



**ERASCA**

# On a Journey to Erase Cancer

**Erasca Corporate Presentation**

**April 2026**

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

# Disclaimer: Forward Looking Statements & Market and Clinical Data

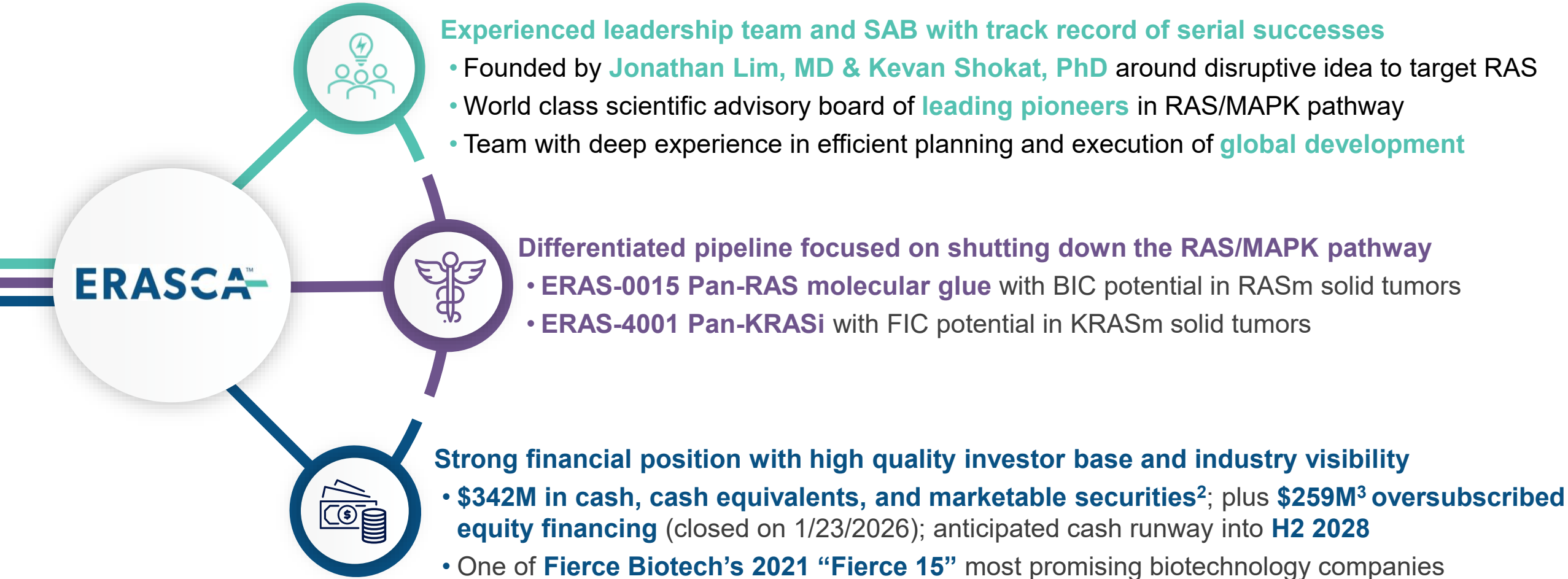
We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation are forward-looking statements, including statements regarding: our future results of operations and financial position, business strategy, research and development plans; the anticipated timing (including the timing of initiation and the timing of data readouts), costs, design and conduct of our ongoing and planned preclinical studies and clinical trials for our product candidates; the potential therapeutic benefits and potential patient population for each of our product candidates; the potential for ERAS-0015 to be best-in-class or serve as backbone therapy for future combination therapies; characterizations of the clinical profile of our product candidates and any comparisons against other products or product candidates in development; our intellectual property protection; and future results of anticipated product development efforts, including anticipated milestones. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negative of these terms or other similar expressions. The inclusion of forward-looking statements should not be regarded as a representation by us that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: the timing of our clinical data readouts, including for the AURORAS-1 and BOREALIS-1 trials may be delayed; our product candidates, including ERAS-0015 and ERAS-4001, may not demonstrate therapeutic benefits that we expect; this presentation includes clinical data generated by our third-party licensor, and such data are presented as received and have not been independently verified by us; topline and preliminary results of a clinical trial are not necessarily indicative of final results and one or more of the clinical outcomes may materially change as patient enrollment continues, following more comprehensive reviews of the data and as more patient data becomes available, including the risk that an unconfirmed partial response to treatment may not ultimately result in a confirmed partial response to treatment after follow-up evaluations; our observations, including those regarding the first dosage level at which a clinical response is detected and the efficacy and safety of ERAS-0015 compared to other products and product candidates (including our internal benchmark RMC-6236), are based on data generated within individual clinical trials, and comparisons of such clinical observations across different trials involve data from separate trials with distinct designs, patient populations, and methodologies, and therefore may not be directly comparable; any forward-looking statements regarding dose-response relationships reflect current expectations and/or assumptions are subject to risks and uncertainties that could cause actual results to differ materially; our approach to the discovery and development of product candidates based on our singular focus on shutting down the RAS/MAPK pathway, a novel and unproven approach; we only have two product candidates in clinical development and all of our other development efforts are in the preclinical stage; our assumptions around which programs may have a higher probability of success may not be accurate, and we may expend our limited resources to pursue a particular product candidate and/or indication and fail to capitalize on product candidates or indications with greater development or commercial potential; potential delays in the commencement, enrollment, data readout, and completion of clinical trials and preclinical studies; our dependence on third parties in connection with manufacturing, research, and preclinical and clinical testing; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval, and/or commercialization, or may result in recalls or product liability claims; unfavorable results from preclinical studies or clinical trials; the inability to realize any benefits from our current licenses, acquisitions, or collaborations, and any future licenses, acquisitions, or collaborations, and our ability to fulfill our obligations under such arrangements; regulatory developments in the United States and foreign countries; our ability to obtain and maintain intellectual property protection for our product candidates and maintain our rights under intellectual property licenses; our ability to fund our operating plans with our current cash, cash equivalents, and marketable securities; and other risks described in our prior filings with the Securities and Exchange Commission (SEC), including under the heading “Risk Factors” in our annual report on Form 10-K for the year ended December 31, 2025, and any subsequent filings with the SEC. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.

