ERASCA

On a Journey to Erase Cancer

Erasca Corporate Presentation

November 2026



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

Disclaimer: Forward Looking Statements & Market and Clinical Data

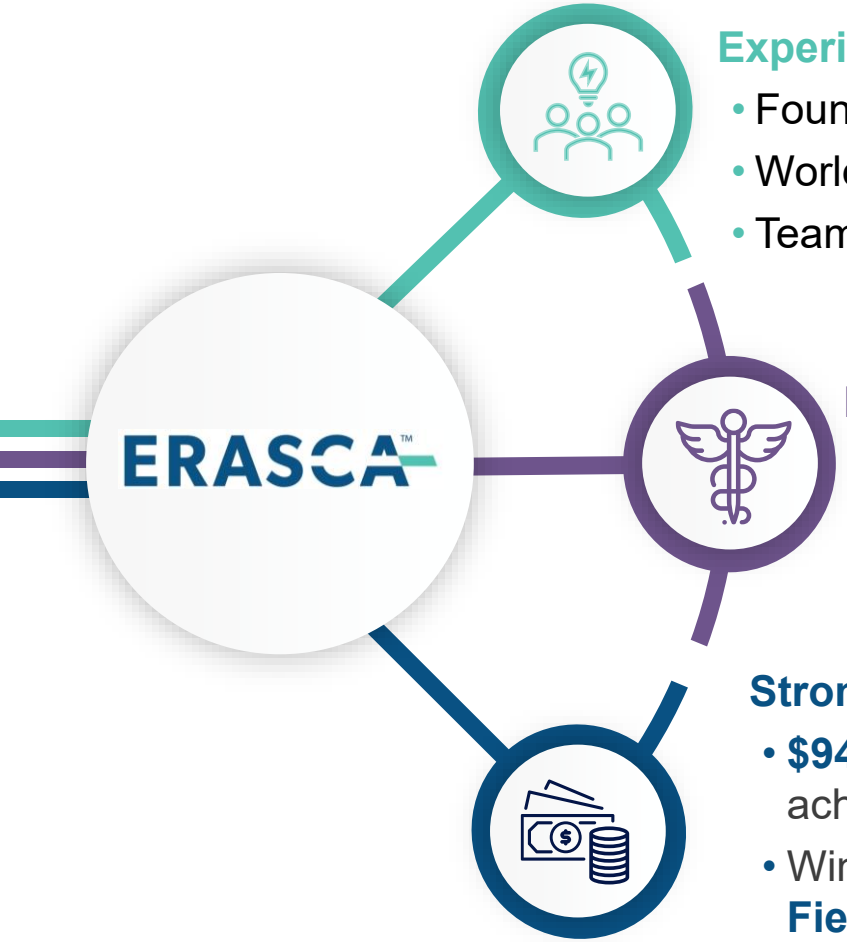
We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation are forward-looking statements, including statements regarding: our future results of operations and financial position, business strategy, 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; 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; interim, 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; FTD may not result in a more expedited development or regulatory review process, and such a designation does not increase the likelihood that ERAS-0015 will receive marketing approval in the United States; 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 is 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; results from preclinical studies not necessarily being predictive of future results; 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; our planned potentially registration-enabling trials may be delayed based on Food and Drug Administration (FDA) feedback or requirements, as the FDA retains broad discretion to require additional clinical data prior to the conduct of a registrational trial or submission for regulatory approval; even if our planned trials are successful, they may not support regulatory approval; unfavorable results from preclinical studies or clinical trials; the inability to realize any benefits from our current licenses, acquisitions, and 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, including our ability to successfully defend against allegations raised by, or any future litigation initiated by, Revolution Medicines (RevMed) that ERAS-0015 infringes patents held by RevMed or was derived from RevMed trade secrets; the sufficiency of our cash, cash equivalents, and marketable securities; we may use our capital resources sooner than we expect; 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 daraxonrasib (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

- **$948M in cash, cash equivalents, and marketable securities[2]**; sufficient capital to achieve our corporate milestones
- Winner of **2026 BioTech Breakthrough R&D Platform of the Year** and named 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 September 30, 2026
FIC: first-in-class; BIC: best-in-class; RASm: RAS mutated; KRASm: KRAS mutated

ERASCA

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

BWH BRIGHAM AND WOMEN'S HOSPITAL



Ryan Corcoran, MD, PhD

World expert in the RAS/MAPK pathway in CRC, elucidating adaptive and acquired resistance mechanisms to RAS/MAPK pathway inhibitors, including pan-(K)RAS inhibitors

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 launch 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 UCL

ERASCA

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-0015 granted US FDA Fast Track Designation (FTD)** for the treatment of patients with metastatic PDAC

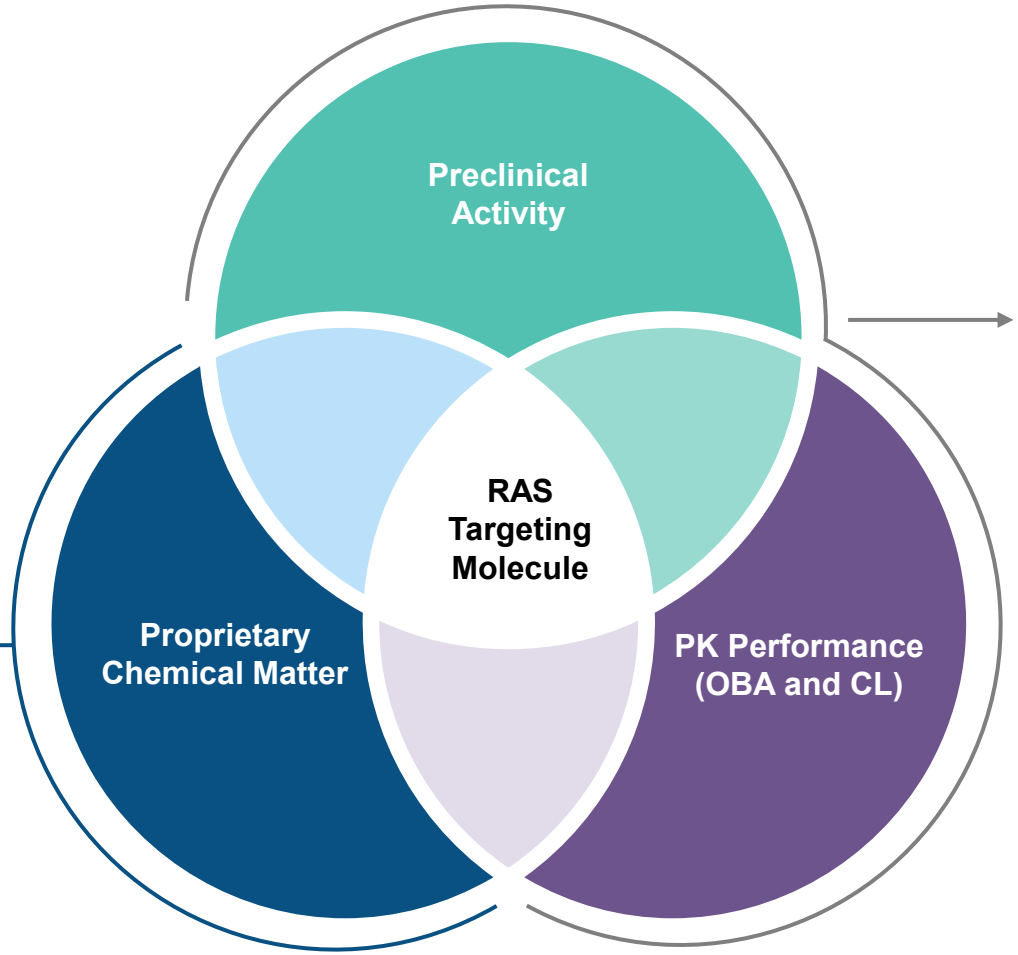
small molecule small molecule molecular glue

PDAC: pancreatic ductal adenocarcinoma
Note: Pipeline also includes ERAS-12, our EGFR D2/D3 bispecific antibody program, for which we are exploring strategic alternatives

ERASCA

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: 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

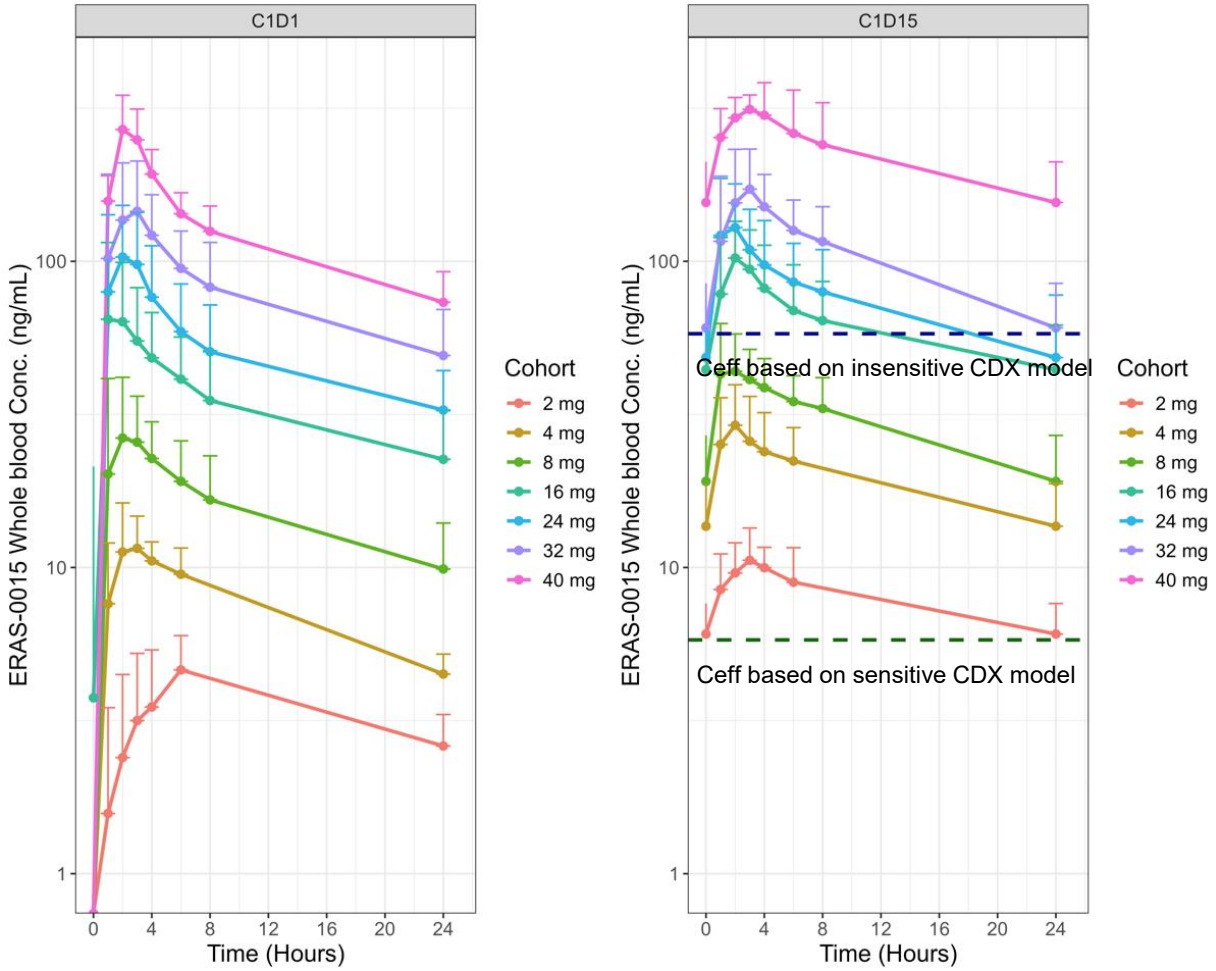
Preclinical Differentiation[1]

