ERASCA

On a Journey to Erase Cancer

Erasca Investor 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, including ERAS-0015 and ERAS-4001; the potential for ERAS-0015 and ERAS-4001 to be used in 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: our ability to achieve the corporate milestones set forth in this presentation within the applicable timeframes; our product candidates, including ERAS-0015 and ERAS-4001, may not demonstrate therapeutic benefits that we expect; 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; the efficacy and safety of ERAS-0015 compared to other products and product candidates (including daraxonrasib (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; 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; our planned potentially registration-enabling trials may be delayed based on U.S. 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, 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 related to, 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 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.

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Erasca Investor Presentation Agenda

ERAS-0015 AURORAS-1 FIH Study	Monotherapy activity in key indications PK/PD, safety, and tolerability Combination development updates in Big 3 indications
ERAS-4001 BOREALIS-1 FIH Study	Preliminary dose escalation safety, PK, and anti-tumor activity
Summary	Conclusions Key milestones and wrap-up

FIH: first-in-human; PK: pharmacokinetics; PD: pharmacodynamics; Big 3 indications include pancreatic ductal adenocarcinoma (PDAC), non-small cell lung cancer (NSCLC), and colorectal cancer (CRC)

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

Preclinical Differentiation[1]

In Vivo Activity	• Comparable anti-tumor activity at 1/10th-1/5th of daraxonrasib dose in multiple mouse models
PK Properties	• Higher oral bioavailability (improved %F) • Lower CL, longer T1/2 • Preferential tumor distribution with longer residence time
Cellular Potency	• Improved potency across RAS-altered cell lines
CYPA Binding	• Improved binding affinity (8-21x)

Clinical findings have generally been consistent with preclinical data

Phase 1 Data

Encouraging interim efficacy results

Robust monotherapy response rates with encouraging combination data

Favorable safety and tolerability results

Generally well-tolerated and combinable within PAD

Potential to become RAS-targeting agent of choice as monotherapy and combination backbone

[1] These data were generated in head-to-head assay and in vivo experiments
CL: clearance; T1/2: half life; CYPA: cyclophilin A; PAD: pharmacologically active dose

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AURORAS-1: Phase 1 clinical trial in patients with KRASm solid tumors

Study Overview

AURORAS-1

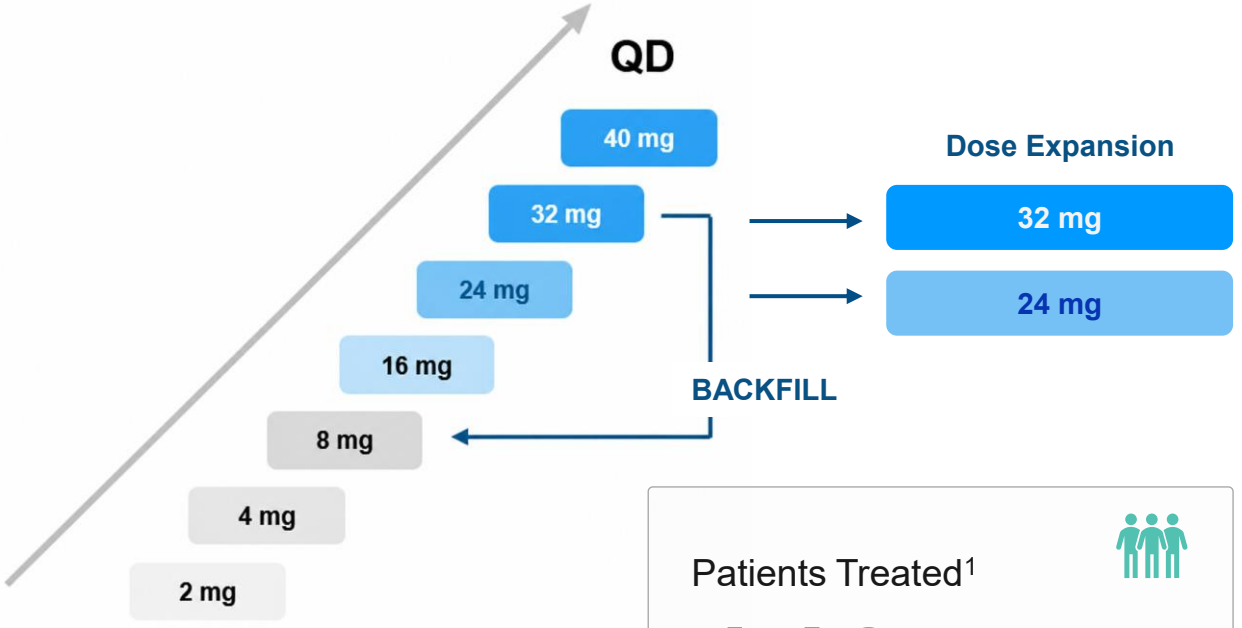
Study Population

- KRAS G12X/G13X NSCLC and select GI tumors; PDAC
- ECOG 0-1
- Measurable disease

Study Endpoints

- Primary: DLTs, Safety, PK
- Secondary: ORR, DCR, TTR, DOR, PFS, OS

Dose Escalation and Expansion



Patients Treated[1]

148

[1] As of 1Sep2026

NSCLC: non-small-cell lung cancer; GI: gastrointestinal; PDAC: pancreatic ductal adenocarcinoma; ECOG: Eastern Cooperative Oncology Group; DLT: dose-limiting toxicity; PK: pharmacokinetics; ORR: objective response rate; DCR: disease control rate: TTR: time to response: DOR: duration of response; PFS: progression free survival; OS: overall survival; QD: once daily

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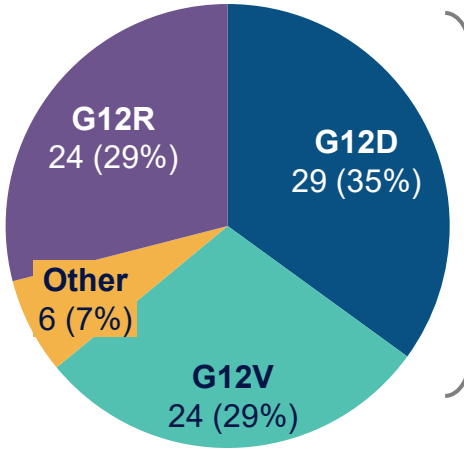
Patient demographics and baseline characteristics

	Total (N=148)
Age, median (range), years	65.0 (36–88)
Female, n (%)	62 (41.9)
ECOG, PS 1, n (%)	109 (73.6)
Tumor type, n (%)	
PDAC	87 (58.8)[1]
NSCLC	38 (25.7)[2]
BTC	13 (8.8)
Other	10 (6.8)

	PDAC (n=83)[1]
Sites of metastatic disease – liver/lung, n (%)	61/31 (73.5/37.3)
Prior systemic therapy[3]	
Regimen containing gemcitabine, n (%)	63 (75.9)
Regimen containing 5-FU, n (%)	77 (92.8)
Median (range) number of prior lines	2.0 (1–8)

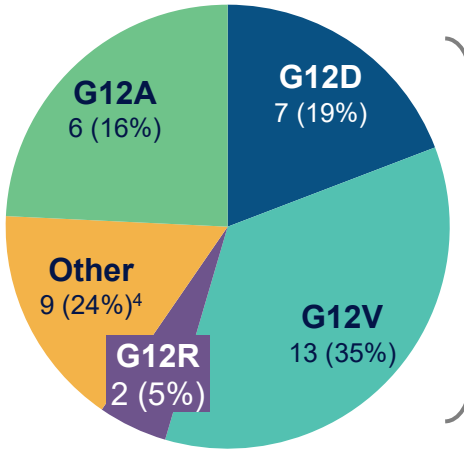
	NSCLC (n=37)[2]
Smoking status, n (%)	
Never/former	10 (27.0) / 24 (64.9)
Current	3 (8.1)
Sites of metastatic disease – brain, n (%)	10 (27.0)
Prior systemic therapy[3]	
ICI, n (%)	35 (94.6)
Platinum-based chemotherapy, n (%)	36 (97.3)
Median (range) number of prior lines	2.0 (0–6)

KRAS Mutation Type (n; %)



PDAC (N=83)

- ~64% of patients analyzed had either a G12D or G12V mutation
- Higher proportion of patients with G12R mutation enrolled relative to historical PDAC incidence of ~15%[5]



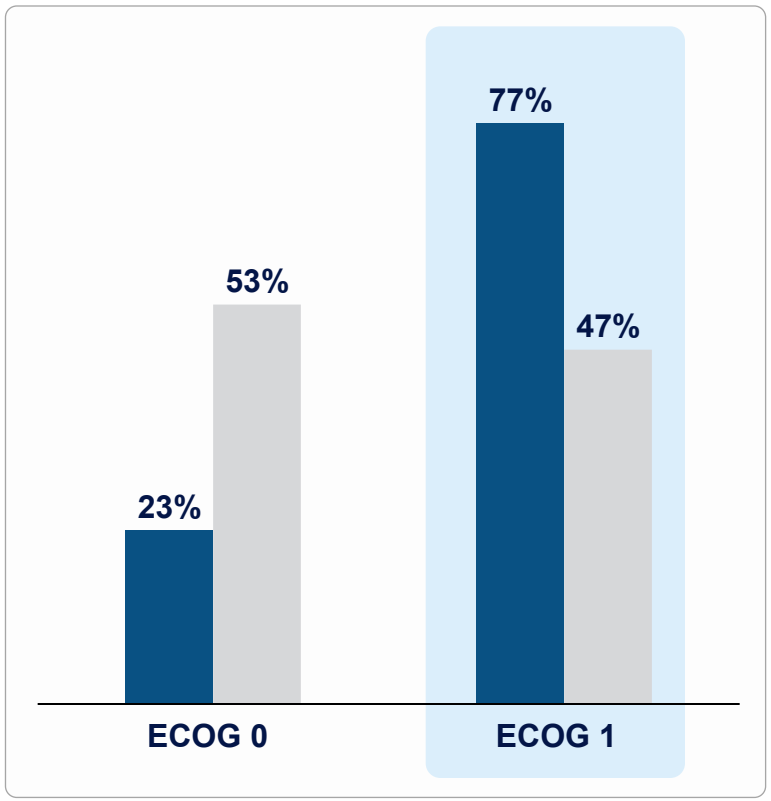
NSCLC (N=37)

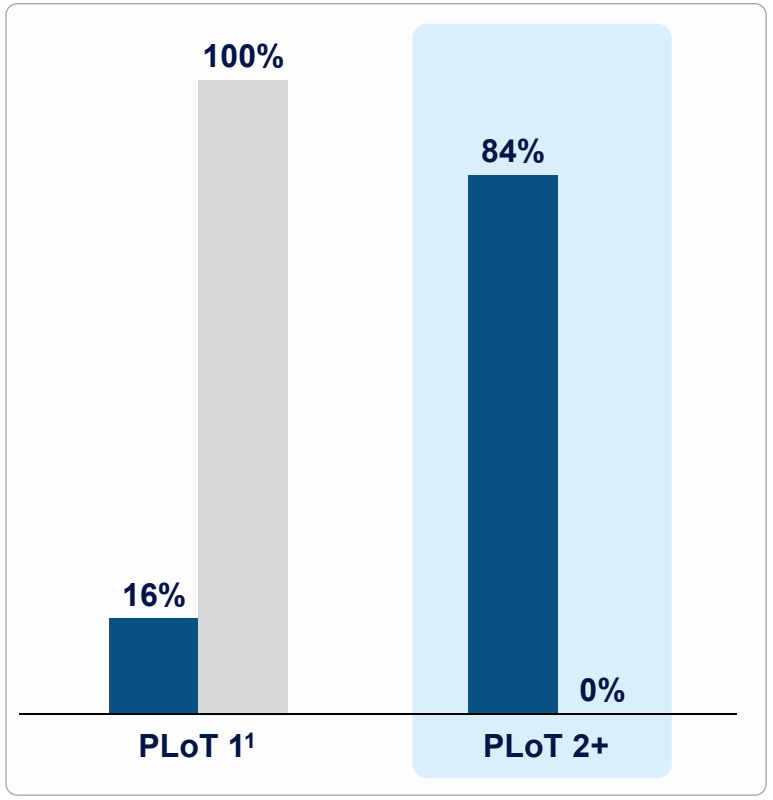
- ~54% of patients analyzed had either a G12D or G12V mutation