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

# Our name is our mission: to erase cancer

Vision to one day erase cancer<sup>1</sup> in at least 100,000 patients annually as a leading global oncology company



<sup>1</sup> 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)

<sup>2</sup> Audited, as of December 31, 2025; <sup>3</sup> Gross proceeds

FIC: first-in-class; BIC: best-in-class; RASm: RAS mutated; KRASm: KRAS mutated

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



**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)

**Kevan Shokat, 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)

**Michael Varney, PhD**



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

**René Bernards, PhD**



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

**Stephen Blacklow MD, PhD**



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

**Karen Cichowski, 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

**Ryan Corcoran, MD, PhD**



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

**George Demetri, MD**



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

**Piro Lito, MD, 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

**Pablo Rodriguez-Viciano, PhD**

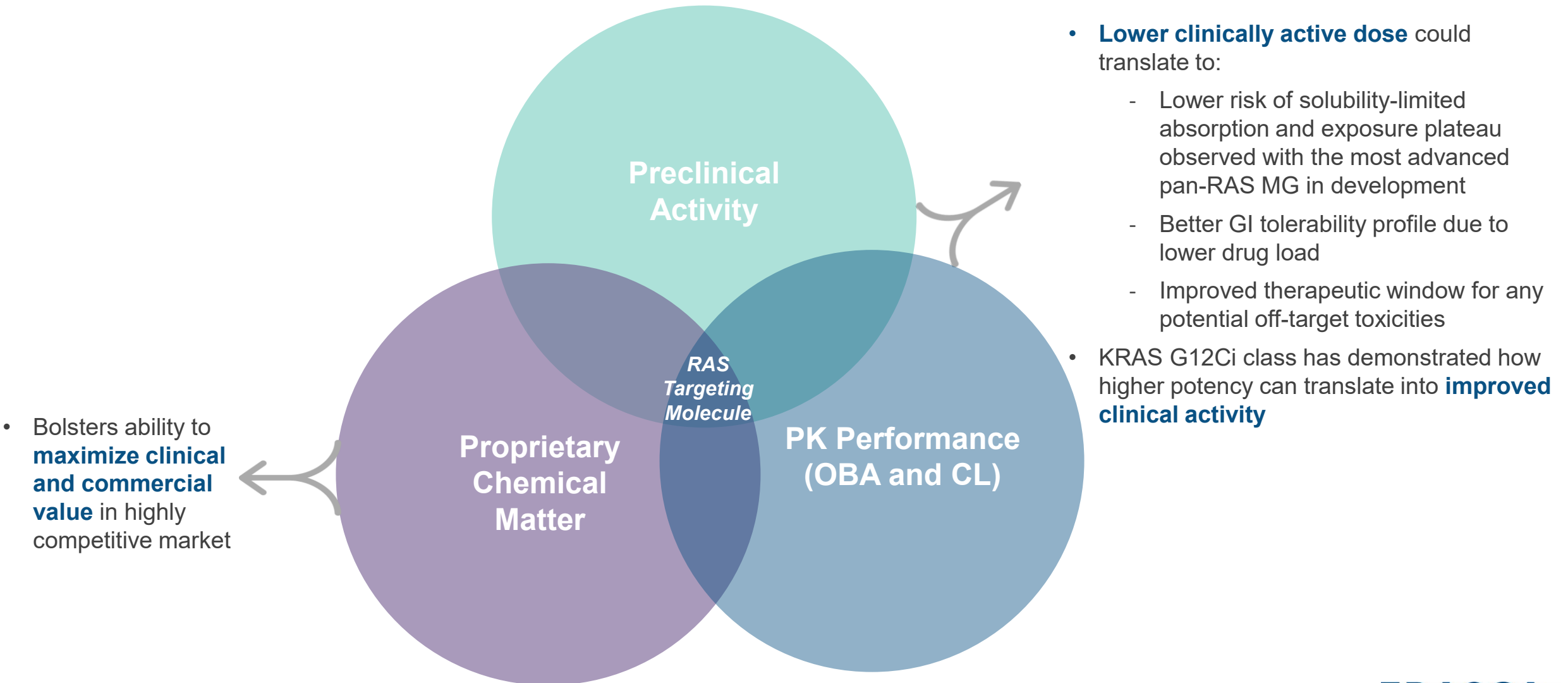


# Focused modality-agnostic RAS/MAPK pipeline

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

# Ideal RAS targeting molecules integrate three key attributes



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

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

