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On a Journey to Erase Cancer

ERAS-0015 Preliminary Phase 1 Data Update

April 27, 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, including our ability to successfully defend against allegations raised by, or any litigation initiated by, Revolution Medicines (RevMed) that ERAS-0015 infringes patents held by RevMed or was derived from RevMed trade secrets; 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.

ERAS-0015 Preliminary Phase 1 Data Update Agenda

ERAS-0015 Overview	Overview, clinical studies, PK/PD Potential differentiation of ERAS-0015 in “Big 3” tumor types and against comparator pan-RAS molecular glue
ERAS-0015 Clinical Data	Preliminary efficacy results Preliminary safety and tolerability results
Closing Remarks	Conclusions Key milestones & wrap-up

Q&A Session

ERAS-0015 overview, clinical studies, and PK/PD

Unmet need remains across RASm tumors despite recent therapeutic advances

NSCLC
~30% RAS-driven[1]

- Improved SOC with KRAS G12C inhibitors, yet needs remain for improved response rates and durability
- Non-G12C mutations remain unaddressed

PDAC
>90% RAS-driven[1]

- Limited durability and significant toxicity across approved therapies
- Relative insensitivity to pan-RAS inhibition vs. NSCLC

CRC
~50% RAS-driven[1]

- KRAS mutations drive resistance to anti-EGFR
- Minimal activity observed with pan-RAS monotherapies

- The most advanced pan-RAS inhibitor in development has demonstrated POC in both NSCLC and PDAC, but opportunities still exist to improve efficacy and tolerability
- ERAS-0015 (potentially best-in-class pan-RAS molecular glue) aims to address these gaps through key differentiating product characteristics, including improved PK and potency

[1] Reidl et al. Cancer Cell (2026)
RASm: RAS mutant; NSCLC: non-small cell lung cancer; PDAC: pancreatic ductal adenocarcinoma; CRC: colorectal cancer; SOC: standard of care; PK: pharmacokinetics

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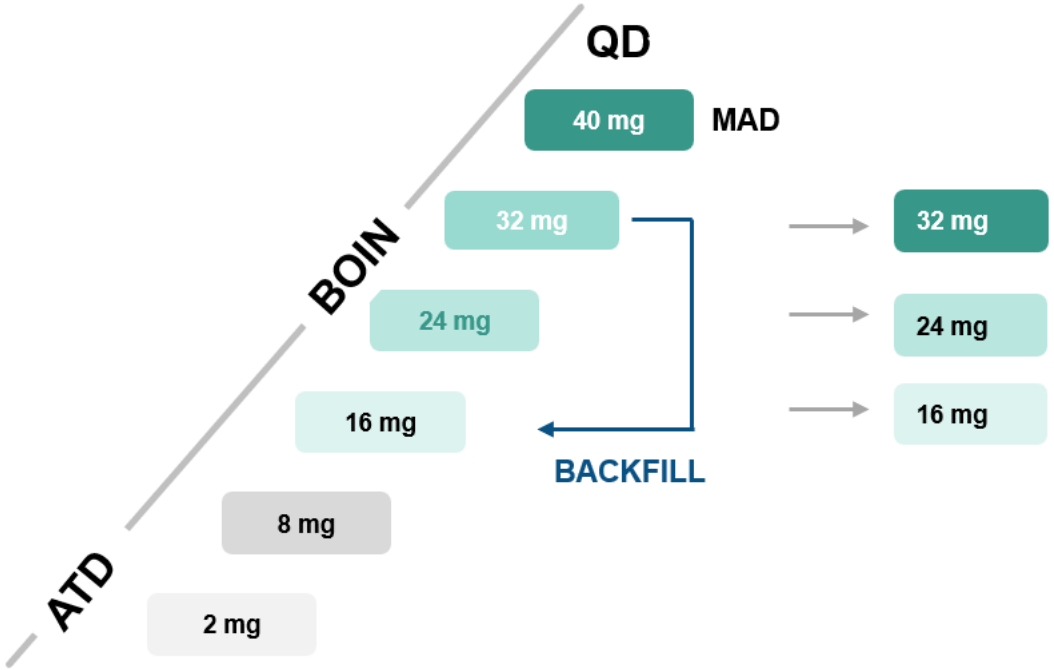
CN and US Trials: Similar trial designs support generalizability between them

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

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



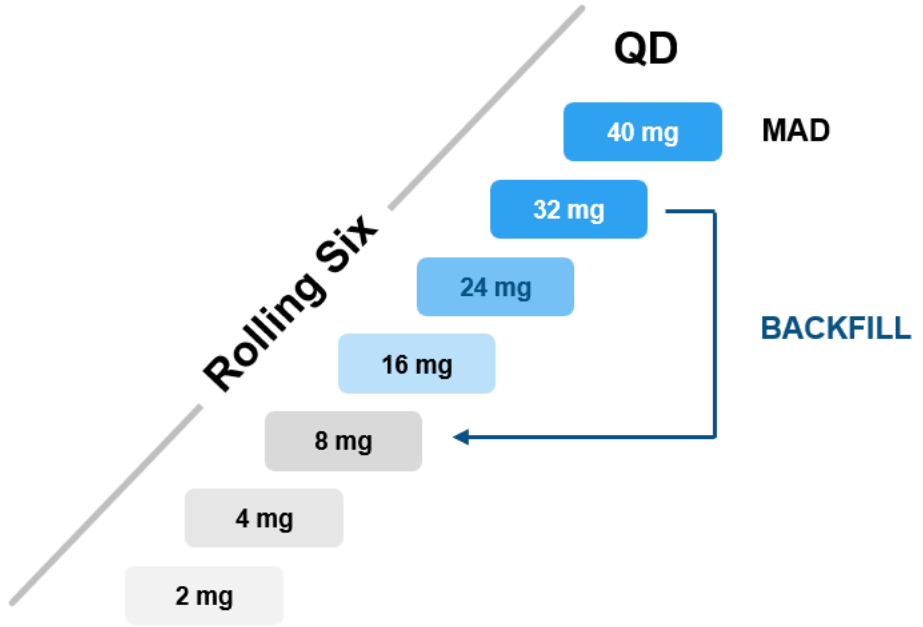
Primary Endpoints: DLTs, Safety assessments

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	
FOLFIRINOX	NA	
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

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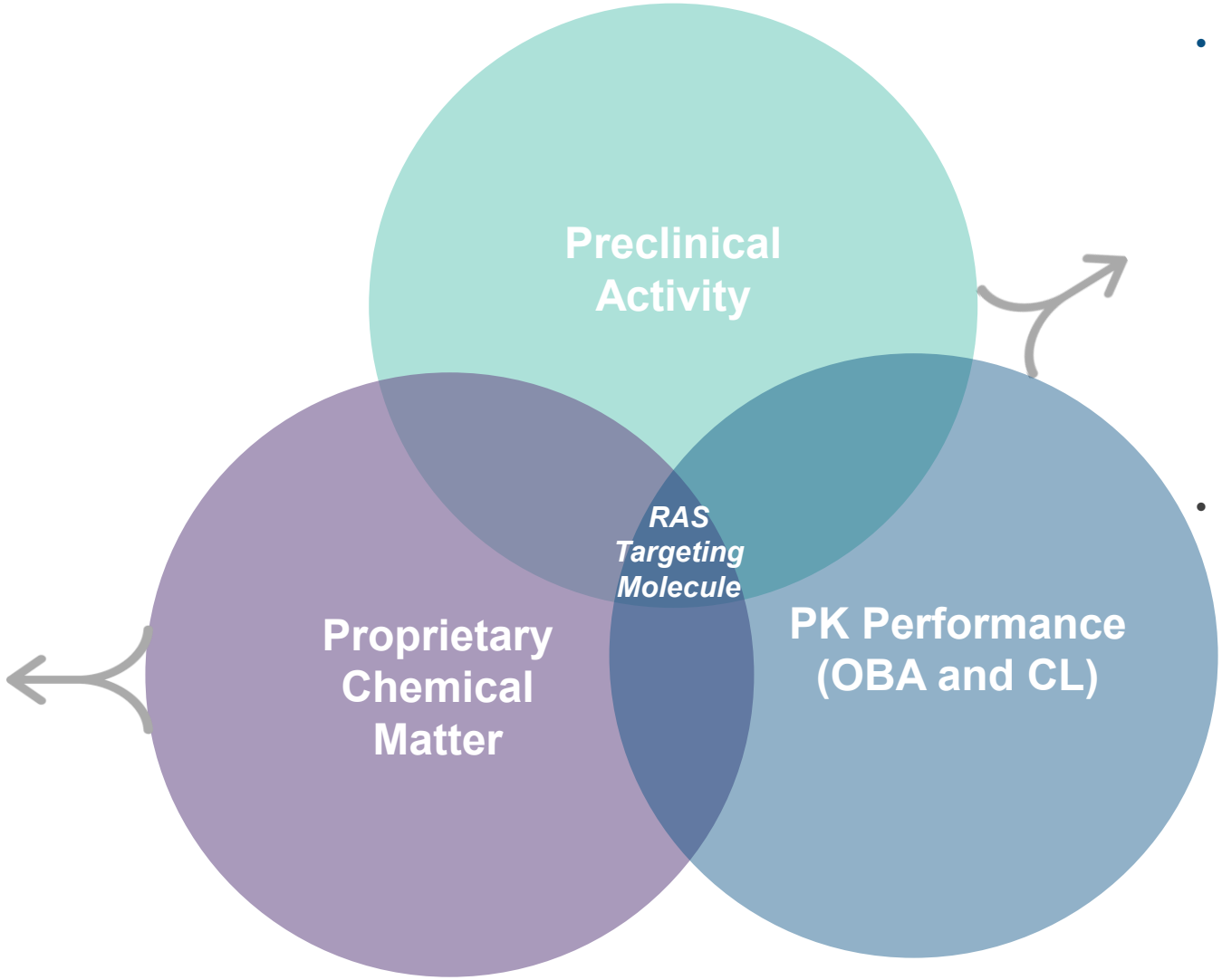
Ideal RAS targeting molecules integrate three key attributes