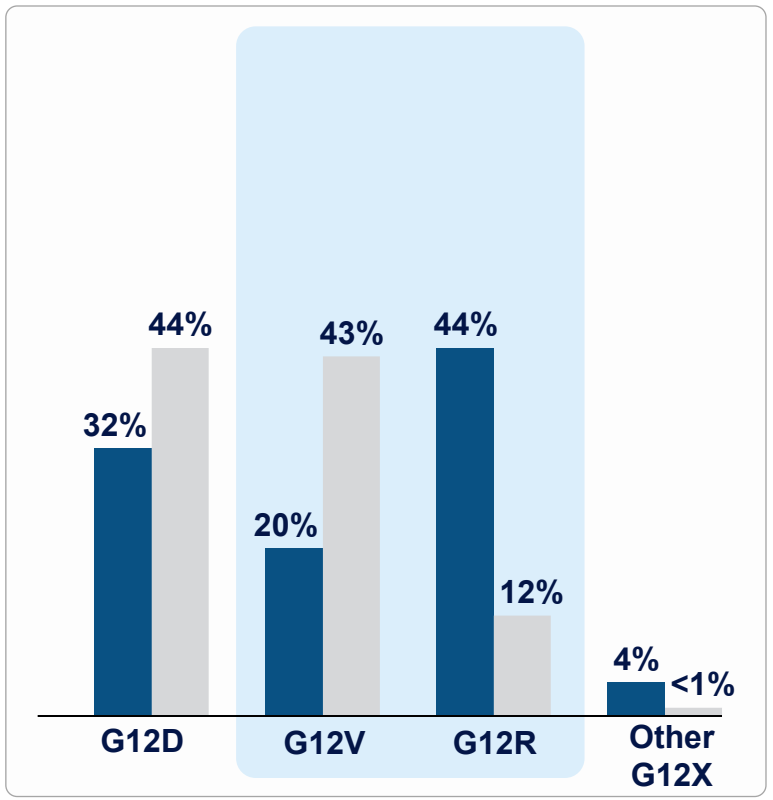
DCO 1Sep2026

[1]4 patients with PDAC who received ERAS-0015 in the first-line setting were not included; [2]1 patient with NSCLC who had received prior KRAS G12C inhibitor therapy was not included; [3]No patients had received prior RAS inhibitor therapy; [4]Includes a patient with G12D and G12V double mutations; [5]Varghese et al (Nat Med 2025) 5-FU, 5-fluorouracil; BTC, biliary tract cancer; ECOG PS, Eastern Cooperative Oncology Group Performance Status; ICI, immune checkpoint inhibitor; NSCLC, non-small cell lung cancer; PDAC, pancreatic ductal adenocarcinoma.

AURORAS-1 PDAC pts at 32 mg had worse ECOG status, more pre-treatment, and lower freq. of G12V/higher freq. of G12R mutations than RASolute 302 pts at 300 mg







AURORAS-1 (32 mg QD) RASolute 302 (300 mg QD)

Cross-study comparisons, not 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*

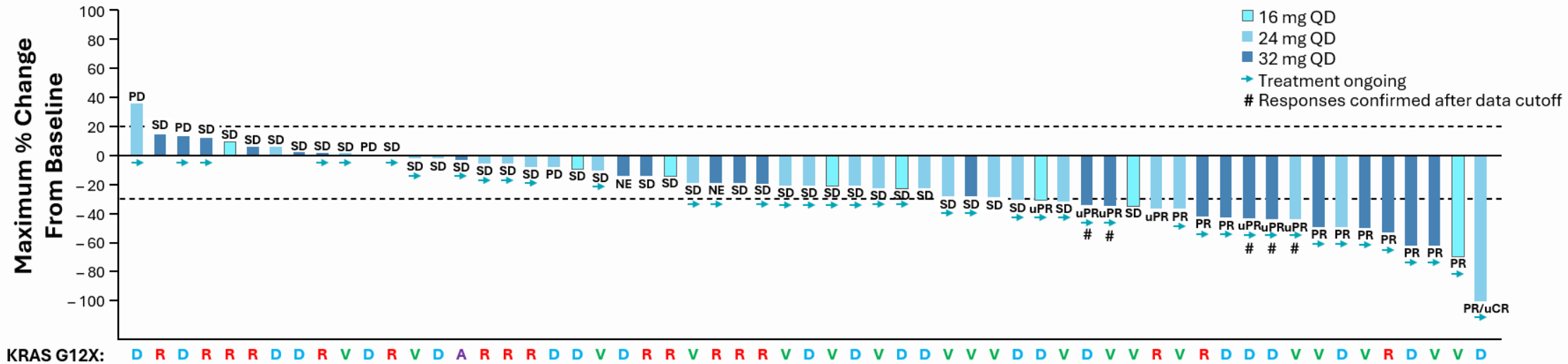
ECOG: ERAS-0015 DCO 1Sep2026 (N=26); Daraxonrasib O'Reilly et al. NEJM 2026 (N=228); Prior line of therapy (PLoT): ERAS-0015 DCO 1Sep2026 (N=25); Daraxonrasib O'Reilly et al. NEJM 2026 (N=228); KRAS G12X Mutation Status: ERAS-0015 DCO 1Sep2026 (N=25; Efficacy-evaluable analysis set); Daraxonrasib FDA CDER (Multi-Disciplinary Review 2026; N=217); % of RAS G12X population

[1]PLoT 1: includes patients described as 0 or 1 prior lines of therapy (PLoT) in the O'Reilly 2026 NEJM reference where patients with 0 prior lines of therapy were defined as being treated in the neoadjuvant/adjuvant setting and whose diagnosis of metastatic disease was <6 months from the last dose

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Encouraging efficacy results across PAD in patients with 2L+ KRAS G12X PDAC

2L+ KRAS G12X PDAC (16-32 mg QD; N=60)



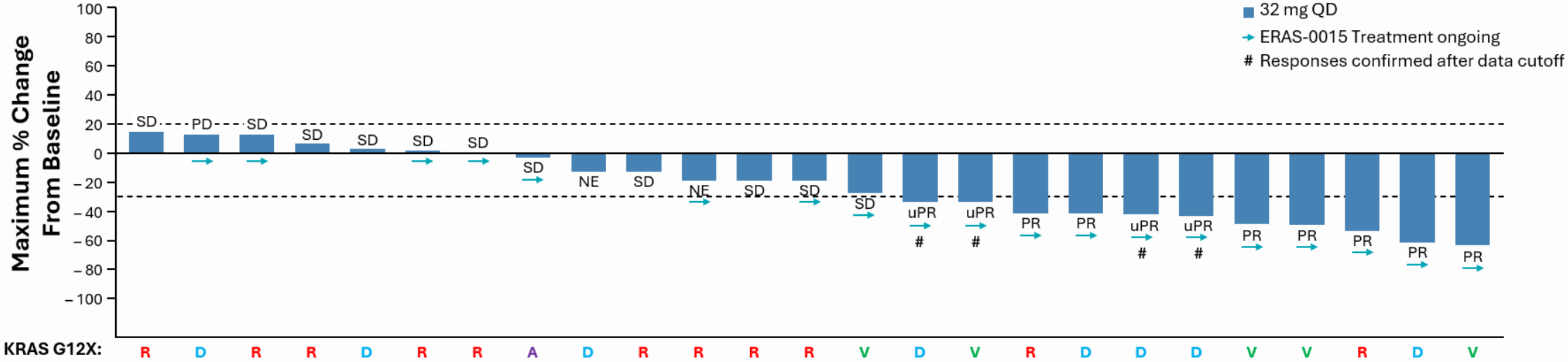
cORR by Dose

Dose	16-24 mg N=35 (%)[2]	32 mg N=25 (%)[2]
cORR[1]	14%	44%
DCR	89%	88%

DCO 1Sept 2026; Efficacy-evaluable analysis set: all participants who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date; One patient is not included because no post-baseline tumor assessment was available. All doses administered QD;
[1] cORR included confirmed responses only; [2]16 mg: one unconfirmed response will not confirm; 24 mg: one of two unconfirmed responses confirmed after DCO, one will not confirm; 32 mg: all four unconfirmed responses confirmed after DCO
DCR: disease control rate; 2L: second line; NE: not evaluable; cORR: confirmed objective response rate; PD: progressive disease; PR: partial response; PDAC: pancreatic ductal adenocarcinoma; QD: once daily; SD: stable disease; uCR: unconfirmed complete response; uPR: unconfirmed PR.

At 32 mg: 44% cORR in KRAS G12X despite high proportion of later line treatment (84% in 3L+) and G12R mutations (44% G12R); 69% cORR in KRAS G12D or G12V

2L+ KRAS G12X PDAC (32 mg QD; N=25)



ERAS-0015 2L+

32 mg	KRAS G12D or V (N=13)	KRAS G12X (N=25)
cORR[1]	69%	44%
DCR	85%	88%

KRAS G12D 95% CI: 24.5%, 91.5%[2]

Of the patients treated at 32 mg:

16% treated in 2L; 84% treated in 3L+

All patients with responses were still on treatment

KRAS Allele-Specific cORR

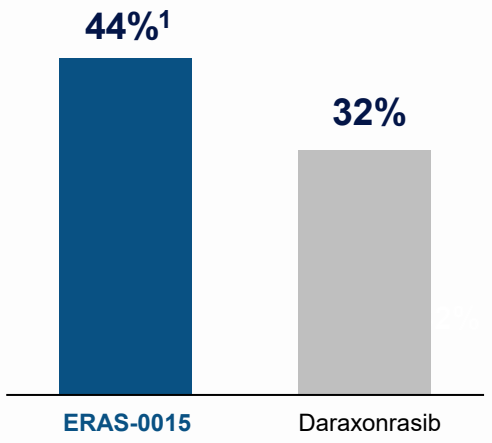
KRAS Mutation	32 mg (%)
G12D (N=8)	63%
G12V (N=5)	80%
G12R (N=11)	18%

DCO 1Sept 2026; Efficacy-evaluable analysis set: all participants who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date; All doses administered QD
[1]cORR is confirmed as all unconfirmed PRs were confirmed after the DCO; [2]The 95% confidence interval (CI) was calculated using the exact Clopper–Pearson method.
2L: second line; 3L: third line; NE: not evaluable; cORR: confirmed objective response rate; PD: progressive disease; PR: partial response; PDAC: pancreatic ductal adenocarcinoma; QD: once daily; SD: stable disease; uPR: unconfirmed PR.

ERAS-0015: Potential best-in-class activity observed across PDAC mutations and allele-specific approaches

KRAS G12X cORR (%)

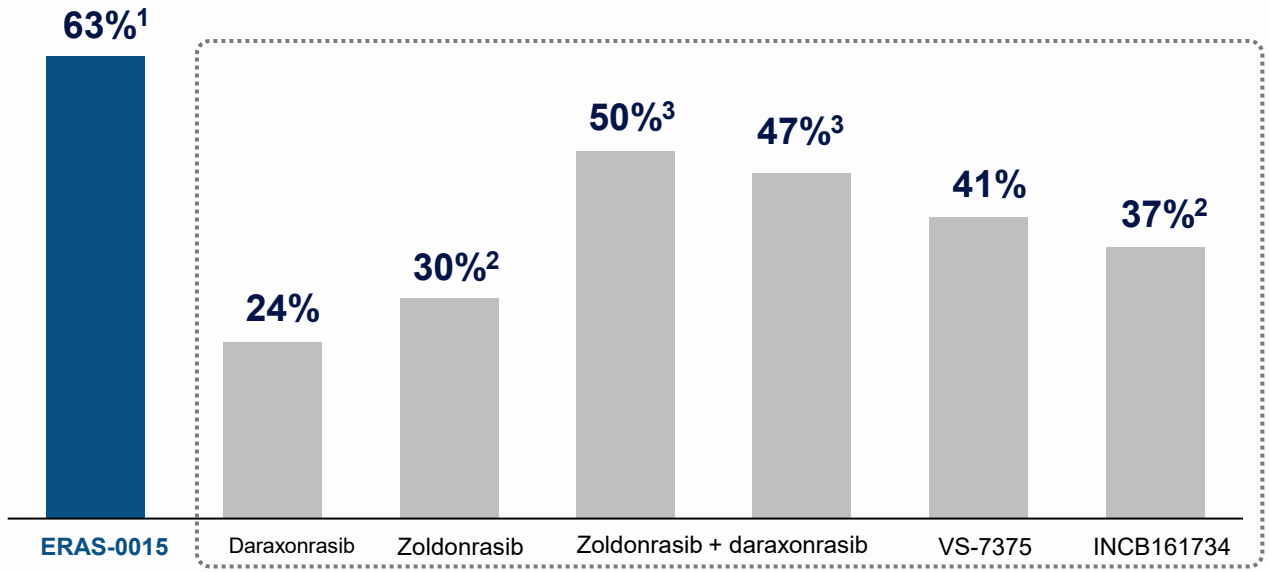
90% of PDAC



Dose	32 mg QD	300 mg QD
N	25	237
Tx Line	2L+	2L
2L / 3L+ (% of pts)	16% / 84%	100% / 0%

