5	**24.5**	7.6	**15.2**	No Data
	Vd_{ss} (L/kg)	**5.3**	1.9	**1.9**	1.9	**3.8**	3.7	**1.8**	No Data
	CL (mL/kg/min)	**12.8**	15.6	**4.6**	19.2	**1.9**	7.9	**1.6**	No Data
	$AUC_{0\text{-last}}$ (nM*h)	**1,337**	1,274	**4,125**	1,123	**7,910**	2,630	**11,479**	No Data
Oral	Dose (mpk)	**10**	10	**10**	10	**5**	5	**5**	No Data
	C_{max} (nM)	**745**	1,443	**1,620**	339	**472**	377	**723**	No Data
	$T_{1/2}$ (h)	**6.3**	1	**6.1**	2.5	**22.4**	7.8	**12.3**	No Data
	$AUC_{0\text{-last}}$ (nM*h)	**6,786**	4,467	**15,213**	1,427	**8,720**	2,755	**10,004**	No Data
	Bioavailability (F%)	**48%**	33%	**38%**	14%	**22%**	21%	**17%**	No Data

These data were generated in head-to-head assay and/or in vivo experiments

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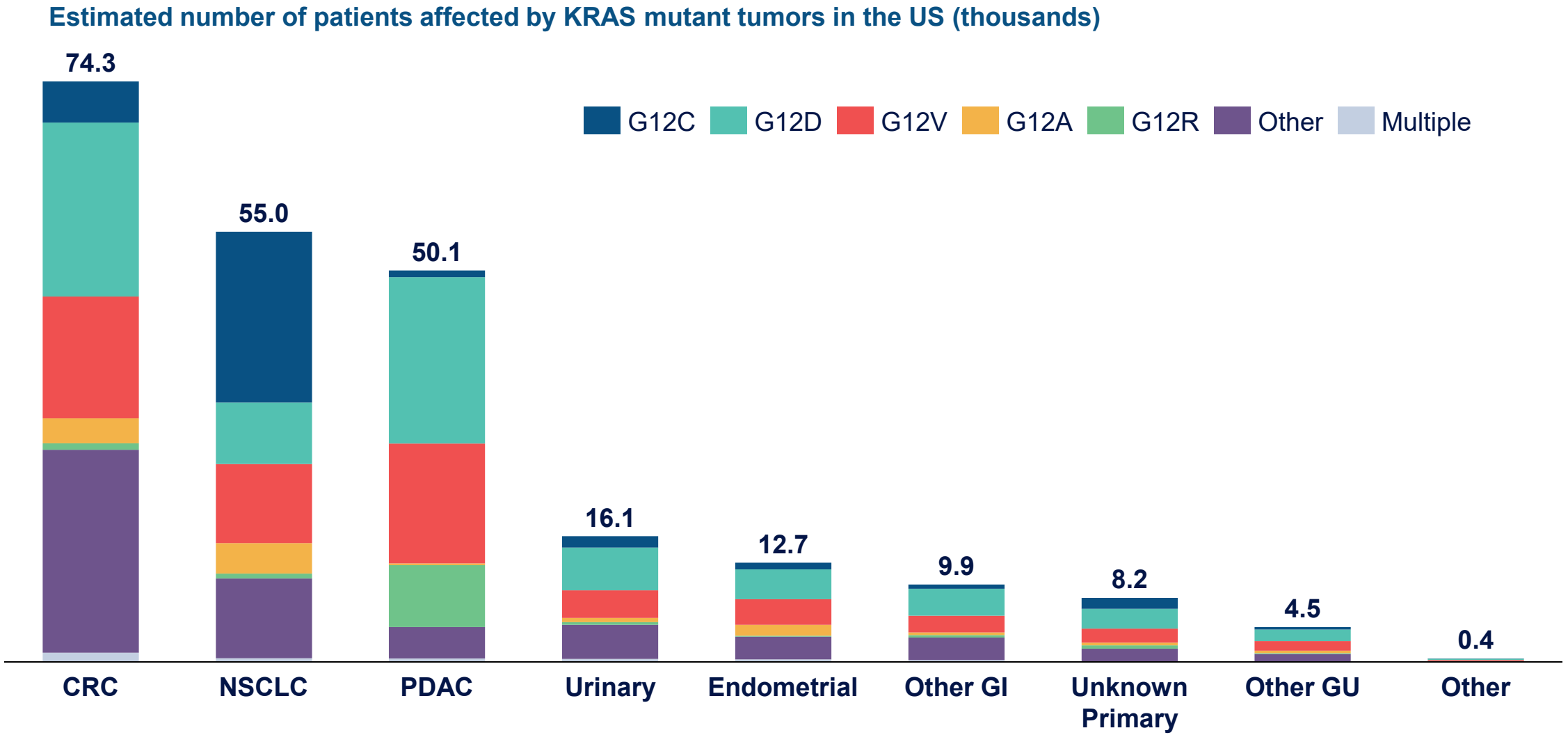
KRAS alterations found most commonly in CRC, PDAC and NSCLC



