ERASCA

On a Journey to Erase Cancer

Erasca Corporate Presentation

March 2026

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

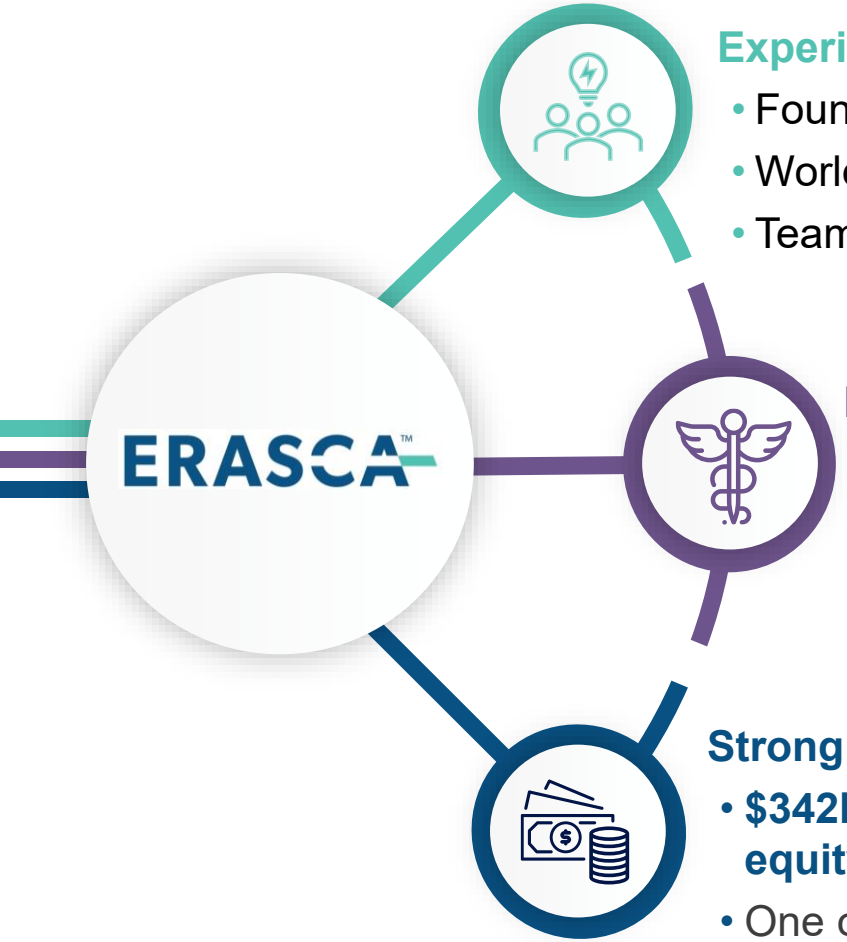
Disclaimer: Forward Looking Statements & Market Data

We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, research and development plans, the anticipated timing (including the timing of 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, our intellectual property protection, the timing and likelihood of success of our plans and objectives, the impact of the deprioritization of certain programs, and future results of anticipated product development efforts, are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negative of these terms or other similar expressions. The inclusion of forward-looking statements should not be regarded as a representation by us that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: our expectations regarding the potential therapeutic benefits of our product candidates, including ERAS-0015 and ERAS-4001, and our planned advancement of our development pipeline, including the anticipated timing of data readouts for the AURORAS-1 and JYP0015M101 trials, and the BOREALIS-1 trial; 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; observations regarding the first dosage level at which a clinical response is detected 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 assumptions about the development potential of ERAS-0015 and ERAS-4001 are based in large part on the preclinical data generated by the licensors and we may observe materially and adversely different results as we conduct our planned studies and trials; the initial data presented from the JYP0015M101 trial will be based upon data generated by the licensor; 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 three 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; results from preclinical studies or early clinical trials not necessarily being predictive of future results; the inability to realize any benefits from our current licenses, acquisitions, or collaborations, and any future licenses, acquisitions, or collaborations, and our ability to fulfill our obligations under such arrangements; regulatory developments in the United States and foreign countries; our ability to obtain and maintain intellectual property protection for our product candidates and maintain our rights under intellectual property licenses; our ability to fund our operating plans with our current cash, cash equivalents, and marketable securities into the second half of 2028; 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.

ERASCA

Our name is our mission: to erase cancer

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



Experienced leadership team and SAB with track record of serial successes

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

Differentiated pipeline focused on shutting down the RAS/MAPK pathway

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

Strong financial position with high quality investor base and industry visibility

- **$342M in cash, cash equivalents, and marketable securities[2]**; plus **$259M[3] oversubscribed equity financing** (closed on 1/23/2026); anticipated cash runway into **H2 2028**
- One of **Fierce Biotech's 2021 "Fierce 15"** most promising biotechnology companies

[1] Number of patients alive and free of cancer or free from cancer progression 2 yrs after starting an Erasca regimen, as measured by disease-free survival (adjuvant setting) and progression-free survival (metastatic setting)
[2] Audited, as of December 31, 2025; [3] Gross proceeds
FIC: first-in-class; BIC: best-in-class; RASm: RAS mutated; KRASm: KRAS mutated