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

ERASCA

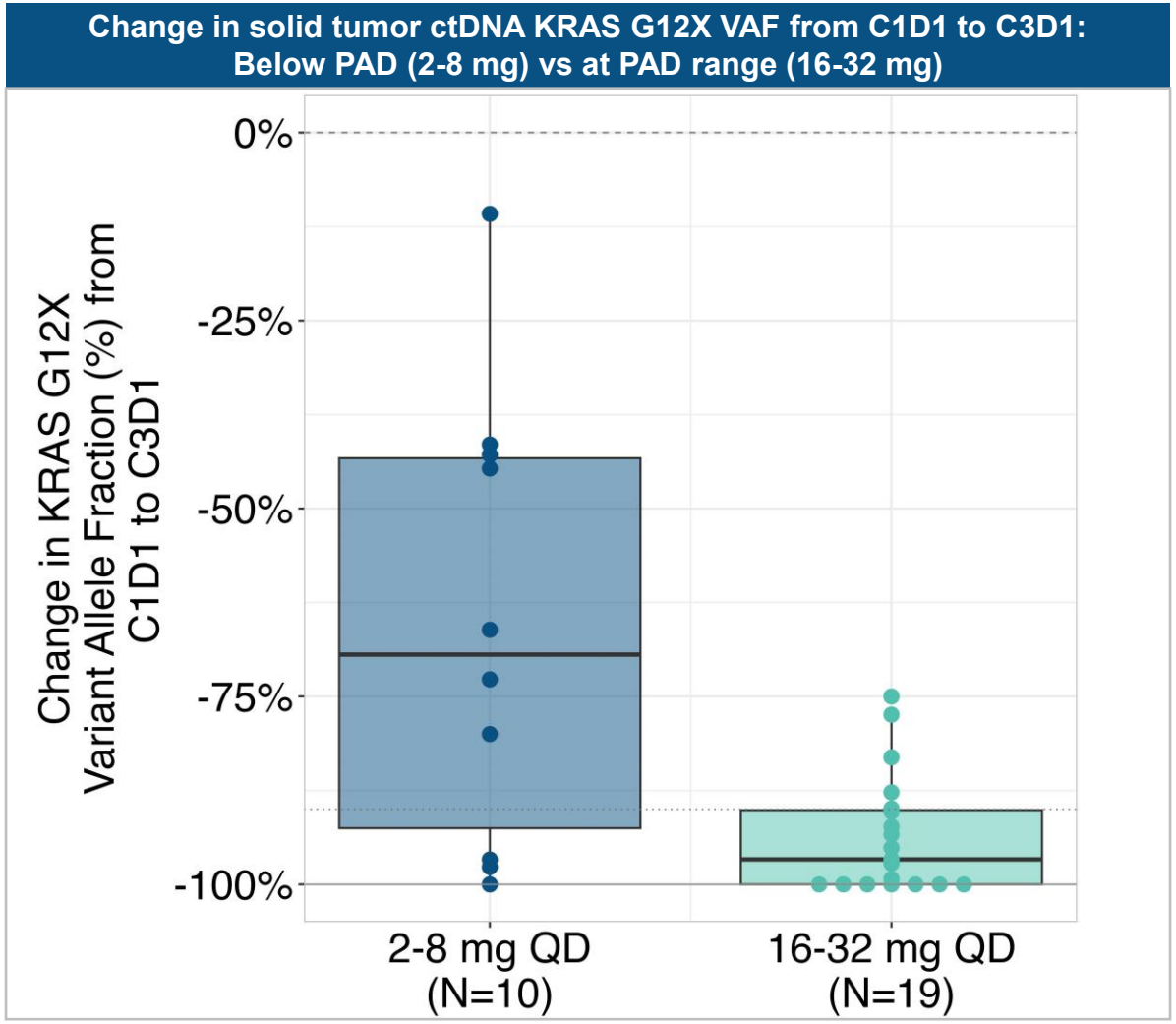
US Trial: Dose-dependent increase in PK exposure up to 40 mg MAD with no exposure plateau observed; PAD defined based on steady-state exposures that exceeded target threshold



- **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

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

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



100% of patients (19/19) at PAD showed at least **75% reduction of KRAS G12X variant allele fraction**, including 74% (14/19) showing at least a 90% 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







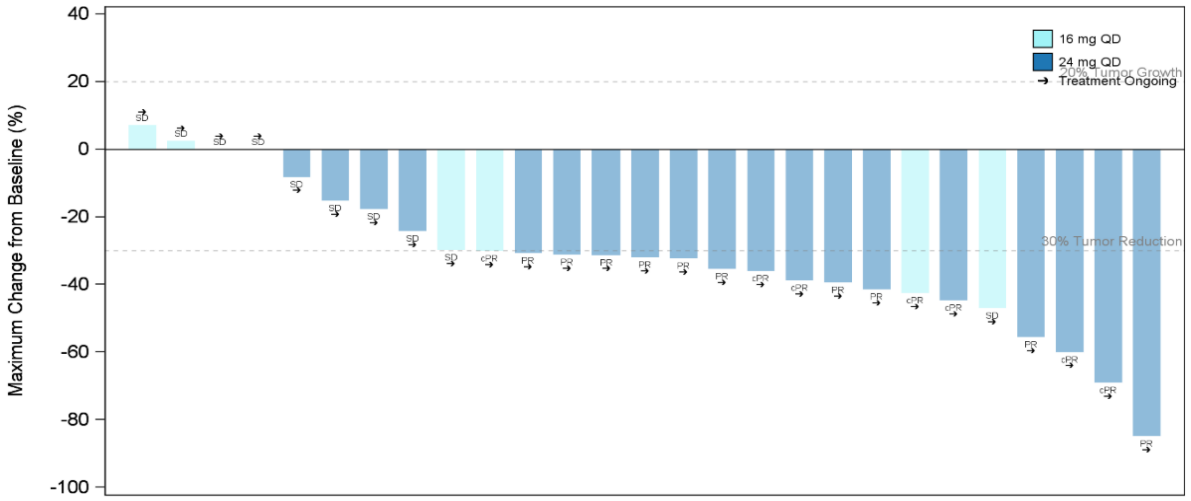
	NSCLC	PDAC	CRC
Relative anticipated sensitivity in the clinic to pan-RAS inhibition	**High**	**Med**	**Low**
Pan-RAS response dynamics	Faster, deeper responses	Up to 50% of patients may exhibit delayed RECIST responses (detectable after ≥2 scans, i.e., ~14 weeks)	Primary and adaptive resistance, often 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

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

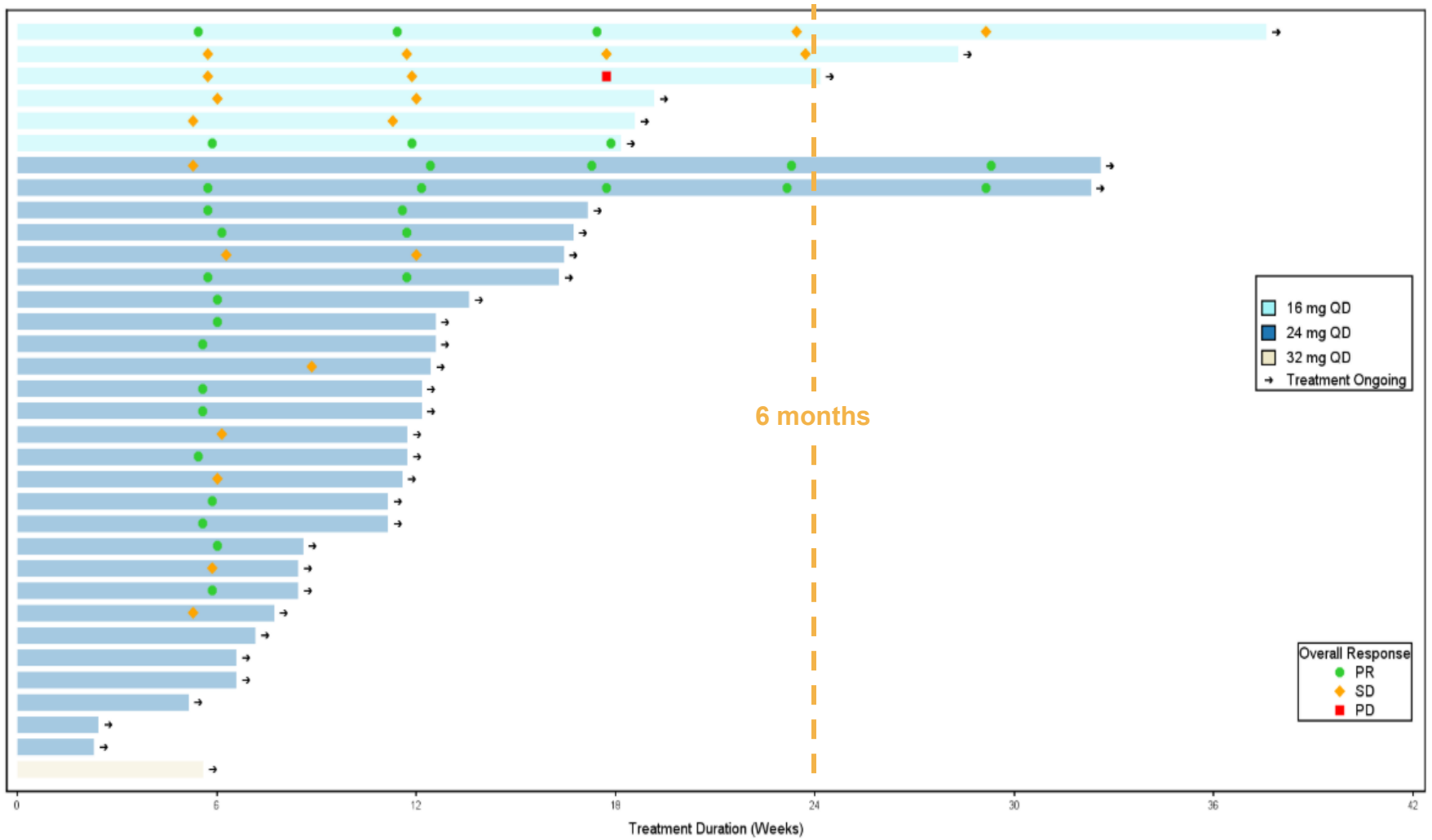
DCO Feb 2026

[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]