KRAS G12D ORR (%)

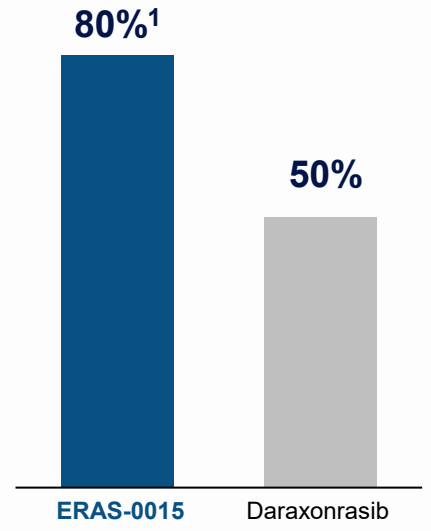
40% of PDAC



32 mg QD	300 mg QD	1200 mg QD	Z:1200 mg QD/D:300 mg QD		900 mg QD	1200 mg QD
8	95	40	30	30	22	41
2L+	2L	1L+	2L	3L+	2L+	2L+

KRAS G12V cORR (%)

30% of PDAC



32 mg QD	300 mg QD
5	94
2L+	2L

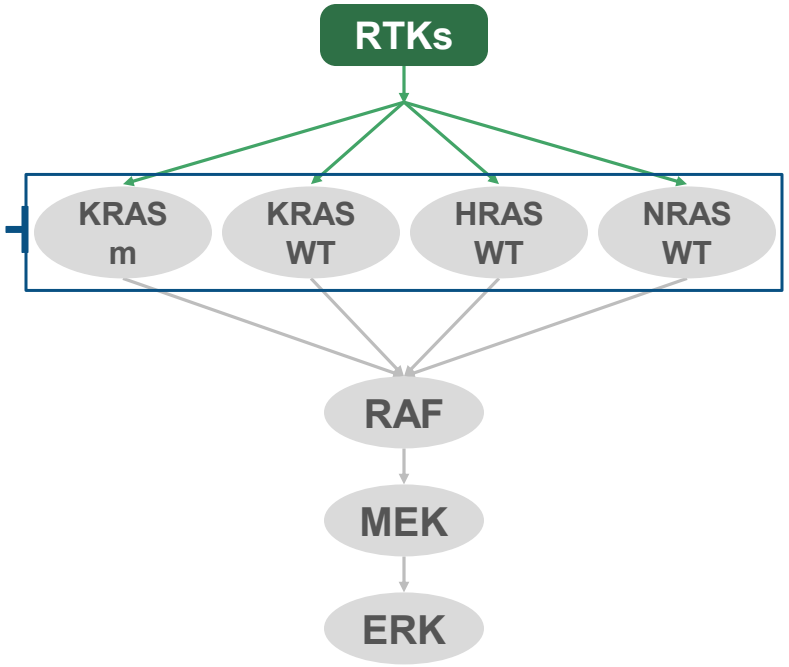
Cross-study comparisons, not head-to-head clinical trials: *Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable*

Sources: Varghese et al (Nat Med 2025) ERAS-0015 (AURORAS-1; DCO 1Sep2026); daraxonrasib (O'Reilly et al. NEJM 2026; FDA CDER Multi-Disciplinary Review 2026); zoldonrasib G12D (ASCO-GI 2025); zoldonrasib + daraxonrasib G12D (ESMO-GI 2026); GFH7375/VS-7375 G12D Corporate Presentation (Oct 2026); Incyte G12D (ASCO-GI 2026); [1] cORR is confirmed as all unconfirmed partial responses were confirmed after the DCO; [2] uORR includes unconfirmed responses; [3] Reported cORR; cORR: confirmed objective response rate; PDAC: pancreatic ductal adenocarcinoma; QD: once daily; Tx Line: treatment line

ERAS-0015 is designed to block MAPK pathway and K/H/NRAS WT RAS bypass pathways known to reactivate MAPK signaling and drive resistance to allele-specific and pan-KRAS inhibitors

ERAS-0015: pan-RAS

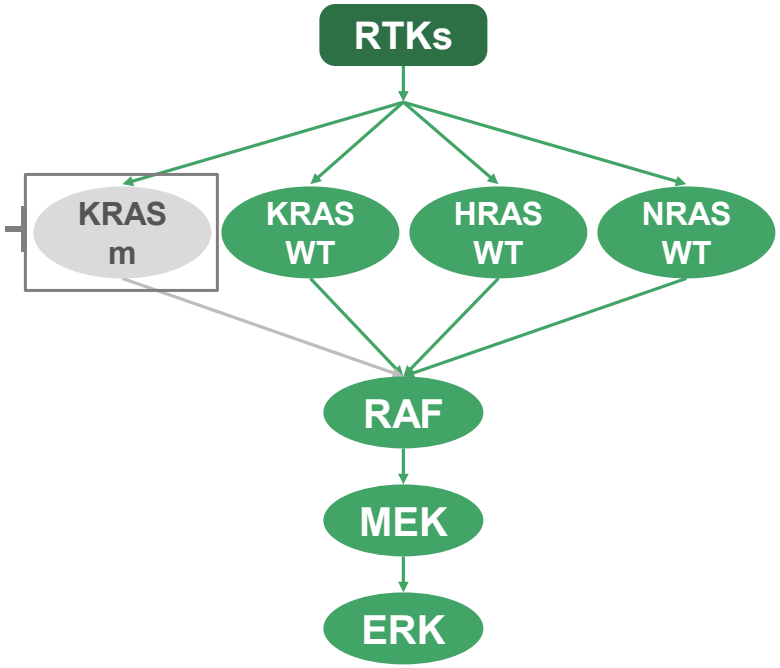
Blocks KRASm and K/H/N RAS WT



Designed to suppress potential resistance route via K/H/N RAS WT

Mutant-selective inhibitors

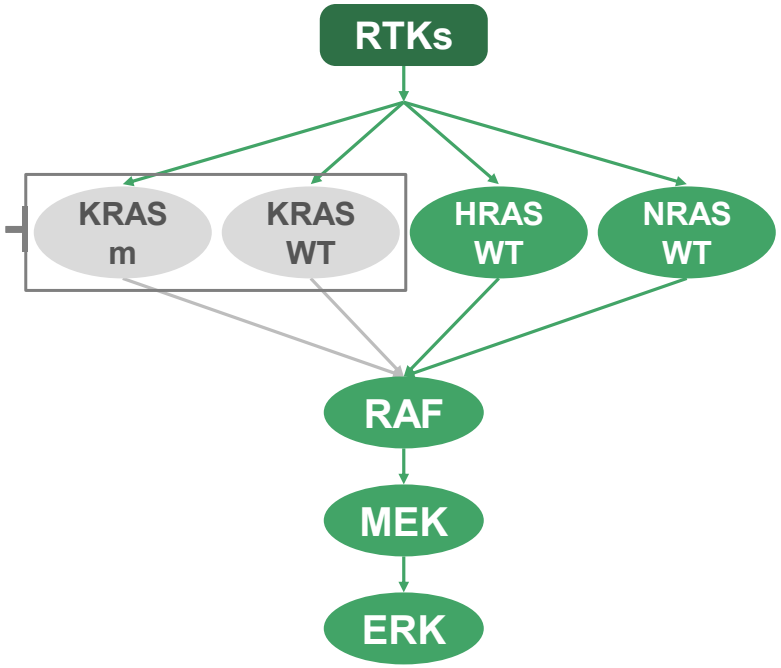
Blocks KRASm



Fails to address signaling that may arise from K/H/N RAS WT

Pan-KRAS inhibitors

Blocks KRASm and KRAS WT



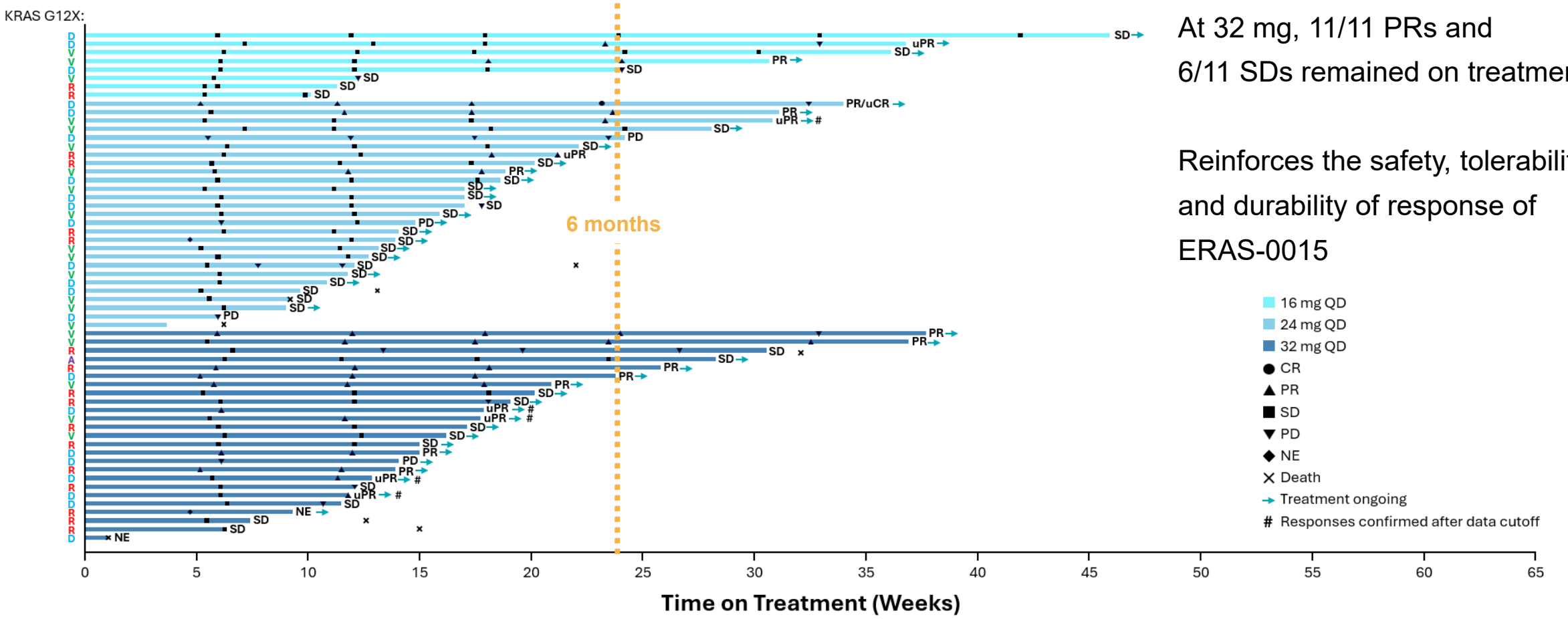
Fails to address signaling that may arise from HRAS/NRAS WT

Adapted from Ryan MB et al. Cell Rep. 2022;39:110993. PMID: 35732135.

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Most patients with 2L+ KRAS G12X PDAC remained on treatment

2L+ PDAC (16-32 mg QD; N=60)



At 32 mg, 11/11 PRs and 6/11 SDs remained on treatment

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

DCO 1Sept2026

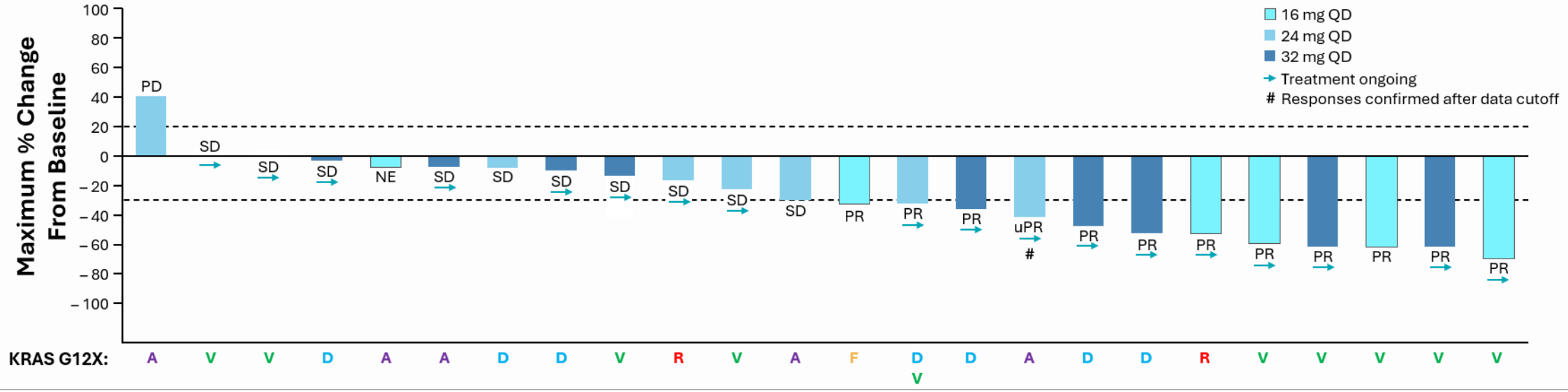
Response at the end of the bar represents the best objective response based on investigator assessment denoted as PR for confirmed PR or uPR for unconfirmed PR.