ERASCA

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

Kevan Shokat, PhD

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

UCSF University of California San Francisco

Michael Varney, PhD

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

Genentech

René Bernards, PhD

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

Netherlands Cancer Institute Antoni van Leeuwenhoek

Stephen Blacklow MD, PhD

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

Harvard Medical School · Dana-Farber Cancer Institute

Karen Cichowski, PhD

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

Harvard Medical School · Brigham and Women's Hospital

Ryan Corcoran, MD, PhD

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

Harvard Medical School · Massachusetts General Hospital Cancer Center

George Demetri, MD

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

Harvard Medical School · Dana-Farber Cancer Institute

Piro Lito, MD, PhD

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

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

Pablo Rodriguez-Viciana, PhD

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

UCL Cancer Institute

ERASCA

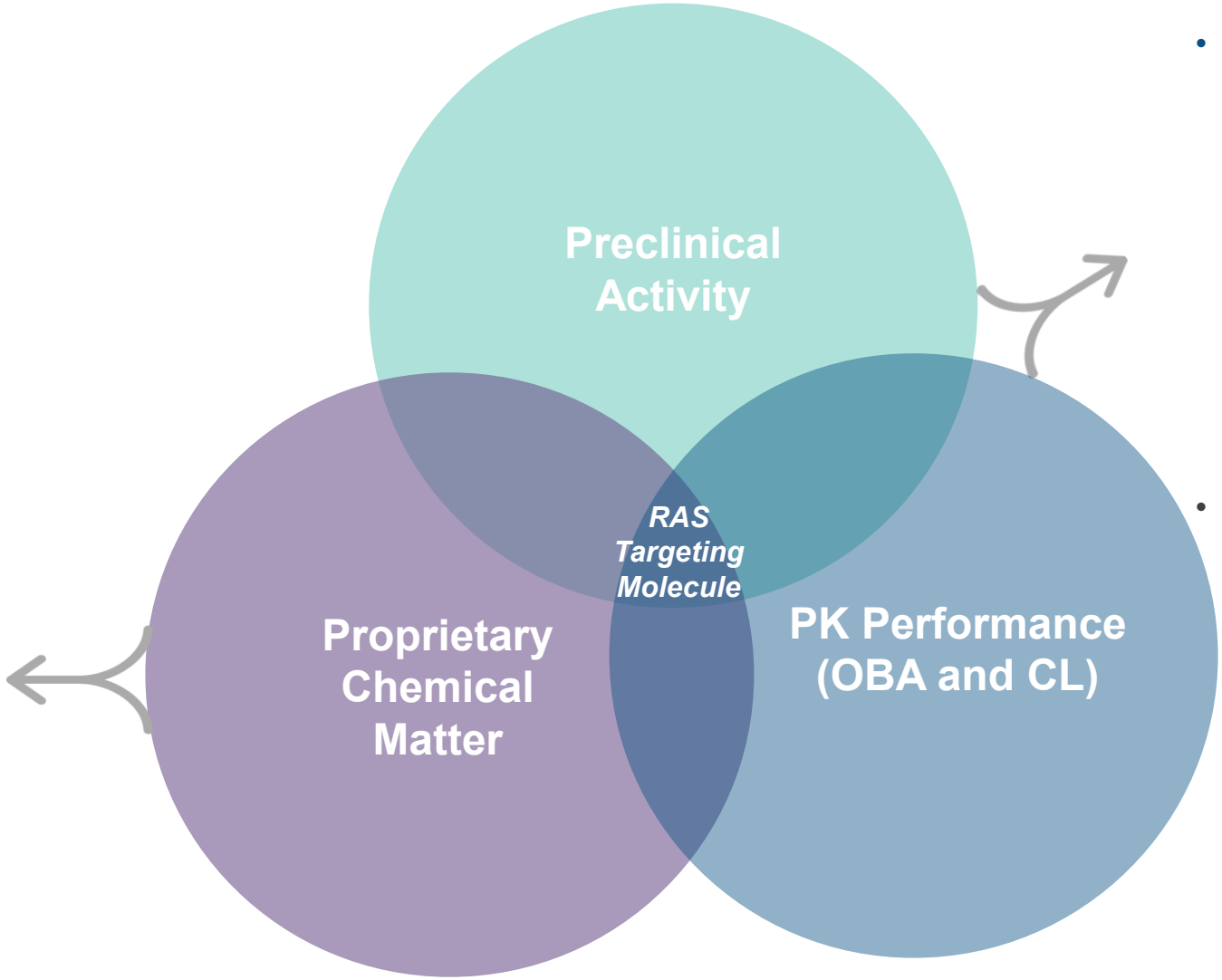
Focused modality-agnostic RAS/MAPK pipeline

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

small molecule small molecule molecular glue large molecule

ERASCA

Ideal RAS targeting molecules integrate three key attributes



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

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

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

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

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

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

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

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

ERASCA

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

ERAS-0015
Pan-RAS
Molecular Glue

Potential best-in-class Pan-RAS molecular glue

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

ERAS-4001
Pan-KRAS
Inhibitor

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

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

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

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

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

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

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

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

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

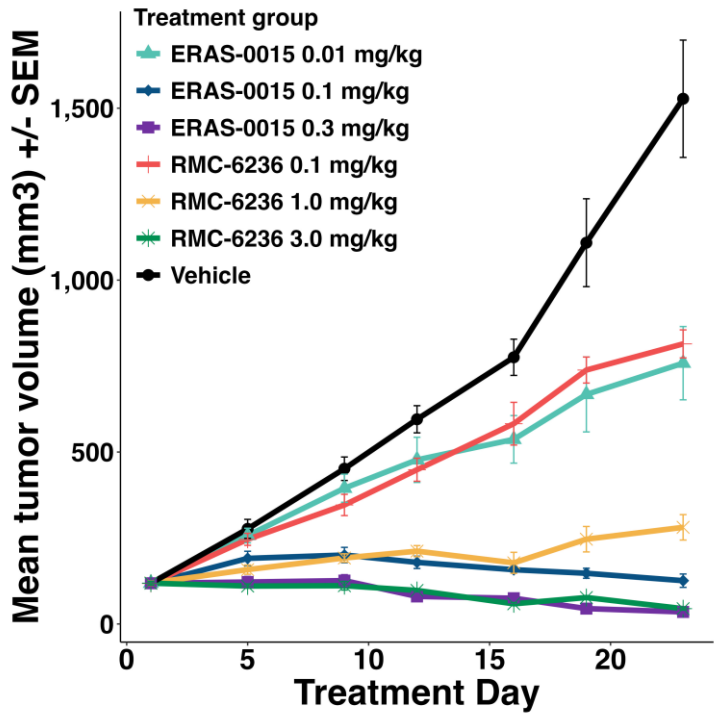
Sub-nM to nM potency against multiple KRAS wildtype, KRAS mutant, and RTK altered cell lines