	Preclinical Activity <sup>1</sup>	OBA <sup>2</sup>	IP <sup>3</sup>
<b>ERAS-0015</b> Pan-RAS Molecular Glue	<ul style="list-style-type: none"> <li>In vitro: <b>0.2 – 13.8 nM IC50</b> in KRAS G12D/V/C/X, G13D, WT; activity in H/NRAS</li> <li>In vivo: Tumor regression in KRAS G12D/V/R CDX models at low doses between <b>0.3 – 5 mpk PO QD</b></li> </ul>	<ul style="list-style-type: none"> <li><b>38 – 48%</b> in small animal species</li> <li><b>17 – 22%</b> in large animal species</li> </ul>	<ul style="list-style-type: none"> <li>IP exclusivity expected through <b>2043</b></li> <li>US patent covering composition of matter issued in Oct. 2025</li> </ul>
<b>ERAS-4001</b> Pan-KRAS Inhibitor	<ul style="list-style-type: none"> <li>In vitro: <b>0.7 – 10.8 nM IC50</b> in KRAS G12D/V/C and KRAS WT; <b>5.8 – 56 nM IC50</b> in KRAS G12X and G13D; no activity in H/NRAS</li> <li>In vivo: Tumor regression in KRAS G12D/V CDX models at doses between <b>30 - 300 mpk PO BID</b></li> </ul>	<ul style="list-style-type: none"> <li><b>Up to 27%</b> in small animal species</li> <li><b>16%</b> in large animal species</li> </ul>	<ul style="list-style-type: none"> <li>IP exclusivity expected through <b>2043</b></li> <li>US patent covering composition of matter issued in Feb. 2026</li> </ul>
<p>Potential BIC <b>Pan-RAS MG</b> 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)<sup>4</sup></p>		<p>Potential FIC/BIC <b>Pan-KRAS</b> or “<b>KRAS-selective</b>” <b>SMi</b> 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)</p>	