CR: complete response; NE: not evaluable; PD: progressive disease; PDAC: pancreatic ductal adenocarcinoma; PR: partial response; QD: once daily; SD: stable disease; uCR: unconfirmed CR; uPR: unconfirmed PR.

Encouraging efficacy results (60% cORR) in patients with post-ICI/platinum (2/3L) KRAS G12X NSCLC at 32 mg

AURORAS-1

2L+ NSCLC (16-32 mg QD; N=24)



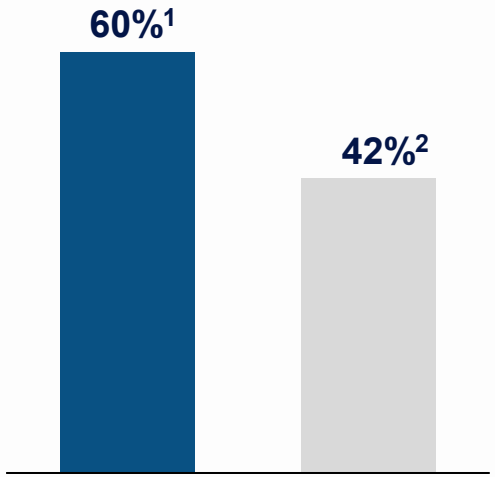
Population	Post-ICI/platinum (2/3L)		2L+	
Dose	**32 mg** N=5	**16-32 mg** N=12	**32 mg** N=10	**16-32 mg** N=24
cORR[1]	**60%**	58%	50%	50%[2]
DCR	100%	92%	100%	92%

DCO 1Sept 2026; Efficacy-evaluable analysis set: all participants who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date; All doses administered QD
[1]cORR is confirmed as all unconfirmed PRs were confirmed after the DCO; [2] The one unconfirmed response was confirmed after the DCO
DCR: disease control rate. ICI: immune checkpoint inhibitor; 2L: second line; 3L: third line; NE: not evaluable; NSCLC: non-small cell lung cancer; cORR: confirmed objective response rate; PD: progressive disease; PR: partial response; QD: once daily; SD: stable disease.

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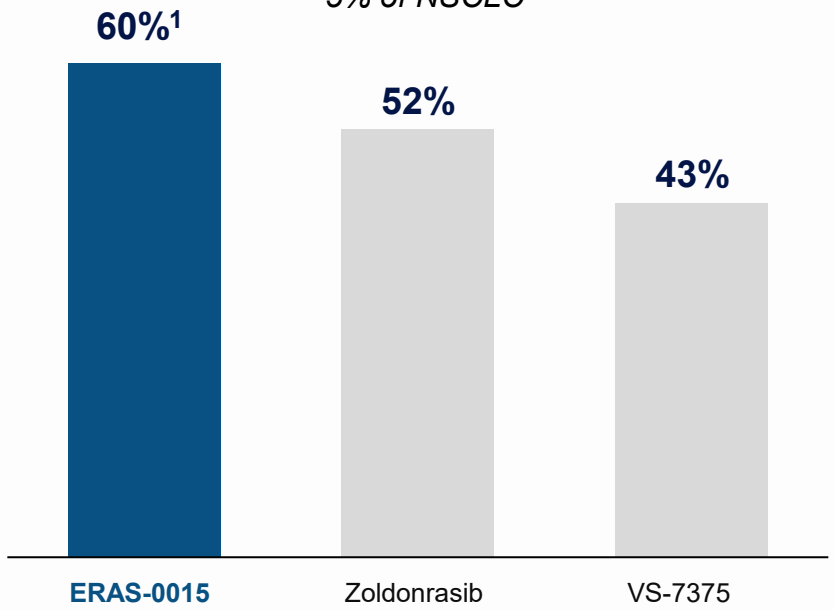
ERAS-0015: Potential best-in-class activity observed across NSCLC mutations and allele-specific approaches

KRAS G12X
30% of NSCLC



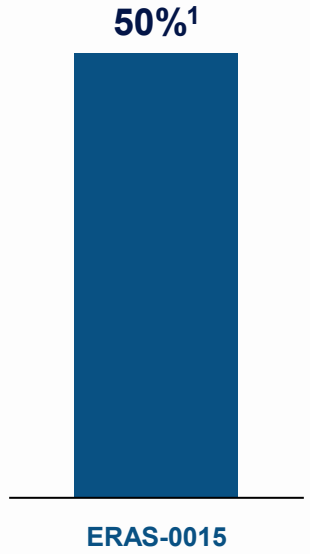
Dose	32 mg QD	160-220 mg QD
N	5	33
Tx Line	2L/3L	2L/3L

KRAS G12D
5% of NSCLC



32 mg QD	1200 mg QD	600 mg QD
5	27	23
2L+	2L/3L	2L+

KRAS G12V
7% of NSCLC



32 mg QD
4
2L+

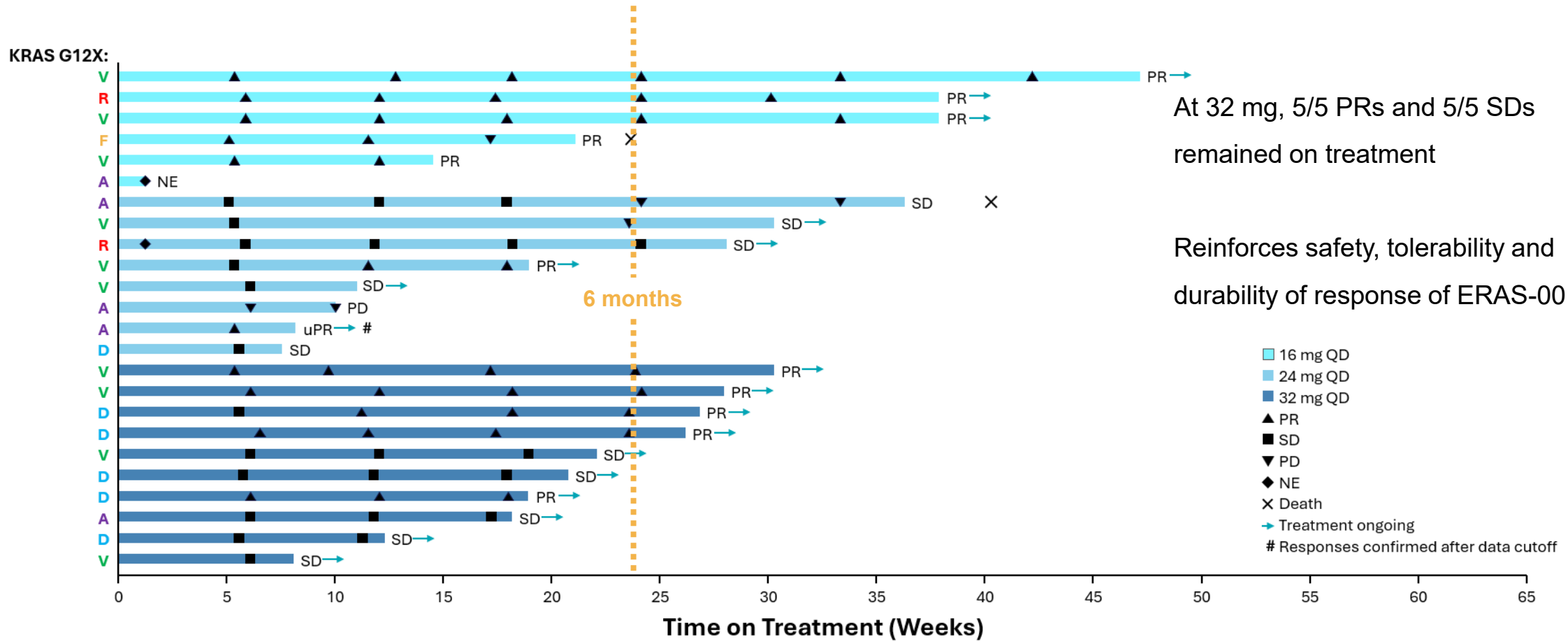
Cross-study comparisons, not head-to-head clinical trials: *Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable*

Sources: NCI SEER Database. ERAS-0015 (AURORAS-1; DCO 1Sep2026); daraxonrasib (Arbour et al. NEJM 2026); zoldonrasib G12D (AACR 2026); GFH7375/VS-7375 G12D Corporate Presentation (Oct 2026)
[1] cORR represents confirmed responses; [2] Single dose data for daraxonrasib in NSCLC are not publicly available; NSCLC: non-small-cell lung cancer; Tx Line: treatment line

Most patients with 2L+ KRAS G12X NSCLC remained on treatment, including all patients at 32 mg QD

AURORAS-1

2L+ NSCLC (16-32 mg QD; N=24)



At 32 mg, 5/5 PRs and 5/5 SDs remained on treatment

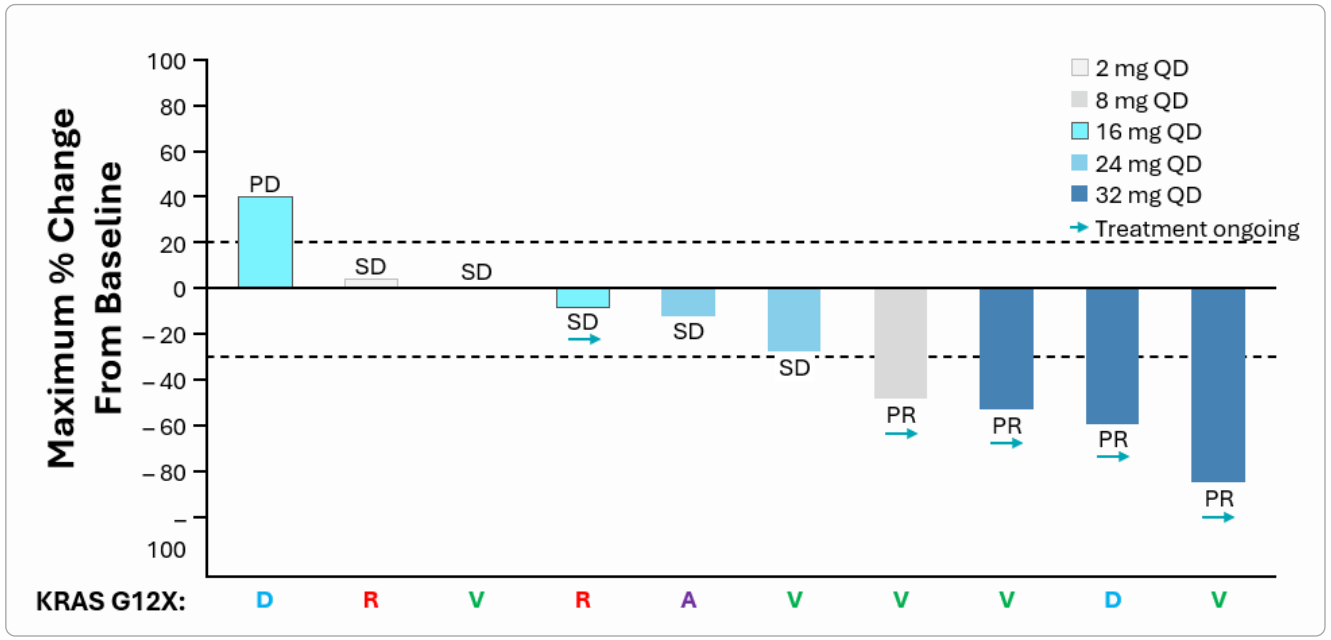
Reinforces safety, tolerability and durability of response of ERAS-0015

DCO 01Sep2026. 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.
NSCLC: non-small cell lung cancer; PD: progressive disease; PR: partial response; QD: once daily; SD: stable disease; uPR:, unconfirmed PR.