RTK: receptor tyrosine kinase

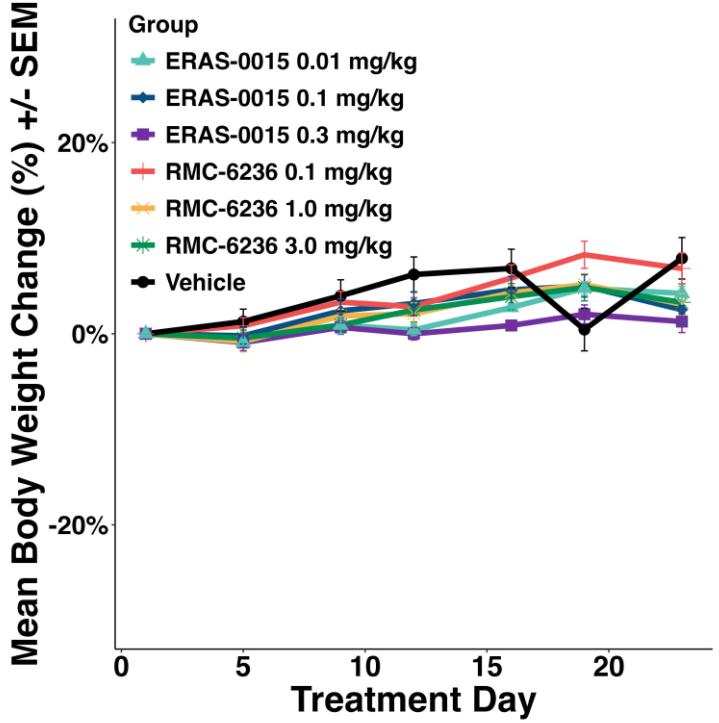
ERASCA

ERAS-0015 demonstrated comparable antitumor activity to RMC-6236 at 1/10th of the dose in a sensitive KRAS G12D PDAC CDX model

TGI in KRAS G12D PK-59



% Body Weight Change



TGI Summary

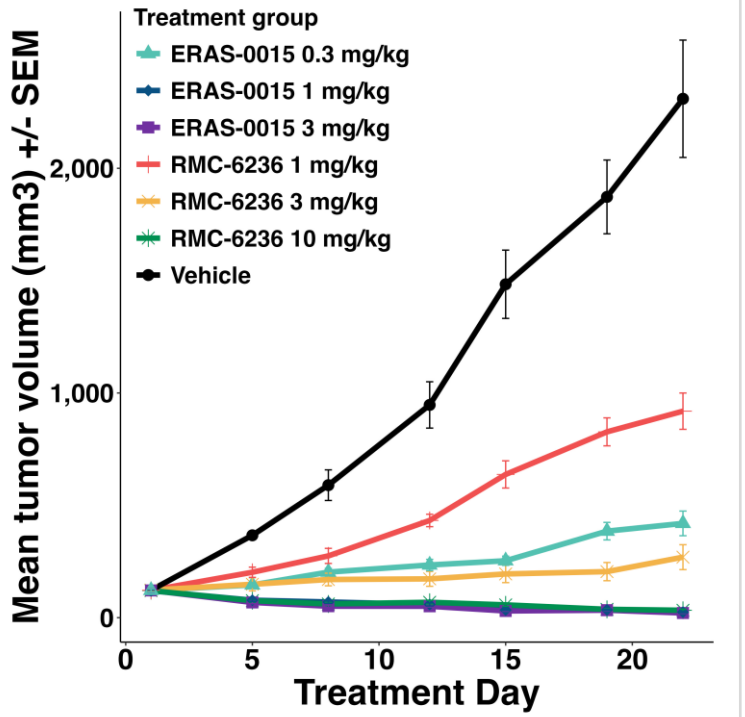
Therapy	Dose	TGI on Day 23
ERAS-0015	0.01 mg/kg	55%
	0.1 mg/kg	99%
	0.3 mg/kg	106%
RMC-6236	0.1 mg/kg	51%
	1.0 mg/kg	89%
	3.0 mg/kg	105%

- No dose reductions or holidays and no body weight loss for all doses of ERAS-0015

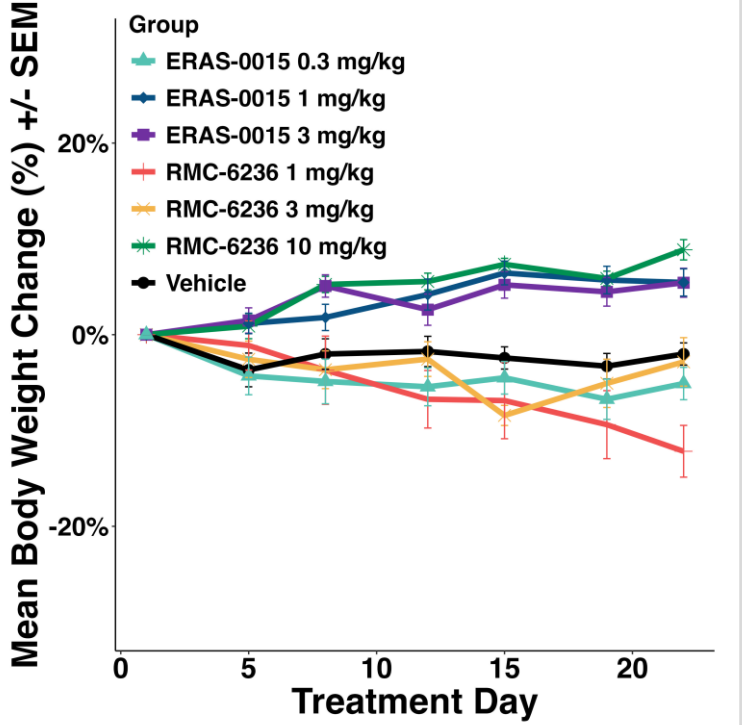
ERAS-0015 and RMC-6236 dosed orally once daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

ERAS-0015 demonstrated comparable antitumor activity to RMC-6236 at 1/10th of the dose in an insensitive KRAS G12V NSCLC CDX model