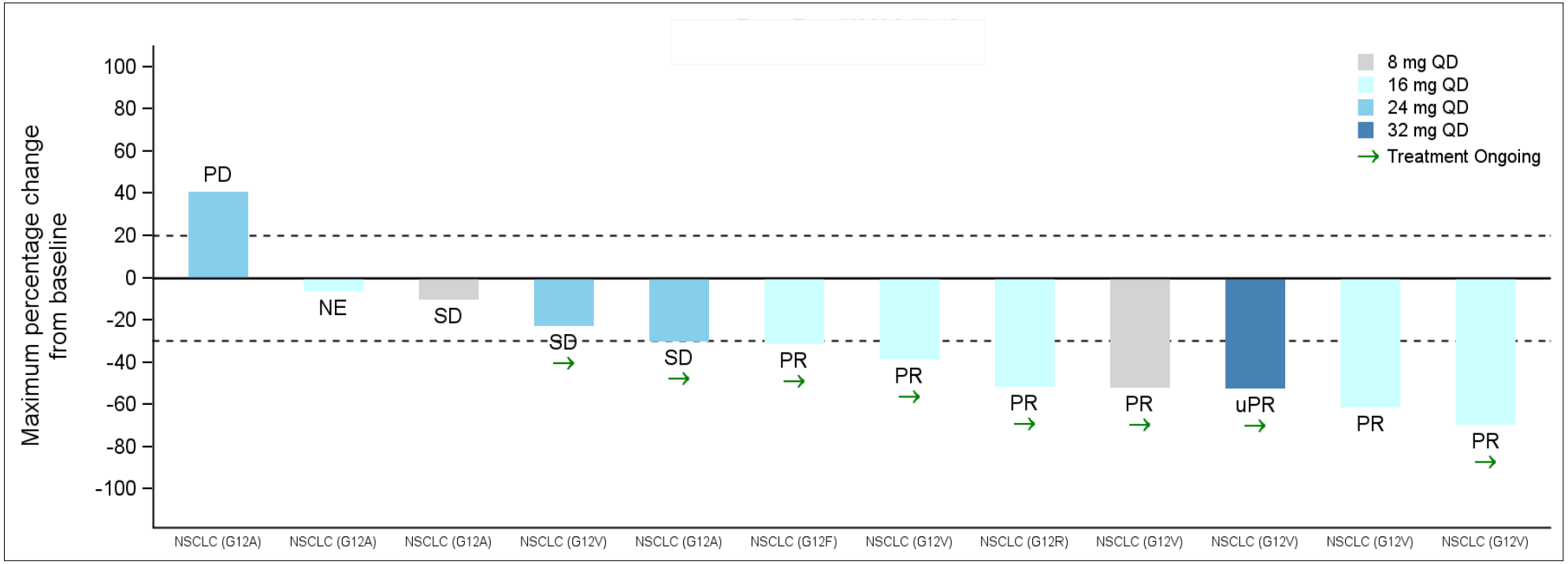
100% of responders – including all patients with uPRs – remain on treatment

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

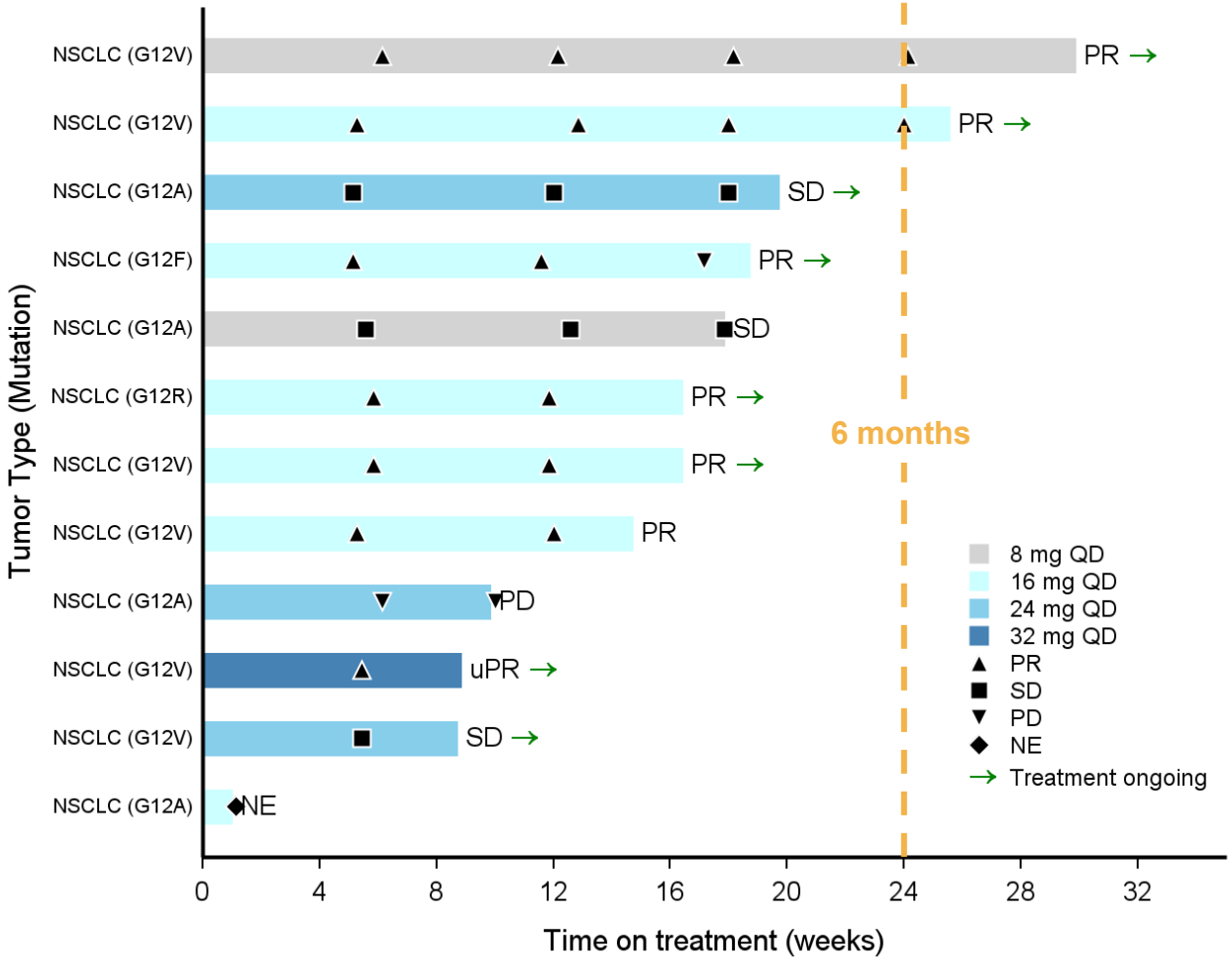
71% uORR observed in post-ICI/platinum (2/3L) KRAS G12X

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

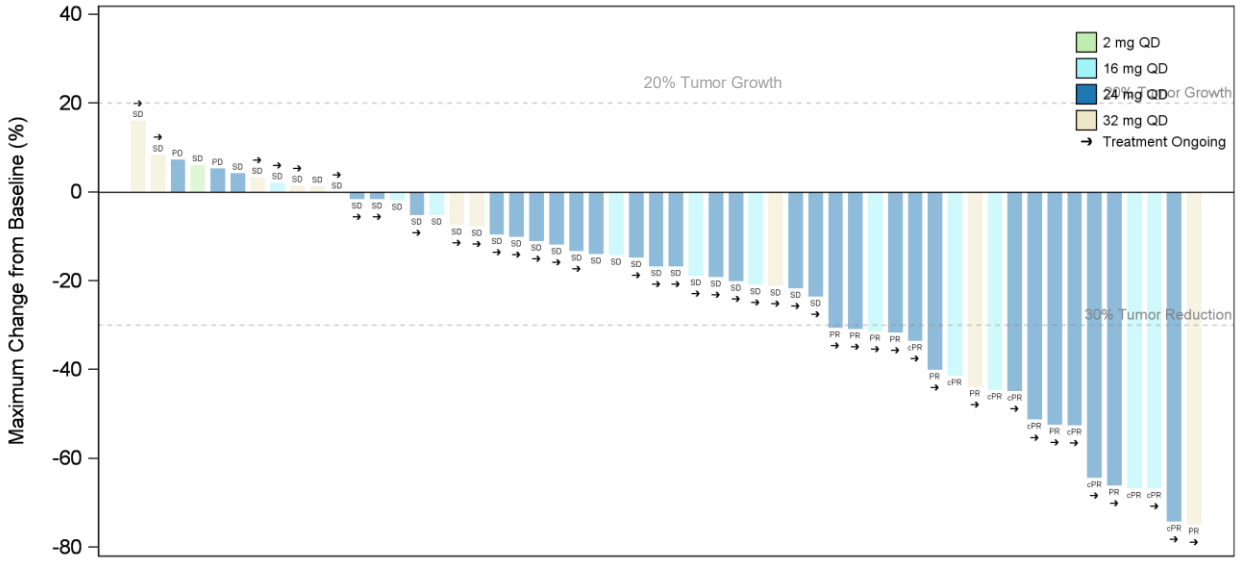
Reinforces the safety, tolerability and potential durability of response of ERAS-0015

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.