- **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**

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

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

ERAS-0015 preclinical profile may lead to differentiated clinical performance

Differentiated Preclinical Profile and PK Properties

Increased potency and improved CYPA binding

Enhanced PK (↑ bioavailability, ↓ clearance, longer $T_{1/2}$)

Preferential tumor distribution, longer residence time

Improved PK Properties

Greater systemic exposure

Reduced risk of solubility-limited exposure plateau

Enhanced Clinical Activity

Higher potency may translate into improved clinical activity

Analogous to KRAS G12Ci

Improved Tolerability Profile

Lower drug load

Reduced GI tox

Potential for wider therapeutic window and improved combinability

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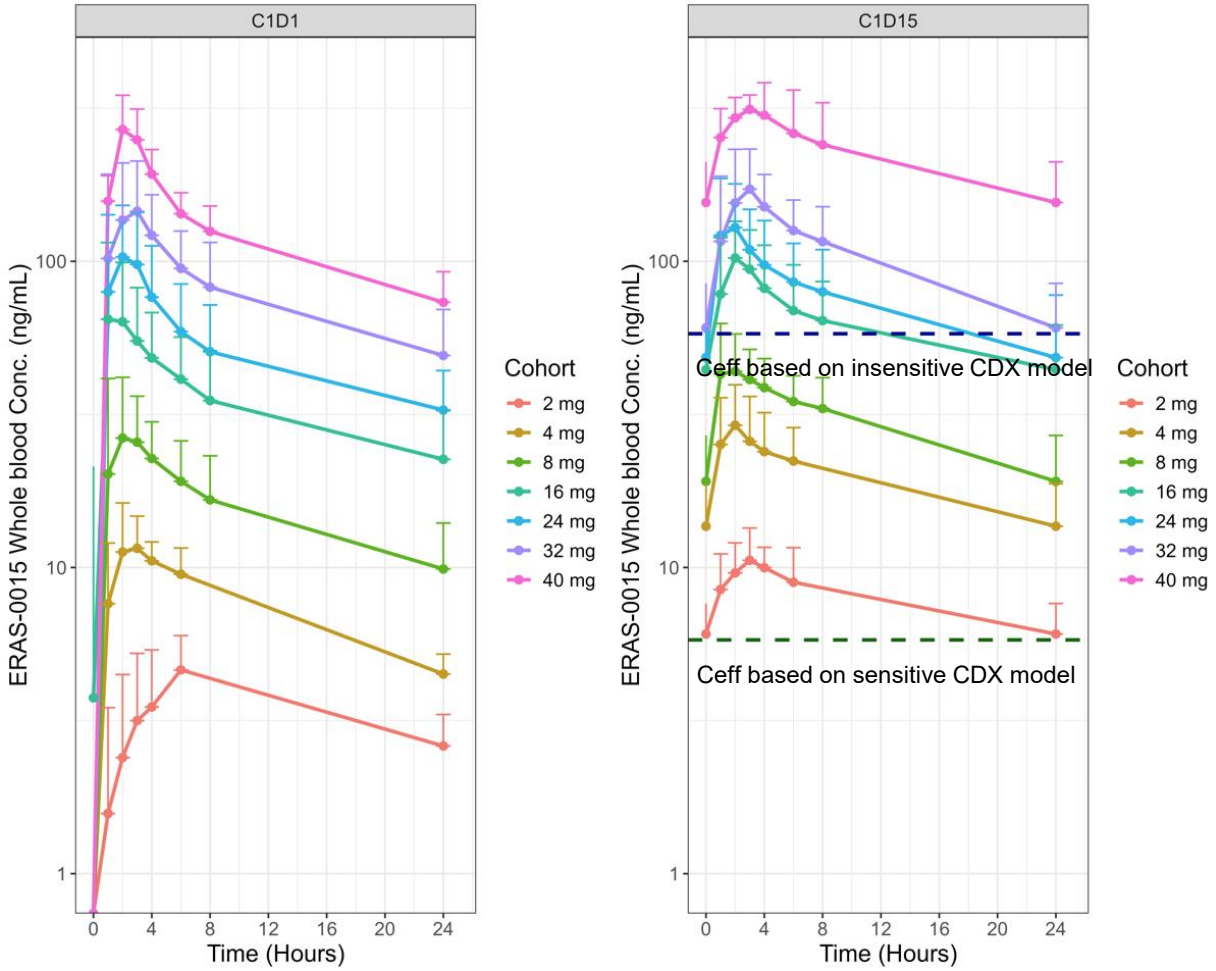
US Trial: Dose-dependent increase in PK exposure up to 40 mg MAD with no exposure plateau observed; PAD defined based on steady-state exposures that exceeded target threshold

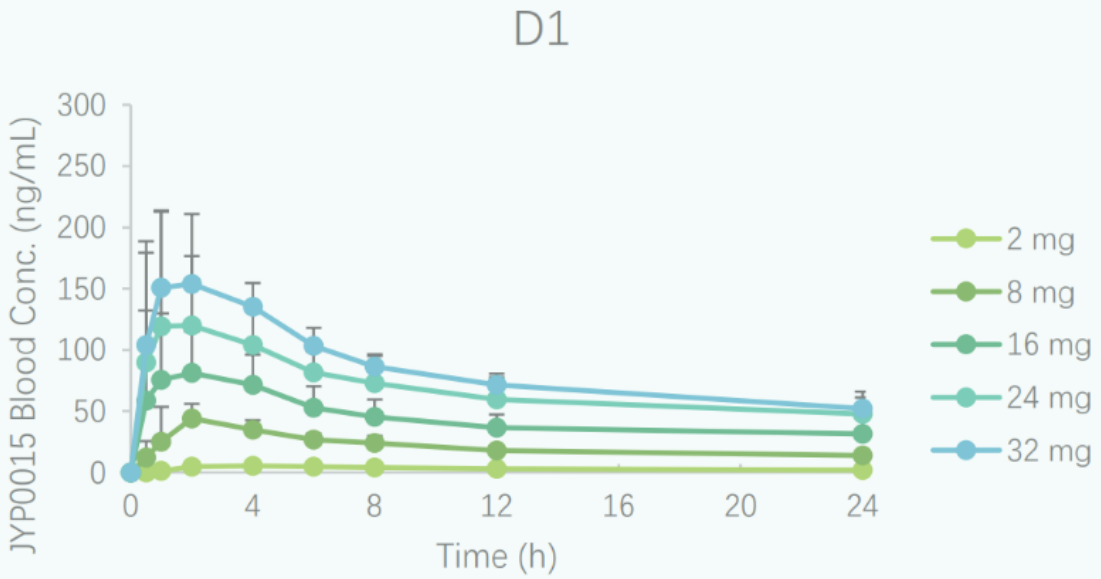


- **PK exposure** increased with dose with low-to-moderate PK variability
- **Pharmacologically active dose (PAD)** range: 16-32 mg QD
  - Mean steady-state average exposure ($C_{avg, ss}$) at ≥16 mg exceeded target exposure threshold (based on insensitive NCI-H727 CDX model)
- **Recommended doses for 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)