TGI in KRAS G12V NCI-H727



% Body Weight Change



TGI Summary

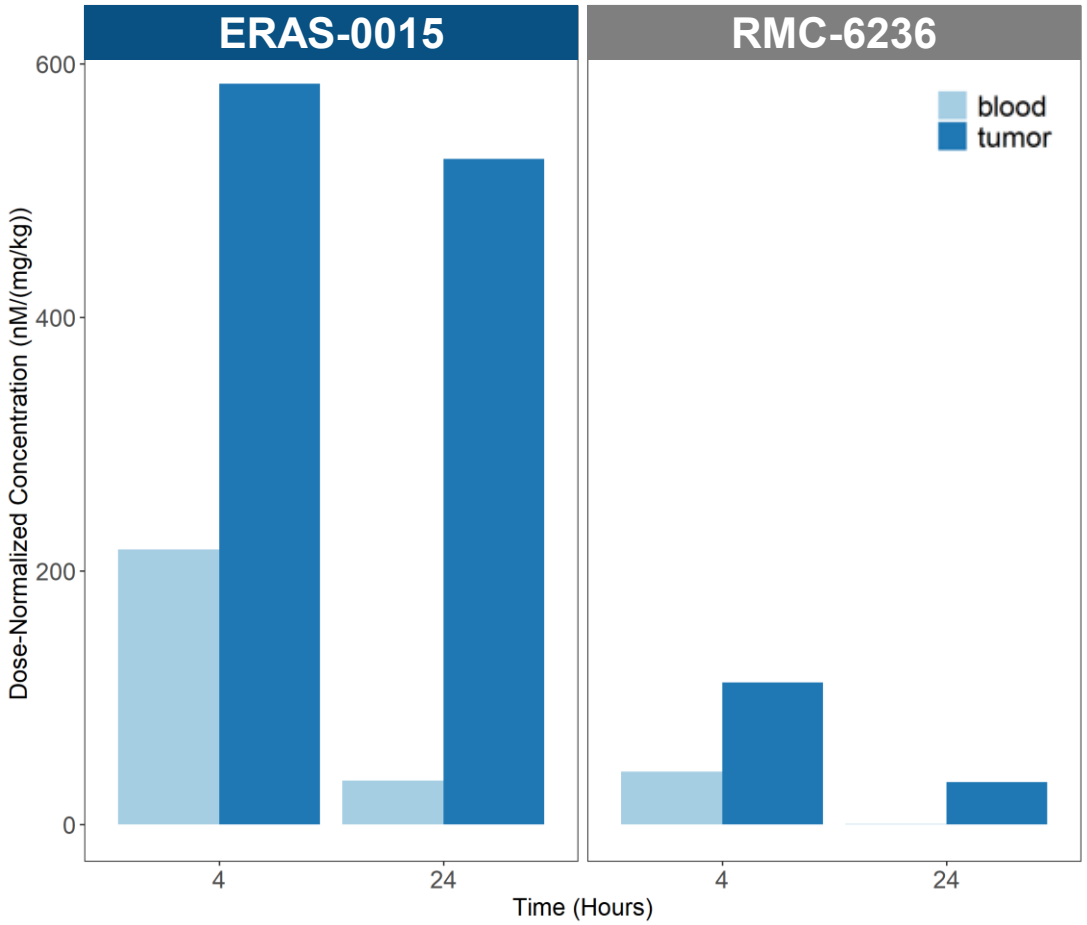
Therapy	Dose	TGI on Day 22
ERAS-0015	0.3 mg/kg	86%
	1 mg/kg	104%
	3 mg/kg	105%
RMC-6236	1 mg/kg	64%
	3 mg/kg	93%
	10 mg/kg	104%

- ERAS-0015 was well tolerated at all doses

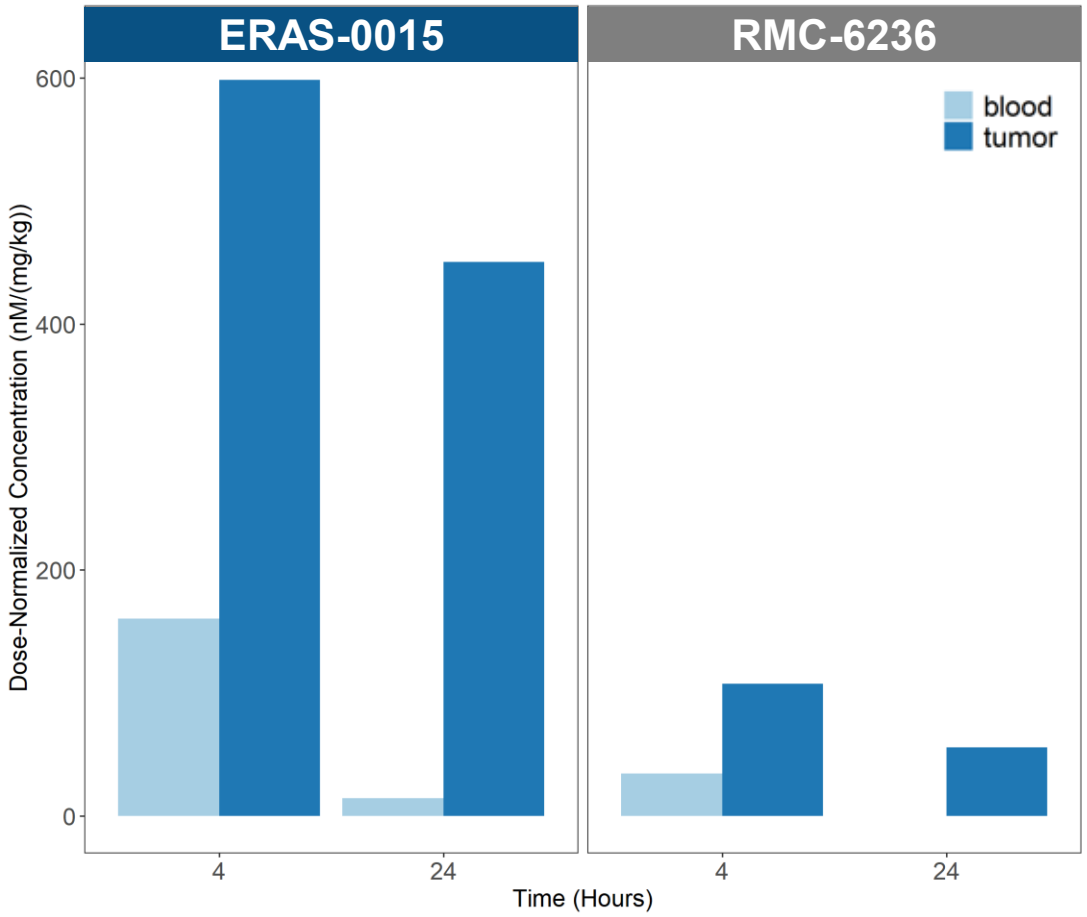
ERAS-0015 and RMC-6236 dosed orally once daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

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

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



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



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

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

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

ERASCA

AURORAS-1: First-in-human Phase 1/1b multicenter open-label trial of ERAS-0015 in RASm solid tumors