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; <sup>1</sup> in vitro potency assessed by CTG 2D and 3D-cell proliferation assay IC50s; <sup>2</sup> OBA assessed by %F; <sup>3</sup> IP includes composition of matter, methods of use, and methods of making licensed compounds; date is absent any patent term adjustments or extensions, <sup>4</sup> These data were generated in head-to-head in vitro assay and/or in vivo experiments

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

## ERAS-0015 Pan-RAS Molecular Glue

### Potential best-in-class Pan-RAS molecular glue

- ~5x – 10x greater antitumor activity vs. most advanced pan-RAS MG in development<sup>1</sup>
- Favorable ADME properties and PK performance in animals vs. most advanced pan-RAS MG in development<sup>1</sup>
- 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

<sup>1</sup> These data were generated in head-to-head in vitro assay and/or in vivo experiments

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

# 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	<b>21x</b>
ITC $K_D$	5.3	44.1	<b>8x</b>

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

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

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

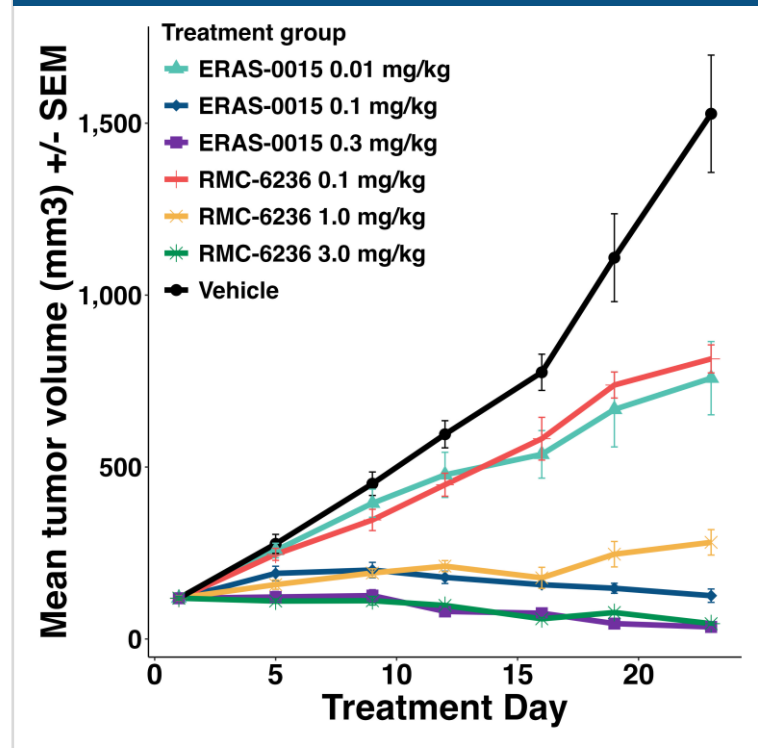
Mutation	Tumor type	Cell line	ERAS-0015 cell growth inhibition (nM)	RMC-6236 cell growth inhibition (nM)	ERAS-0015:RMC-6236 Fold Potency
KRAS G12C	NSCLC	H358 (adagrasib-resistant)	0.8	3.6	4.5x
	NSCLC	LU99	1.4	5.4	3.9x
KRAS G12D	NSCLC	A-427	13.3	59.2	4.5x
	CRC	SW620	0.2	1.3	6.5x
	CRC	GP2d	0.9	4.6	5.1x
	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