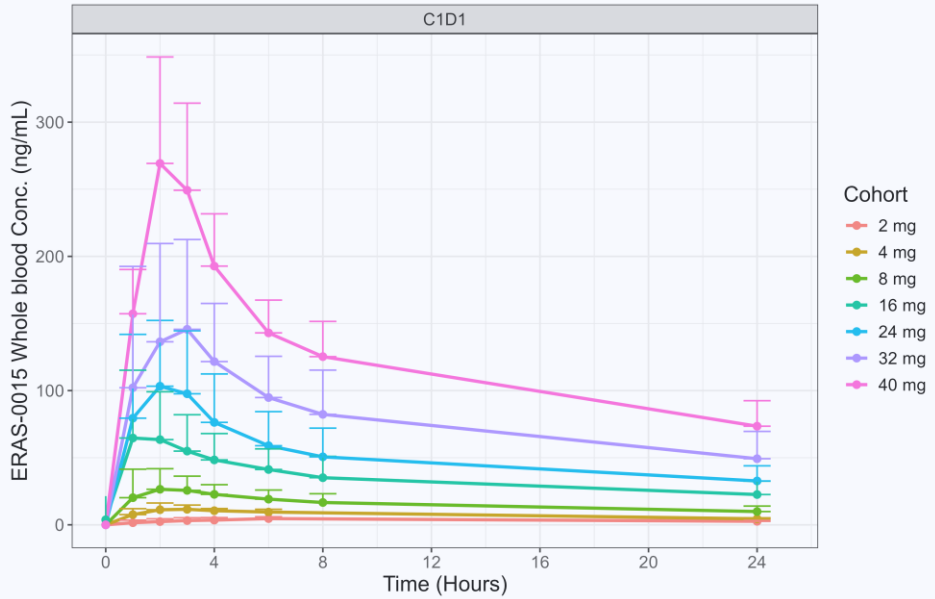
AURORAS-1 PK data are as of April 2, 2026; data points in the figures are presented as Mean + SD
Note: MAD: maximum administered dose; $C_{avg,ss}$: average concentration at steady-state

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

CN Trial (JYP0015M101)[1]



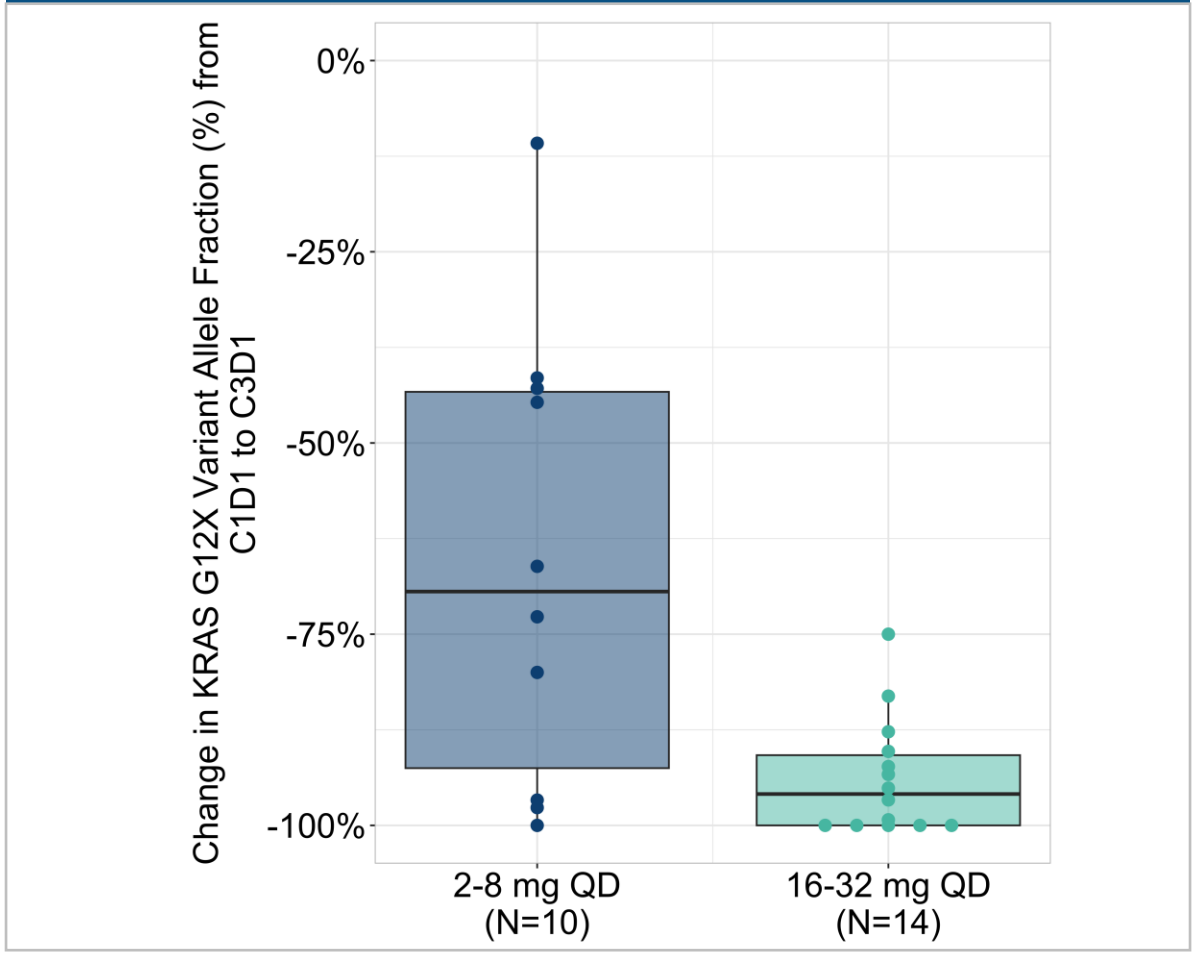
US Trial (AURORAS-1)[2]



[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)

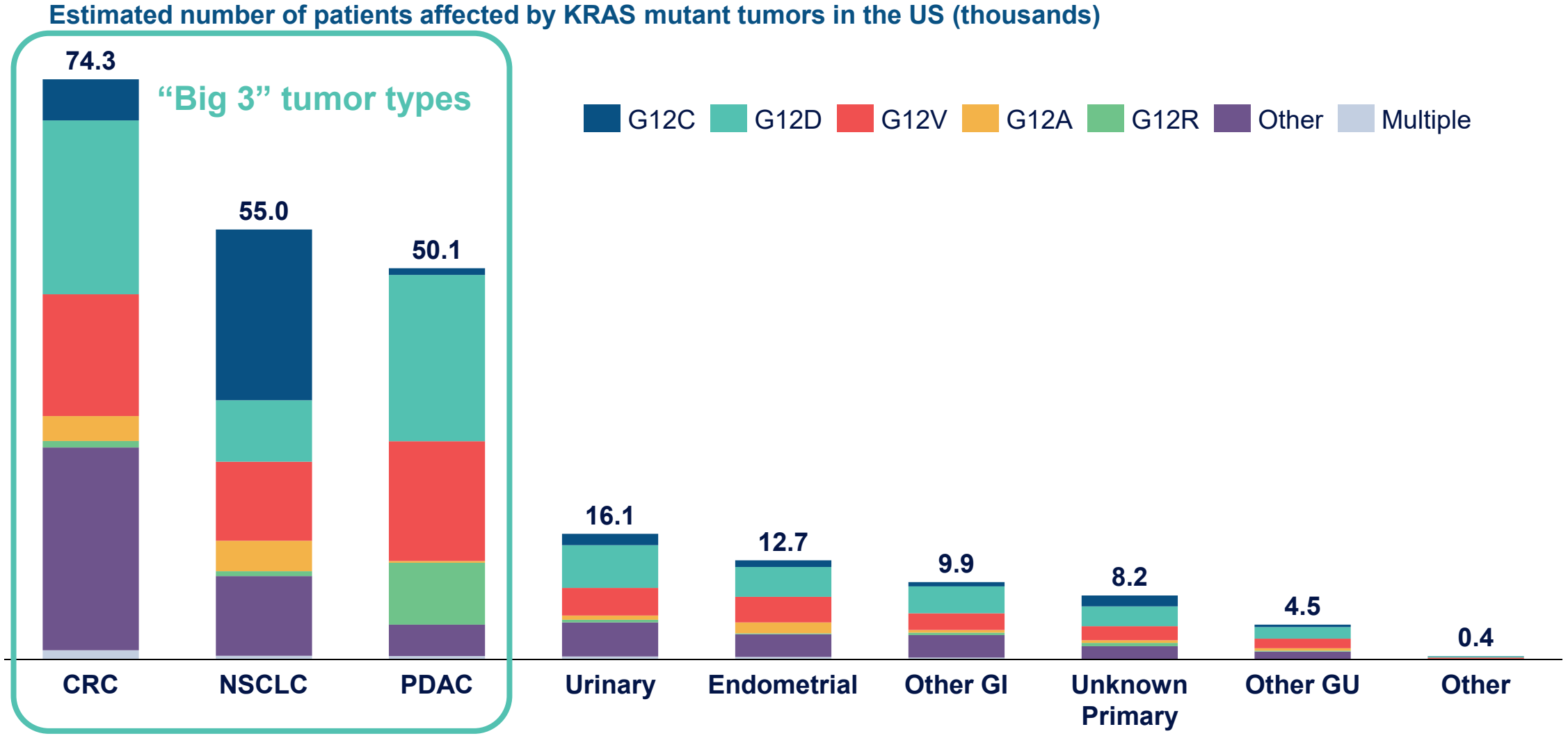


100% of patients (14/14) at PAD showed at least **75% reduction of KRAS G12X variant allele fraction**, including 5 out of 14 patients showing 100% reduction

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

Potential differentiation of ERAS-0015 in “Big 3” tumor types and against comparator pan-RAS molecular glue