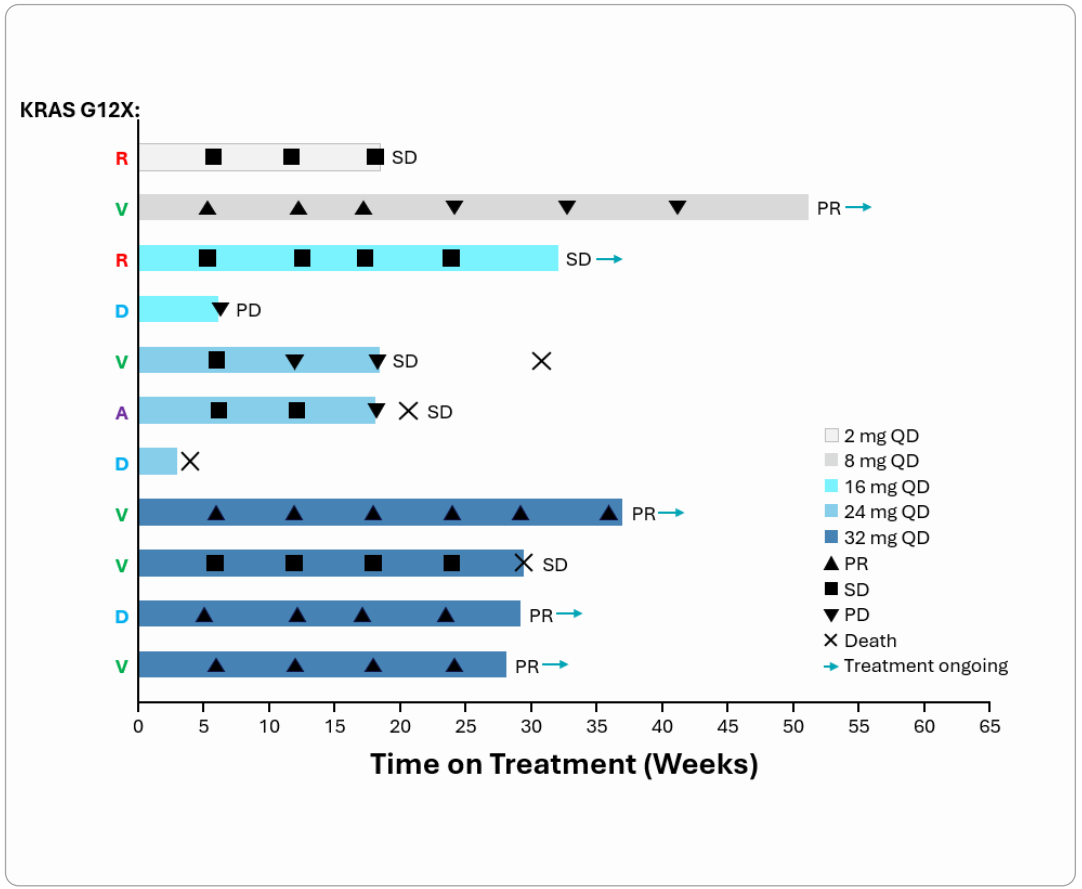
ERASCA

Encouraging signs of efficacy in KRAS G12X biliary tract cancer (BTC)

1L+ KRAS G12X BTC (2-32 mg QD; N=11)[1]



1L+ KRAS G12X BTC (2-32 mg QD; N=11)



Population	1L+	
Dose	**32 mg** N=4	**8-32 mg** N=10
cORR[2]	75%	40%

DCO 1 Sept 2026; [1]Efficacy-evaluable analysis set: all participants who received first dose of ERAS-0015 at least 8 weeks prior to the data cutoff date; [1] One patient is not displayed in the waterfall plot because no post-baseline tumor assessment was available [2] cORR is confirmed objective response rate; [3] One patient declined SOC and was treated with ERAS-0015 in 1L; remainder of the patients were treated in 2L+
BTC: biliary tract cancer; 1L: first line; 2L: second line; ORR: objective response rate; PD: progressive disease; PR: partial response; QD: once daily; SD: stable disease.

RASm BTC: Opportunity to address substantial unmet need

Estimated annual RASm BTC diagnoses[1]

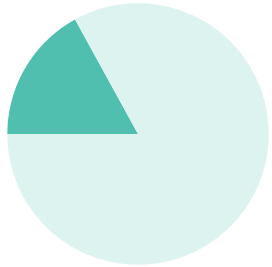
Illustrative estimates pending validation

~5,000
United States

~45,000
Ex-United States

KRAS-Mutation Prevalence

~17% of BTC[2]



1L Standard-of-Care Benchmark

29% ORR

Pembrolizumab + gemcitabine/cisplatin[3]

Five-Year Relative Survival

~2–4%

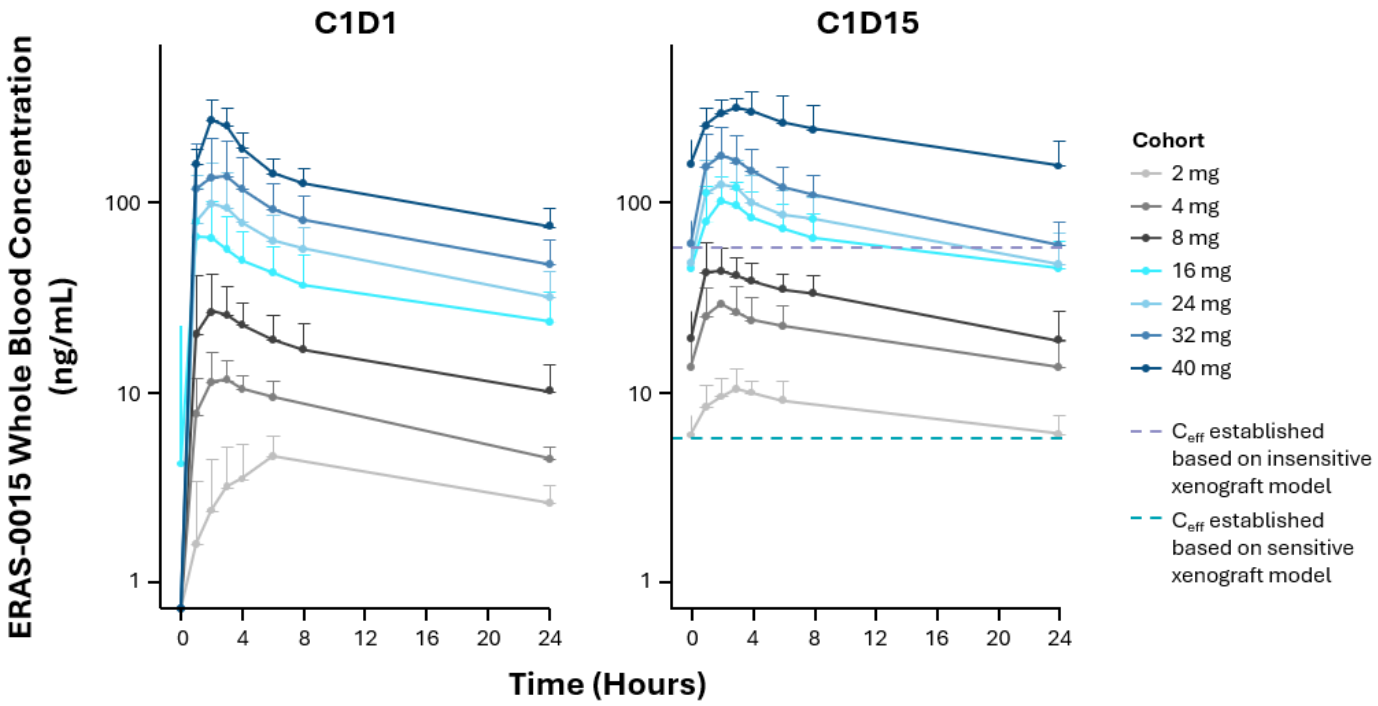
in distant-stage BTC[4]

[1] US: NCI SEER Training Module Biliary Tract Cancer (Accessed Nov 2026); Global: GBD 2023 Cancer Collaborators (Lancet 2025), IARC GLOBOCAN 2024, Rumgay et al. Eur J of Cancer (2022)
[2] Moffatt et al. JCO 2023
[3] Kelley RK, et al. Lancet. 2023;401:1853–1865 (KEYNOTE-966, unselected BTC).
[4] American Cancer Society. Bile duct and gallbladder cancer survival rates (2025). SEER diagnoses 2015–2021. Range across BTC subtypes, not a pooled estimate.

ERASCA

Well-behaved pharmacokinetics (PK)

Mean PK Profiles



- **PK exposure** increased with dose with low-to-moderate PK variability
- **Pharmacologically active dose (PAD)** range: 16-32 mg QD

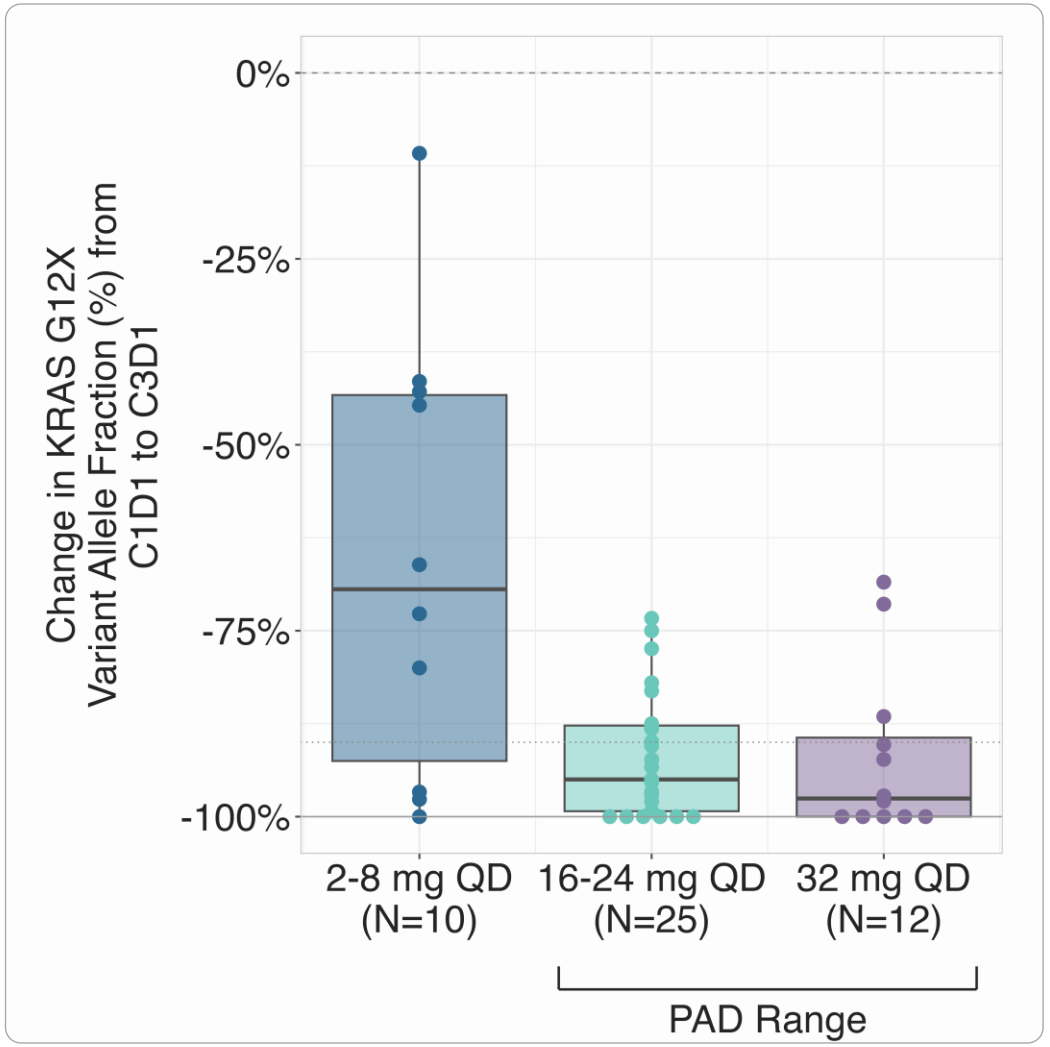
  Mean steady-state average exposure ($C_{avg, ss}$) at ≥16 mg exceeded target exposure threshold (based on insensitive NCI-H727 CDX model)
- **Current clinical data suggest 32 mg QD has potential favorable benefit/risk profile**

  >95% of patients projected to have steady-state PK exceeding Ceff and providing maximal PK/ pharmacodynamic coverage at 32 mg QD

AURORAS-1 PK data are as of 18Sep2026; data points in the figures are presented as mean ± standard deviation
PK: pharmacokinetics; QD: once daily; CDX: cell line-derived xenograft

Maximal KRAS G12X ctDNA reduction at 32 mg QD dose

Change in solid tumor ctDNA KRAS G12X VAF from C1D1 to C3D1



AURORAS-1 patients receiving ERAS-0015 at 32 mg QD showed **reductions in KRAS G12X variant allele fraction**:

- 83% (10/12) showed at least 75% reduction
- 75% (9/12) showed at least 90% reduction
- 42% (5/12) showed 100% reduction

ctDNA data as of October 2, 2026; data points in the figures are presented as median ± IQR/range.
C, cycle; Ceff, effect-site concentration; ctDNA, circulating tumor deoxyribonucleic acid
D: day; IQR: interquartile range; PAD: pharmacologically active dose; VAF: variant allele fraction.