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

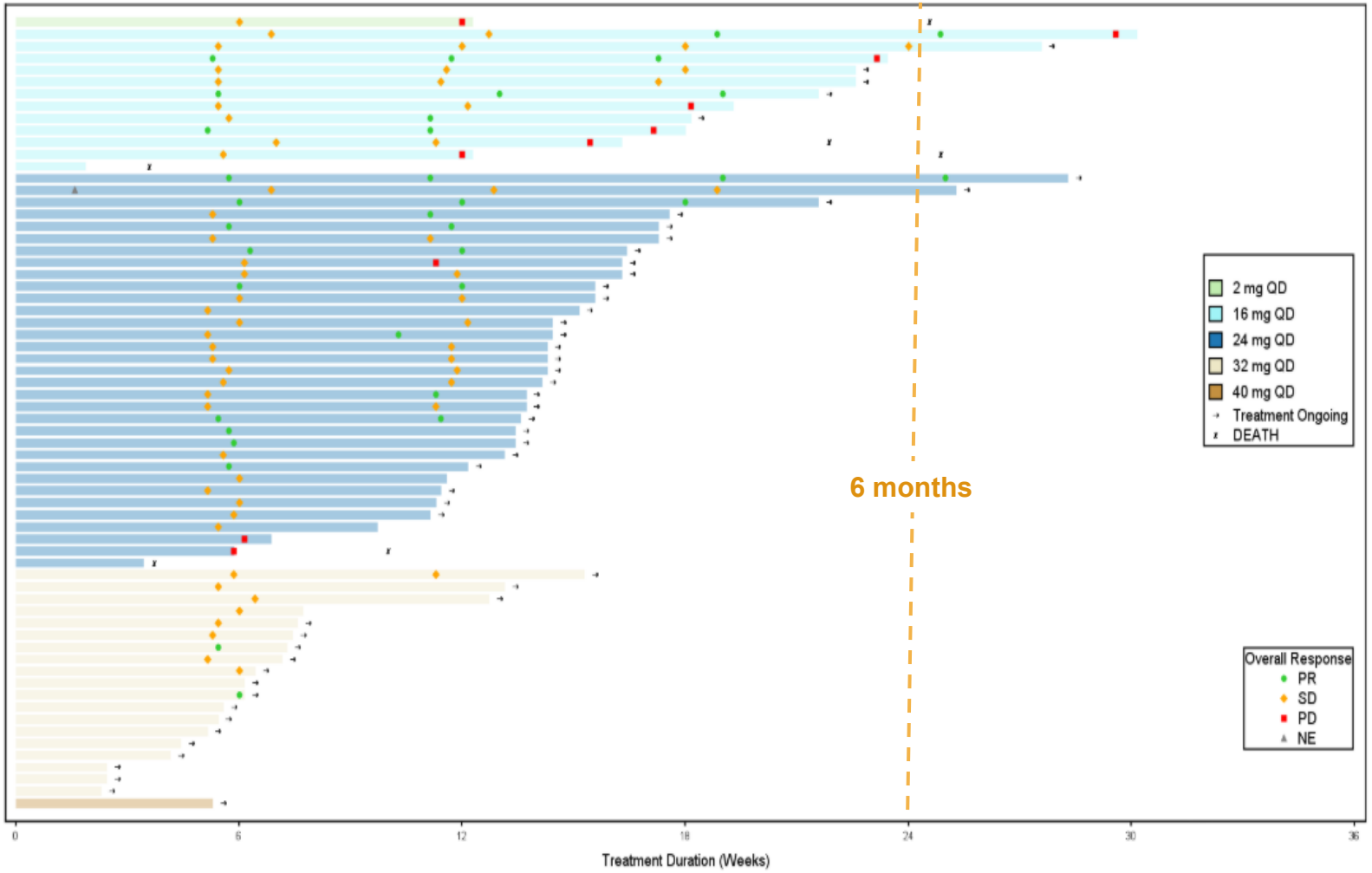
[1] Efficacy evaluable analysis set: Patients with at least one post-dose tumor assessment

[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

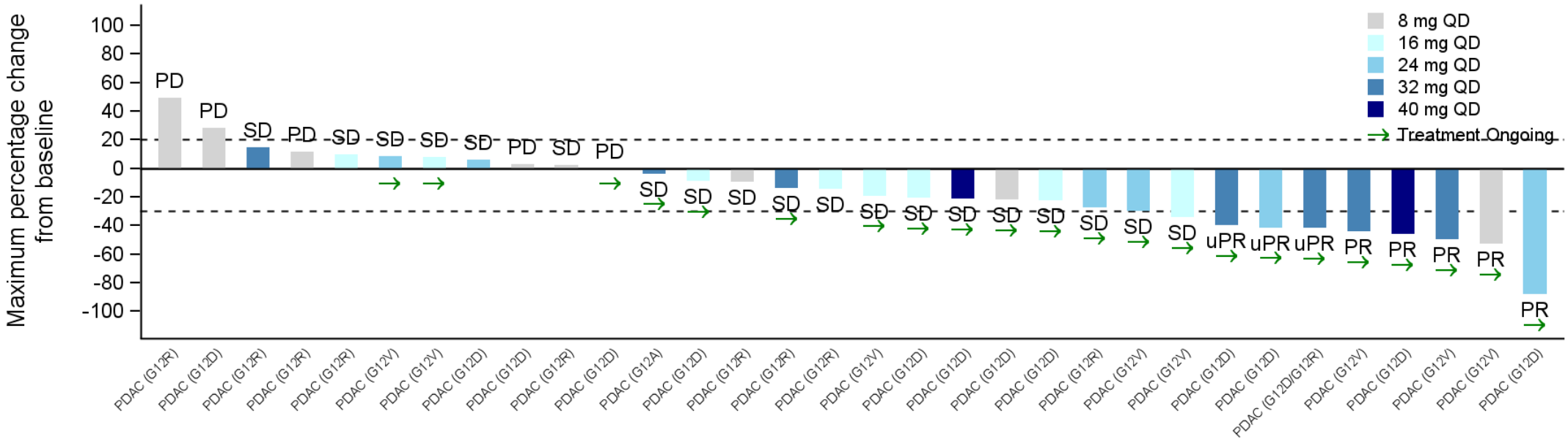
Reinforces the safety, tolerability and durability of response of ERAS-0015

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, including 57% uORR at 32 mg



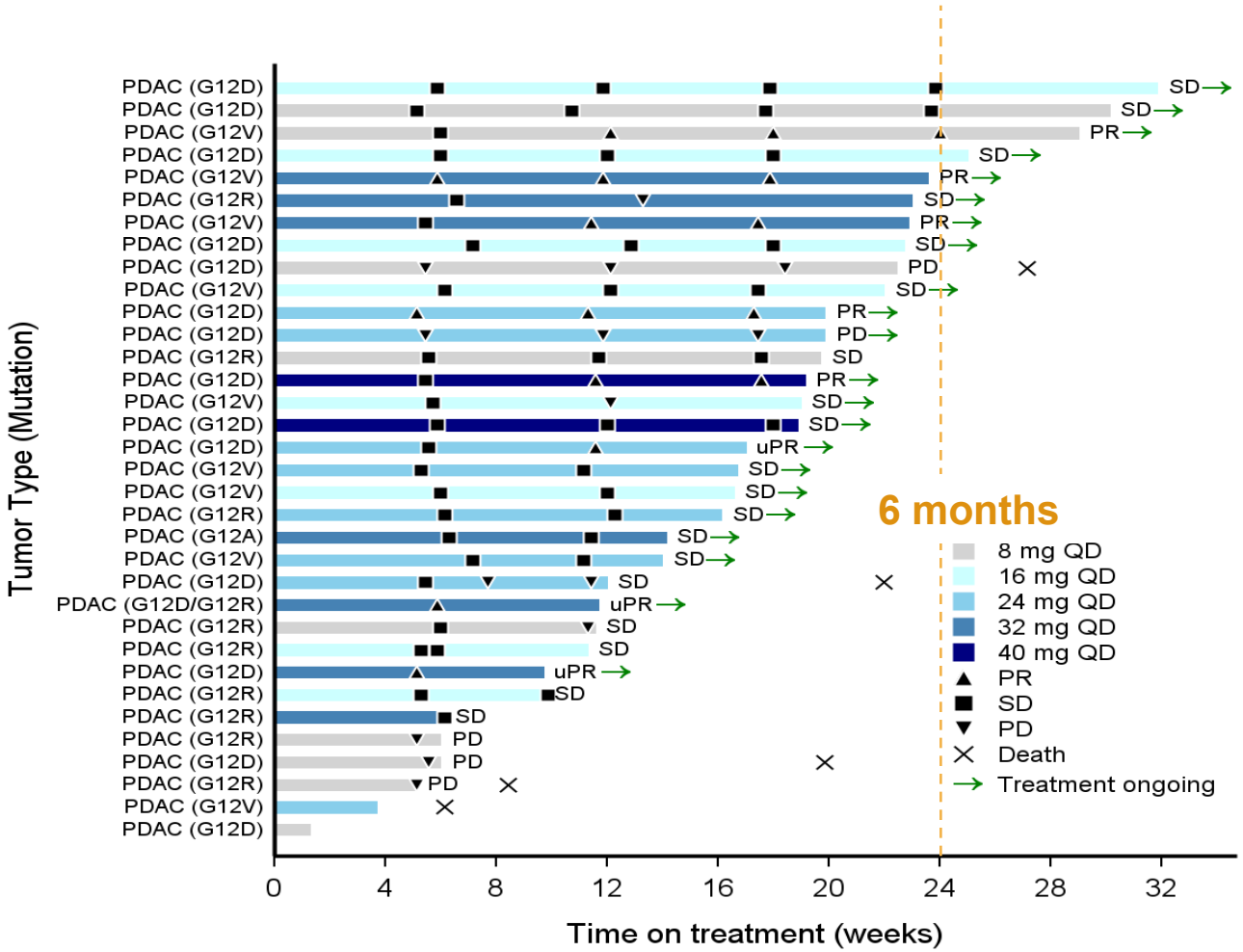
ERAS-0015 2L+ KRAS G12X PDAC[1]	8 mg QD N=9	16 mg QD N=8	24 mg QD N=8	32 mg QD N=7	40 mg QD N=2
uORR[1], n (%)	1 (11)	0	2 (25)	4 (57)	1 (50)
DCR, n (%)	4 (44)	8 (100)	6 (75)	7 (100)	2 (100)

Of the patients treated at RDEs of 24 mg and 32 mg:

- **27% were 2L**
- **73% were 3L+**

[1]uORR includes confirmed and unconfirmed responses; DCO 25May2026; 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; uPR: unconfirmed PR; PR: partial response; PDAC: pancreatic ductal adenocarcinoma; PD: progressive disease; QD: once daily; SD: stable disease; RDEs: recommended doses for expansion

US trial: Time on treatment for patients with 2L+ KRAS G12X PDAC



All patients with either confirmed or unconfirmed responses remain on treatment

At RDE of 32 mg QD, 6 of 7 enrolled patients remained on treatment; at RDE of 24 mg QD, 6 of 8 enrolled patients remained on treatment

DCO 25May2026; PDAC=pancreatic ductal adenocarcinoma; PD=progressive disease; PR=partial response; QD=once daily; RDE=recommended dose for expansion; 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 PR for confirmed PR or uPR for unconfirmed PR.
Note: see Disclaimer slide regarding Cross-Study Comparisons.