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

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

ERAS-0015 showed robust early efficacy in 2L+ KRAS^{G12X} NSCLC

	ERAS-0015 Preliminary Phase 1 Data FPD to DCO: ~9.5 months (US) – 10.5 months (CN)		
	$uORR_{8wk}$[1] for ERAS-0015		
	≥8 mg	16 - 32 mg	24 - 32 mg
		Pharmacologically Active Dose	*Recommended Doses for Expansion*
NSCLC 2L+ KRAS^{G12X}	**62%** N=39	**62%** N=37	**64%** N=25
	US: 58%; 6 cPR, 1 uPR, N=12	**US:** 60%; 5 cPR, 1 uPR; N=10	**US:** 25%; 1 uPR, N=4
	CN: 63%; 7cPR, 10 uPR, N=27	**CN:** 63%; 7 cPR, 10 uPR, N=27	**CN:** 71%; 5 cPR, 10 uPR, N=21

KRAS^{G12X} NSCLC ***Post-ICI/plat (2/3L) Only***
75% N=16
US: 80%; 3 cPR, 1 uPR, N=5
CN: 73%; 2 cPR, 6 uPR, N=11

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[1]$uORR_{8wk}$=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 tumor assessment (CN trial)
Note: see Disclaimer slide regarding Cross-Study Comparisons
FPI: first patient in; DCO: data cut-off; US=United States; CN=China; ICI: immune-checkpoint inhibitor; plat: platinum; cPR: confirmed partial response; uPR: unconfirmed partial response

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
Note: Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons
NSCLC: non-small-cell lung cancer; ORR: overall response rate; QD: once daily; BID: twice daily; ICI: immune checkpoint inhibitor; plat: platinum

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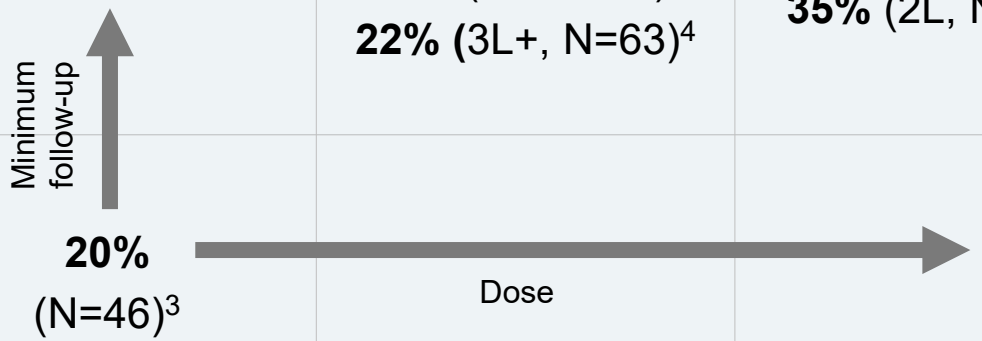
Predecessor pan-RAS molecular glue showed increased response rates in 2L+ KRAS G12X PDAC with longer minimum follow-up and earlier line of treatment

RMC-6236 uORR by dose, minimum follow-up, and line of treatment in 2L+ KRAS G12X PDAC

Time of Assessment	≥80 mg	160-300 mg	300 mg only
$uORR_{14wk}$[1] (received first dose at least 14 weeks prior to DCO)		**29%** (2L, N=42)[4] **22%** (3L+, N=63)[4]	**35%** (2L, N=26)[5]
$uORR_{8wk}$[2] (received first dose at least 8 weeks prior to DCO)	**20%** (N=46)[3]		



According to RVMD [6], "among patients with a response (confirmed or unconfirmed), 50% of first response occurred after 2 months of RMC-6236 treatment"

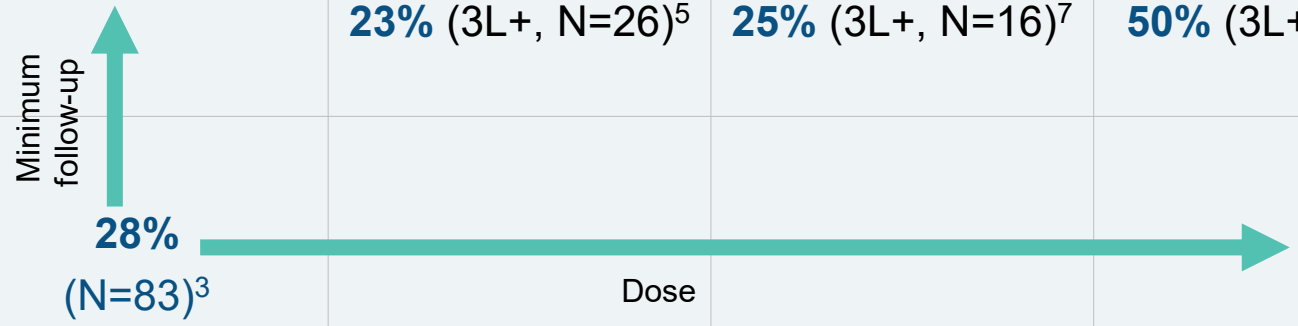
Note: the data presented above are based on prior Revolution Medicines' presentations. RMC-6236 has been evaluated and continues to be evaluated in subsequent clinical trials, including its registrational studies, and the data from prior studies may not be indicative of subsequent clinical data.

[1]$uORR_{14wk}$=Objective Response Rate (confirmed and unconfirmed responses) for patients who received first dose of RMC-6236 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 RMC-6236 at least 8 weeks prior to data extract date; [3]Arbour et al, ESMO 2023, DCO Oct. 2023: PDAC (≥80 mg): 5 cPR, 4 uPR; [4]ENA 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] Wolpin et al. ENA2024

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

ERAS-0015: Preliminary PDAC data suggest differentiated preclinical characteristics could potentially drive improved clinical activity, consistent with NSCLC data

First-in-class Pan-RAS MG

25%

RMC-6236 uORR
in 2L+ KRAS G12X PDAC,
160-300 mg QD, N=105[1]

29%

in 2L only
KRAS G12X PDAC
160-300 mg QD
N=42[1]

Potentially best-in-class Pan-RAS MG

30%

ERAS-0015 uORR
in 2L+ KRAS G12X PDAC,
16-32 mg QD, N=46[2]

40%

in 2L only
KRAS G12X PDAC
16-32 mg QD
N=20[3]

[1]Wolpin et al, ENA 2024
[2]ERAS-0015 US Trial: 1 cPR, 1 uPR, N=10, CN Trial: 9 cPR, 3 uPR, N=36
[3]ERAS-0015 US Trial: 1 uPR, N=5, CN Trial: 4 cPR, 3 uPR, N=15
Note: Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons. 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

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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)

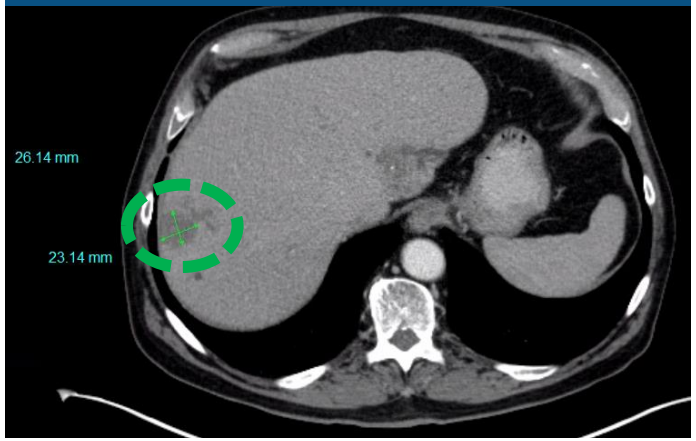
[1] As of 31Mar2026
DLT: dose-limiting toxicity

ERASCA

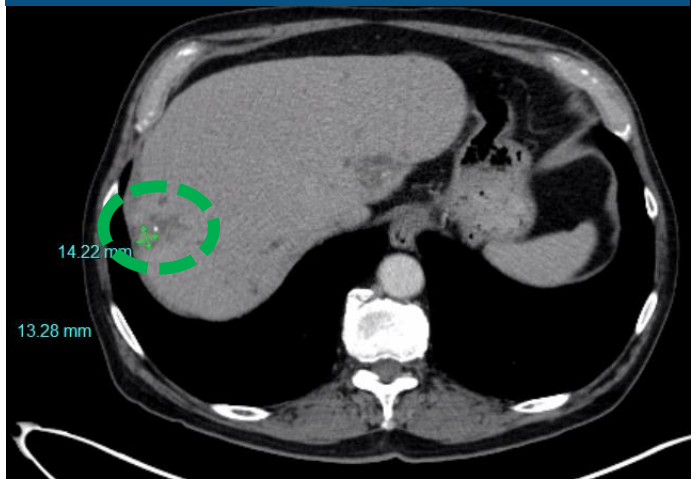
AURORAS-1 combination case study in KRAS G12D CRC: Ongoing uPR in 77-year-old male treated with ERAS-0015 16 mg + panitumumab