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ERAS-0015 was generally well-tolerated with mostly low-grade TRAEs

Summary of TRAEs Occurring in ≥10% of Patients

All Patients Treated at PAD of ERAS-0015 16-32 mg QD (N=122)

TRAEs, n (%)	Grade 1	Grade 2	Grade 3[2]	Grade 4	Grade 5	All Grades
Rash[1], n (%)	61 (50.0)	34 (27.9)	4 (3.3)	0	0	**99 (81.1)**
Diarrhea, n (%)	30 (24.6)	8 (6.6)	1 (0.8)	0	0	**39 (32.0)**
Nausea, n (%)	22 (18.0)	7 (5.7)	0	0	0	**29 (23.8)**
Stomatitis, n (%)	15 (12.3)	8 (6.6)	3 (2.5)	0	0	**26 (21.3)**
TRAEs leading to dose interruptions, n (%)						**15 (12.3)**
TRAEs leading to dose reductions, n (%)						**13 (10.7)**
TRAEs leading to dose discontinuations, n (%)						**2 (1.6)**

No prophylaxis recommended in the protocol

DCO 1Sep2026; Safety set includes all patients that have received at least one dose of ERAS-0015

[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).

PAD: pharmacologically active dose; TRAE: treatment-related adverse event

[2] As disclosed by the Company in April 2026, 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. No additional Grade 5 TRAEs have occurred.

ERAS-0015 was generally well-tolerated at 32 mg QD dose

Summary of TRAEs Occurring in ≥10% of Patients

All Patients Treated at PAD (N=122) and 32 mg (N=54) of ERAS-0015

TRAEs, n (%)	16-32 mg QD (N=122)	32 mg QD (N=54)
Rash[1], n (%)	99 (81.1)	43 (79.6)
Diarrhea, n (%)	39 (32.0)	19 (35.2)
Nausea, n (%)	29 (23.8)	12 (22.2)
Stomatitis, n (%)	26 (21.3)	14 (25.9)
TRAEs leading to dose interruptions, n (%)	15 (12.3)	7 (13.0)
TRAEs leading to dose reductions, n (%)	13 (10.7)	9 (16.7)
TRAEs leading to dose discontinuations, n (%)	2 (1.6)	0

No prophylaxis recommended in the protocol

DCO 1Sep2026

[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).

PAD: pharmacologically active dose; QD: once daily; TRAE: treatment-related adverse event.

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100% Median Relative Dose Intensity at PAD and 32 mg QD dose

Patients Treated With ERAS-0015 at PAD and 32 mg

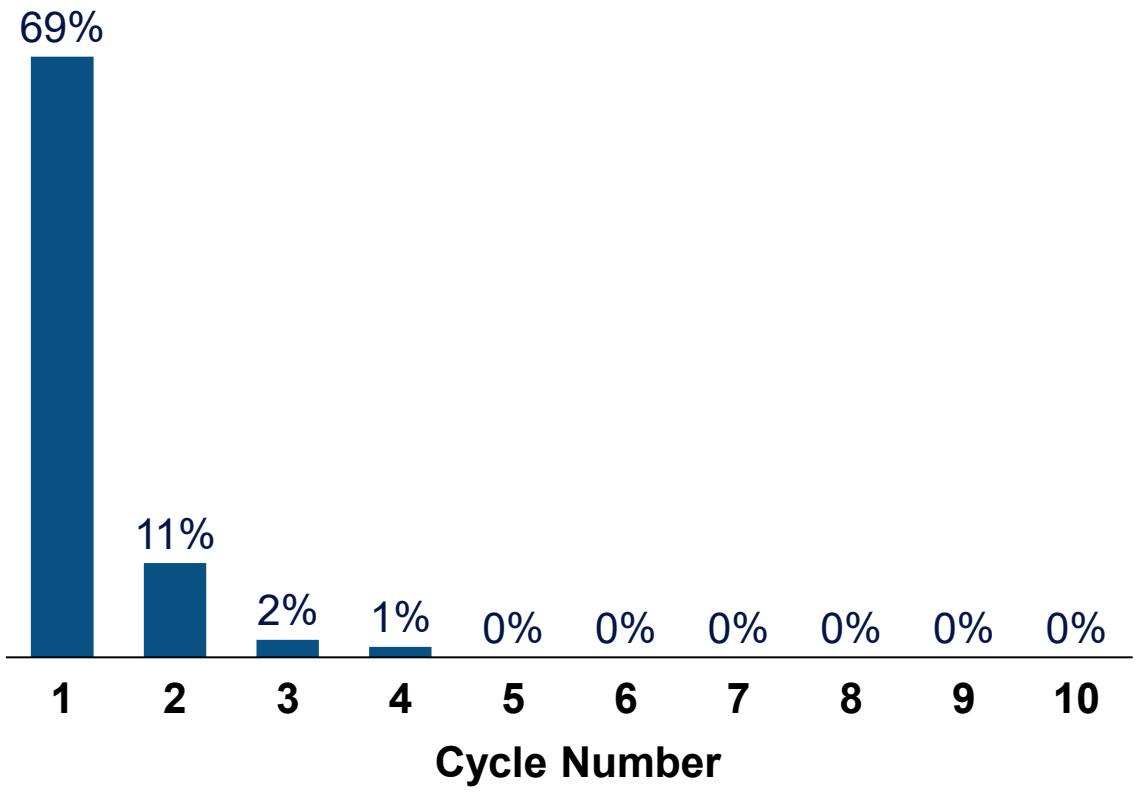
	16-32 mg QD (N=122)	32 mg QD (N=54)
Duration of ERAS-0015 Exposure (weeks)		
Median	14.4	14.5
Min, max	0.7, 47.0	0.9, 37.7
Relative dose intensity of ERAS-0015 (%)		
n	119	51
Median	**100.0**	**100.0**
Mean (SD)	94 (15.0)	91 (15.5)

DCO 1Sep2026
PAD: pharmacologically active dose; QD: once daily; SD: standard deviation

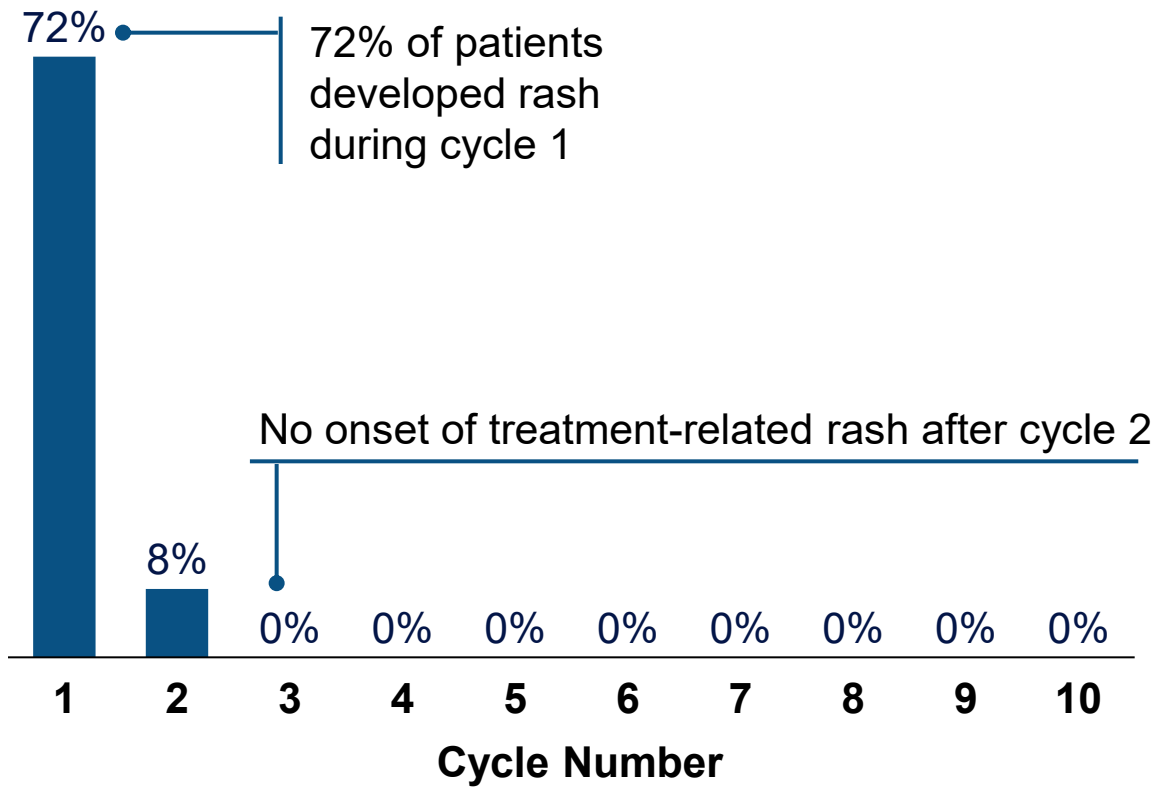
Onset of treatment-related rash occurred principally within the first 2 cycles

Time to onset (first incidence) of rash

ERAS-0015 (16-32 mg QD; N=122)



ERAS-0015 (32 mg QD; N=54)



DCO 1Sep2026; Safety set includes all patients that have received at least one dose of ERAS-0015
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); Percentages represent patients with first treatment-related rash onset during each cycle divided by the number of patients who started that cycle. Denominators vary by cycle

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

ERAS-0015 data support combination development in Big 3 indications

+ gemcitabine / nab-paclitaxel (D1,15)

24 mg QD dose escalation cohort currently being dosed

16 mg cohort DLT window cleared

Generally well tolerated with favorable safety data; no unexpected safety signals as of DCO

Efficacy data not yet available



Support further development in 1L PDAC

+ pembrolizumab

32 mg QD DLT window cleared; no DLTs

Generally well tolerated with favorable safety data; no unexpected safety signals as of DCO

Multiple responses observed in multiple solid tumor types in dose escalation



Support further development in 1L NSCLC, including combo with carbo/pem/pembro (initiated in Q4)

+ panitumumab

16 mg QD generally well tolerated with favorable safety data, and within PAD

No unexpected safety signals as of DCO

Multiple responses observed in dose escalation:

- Ongoing confirmed PRs in 2 out of 2 patients at 24 mg QD then dose reduced to 16 mg QD[1]
- uPR in 1 out of 4 patients at 16 mg QD[2]



Combo dose exploration ongoing; plan to make dose selection in 2027

DCO 14Oct2026

[1]Out of two efficacy-evaluable KRAS G12X CRC patients; both patients started at 24 mg and reduced to 16 mg QD; both of the 2 uPRs confirmed after DCO; [2]Out of four efficacy-evaluable KRAS G12X CRC patients dosed at 16 mg QD

DLT: dose-limiting toxicity; QD: once daily; PAD: pharmacologically active dose; carbo/pem/pembro: carboplatin/pemetrexed/pembrolizumab; PoC: proof of concept

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ERAS-0015: emerging data suggest differentiated safety, tolerability, combinability

Differentiated safety and tolerability

ERAS-0015 32 mg QD (N=54) vs. daraxonrasib 300 mg QD (N=241)

TRAEs occurring in ≥ 10% of patients	ERAS-0015 All Grade	Daraxonrasib All Grade	ERAS-0015 Grade 3+	Daraxonrasib Grade 3+
Rash[1]	**79.6%**	**85.5%**	**5.6%**	**13.7%**
Diarrhea	**35.2%**	**58.1%**	**0%**	**5.4%**
Stomatitis	**25.9%**	**53.1%**	**1.9%**	**12.0%**
Nausea	**22.2%**	**46.5%**	**0%**	**2.1%**
Fatigue	**13.0%**	**23.2%**	**0%**	**3.7%**
All other TRAEs occurring in ≥10% patients	Pruritus (11.1%)	vomiting, anemia, decreased appetite, paronychia, dry skin, AST increased, asthenia, thrombocytopenia	-	-