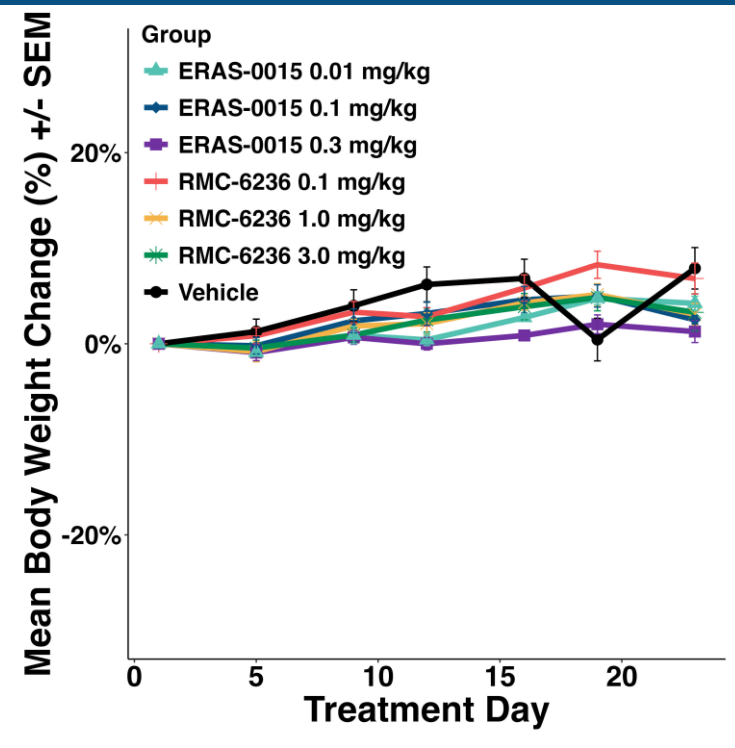
These data were generated in head-to-head in vitro assay and/or in vivo experiments. RTK: receptor tyrosine kinase

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

## TGI in KRAS G12D PK-59



## % Body Weight Change



## TGI Summary

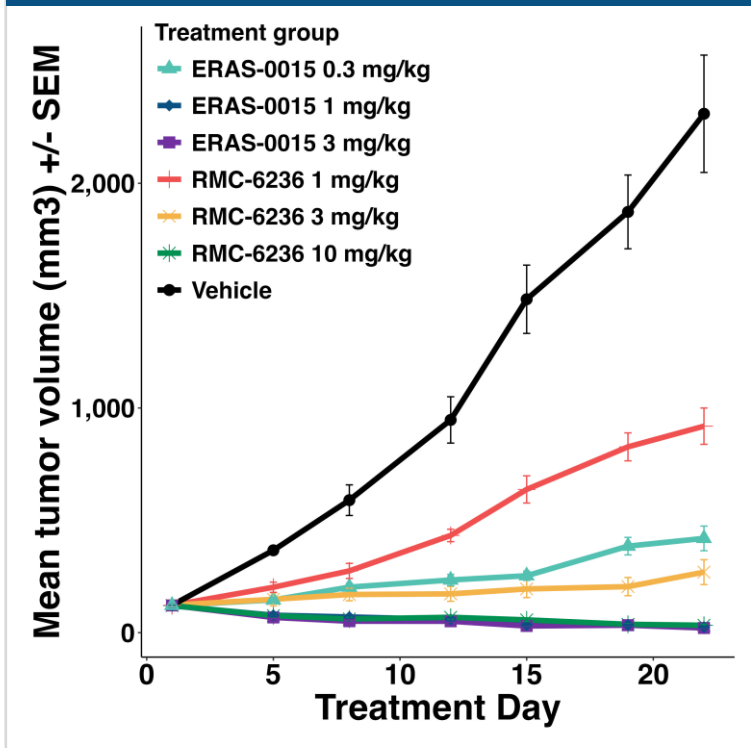
Therapy	Dose	TGI on Day 23
ERAS-0015	0.01 mg/kg	55%
	0.1 mg/kg	99%
	0.3 mg/kg	106%
RMC-6236	0.1 mg/kg	51%
	1.0 mg/kg	89%
	3.0 mg/kg	105%