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

Early data suggest promising ERAS-0015 safety & tolerability

TRAE	ERAS-0015 16-32 mg PAD NSCLC and PDAC (N=43)[1]		ERAS-0015 8-40 mg NSCLC and PDAC (N=59)[1]	
	All Grades	Grade ≥3[3]	All Grades	Grade ≥3[3]
Rash[2]	29 (67%)	1 (2%)	42 (71%)	3 (5%)
Stomatitis/mucositis	7 (16%)	0	9 (15%)	0
Diarrhea	13 (30%)	0	17 (29%)	0
Nausea	5 (12%)	0	10 (17%)	0
Vomiting	1 (2%)	0	3 (5%)	0
Fatigue	2 (5%)	0	4 (7%)	0
Events leading to dose interruption	**5 (12%)**		**10 (17%)**	
Events leading to dose reduction	**3 (7%)**		**4 (7%)**	
Events leading to dose discontinuation	**0**		**0**	

Promising safety and tolerability of ERAS-0015 evidenced by low frequency of TRAEs leading to dose modifications and no discontinuations

[1] Safety analysis set with DCO Apr 2026: all patients with NSCLC or PDAC that received at least one dose of ERAS-0015
[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).
[3] 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.
ND=not disclosed; Note: see Disclaimer slide regarding Cross-Study Comparisons

Observed safety and tolerability of comparator pan-RAS molecular glue

TRAE	RMC-6236 300 mg 2L+ PDAC (N=83)[1]		RMC-6236 120-220 mg NSCLC (N=73)[2]		RMC-6236 80-400 mg NSCLC + PDAC (N=111)[3]	
	All Grades	Grade ≥3	All Grades	Grade ≥3	All Grades	Grade ≥3
Rash[4]	75 (90%)	6 (7%)	66 (90%)	5 (7%)	90 (81%)	7 (6%)
Stomatitis/mucositis	45 (54%)	3 (4%)	25 (34%)	0	24 (22%)	2 (2%)
Diarrhea	43 (52%)	3 (4%)	46 (63%)	1 (1%)	43 (39%)	1 (1%)
Nausea	32 (39%)	0	36 (49%)	0	51 (46%)	0
Vomiting	30 (36%)	0	29 (40%)	2 (3%)	37 (33%)	0
Fatigue	14 (17%)	1 (1%)	8 (11%)	0	17 (15%)	0
Events leading to dose interruption	**36 (43%)**		**25 (34%)**		**ND**	
Events leading to dose reduction	**25 (30%)**		**15 (21%)**		**15 (14%)**	
Events leading to dose discontinuation	**0 (0%)**		**3 (4%)**		**1 (1%)**	

Note: 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.

[1] Revolution Medicines Investor Deck (Nov 2025); DCO Jun 2025 [2] Revolution Medicines Investor Deck (Nov 2025); DCO Sep 2024 [3] Arbour et al. (ESMO 2023); DCO Oct 2023 [4] Rash includes the preferred terms dermatitis acneiform, rash maculopapular, rash pustular, erythema, and rash erythematous (Arbour 2023) ND: Not Disclosed

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: early clinical data suggest potential for Paradigm-Shifting Scenario

Based on performance in:

Both efficacy attributes	↑ **11-15% point ORR** in 2L only KRAS G12X PDAC ↑ **24-37% point ORR** in 2L+ KRAS G12X NSCLC
All 5 safety and tolerability attributes	↓ **Rash TRAEs** frequency and severity ↓ **GI TRAEs** frequency and severity ↓ **Stomatitis/mucositis TRAEs** frequency and severity ↓ **Dose modifications/discontinuations** from TRAEs frequency **Combo w/ anti-EGFR tolerated thus far with no DLTs to date[1] and uPR** observed in first patient, first scan in CRC

[1]As of 31 Mar. 2026 | There is no guarantee that ERAS-0015 can be "paradigm shifting" even if the final data meets these internal benchmarks and rather will depend on FDA approval and market acceptance of this product candidate. 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.

ERASCA

CN and US trials: Preliminary efficacy results

NSCLC and PDAC

ERAS-0015 preclinical profile may lead to differentiated clinical performance

Differentiated Preclinical Profile and PK Properties

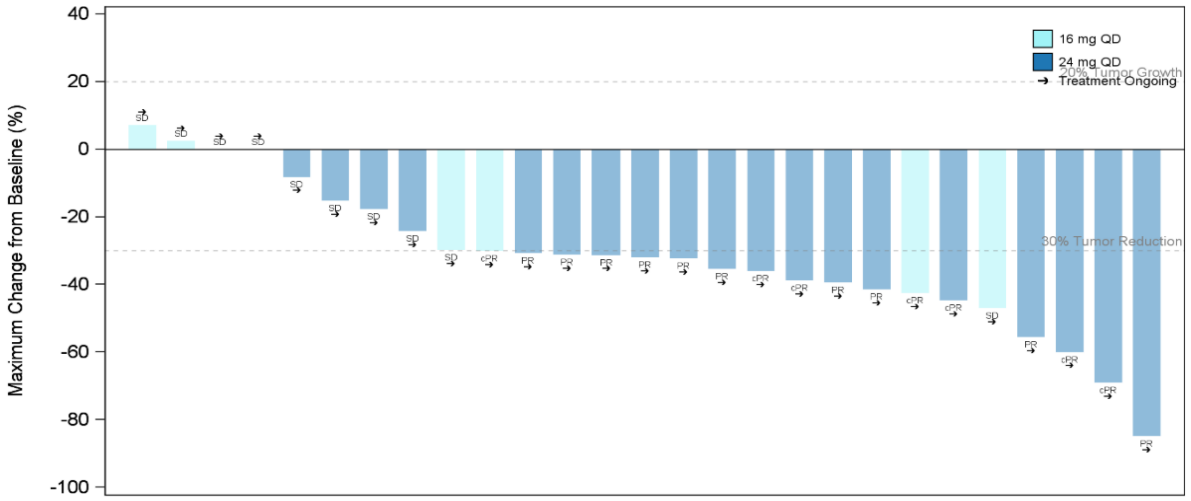
Increased potency and improved CYPA binding	Enhanced PK (↑ bioavailability, ↓ clearance, longer $T_{1/2}$)	Preferential tumor distribution, longer residence time

Lower Clinically Active Dose	Enhanced Clinical Activity	Improved Tolerability Profile
Greater systemic exposure Reduced risk of solubility-limited exposure plateau	Higher potency may translate into improved clinical activity Analogous to KRAS G12Ci	Lower drug load Reduced GI tox Potential for wider therapeutic window and improved combinability