Single Agent Dose Escalation

Key Eligibility

Advanced RASm solid tumors

Received prior therapy

No previous treatment with RAS inhibitor

ERAS-0015

Single agent

Oral once daily dosing

Single Agent Goals

- Signs of clinical efficacy
- Well-behaved PK characteristics
- Safe and well-tolerated
- Inform combination therapy strategy

AURORAS-1 was designed to evaluate ERAS-0015's potential for **single agent differentiation** and to be the **best-in-class RAS-targeting molecule for combo development** across multiple solid tumors

Dose escalation has advanced faster than anticipated, underscoring **significant unmet need** and **high investigator/patient enthusiasm**

PK: pharmacokinetics

ERAS-0015's differentiation potential: Clinical responses first observed at 8 mg QD (1/10th of the dose observed with RMC-6236) coupled with promising safety and PK

Preclinical Differentiation	
In Vivo Activity	Comparable efficacy at 1/10th-1/5th of RMC-6236 dose in multiple mouse models
PK Properties	• 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)

ERAS-0015 Clinical Data[1]	
Ongoing Confirmed and Unconfirmed Responses	In multiple patients with differing tumor types and RAS mutations - Ongoing responses (2 cPRs, 1 uPR) in patients with different tumor types and RAS mutations at low dose of 8 mg QD - Additional ongoing responses in multiple patients at > 8 mg QD
Favorable Safety/Tolerability	• No DLTs and predominantly low-grade AEs observed at all doses evaluated • Patients have been dosed at 40 mg QD[2]
Well-Behaved, Linear PK	Across all doses evaluated; no evidence of exposure plateau

ERAS-0015 has the potential to become preferred RAS-targeting backbone for combination therapy

Topline safety, tolerability, PK, and initial efficacy data for dozens of patients planned in H1 2026

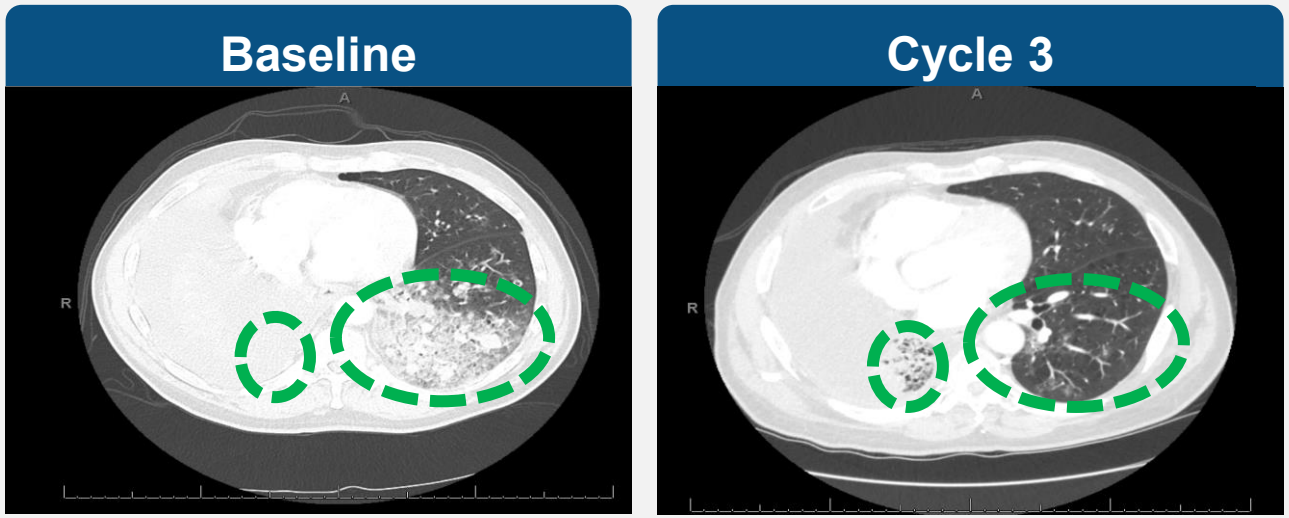
[1] As of January 7, 2026; [2] Dosing occurred after the January 7, 2026 data cutoff date

%F = bioavailability; Cl = clearance; $T_{1/2}$ = half-life; CYPA: cyclophilin A; PR = partial response (cPR = confirmed PR, uPR = unconfirmed PR); QD = once daily; DLT = dose-limiting toxicity; AE = adverse event

ERASCA

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

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 patient's dose was escalated to 16 mg QD • Patient continues on treatment[1]



- Patient was able to come off supplemental oxygen after first week of ERAS-0015 8 mg QD treatment
- Patient is currently asymptomatic from disease and from treatment[1]

[1] As of January 7, 2026
NSCLC: non-small cell lung cancer; QD: once daily

ERAS-0015 1H26 data readout: potential scenarios vs. RMC-6236 benchmark

	Potential differentiation attributes	RMC-6236 (≥80 mg) benchmark[1] FPD (06/22) to DCO (10/23): 16 months			
Efficacy	≥10% point increase in ORR in **2L+ KRAS^{G12X} PDAC**	19.6% uORR (5 PR, 4 uPR, N=46)			
	≥10% point increase in ORR in **2L+ KRAS^{G12X} NSCLC**	37.5% uORR (1 CR, 11 PR, 3 uPR, N=40)			
Safety and Tolerability	Reduced frequency and/or severity of **rash[2] TRAEs**	All Grades	81%		
		Gr 1	52%		
		Gr 2	23%		
		Gr 3+	6%		
	Reduced frequency and/or severity of **GI TRAEs**		Nausea	Vomiting	Diarrhea
		All Grades	46%	33%	39%
		Gr 1	36%	27%	25%
		Gr 2	10%	6%	13%
		Gr 3+	0%	0%	1%
	Reduced frequency and/or severity of **stomatitis/mucositis TRAEs**	All Grades	22%		
		Gr 1	12%		
		Gr 2	8%		
		Gr 3+	2%		
	Combinability with SOC agents (ERAS-0015 combo data guided to 2027)	CRC: Anti-EGFR combo not shown to be tolerated PDAC: Dose modification required for chemo			