US trial: 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
- No DLTs observed in the 16 mg cohort during dose escalation; enrolling backfill
- Combo dose escalation data anticipated in H1 2027

24 mg ERAS-0015 plus panitumumab (at approved dose) combo cohort enrolling

Toxicity manageable to date with AEs consistent with the known profile of ERAS-0015 and panitumumab (per investigator feedback)

[1] As of 6July2026
DLT: dose-limiting toxicity

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=72)					
TRAEs, n (%)	**Grade 1**	**Grade 2**	**Grade 3[1]**	**Grade 4**	**All Grades**
Rash[2], n (%)	37 (51)	13 (18)	2 (3)	0	52 (72)
Diarrhea, n (%)	17 (24)	5 (7)	1 (1)	0	23 (32)
Stomatitis, n (%)	9 (13)	3 (4)	2 (3)	0	14 (19)
Nausea, n (%)	9 (13)	1 (1)	0	0	10 (14)
TRAEs leading to dose interruptions, n (%)					9 (13)
TRAEs leading to dose reductions, n (%)					6 (8)
TRAEs leading to dose discontinuations, n (%)					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 25May2026

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=72)				
TRAEs, n (%)	**16 mg QD (N=16)**	**24 mg QD (N=28)**	**32 mg QD (N=28)**	**16-32 mg (N=72)**
Rash[1], n (%)	13 (81)	21 (75)	18 (64)	52 (72)
Diarrhea, n (%)	5 (31)	11 (39)	7 (25)	23 (32)
Stomatitis, n (%)	4 (25)	5 (18)	5 (18)	14 (19)
Nausea, n (%)	3 (19)	3 (11)	4 (14)	10 (14)
TRAEs leading to dose interruptions, n (%)	1 (6)	6 (21)	2 (7)	9 (13)
TRAEs leading to dose reductions, n (%)	1 (6)	2 (7)	3 (11)	6 (8)
TRAEs leading to dose discontinuations, n (%)	0	0	0	0

[1] 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 25May2026

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US trial: Relative Dose Intensity supports tolerability of doses across the PAD

Relative Dose Intensity (RDI)				
Patients with RASm NSCLC and PDAC Treated at PAD (16-32mg) ERAS-0015 (N=72)				
	16 mg QD (N=16)	24 mg QD (N=28)	32 mg QD (N=28)	16-32 mg QD (N=72)
RDI of ERAS-0015 (%)				
n[1]	16	27	27	70
Mean (SD)	101.3 (13.3)	95.0 (11.3)	93.5 (13.7)	95.9 (12.9)
Median	100.0	100.0	100.0	100.0

DCO 25May2026

[1] Data were unavailable for subset of patients who were therefore not included in the RDI calculation

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

Emerging efficacy differentiation from Ph 1 clinical data strengthens rationale for potential registration-enabling trials in PDAC and NSCLC

PDAC

	ERAS-0015			
	Apr 2026		25 May 2026	
Dose	N	uORR	N	uORR
24 mg (2L+)	7	14%	8	25%
32 mg (2L+)	4	50%	7	57%
40 mg (2L+)	2	0%	2	50%
24-32 mg (2L+)[4]	11	27%	15	40%

~40-57%

uORR for ERAS-0015

in 2L+ KRAS G12X PDAC (US)

	RMC-6236			
	ENA 2024[1]		Sep 2025[2]	
Dose	N	uORR	N	ORR
300 mg (2L)	-	-	26	35%
160-300 mg (2L+)[4]	105	25%	-	-

~25-35%

uORR/ORR for RMC-6236

in 2L+/2L KRAS G12X PDAC (US)

NSCLC

	ERAS-0015		
	Apr 2026		
Dose	N	uORR	
16-32 mg	16	75%	

75%

uORR for ERAS-0015

in Post-ICI/plat (2/3L) KRAS G12X NSCLC (US, CN)

	RMC-6236		
	JTO 2025[3]		
Dose	N	ORR	
120-220 mg	40	38%	

38%

ORR for RMC-6236

in Post-ICI/plat (2/3L) KRAS G12X NSCLC (US)

PDAC: pancreatic ductal adenocarcinoma; NSCLC: non-small cell lung cancer; uORR: unconfirmed overall response rate; ORR: overall response rate; ICI: immune checkpoint inhibitor; plat: platinum

[1]Wolpin et al, ENA 2024; [2]Revolution Medicines Corporate Presentation, Sep 2025; [3]Punekar, JTO 2025; [4]27% of patients were 2L for ERAS-0015, 40% of patients were 2L for RMC-6236

Note: Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons

ERASCA

Emerging safety and tolerability differentiation from Ph 1 clinical data strengthens rationale for potential registration-enabling trials in PDAC and NSCLC

	ERAS-0015		RMC-6236				
	Ph 1 16-32 mg Apr 2026	**Ph 1 16-32 mg** 25 May 2026	**Ph 1 ≥80mg** (ESMO 2023)[1]	**Ph 1 160-300mg** (ENA 2024)[2]	**Ph 1 300mg** (NEJM 2026)[3]	**Ph 1 All doses** (NEJM 2026)[3]	**Ph 3 300 mg** (NEJM 2026)[4]
n	43	72	111	127	83	168	241
All Grade							
Rash	67%	72%	81%	91%	90%	88%	86%
Diarrhea	30%	32%	39%	48%	52%	46%	58%
Stomatitis	16%	19%	22%	31%	54%	40%	53%
Nausea	12%	14%	46%	43%	39%	42%	47%
Grade 3+							
Rash	2%	3%	6%	8%	7%	7%	14%
Diarrhea	0%	1%	1%	2%	4%	3%	5%
Stomatitis	0%	3%	2%	3%	4%	4%	12%
Nausea	0%	0%	0%	0%	0%	0%	2%
Dose Interruptions	12%	13%	-	34%	43%	37%	56%
Dose Reductions	7%	8%	14%	19%	30%	21%	36%
Dose Discontinuations	0%	0%	1%	0%	0%	1%	1%

Cell shading reflects the relative incidence of each adverse event within that AE category only; color intensity should not be compared across different AE categories.

[1]Arbour et al, ESMO 2023; [2]Wolpin et al, ENA 2024; [3]Wolpin et al, NEJM 2026; [4]O'Reilly et al, NEJM 2026
Note: Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons

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

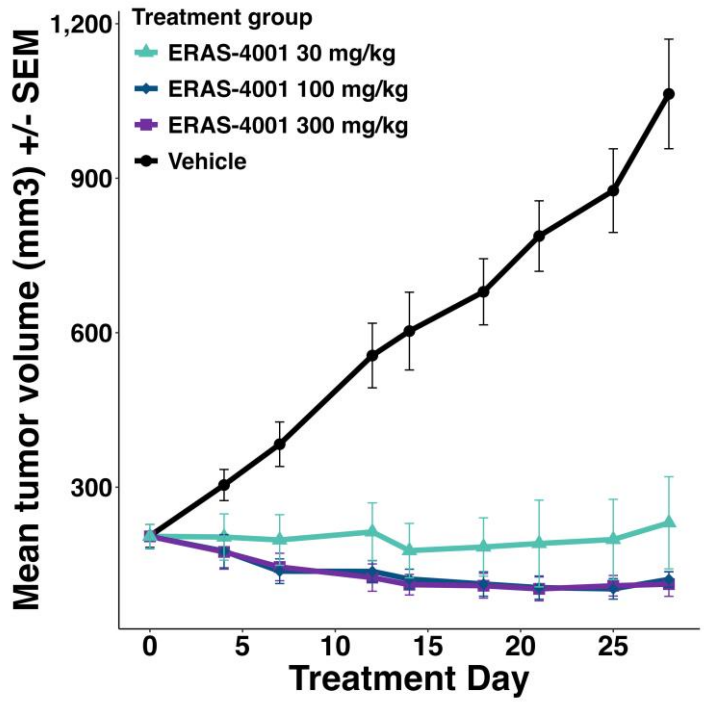
ERAS-4001 potently and selectively inhibited cell growth in KRAS G12X, G13D and WT cell lines

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**

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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

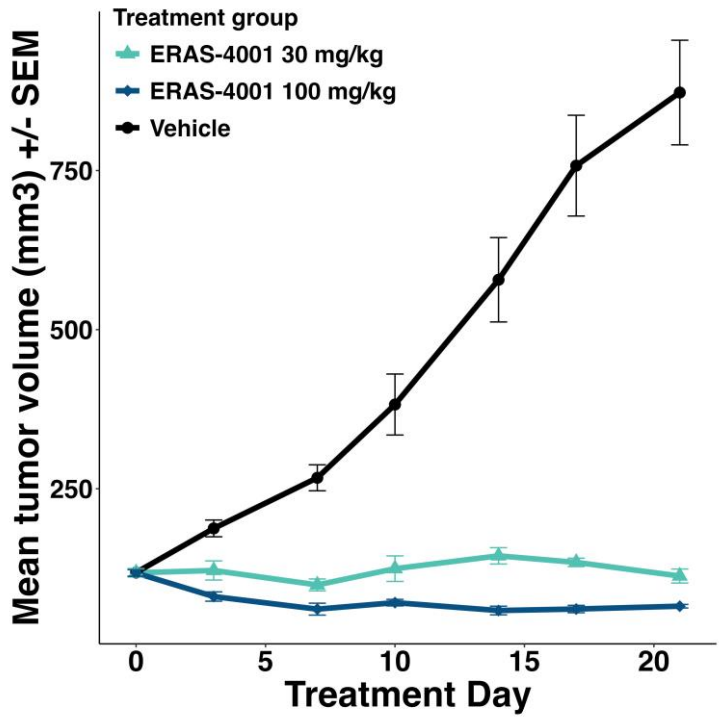
Therapy	Dose	TGI on Day 28	pERK Inhibition at 8 hr	$AUC_{0\text{-last}}$ (nmol/L·h)
ERAS-4001	30 mg/kg	97%	17%	1,547
	100 mg/kg	110%	64%	5,153
	300 mg/kg	111%	80%	12,971

- 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)