Protocol prophylaxis

ERAS-0015: No prophylaxis recommended in the protocol

Daraxonrasib: Rash, nausea and vomiting, and stomatitis prophylaxis recommended in the protocol

Cross-study comparisons, not 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*

DCO 1Sep2026; O'Reilly et al. NEJM 2026; 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); TRAE: treatment-related adverse event; SOC: standard of care; pembro: pembrolizumab; GnP: gemcitabine + nab-paclitaxel

Dose & combinability

Monotherapy Dose

ERAS-0015: Favorable benefit/risk data at 32 mg in PDAC and NSCLC

Daraxonrasib: Benefit/risk profile varies by tumor type (300 mg in PDAC; 200 mg in NSCLC)

Combinability

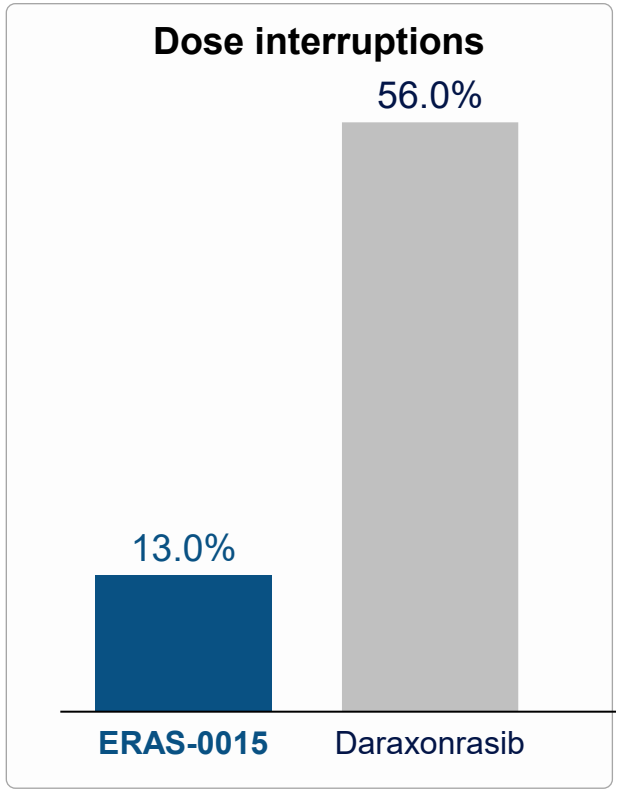
ERAS-0015: Data support combination with pembro (32 mg), GnP (D1,15; cleared 16 mg) and panitumumab (cleared 16 mg; dose exploration and selection ongoing)

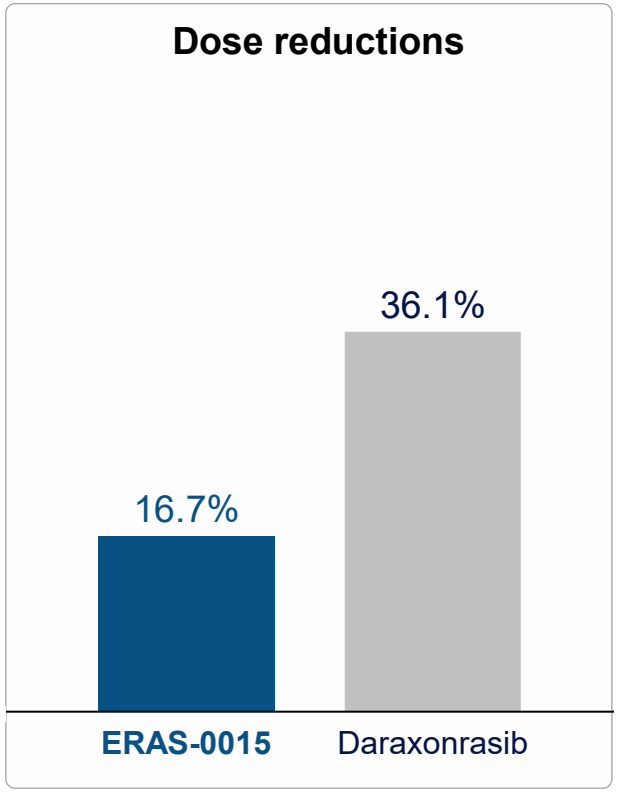
Daraxonrasib: 200 mg selected with pembro and with GnP; anti-EGFR combo not shown to be tolerated

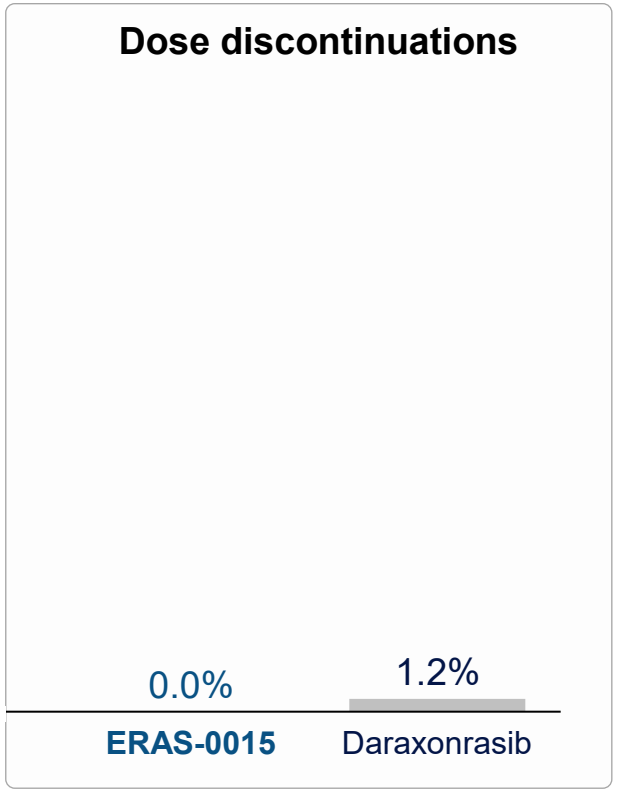
ERASCA

ERAS-0015: fewer reported TRAE-related treatment modifications

ERAS-0015 32 mg QD (N=54) vs. daraxonrasib 300 mg QD (N=241)







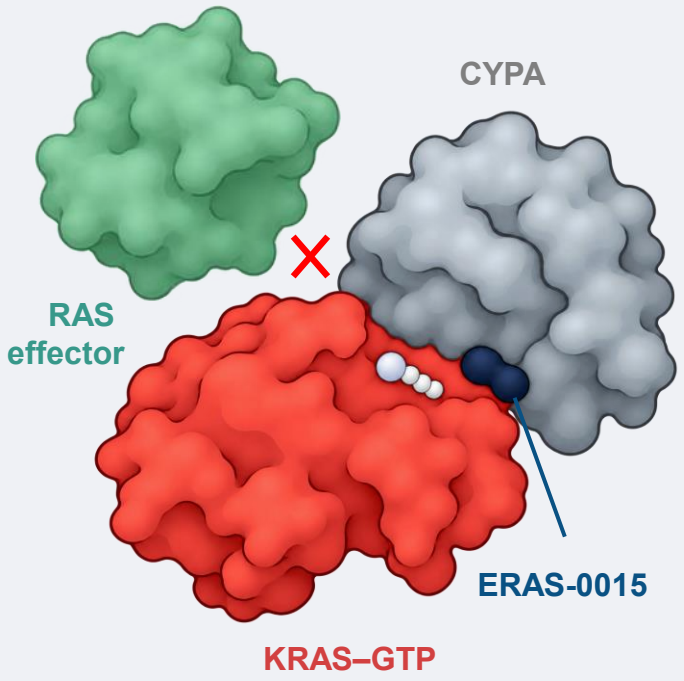
Cross-study comparisons, not head-to-head clinical trials: *Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable*

Sources: ERAS-0015 (AURORAS-1; DCO 1Sep2026); daraxonrasib (O'Reilly et al. NEJM 2026)
QD: once daily; TRAE: treatment-related adverse event

ERAS-0015 + ERAS-4001 combination potential: 0015 increases hydrolysis of KRAS-GTP → KRAS-GDP; 4001 locks accumulated KRAS-GDP in inactive state

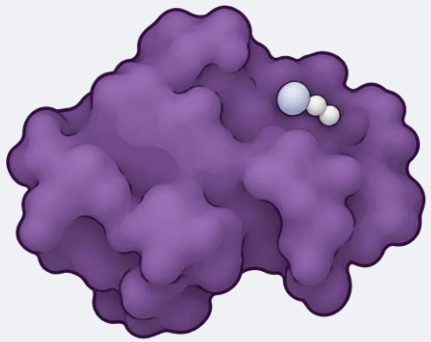
RAS Effector Engagement Blocked

ERAS-0015 binds CYPA, forms a ternary complex with GTP-bound RAS that blocks effector binding



Accelerated GTP Hydrolysis

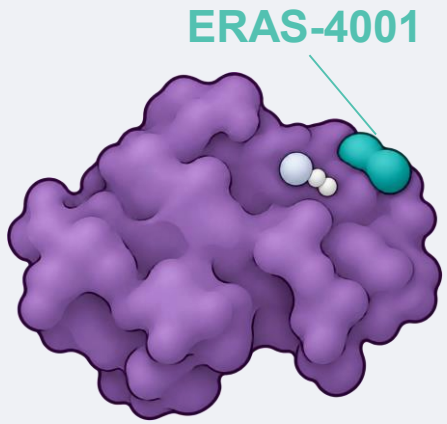
ERAS-0015 ternary complexes accelerate RAS GTP hydrolysis in a subset of KRAS mutants



KRAS–GDP
Inactive state

ERAS-4001 Binding Locks RAS Inactive

ERAS-4001 binds and locks RAS in its GDP-bound inactive state



KRAS–GDP
Inactive state

Illustration generated by OpenAI GPT-6 Astra

ERAS-4001 combination potential: Encouraging safety/tolerability and PK results coupled with preliminary monotherapy antitumor activity

BOREALIS-1 Trial Status[1]

- 36 patients enrolled
- Completed QD dose escalation from 20 mg to 600 mg
- Cleared 300 mg BID
- Currently evaluating 400 mg BID

ERAS-4001 Interim Clinical Data[1]

- Favorable safety/tolerability results with **predominantly low-grade AEs** (mostly GI) observed at all doses evaluated
- **No TRAEs have led to dose discontinuation**
- **Encouraging PK** with mean steady-state average exposure at dosage ≥240 mg QD exceeding target efficacious concentration for sensitive preclinical xenograft models; dosage at ≥400 mg BID is predicted to achieve target efficacious concentration for insensitive preclinical xenograft models
- **Preliminary monotherapy antitumor activity** supports ERAS-4001's potential as combo partner for ERAS-0015

ERAS-4001 + ERAS-0015 combo dose escalation planned for 2027

[1] As of 9Nov2026

QD: once daily; PK: pharmacokinetics; BID: twice a day; AE: adverse event

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

Encouraging interim efficacy results

Robust monotherapy response rates with encouraging combination data

PDAC: 44% cORR at 32 mg QD in patients with 2L+ KRAS G12X PDAC despite heavy pretreatment and high proportion of KRAS G12R; **69% cORR** in KRAS G12D or G12V

NSCLC: 60% cORR at 32 mg QD in patients with post-ICI/platinum (2/3L) KRAS G12X NSCLC

BTC: 75% cORR at 32 mg QD in patients with 1L+ KRAS G12X BTC

CRC: Initial efficacy signal observed in PAD range + panitumumab

Favorable safety and tolerability results

Generally well-tolerated with favorable safety data, and combinable within pharmacologically active dose (PAD)

Low rate of TRAE-related dose interruptions, reductions, and discontinuations at 32 mg QD (13%, 17%, and 0%, respectively)

Generally **well-tolerated with mostly low-grade AEs and favorable safety data**

Tolerability in combination with GnP, pembrolizumab, and panitumumab **supports combination development in Big 3 indications**

Potential to become RAS-targeting agent of choice as monotherapy and combination backbone

Sources: ERAS-0015 (AURORAS-1; monotherapy DCO 1Sep2026 and combination DCO 14Oct2026)
PDAC: pancreatic ductal adenocarcinoma; cORR: confirmed objective response rate; QD: once daily; NSCLC: non-small-cell-lung cancer; CRC: colorectal cancer; PAD: pharmacologically active dose; AE: adverse event; GnP: gemcitabine plus nab-paclitaxel; Big 3 indications: PDAC, NSCLC, CRC