ERAS-0015 1H26 Data
FPD to DCO <12 months

"Differentiated" Scenario

Differentiated Efficacy* ≥ 1 Efficacy Attribute ***OR*** ***Differentiated Safety**** ≥ 2 Safety and Tolerability Attributes

"Paradigm-Shifting" Scenario

Differentiated Efficacy* ≥ 1 Efficacy Attribute ***AND*** ***Differentiated Safety**** ≥ 2 Safety and Tolerability Attributes

*** Compared to RMC-6236 benchmark**

[1] Arbour et al, ESMO 2023; RMC-6236 benchmark pop for safety & tolerability: N=111 patients with NSCLC, PDAC; [2] 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
Note: TRAE = treatment-related adverse event; Gr = grade; pts = patients; NSCLC = non-small cell lung cancer; PDAC = pancreatic ductal adenocarcinoma; SOC = standard of care; mAb = monoclonal antibody; ORR = objective response rate; uORR = unconfirmed objective response rate; CR = confirmed complete response; PR = confirmed partial response; uPR = unconfirmed partial response; FPD = first patient dosed; DCO = data cutoff

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

ERAS-0015
Pan-RAS
Molecular Glue

Potential best-in-class Pan-RAS molecular glue

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

ERAS-4001
Pan-KRAS
Inhibitor

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

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

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

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

SPR-based kinetic biophysical binding characterization of ERAS-4001

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

SPR = surface plasmon resonance

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

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

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

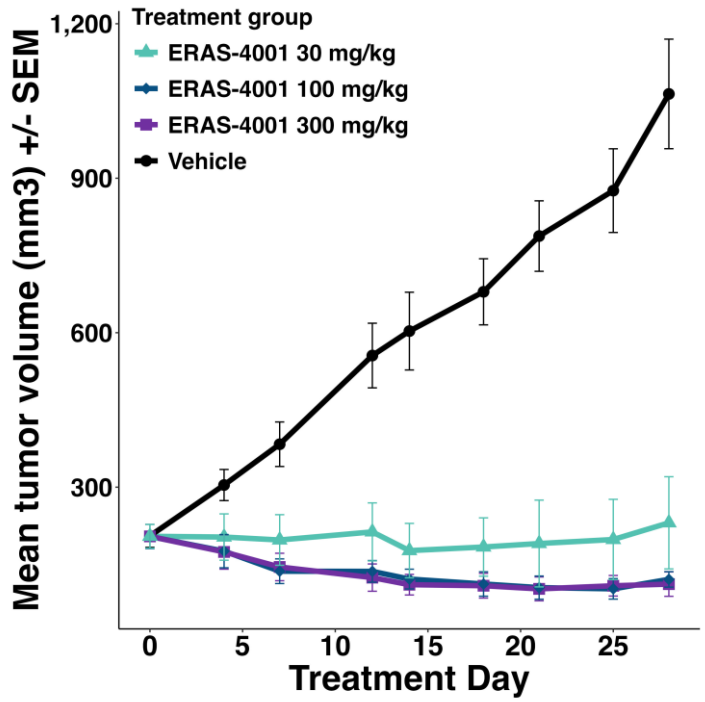
ERAS-4001 potently and selectively inhibited cell growth in KRAS G12X, G13D and WT cell lines

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

ERASCA

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

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



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

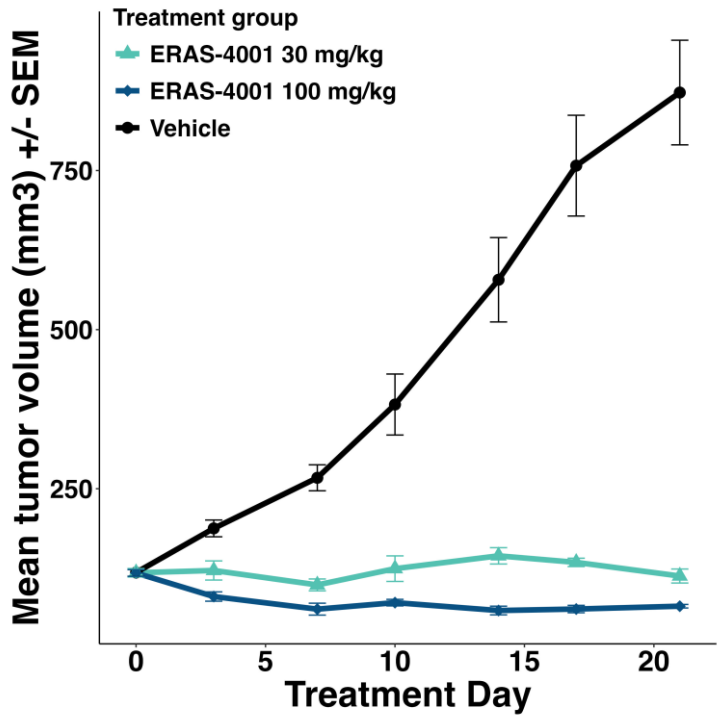
Therapy	Dose	TGI on Day 28	pERK Inhibition at 8 hr	$AUC_{0\text{-last}}$ (nmol/L·h)
ERAS-4001	30 mg/kg	97%	17%	1,547
	100 mg/kg	110%	64%	5,153
	300 mg/kg	111%	80%	12,971

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

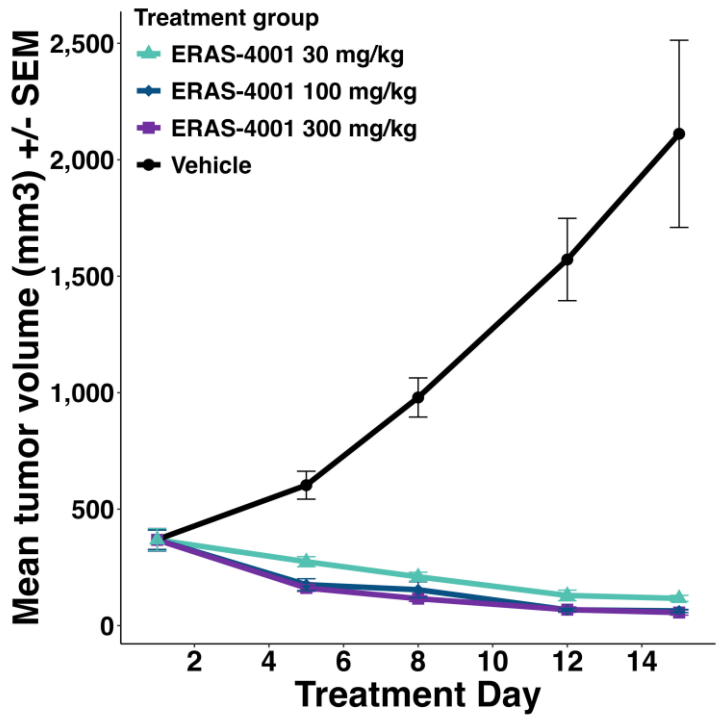
ERAS-4001 dosed orally twice daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