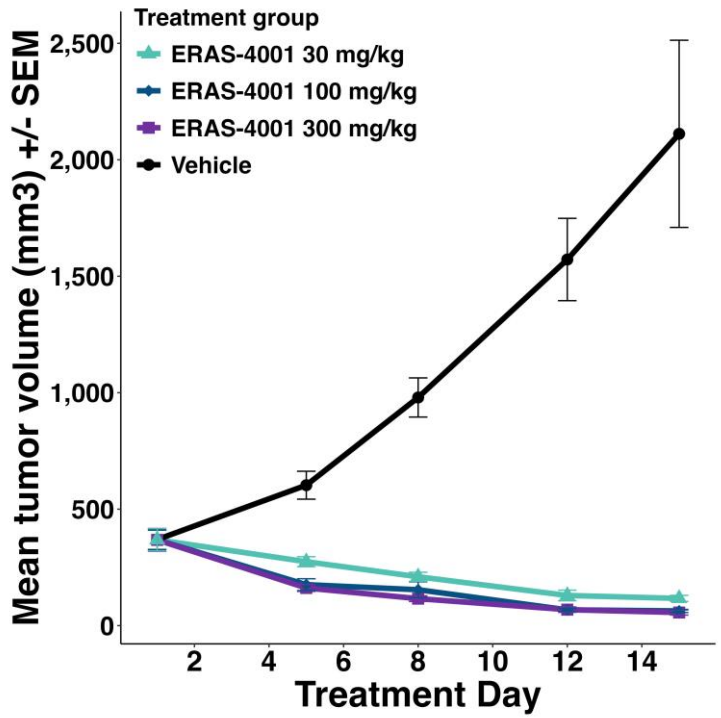
ERAS-4001 dosed orally twice daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

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

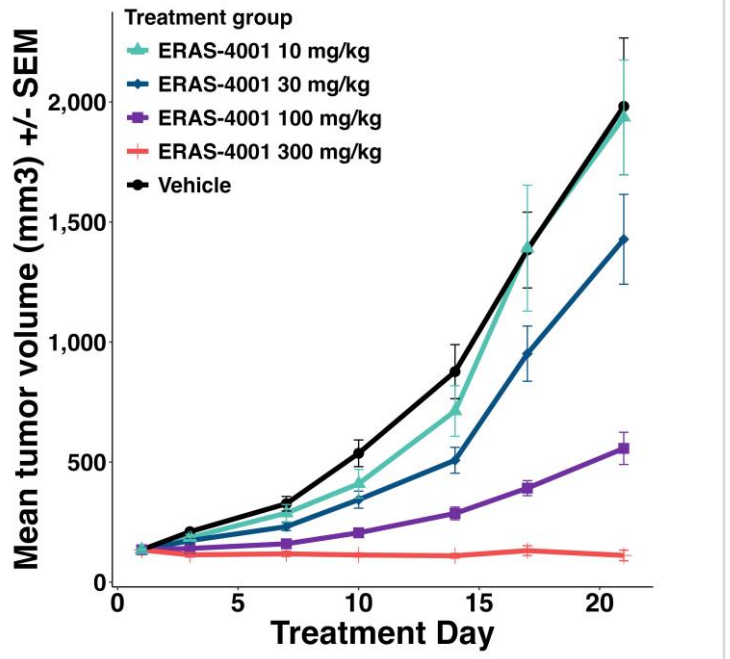


- ERAS-4001 was well tolerated in both studies at doses up to 300 mg/kg BID (i.e., no dose reductions or holidays; no body weight loss or significant health observations)

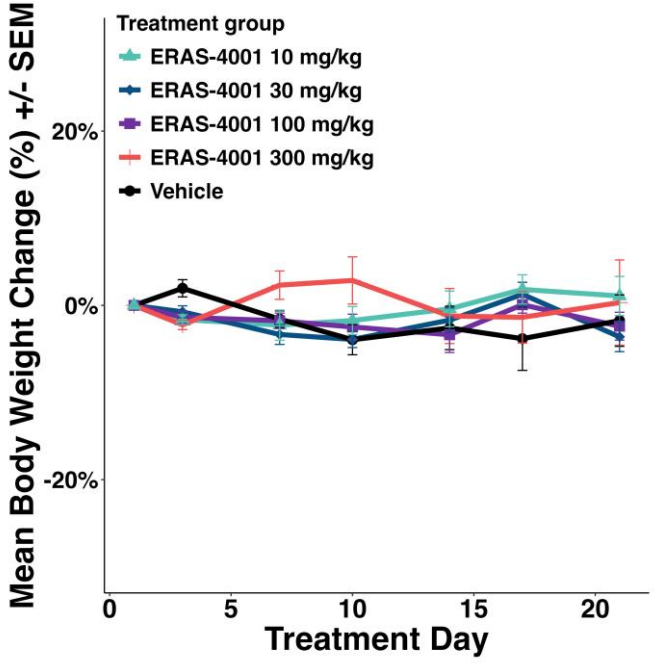
ERAS-4001 dosed orally twice daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

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 to -3** *RASm solid tumors*	• **1H27:** - Monotherapy expansion data - Combination dose escalation data, including panitumumab combination - Initiate potentially registration-enabling trial in 2L+ NSCLC • **2027:** - Initiate 1L PDAC Ph 3 - Initiate confirmatory RASm NSCLC Ph 3 in 2H27-1H28
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

NSCLC: non-small cell lung cancer; PDAC: pancreatic ductal adenocarcinoma; RASm: RAS-mutated

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


ERASCA
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 daraxonrasib
Pan-KRAS	Treeline TLN-372	KESTREL THERAPEUTICS KST-6051	AMGEN AMG-410	ERASCA **ERAS-4001**	
	Lilly LY4066434	BBOT BBO-11818	Pfizer PF-07934040 (G12X)	AstraZeneca JAB-23E73	
Mutant Selective[1]	RANOK Therapeutics RNK08954 (G12D)	Lilly LY3962673 (G12D)	Roche RG6620/GDC-7035 (G12D)	REVOLUTION MEDICINES zoldonrasib	
	QF PHARMA GROUP DN022150 (G12D)	Quanta THERAPEUTICS INC. QTX3034 (KRAS G12D)	Incyte INCB161734 (G12D)	VERASTEM ONCOLOGY VS-7375 (G12D)	
		Tyligand TSN1611 (G12D)	astellas setidegrasib (G12D degrader)	REVOLUTION MEDICINES RMC-5127 (G12V)	

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

[1] Mutant selective beyond KRAS G12C inhibitors

ERASCA

ERAS-0015 early clinical differentiation vs. comparator pan-RAS molecular glue

	Potential differentiation attributes		RMC-6236 benchmark FPD to DCO[3]: 16 months			ERAS-0015 Preliminary Phase 1 Data FPD to DCO[6]: ~9.5 months (US) – 10.5 months (CN)			
			Efficacy in Pts with PDAC and NSCLC			**Efficacy in Pts with PDAC and NSCLC**			
			≥80 mg	160 - 300 mg	300 mg	≥8 mg	16 - 32 mg PAD	24 - 32 mg RDE	32 mg
Efficacy	≥10% point increase in ORR in **2L+ KRAS^{G12X} PDAC**	uORR$_{14wk}$[1]		**29%** (2L, N=42)[4] **22%** (3L+, N=63)[4]	**35%** (2L, N=26)[5]		**40%** (2L, N=20)[7] **23%** (3L+, N=26)[7]	**42%** (2L, N=12)[8] **25%** (3L+, N=16)[8]	**50%** (2L, N=2)[9] **50%** (3L+, N=2)[9]
	≥10% point increase in ORR in **2L+ KRAS^{G12X} NSCLC**	uORR$_{8wk}$[2]	**38%** (N=40)[3]			**62%** (N=39)[10]	**62%** (N=37)[11] **75%** (Post-ICI/plat 2/3L only, N=16)[12]	**64%** (N=25)[13]	
Safety and Tolerability	**Rash[14] TRAEs**		**Safety in Pts with PDAC and NSCLC (≥80 mg, N=111)**			**Safety in Pts with PDAC and NSCLC (PAD, N=43)**			
	All Grades		81%			67%			
	Gr 1		52%			47%			
	Gr 2		23%			19%			
	Gr 3+		6%			2%			
	GI TRAEs		**Nausea**	**Vomiting**	**Diarrhea**	**Nausea**	**Vomiting**	**Diarrhea**	
	All Grades		46%	33%	39%	12%	2%	30%	
	Gr 1		36%	27%	25%	9%	2%	28%	
	Gr 2		10%	6%	13%	2%	0%	2%	
	Gr 3+		0%	0%	1%	0%	0%	0%	
	Stomatitis/Mucositis TRAEs								
	All Grades		22%			16%			
	Gr 1		12%			14%			
	Gr 2		8%			2%			
	Gr 3+		2%			0%			
	TRAEs leading to dose interruptions | reductions | discontinuations		ND | 14% | 1%			12% | 7% | 0%			
	Combinability with SOC		**CRC:** Anti-EGFR combo not shown to be tolerated **PDAC:** Dose modification required for chemo			**CRC:** ERAS-0015 at 16 mg+pani tolerated thus far w/ no DLTs to date (N=3); 100% uORR (1 uPR, N=1)[15]			

ERAS-0015: early clinical data suggest potential for **Paradigm-Shifting Scenario**

Based on performance in:
both efficacy attributes
AND
all 5 safety/tolerability attributes

- ↑ **11-15% point ORR** in 2L only KRAS G12X PDAC
- ↑ **24-37% point ORR** in 2L+ KRAS G12X NSCLC
- ↓ **rash, GI, and stomatitis/mucositis TRAEs** frequency and severity
- ↓ **dose modifications/discontinuations from TRAEs** frequency
- **Combo w/ anti-EGFR tolerated thus far with no DLTs to date and uPR** observed in first patient, first scan in CRC[15]

Note: Data presented are not based on head-to-head studies.

[1]uORR$_{14wk}$=Objective Response Rate (confirmed and unconfirmed responses) for patients who received first dose of RMC-6236 or ERAS-0015 (US, CN) at least 14 weeks prior to data extract date; [2]uORR$_{8wk}$=Objective Response Rate (confirmed and unconfirmed responses) for patients 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 assessment (CN trial); [3]Arbour et al, ESMO 2023, DCO Oct. 2023: PDAC (≥80 mg): 5 cPR, 4 uPR, PDAC (160-300 mg): 3 cPR, 3 uPR, NSCLC (≥80 mg): 1 cCR, 11 cPR, 3 uPR, NSCLC (160-300 mg): 1 cCR, 6 cPR, 3 uPR; [4]EORTC-NCI-AACR 2024, DCO Jul. 2024: PDAC (3L+): 14 c/uPR; PDAC (2L): 1 uCR, 11 c/uPR; [5]9/10/25 press release, DCO Jun. 2025: 1 cCR, 8 cPR; [6]DCO Apr. 2026 (US) and Feb. 2026 (CN); [7]US: 1 cPR, 1 uPR, N=10, CN: 9 cPR, 3 uPR, N=36; [8]US: 1 cPR, 1 uPR, N=4, CN: 5 cPR, 2 uPR, N=24; [9]US: 1 cPR, 1 uPR, N=3, CN: 0 c/uPRs, N=1; [10]US: 6 cPR, 1 uPR, N=12, CN: 7 cPR, 10 uPR, N=27; [11]US: 5 cPR, 1 uPR, N=10, CN: 7 cPR, 10 uPR, N=27; [12]US: 3 cPR, 1 uPR, N=5, CN: 2 cPR, 6 uPR, N=11; [13]US: 1 uPR, N=4, CN: 5 cPR, 10 uPR, N=21; [14]Includes preferred terms of dermatitis acneiform, rash maculopapular, rash, rash pustular, erythema, rash erythematous; multiple types of rash may have occurred in the same patient [15]As of 31 Mar. 2026 Note: FPD=first patient dosed; DCO=data cutoff; US=United States; CN=China; PAD=pharmacologically active dose; RDE=recommended dose for expansion; ORR=objective response rate; TRAE=treatment-related adverse event; Pts=patients; Gr=grade; ND=not disclosed; SOC=standard of care; pani=panitumumab; cCR=confirmed complete response; cPR=confirmed partial response; uPR=unconfirmed partial response; Note: Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons. In addition, the data presented above are based on prior Revolution Medicines' presentations. RMC-6236 has been evaluated and continues to be evaluated in subsequent clinical trials, including its registrational studies, and the data from prior studies may not be indicative of subsequent clinical data. There is no guarantee that ERAS-0015 can be "paradigm shifting" even if the final data meet these internal benchmarks and rather will depend on FDA approval and market acceptance of this product candidate.