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

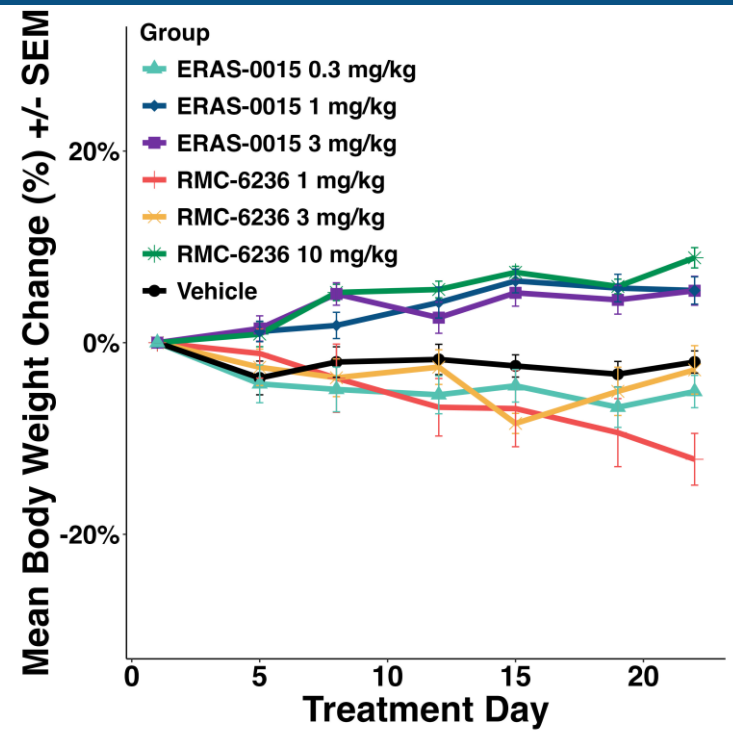
These data were generated in head-to-head in vitro assay and/or in vivo experiments  
 ERAS-0015 and RMC-6236 dosed orally once daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

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

## TGI in KRAS G12V NCI-H727



## % Body Weight Change



## TGI Summary

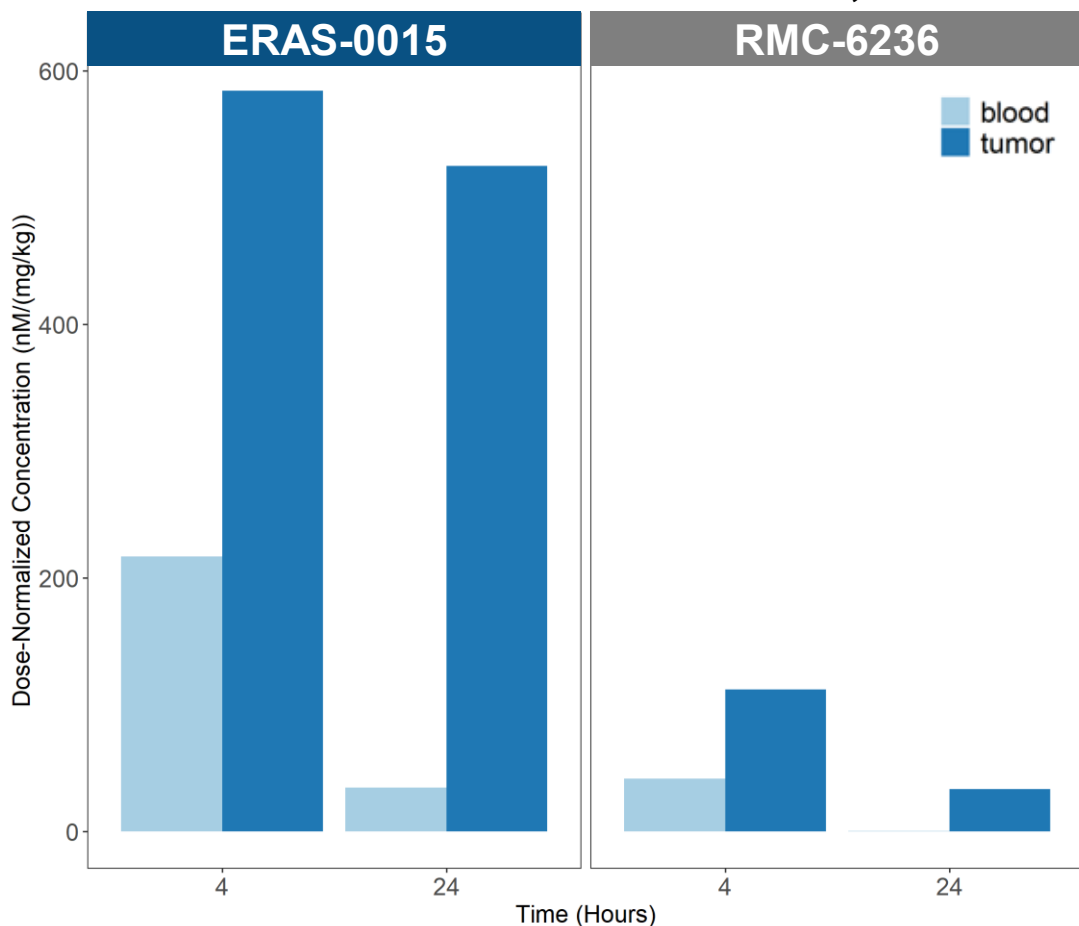
Therapy	Dose	TGI on Day 22
ERAS-0015	0.3 mg/kg	86%
	1 mg/kg	104%
	3 mg/kg	105%
RMC-6236	1 mg/kg	64%
	3 mg/kg	93%
	10 mg/kg	104%