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

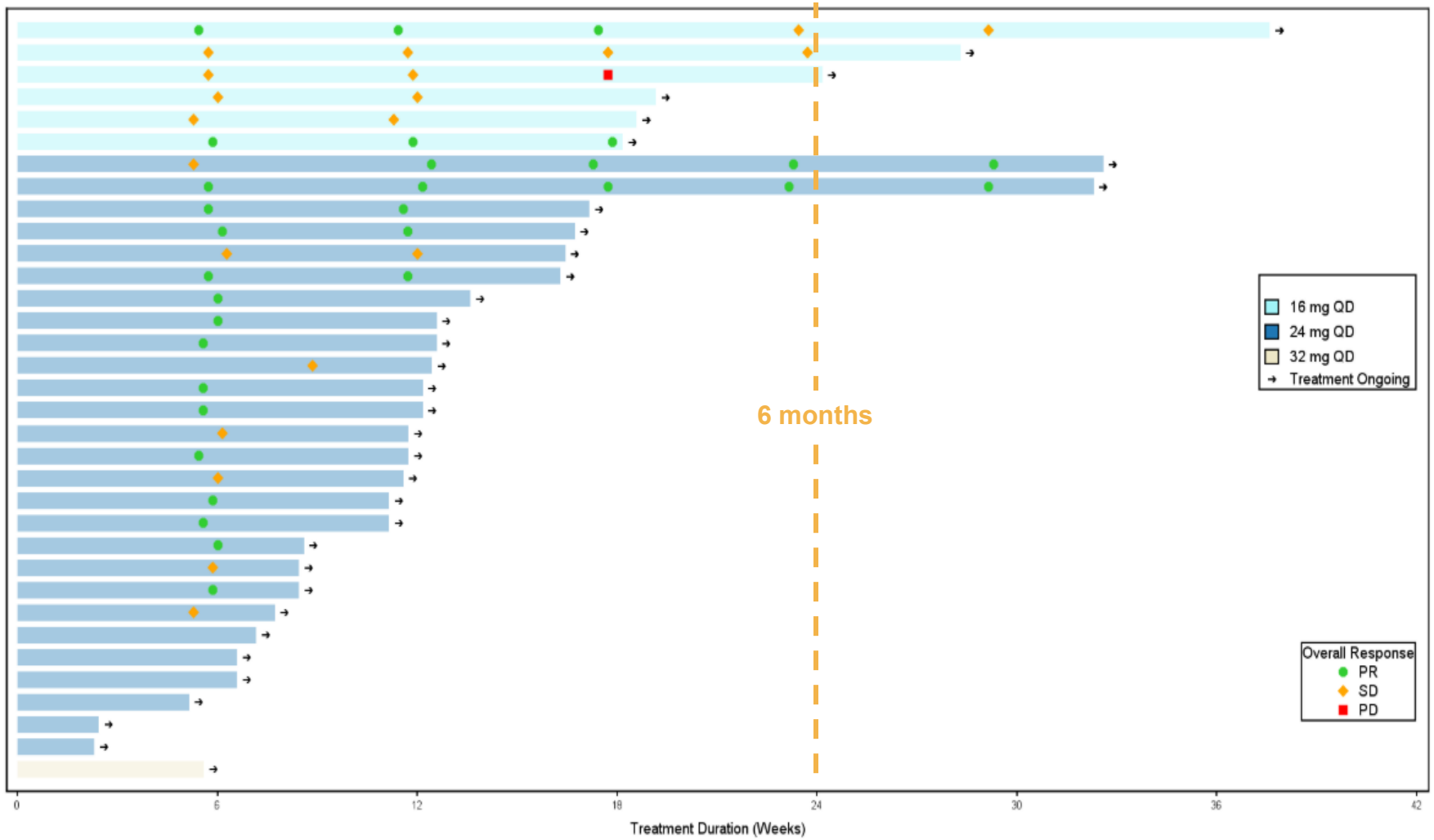
DCO Feb 2026

[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]



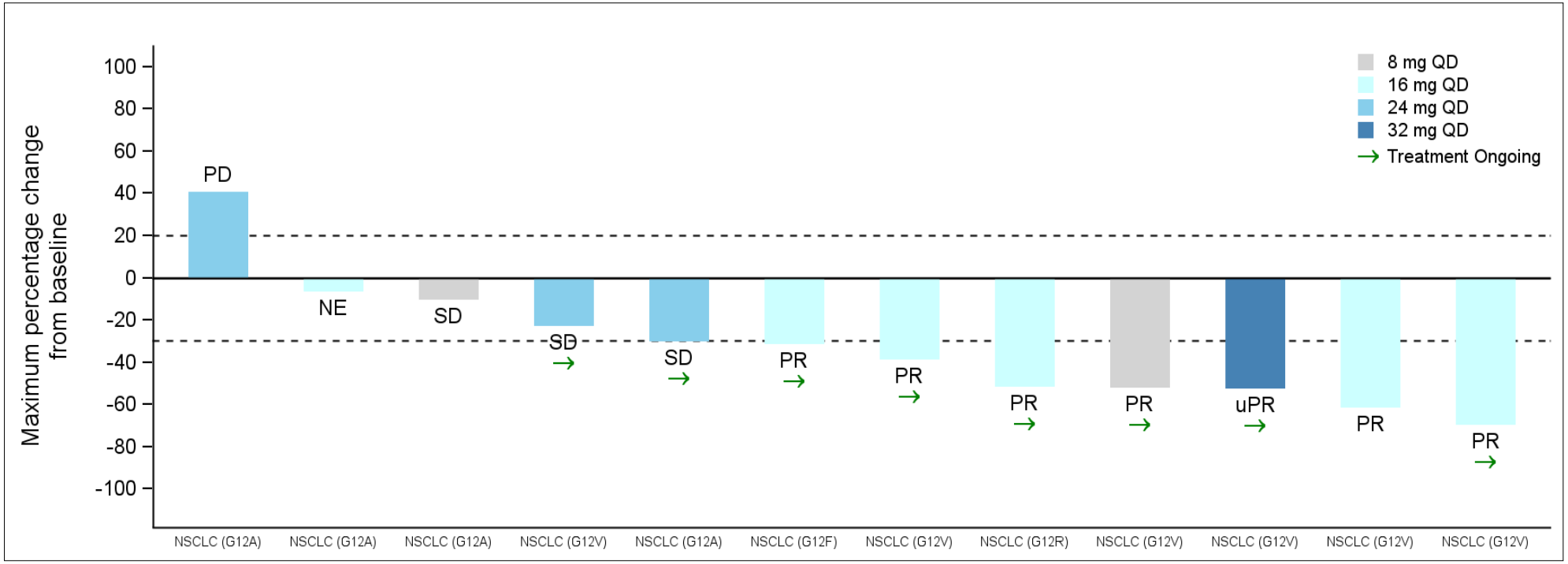
100% of responders – including all patients with uPRs – remain on treatment

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

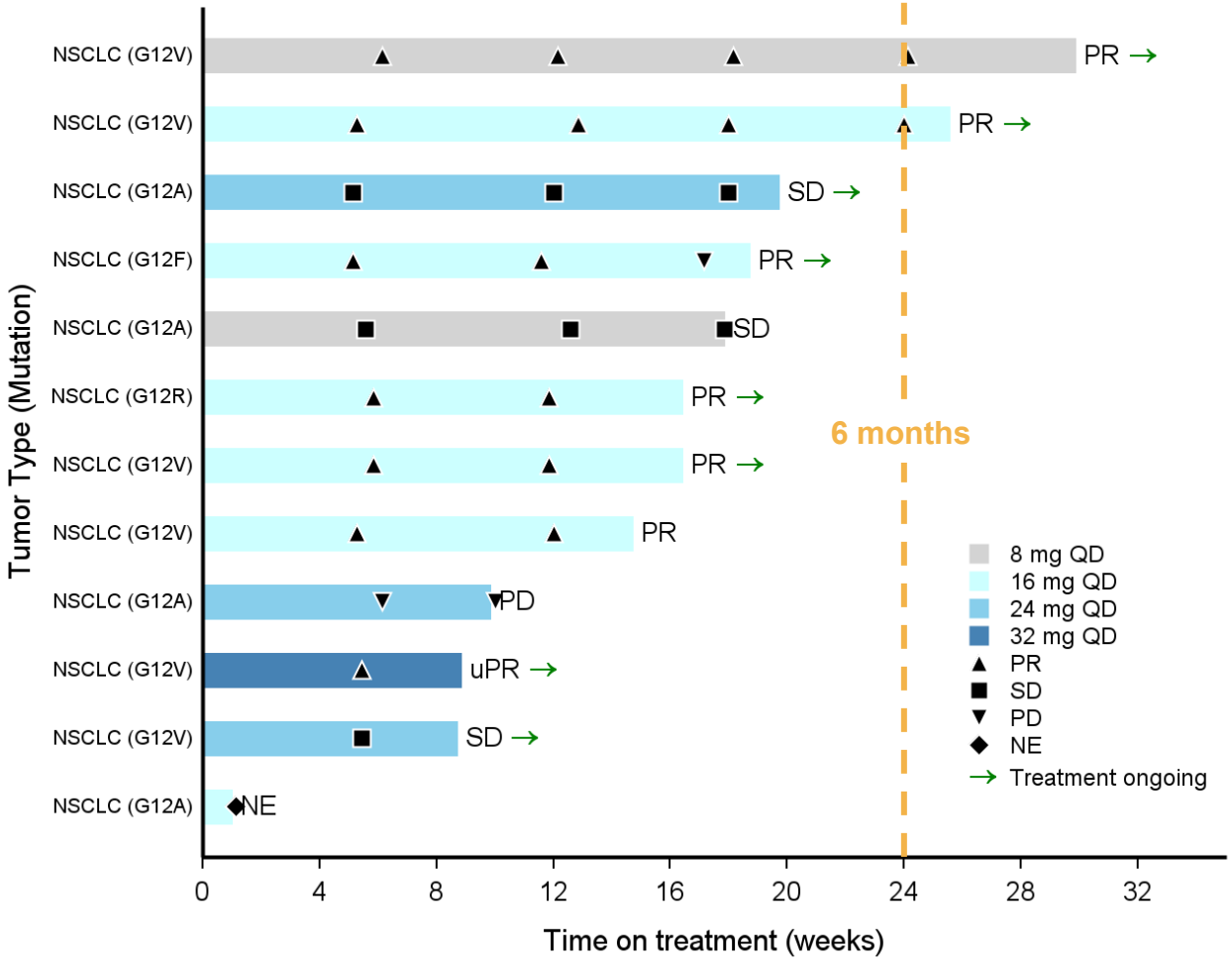
71% uORR observed in post-ICI/platinum (2/3L) KRAS G12X

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



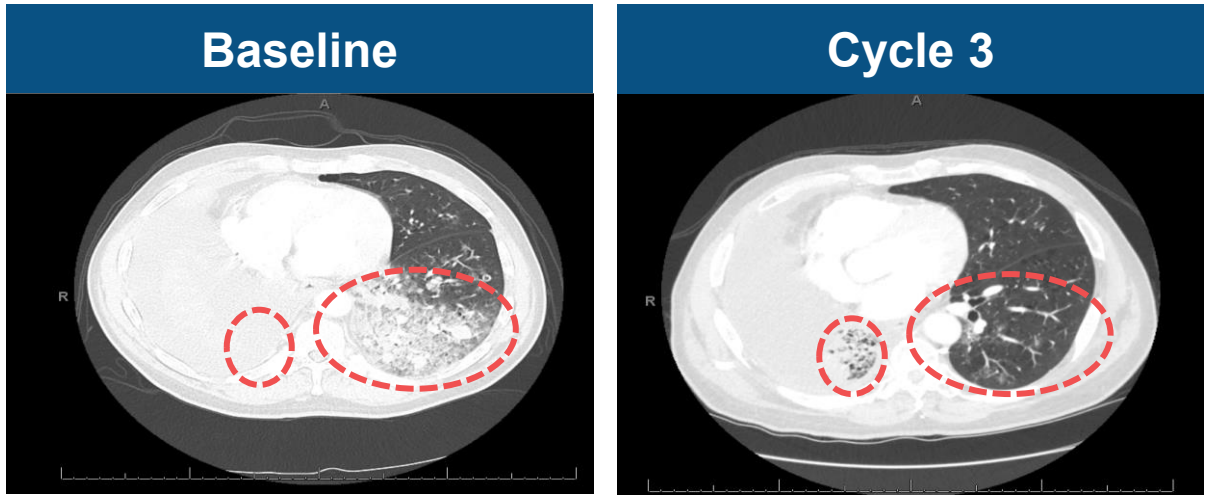
6 out of 7 responders—including all patients with uPRs—remain on treatment

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.