ERASCA

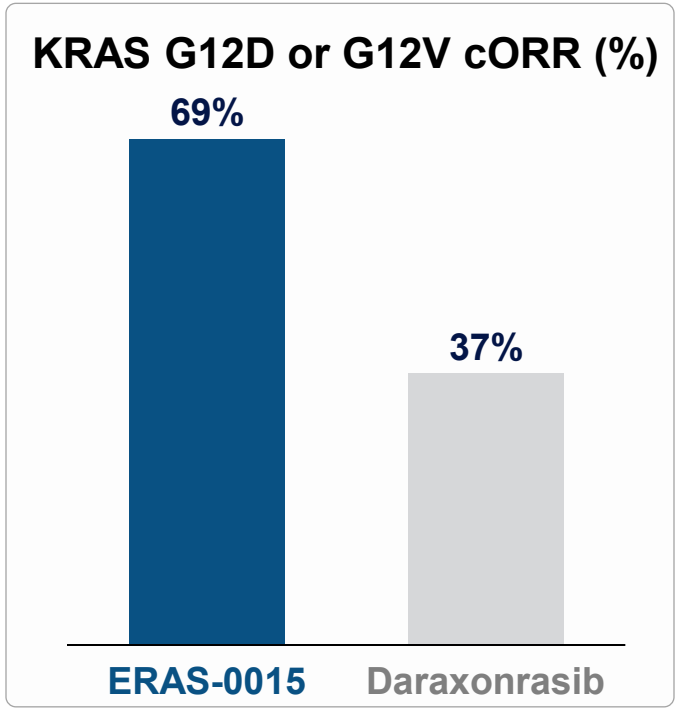
Anticipated key milestones in 2027

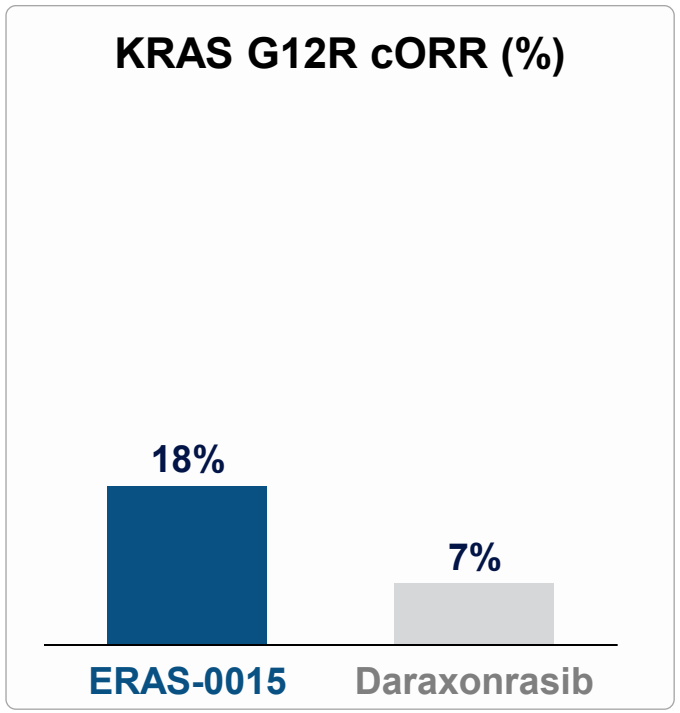
Program *Mechanism*	**Trial Name** *Indication*	**Anticipated Milestones**
ERAS-0015 *Pan-RAS molecular glue*	**AURORAS-1 to -3** *RASm solid tumors*	• **1H27:** - Monotherapy expansion data – **Completed ahead of schedule** - Initiate potentially registration-enabling trial in 2L+ RASm NSCLC - Initiate 1L PDAC Ph 3 – **Accelerated to 1H27** • **2027:** - Combination dose escalation and expansion data - Initiate 1L RASm NSCLC Ph 3 in 2H27-1H28
ERAS-4001 *Pan-KRAS inhibitor*	**BOREALIS-1** *KRASm solid tumors*	• **2027:** Initiate combination dose escalation with ERAS-0015

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


ERASCA
THANK YOU!

ERAS-0015: Potential best-in-class activity observed in the most common PDAC mutations





Dose	32 mg QD	300 mg QD
n/N	9/13	70/189
2L / 3L+ (% of pts)	23% / 77%	100% / 0%

32 mg QD	300 mg QD
2/11	2/27
9% / 91%	100% / 0%

KRAS G12D, G12V, and G12R mutations **occur in over 80% of patients with PDAC**

ERAS-0015 at 32 mg QD demonstrated **69% cORR (9/13) in KRAS G12D or G12V PDAC which occur in nearly 70% of patients with PDAC**

Activity observed in a heavily pre-treated population **77% in this KRAS G12D or G12V subset were 3L+**

Promising efficacy in three most common PDAC mutations support **ERAS-0015's best-in-class potential**

Cross-study comparisons, not 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*

Sources: ERAS-0015 (AURORAS-1; DCO 1Sep2026); daraxonrasib (O'Reilly et al. NEJM 2026; FDA CDER Multi-Disciplinary Review 2026); Varghese et al (Nat Med 2025)
cORR: confirmed objective response rate; PDAC: pancreatic ductal adenocarcinoma; QD: once daily

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Higher ORR was associated with longer durability and clinical benefit across multiple targeted therapies and multiple tumor types

Targeted therapy comparisons and PDAC chemotherapy benchmarks

Target	Trial	Investigational	Comparator	ORR	DOR (mo)	Median PFS (mo)	Median OS (mo)
KRAS G12C (NSCLC)	KRAScendo 1[1]	**Divarasib**	Sotorasib or adagrasib	58.4% vs 32.3%	11.9 vs 6.9	9.8 vs 5.7	17.7 vs 12.0
Cytotoxic (PDAC)	PRODIGE 4/ACCORD 11 1L metastatic[2]	**FOLFIRINOX**	Gemcitabine	32% vs 9%	n/a	6.4 vs 3.3	11.1 vs 6.8
	MPACT 1L metastatic[3]	**Nab-paclitaxel + gemcitabine**	Gemcitabine	23% vs 7%	n/a	5.5 vs 3.7	8.5 vs 6.7
	NAPOLI-3 1L metastatic[4]	**NALIRIFOX**	Nab-paclitaxel + gemcitabine	42% vs 36%	n/a	7.4 vs 5.6	11.1 vs 9.2
	NAPOLI-1 2L[5]	**nal-IRI + 5-FU/LV**	5-FU/LV	8% vs 1%	n/a	3.1 vs 1.5	6.1 vs 4.2
EGFR (NSCLC)	FLAURA[6]	**Osimertinib**	Gefitinib/erlotinib	77% vs 69%	17.6 vs 9.6	18.9 vs 10.2	38.6 vs 31.8
EGFR (NSCLC)	ARCHER 1050[7]	**Dacomitinib**	Gefitinib	74.9% vs 71.6%	14.8 vs 8.3	14.7 vs 9.2	34.1 vs 26.8
ALK (NSCLC)	ALEX[8]	**Alectinib**	Crizotinib	82.9% vs 75.5%	42.3 vs 11.1	34.8 vs 10.9	81.1 vs 54.2*
ALK (NSCLC)	CROWN[9]	**Lorlatinib**	Crizotinib	81% vs 63%	NR vs 9.2	NR vs 9.1	NR†
ALK (NSCLC)	ALTA-1L[10]	**Brigatinib**	Crizotinib	74% vs 62%	33 vs 14	30.8 vs 9.2	NR†
HER2 (Breast cancer)	DESTINY-Breast03[11]	**Trastuzumab deruxtecan**	T-DM1	78.9% vs 36.9%	30.5 vs 17.0	29.0 vs 7.2	52.6 vs 42.7
BRAF (Melanoma)	COLUMBUS[12]	**Encorafenib + binimetinib**	Vemurafenib	63–64% vs 40–41%	16.6 vs 12.3	14.9 vs 7.3	33.6 vs 16.9
BRAF(Melanoma)	COMBI-v[13]	**Dabrafenib + trametinib**	Vemurafenib	64% vs 51%	13.8 vs 7.5	11.4 vs 7.3	26.1 vs 17.8

NR = not reached. n/a: not available; NSCLC = non-small cell lung cancer. PDAC = pancreatic ductal adenocarcinoma.
Selected examples do not establish ORR as a survival surrogate. *ALEX OS was not statistically significant. †OS immature. Assessment methods and cutoffs vary by endpoint.
[1] Divarasib vs sotorasib/adagrasib: Skoulidis F, et al. ESMO 2026. LBA5. [2] FOLFIRINOX vs gemcitabine: Conroy T, et al. ASCO 2010. 4010 (final presentation).
[3] Nab-paclitaxel + gemcitabine vs gemcitabine: Von Hoff DD, et al. ASCO GI 2013. LBA148. [4] NALIRIFOX vs nab-paclitaxel + gemcitabine: Wainberg ZA, et al. ASCO GI 2023. LBA661.
[5] nal-IRI + 5-FU/LV vs 5-FU/LV: Ipsen. ONIVYDE US prescribing information. §14.2. [6] Osimertinib vs gefitinib/erlotinib: AstraZeneca. TAGRISSO US prescribing information. §14.1.
[7] Dacomitinib vs gefitinib: Wu YL, et al. ASCO 2017. LBA9007; Mok T, et al. ASCO 2018. 9004. [8] Alectinib vs crizotinib: Shaw AT, et al. ASCO 2017. LBA9008; Mok T, et al. ESMO 2025. ALEX final OS.
[9] Lorlatinib vs crizotinib: Solomon BJ, et al. ASCO 2024. LBA8503. [10] Brigatinib vs crizotinib: Popat S, et al. ESMO 2021. 1195P.
[11] Trastuzumab deruxtecan vs T-DM1: Hamilton EP, et al. ASCO 2024. 1025. [12] Encorafenib + binimetinib vs vemurafenib: Dummer R, et al. ASCO 2018. 9504; Pfizer. MEKTOVI US prescribing information. §14.1.
[13] Dabrafenib + trametinib vs vemurafenib: Robert C, et al. ESMO 2014. LBA4; ESMO 2016. LBA40.

ERASCA

ERAS-0015 data suggest potential best-in-class safety, tolerability, combinability

Summary of Safety, Tolerability, Monotherapy Dose, and Combinability((

TRAEs occurring in ≥10% of patients	All Grade		Grade 3+	
TRAEs (%)	**ERAS-0015 at 32 mg (N=54)**	**Daraxonrasib at 300 mg (N=241)**	**ERAS-0015 at 32 mg (N=54)**	**Daraxonrasib at 300 mg (N=241)**
Rash[1]	79.6%	85.5%	5.6%	13.7%
Diarrhea	35.2%	58.1%	0%	5.4%
Stomatitis	25.9%	53.1%	1.9%	12.0%
Nausea	22.2%	46.5%	0%	2.1%
Fatigue	13.0%	23.2%	0%	3.7%
All other TRAEs occurring in ≥10% patients	pruritus (11.1%)	vomiting, anemia, decreased appetite, paronychia, dry skin, AST increased, asthenia, thrombocytopenia		

TRAEs leading to… (%)	ERAS-0015 at 32 mg	Daraxonrasib at 300 mg
Dose interruptions	13.0%	56.0%
Dose reductions	16.7%	36.1%
Dose discontinuations	0%	1.2%

Monotherapy Dose & Combinability	ERAS-0015	Daraxonrasib
Monotherapy dose	Favorable benefit/risk data at 32 mg in PDAC and NSCLC	Benefit/risk profile varies by tumor type (300 mg in PDAC; 200 mg in NSCLC)
Combinability with SOC in NSCLC, PDAC, and CRC	Data support combination with pembro (32 mg), GnP (D1,15; cleared 16 mg) and panitumumab (cleared 16 mg; dose exploration and selection ongoing)	200 mg selected with pembro and with GnP; anti-EGFR combo not shown to be tolerated
Protocol prophylaxis	**ERAS-0015:** No prophylaxis recommended in the protocol **Daraxonrasib:** Rash, nausea and vomiting, and stomatitis prophylaxis recommended in the protocol	

Cross-study comparisons, not 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*

DCO 1Sep2026; O'Reilly et al. NEJM 2026; [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
TRAE: treatment-related adverse event; SOC: standard of care; pembro: pembrolizumab; GnP: gemcitabine + nab-paclitaxel