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

TGI in KRAS G12D PDAC CDX PK-59



TGI in KRAS G12V Ovarian CDX RKN

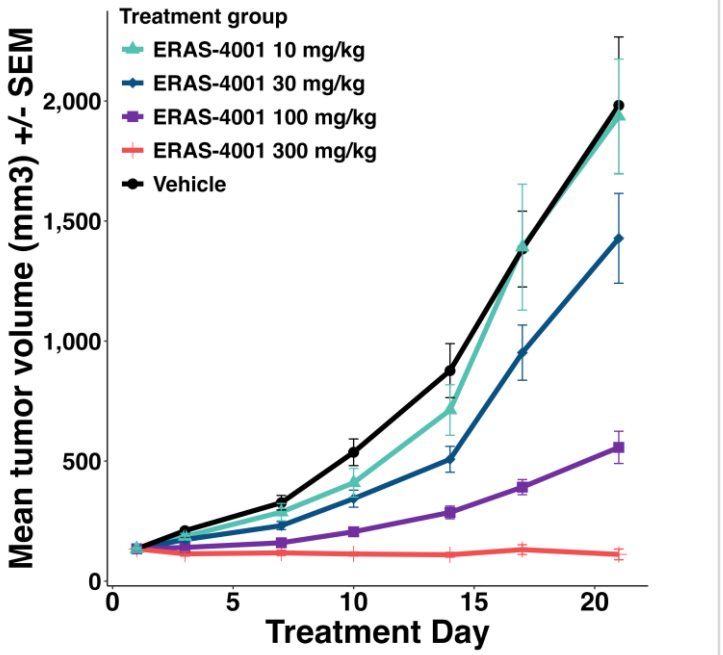


- ERAS-4001 was well tolerated in both studies at doses up to 300 mg/kg BID (i.e., no dose reductions or holidays; no body weight loss or significant health observations)

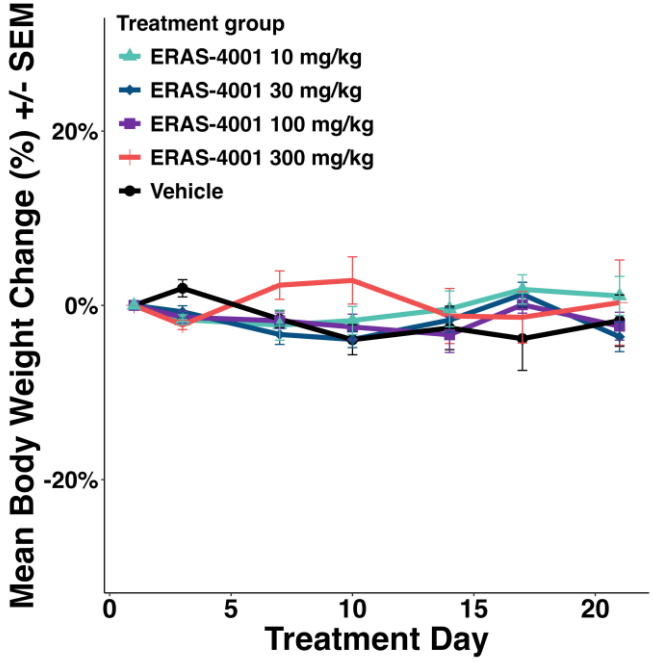
ERAS-4001 dosed orally twice daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

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

TGI in KRAS G12V NCI-H727



% Body Weight Change



TGI Summary

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

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

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

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

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

Anticipated key milestones in 2026-2027

Program *Mechanism*	**Trial Name** *Indication*	**Anticipated Milestone**
ERAS-0015 *Pan-RAS molecular glue*	**AURORAS-1** *RASm solid tumors*	• **H1 2026:** Ph 1 monotherapy data[1] • **H2 2026:** - 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*	• **H2 2026:** Ph 1 monotherapy data • **2027:** - Initiate monotherapy expansion cohorts - Initiate combination dose escalation cohorts

[1] This data readout will also include data from JYP0015M101, a clinical trial in China sponsored by Joyo that is assessing ERAS-0015 in adult patients with advanced solid tumors harboring specific RAS mutations

Compelling investment thesis

EXPERIENCED TEAM WITH TRACK RECORD OF SERIAL SUCCESSES

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

WORLD-CLASS SCIENTIFIC ADVISORY BOARD

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

PROMISING PIPELINE TARGETS LARGE, UNDERSERVED MARKETS ACROSS MULTIPLE TUMOR TYPES

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

CLINICAL ADVANCEMENT OF INDUSTRY LEADING RAS-TARGETING FRANCHISE

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

MULTIPLE POTENTIAL NEAR-TERM AND LONG-TERM VALUE DRIVERS

Focused clinical development plan with near-term clinical readouts

ERASCA


ERASCA
THANK YOU!