ERASCA

AURORAS-1 monotherapy case study in KRAS G12V NSCLC: Ongoing confirmed partial response in 70-year-old male treated with ERAS-0015 (8 mg QD)



Patient was able to come off supplemental oxygen after first week of ERAS-0015 8 mg QD treatment

Patient is currently asymptomatic from disease[1]

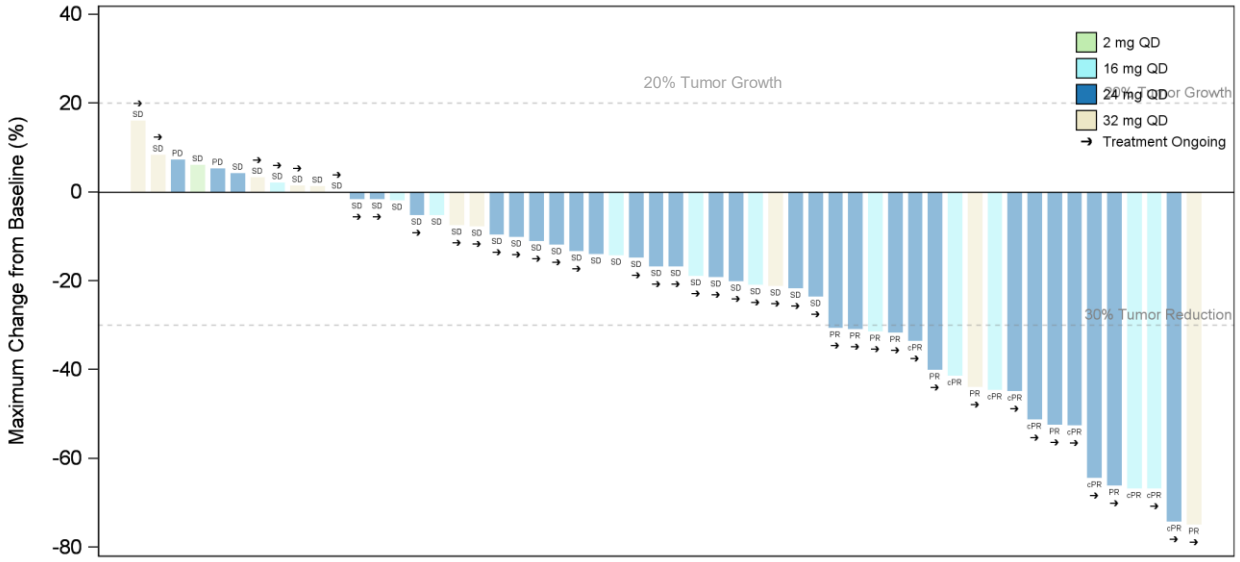
Diagnosis	Stage IV NSCLC; KRAS G12V
Prior Therapy	Carboplatin/pemetrexed/pembrolizumab (Aug – Oct 2023) Carboplatin/paclitaxel (Oct 2023 – Mar 2024) Durvalumab (May – Dec 2024)
ERAS-0015 Treatment	**Cycle 1:** 8 mg QD **Cycle 3:** Restaging CT (-37% per RECIST) **Cycle 5:** Confirmed Partial Response (-41% per RECIST) at 8 mg QD, after which dose was escalated to 16 mg QD **Cycle 7**: Deepening of Confirmed Partial Response (-48% per RECIST) **Cycle 9**: Deepening of Confirmed Partial Response (-52% per RECIST) Patient continues on treatment[1]
Treatment Related Adverse Events	Grade 1 Rash

[1] As of 17Mar2026
NSCLC: non-small cell lung cancer; QD: once daily

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

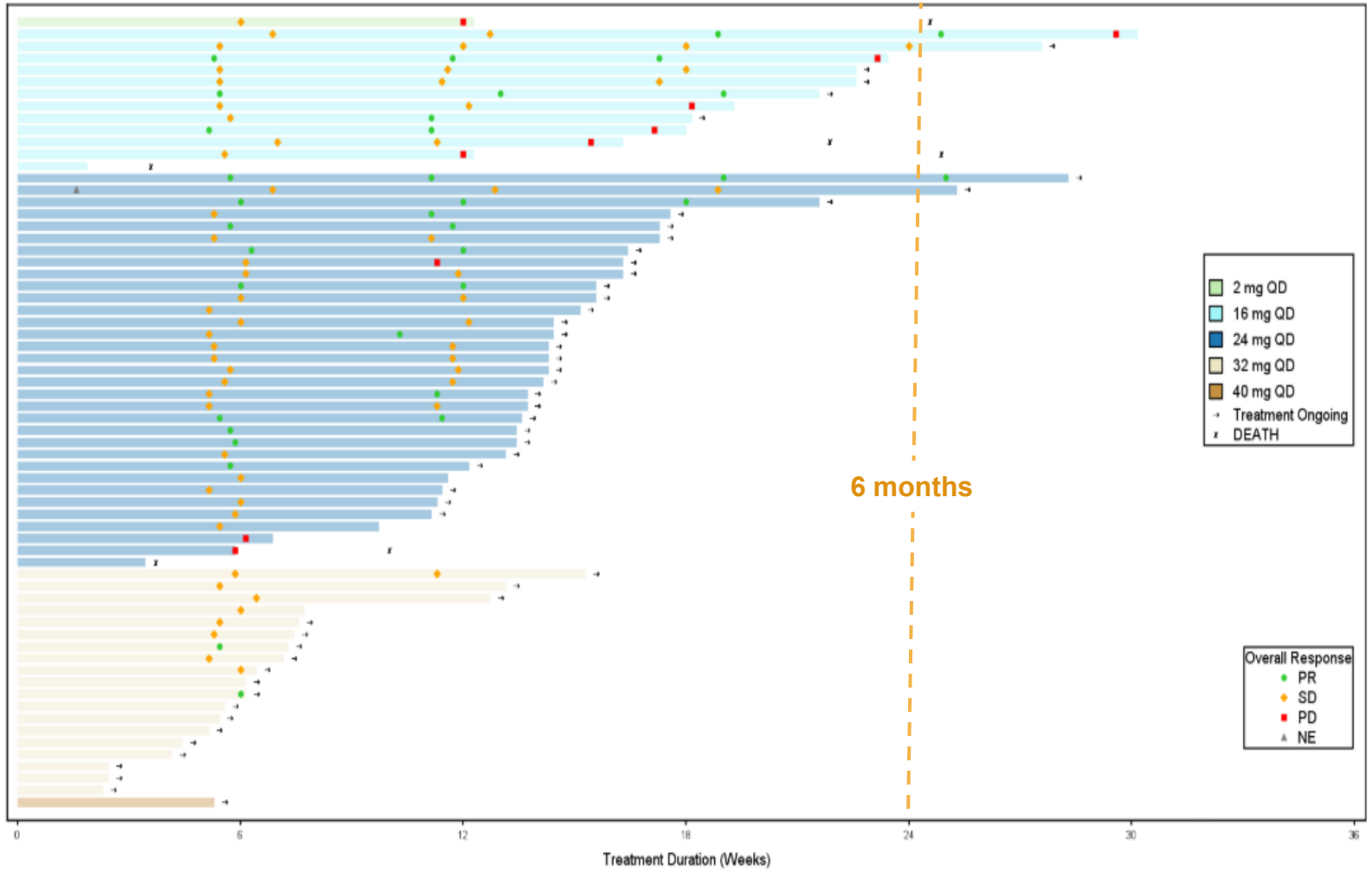
[2] 7 ongoing cPRs, 3 cPRs that have discontinued treatment, and 9 ongoing uPRs out of 53 patients with KRAS G12X mutations

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;