Adapted from Lee J., Sivakumar S., Schrock A., et al. "Comprehensive pan-cancer genomic landscape of KRAS altered cancers and real-world outcomes in solid tumors." NPJ Precision Oncology, 2022. PMID: 36494601.
CRC: colorectal cancer; NSCLC: non-small cell lung cancer; PDAC: pancreatic ductal adenocarcinoma; GI: gastrointestinal; GU: genitourinary

Erasca believes that the claims in RVMD's April 24, 2026 letter[1] are without merit

Claim 1: Patent Infringement under the Doctrine of Equivalents

- **No literal infringement** has been claimed by RevMed
- Courts can be cautious when applying the doctrine of equivalents in the biotech industry, since a small structural difference can lead to (among other things):
  - **Different binding**
  - **Different PK/PD**
  - **Different safety or efficacy**
- The doctrine of equivalents is a **narrow, fact-intensive analysis** and we believe that **ERAS-0015 is a distinct scientific approach from all prior art that produces differentiated results**

Claim 2: Trade Secret Misappropriation by a Third Party (not Erasca)

Medshine[2] is one of several companies that filed patent applications following the publication of the RMC-6236 patent

First Disclosure of RMC-6236
WO2022060836 (Revolution Medicines)
PRIORITY DOC FILED Sep 15, 2020 and PUBLISHED Mar 24, 2022

Patent Application
WO2023025832 (Hoffmann-La Roche)
PRIORITY DOC FILED Jun 13, 2022

Patent Application
CN 117720554 (Adlai Nortye)
PRIORITY DOC FILED Sep 19, 2022

Patent Application
WO2023086341 (Biomea Fusion)
PRIORITY DOC FILED Sep 19, 2022

First Priority Patent Application – Discloses Compounds Analogous to ERAS-0015
CN 202211213222.2 (Medshine)
PRIORITY DOC FILED Sep 29, 2022

Patent Application
WO2024153208 (Genfleet Therapeutics)
PRIORITY DOC FILED Jan 19, 2023

Second Priority Patent Application – First Disclosure of ERAS-0015
CN 202310132699.6 (Medshine)
PRIORITY DOC FILED Feb 17, 2023

At least 7 other companies have initial patent filings with priority dates in 2022 or 2023 that disclose compounds that modified RMC-6236 or the RMC-6236 scaffold

Of these companies, **4 (shown here) have a priority date that is before when Medshine made its initial filing that disclosed ERAS-0015 (none of the four applications claim ERAS-0015)**

[1] See Erasca Form 8-K (filed April 27, 2026) for details; [2] Medshine is a business division of WuXi AppTec. Medshine assigned ERAS-0015 to Guangzhou Joyo Pharmatech Co. (Joyo), and Joyo licensed ERAS-0015 to Erasca