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

Pan-RAS	Adlai Nortye AN9025	GENFLEET THERAPEUTICS GF-276	Roche RO7673396	ERASCA **ERAS-0015**	REVOLUTION MEDICINES RMC-6236
Pan-KRAS		AMGEN AMG-410	Lilly LY4066434 (G12X)	AstraZeneca JAB-23E73	ERASCA **ERAS-4001**
			BBOT BBO-11818	Pfizer PF-07934040 (G12X)	Alterome THERAPEUTICS ALTA-3263
Mutant Selective[1]	Lilly LY3962673 (G12D)	Quanta THERAPEUTICS INC. QTX3034 (KRAS G12D)	Astellas ASP3082 (G12D degrader)	Incyte INCB161734 (G12D)	REVOLUTION MEDICINES RMC-9805 (G12D)
		Quanta THERAPEUTICS INC. QTX3054 (KRAS G12V)	REVOLUTION MEDICINES RMC-5127 (G12V)	VERASTEM ONCOLOGY VS-7375 (G12D)	Boehringer Ingelheim BI 3706674 (wt amp, G12V)

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

[1] Mutant selective beyond KRAS G12C inhibitors

ERASCA

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

	Preclinical (in vitro and in vivo) Activity[1]		OBA[2]	IP[3]
ERAS-0015 Pan-RAS Molecular Glue	KRAS G12D: 0.2 – 13.3 nM KRAS G12V: 0.4 – 2.5 nM KRAS G12C: 0.8 – 1.4 nM KRAS G12X: 4.1 – 7.4 nM KRAS G13D: 2.8 – 5.5 nM KRAS WT: 4.1 – 13.8 nM H/NRAS: Active	***KRAS G12D:*** Tumor regression in PK-59 CDX model at 0.3 mpk PO QD ***KRAS G12V:*** Tumor regression in NCI-H727 CDX model at 1 mpk PO QD ***KRAS G12R:*** Tumor regression in PSN1 CDX model at 5 mpk PO QD	Mouse: 48% Rat: 38% Dog: 22% Monkey: 17%	• IP exclusivity expected through **2043** • US patent covering composition of matter issued in Oct. 2025
ERAS-4001 Pan-KRAS Inhibitor	KRAS G12D: 1.0 – 2.6 nM KRAS G12V: 0.7 – 9.1 nM KRAS G12C: 1.1 – 4.5 nM KRAS G12X: 6.5 – 37.7 nM KRAS G13D: 5.8 – 56.0 nM KRAS WT: 3.6 – 10.8 nM H/NRAS WT: No activity	***KRAS G12D:*** Tumor regression in Panc04.03, PK-59, and LU-01-1381 CDX/PDX models at 30 – 100 mpk PO BID; combo with anti-PD-1 achieved complete disappearance of tumors in all mice (7/7) on D31 at 100 mpk PO BID ***KRAS G12V:*** Tumor regression in RKN and NCI-H727 CDX models at 30 – 300 mpk PO BID	Mouse: 27% Rat: 5 – 27% (variable PK in rat) Dog: 16%	• IP exclusivity expected through **2043** • US patent covering composition of matter issued in Feb. 2026

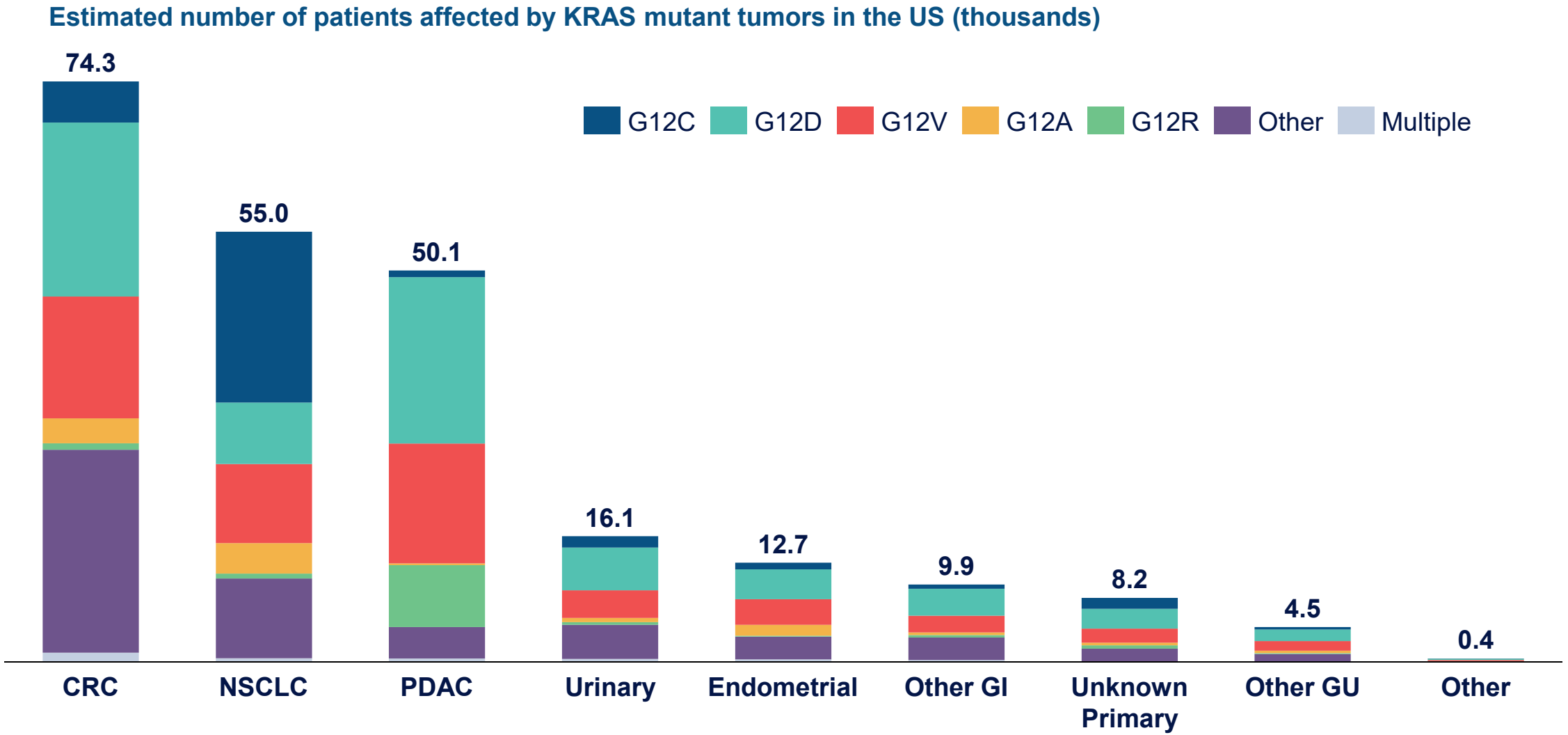
Potential BIC **Pan-RAS MG** for RASm solid tumors, which showed ~5x – 10x greater antitumor activity and favorable ADME properties and PK performance in animal species (vs. most advanced Pan-RAS MG in development)

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

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

ERASCA

KRAS alterations found most commonly in CRC, PDAC and NSCLC



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