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

ERASCA

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.

ERASCA

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

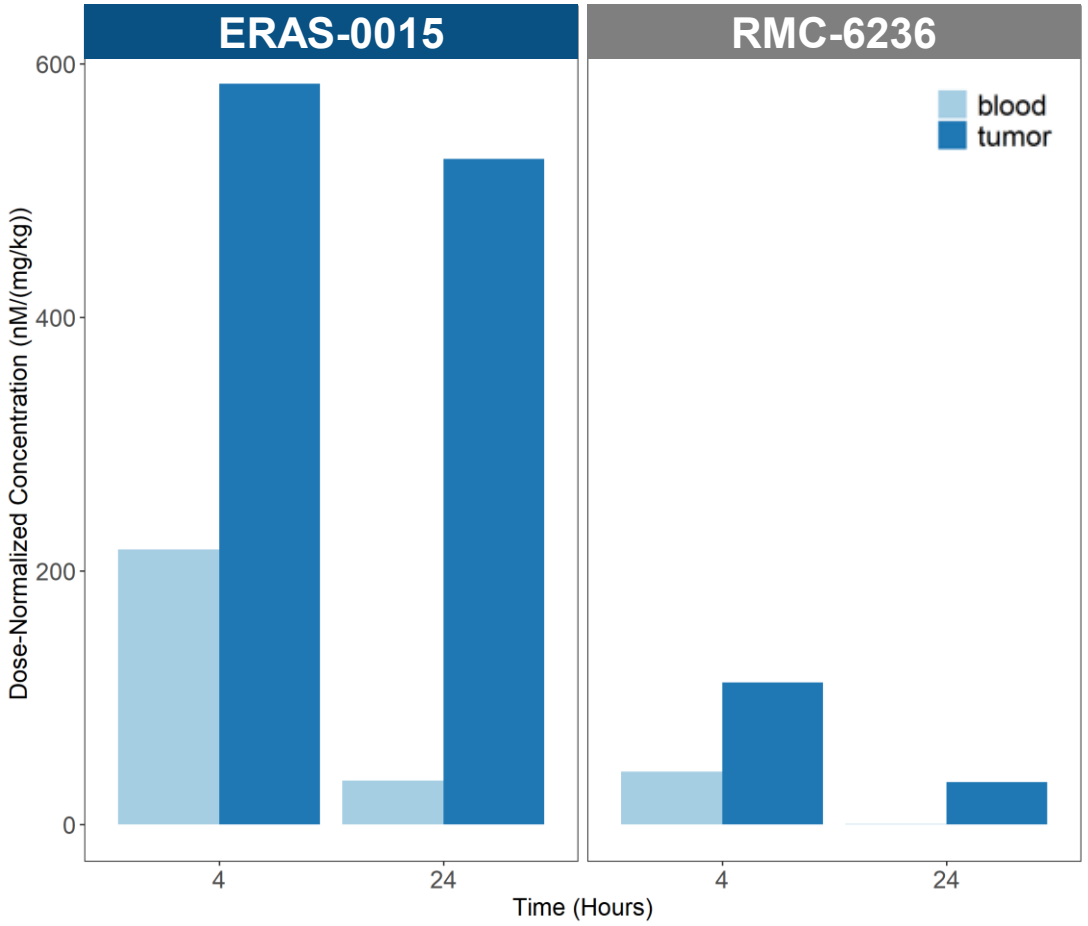
Model	KRAS Mutation	Tumor Origin	Dose achieving tumor regression[1]	
			ERAS-0015	RMC-6236
PK-59: CDX	G12D	PDAC	**0.3 mg/kg**	3 mg/kg
NCI-H727: CDX	G12V	NSCLC	**1 mg/kg**	10 mg/kg
SW620: CDX	G12V	CRC	**3 mg/kg**	25 mg/kg
PSN-1: CDX	G12R	PDAC	**5 mg/kg**	25 mg/kg
CT26: Syngeneic	G12D	CRC	**3 mg/kg**	NA[2]

[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.

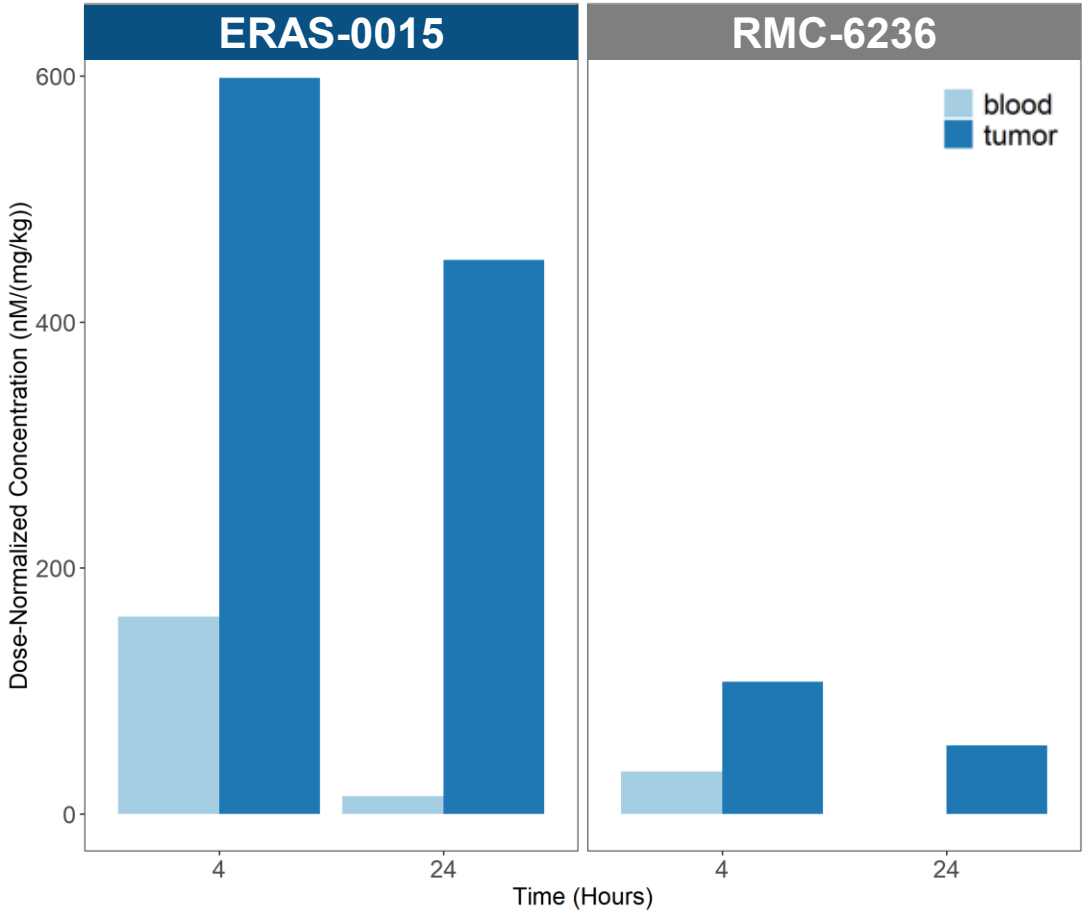
ERASCA

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

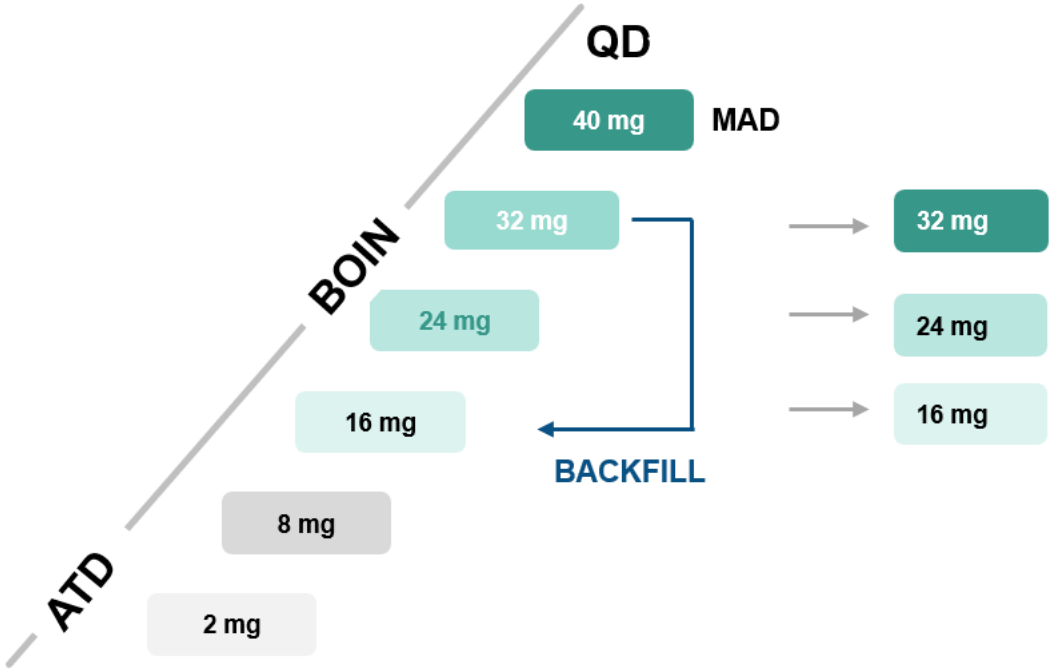
These data were generated in head-to-head assay and/or in vivo experiments