ERASCA

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

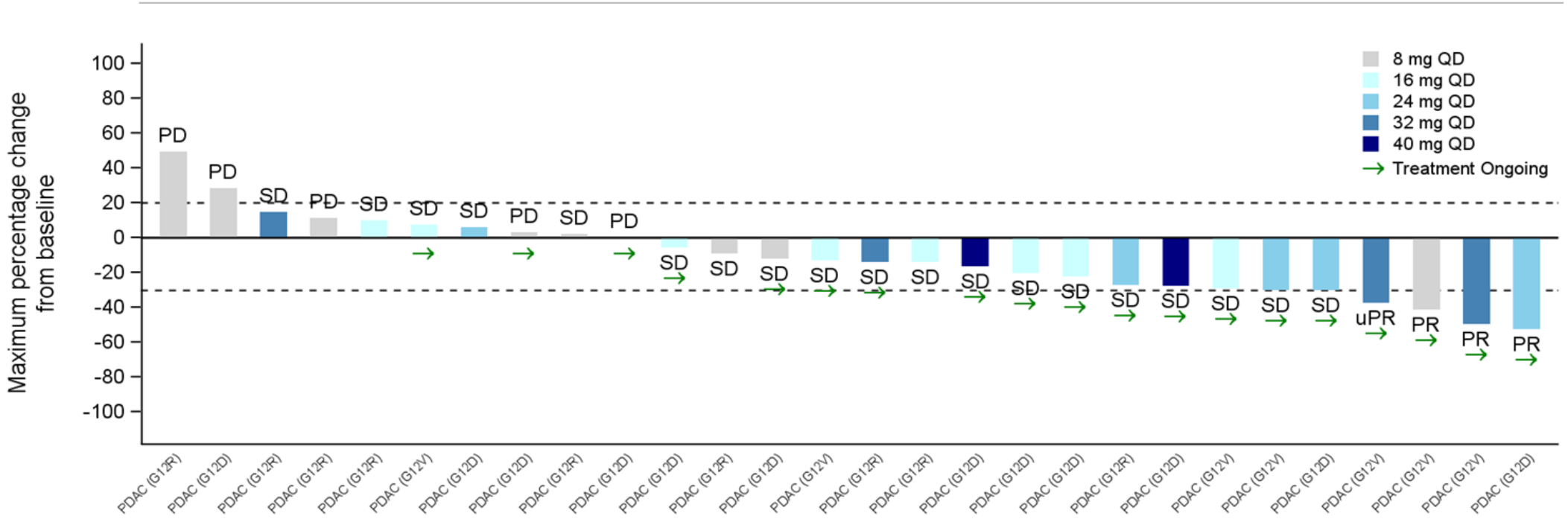
Reinforces the safety, tolerability and durability of response of ERAS-0015

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…

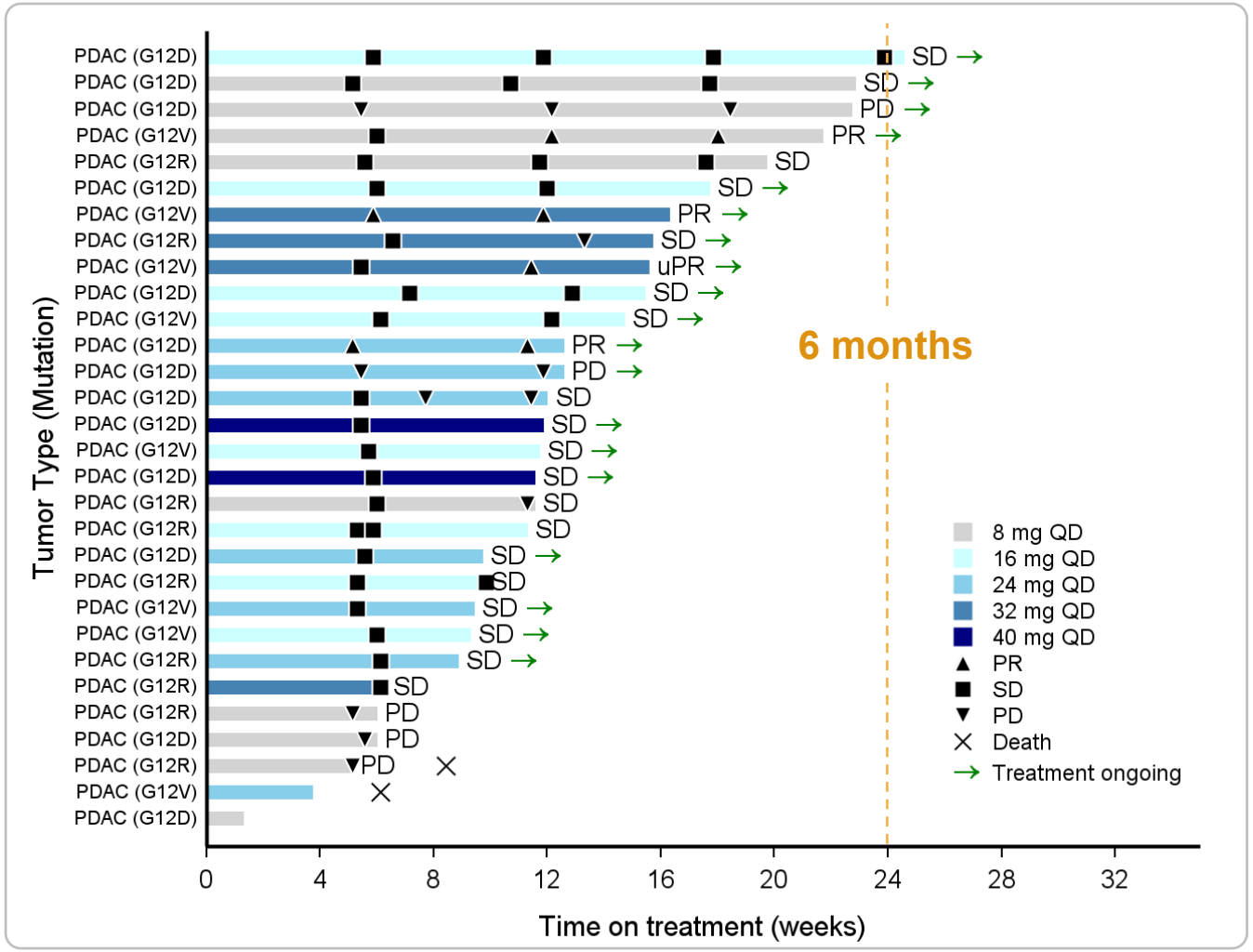
ERAS-0015 2L+ KRAS G12X PDAC[1]	8 mg QD N=9	16 mg QD N=8	24 mg QD N=7	32 mg QD N=4	40 mg QD N=2	Total N=30
uORR[1] , n (%)	1 (11%)	0	1 (14%)	2 (50%)	0	4 (13%)
DCR, n (%)	4 (44%)	8 (100%)	5 (71%)	4 (100%)	2 (100%)	23 (77%)

…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.

AURORAS-1 monotherapy case study in KRAS G12D PDAC: Ongoing partial response in 75-year-old male after intra-subject dose escalation

Diagnosis	Stage IV PDAC; KRAS G12D
Prior Therapy	Gemcitabine + nab-paclitaxel (Sept to Dec 2024) FOLFIRINOX (Jan to Apr 2025) Radiation (May 2025)
ERAS-0015 Treatment	**C1D1** Patient started on 8 mg QD **Week 6** scan on C2D17 revealed new liver lesion and target lesion increase of sum of diameters (SOD) of +3% → progressive disease Patient is treated beyond progression and continues on 8 mg QD **C4D1** Dose is escalated to 16 mg QD **Week 12** scan on C4D20 reveals absence of new liver lesion and target lesion SOD decrease of -17% **Week 18** scan on C7D6 reveals continued absence of new liver lesion and target lesion SOD decrease of -33%; ***ongoing partial response***[1]
Treatment-related adverse events	G1 rash, diarrhea and nausea[1]

[1] As of 19 Mar 2026
PDAC: pancreatic cancer; QD: once daily

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US trial: Preliminary safety and tolerability results

ERAS-0015 preclinical profile may lead to differentiated clinical performance

Differentiated Preclinical Profile and PK Properties

Increased potency and improved CYPA binding	Enhanced PK (↑ bioavailability, ↓ clearance, longer $T_{1/2}$)	Preferential tumor distribution, longer residence time

Lower Clinically Active Dose	Enhanced Clinical Activity	Improved Tolerability Profile
Greater systemic exposure Reduced risk of solubility-limited exposure plateau	Higher potency may translate into improved clinical activity Analogous to KRAS G12Ci	Lower drug load Reduced GI tox Potential for wider therapeutic window and improved combinability

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

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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

Conclusions and key milestones

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

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Anticipated key milestones in 2026-2027

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

COMPLETED per guidance

COMPLETED ahead of guidance:
- Initiated US mono expansions in 2Q26
- Initiated US anti-EGFR mAb combo in 1Q26

Narrowing guidance to 1H27
for select monotherapy expansions and combination dose escalation(s)

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


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THANK YOU!