ERASCA

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

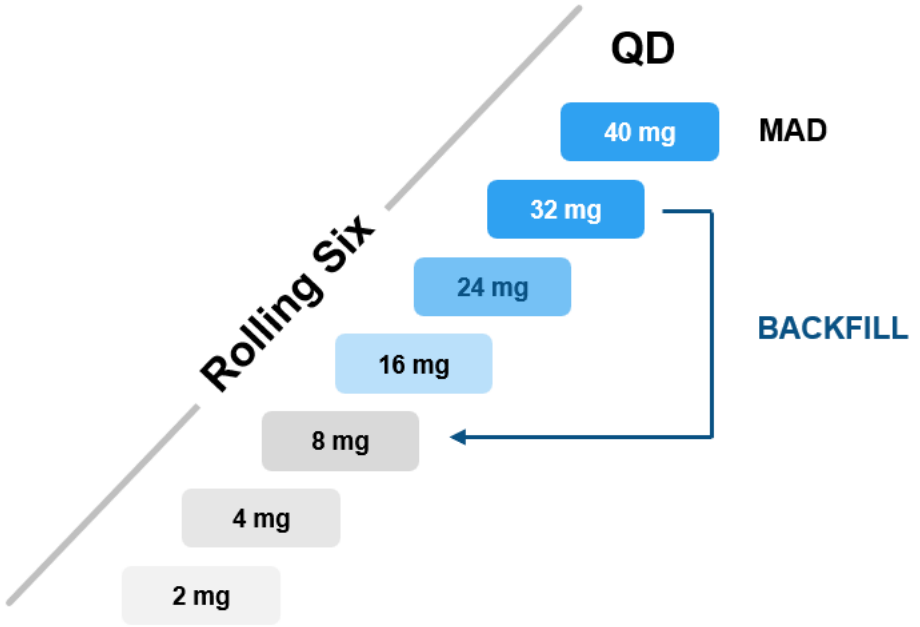
Primary Endpoints: DLTs, Safety assessments

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)

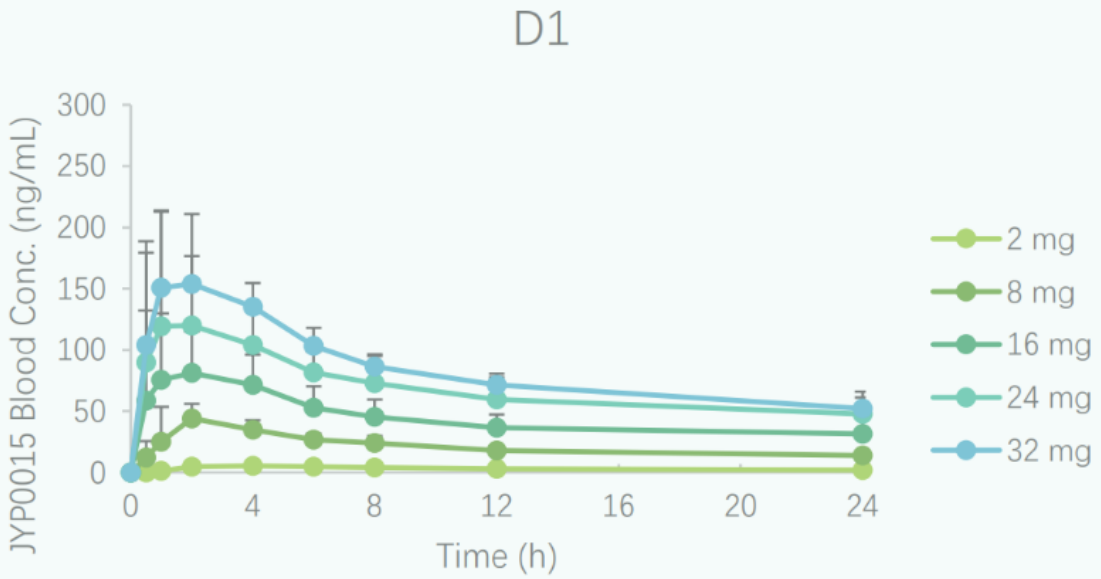
CN Trial (JYP0015M101)	RASm PDAC (2, 8, 16, 24, 32, 40 mg)	RASm NSCLC[1] (16, 24, 32 mg)
	N=78	N=42
Median Age (range)	64.5 years (35 – 79)	65.0 years (36 - 75)
Female	36% (28/78)	36% (15/42)
ECOG PS		
0	9% (7/78)	2% (1/42)
1	91% (71/78)	98% (41/42)
Smoking Status		
Current	NA	5% (2/42)
Past	NA	48% (20/42)
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-based therapy	68% (53/78)	
5FU-based therapy	77% (60/78)	
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)

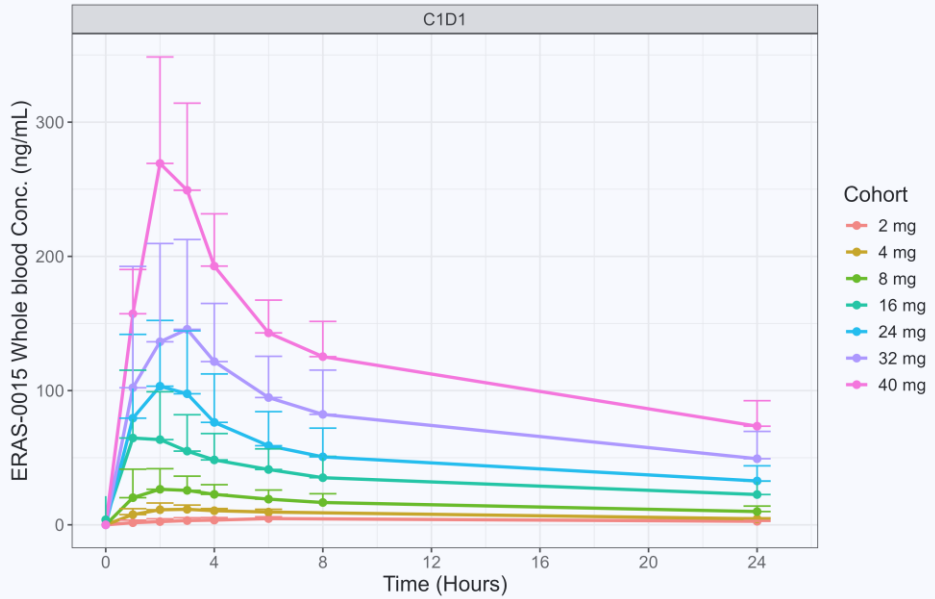
CN Trial DCO: Feb 2026 / US Trial DCO: Apr 2026
[1] 2 and 8 mg cohorts did not enroll patients with NSCLC
[2] Safety analysis set: all patients with PDAC or NSCLC that received at least one dose of ERAS-0015
[3] 2 and 4 mg cohorts did not enroll patients with NSCLC
PDAC: pancreatic ductal carcinoma; NSCLC: non-small cell lung cancer; NA=not available

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

CN Trial (JYP0015M101)[1]

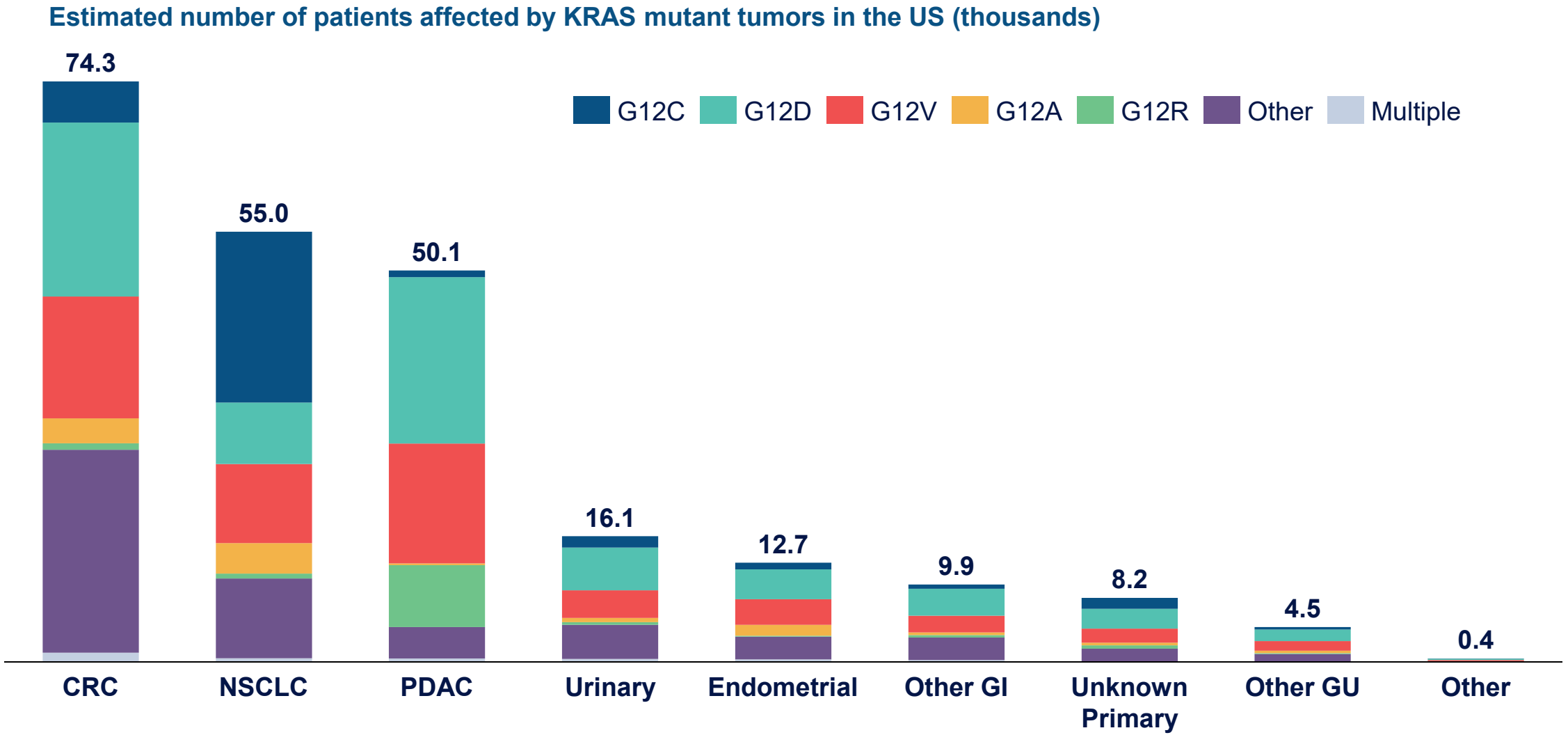


US Trial (AURORAS-1)[2]



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

KRAS alterations found most commonly in CRC, NSCLC, and PDAC



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