- ERAS-0015 was well tolerated at all doses

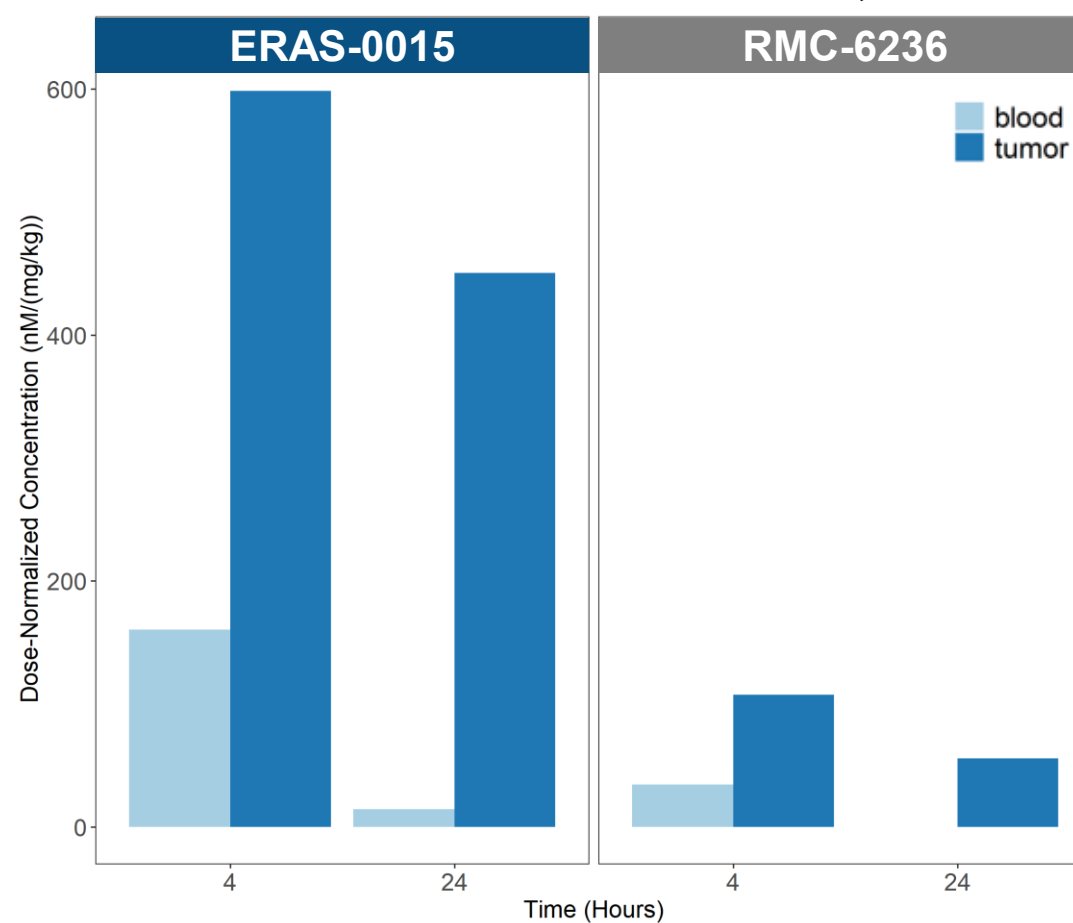
These data were generated in head-to-head in vitro assay and/or in vivo experiments  
ERAS-0015 and RMC-6236 dosed orally once daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

# 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



These data were generated in head-to-head in vitro assay and/or in vivo experiments  
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 <sub>1/2</sub> (h)	5.0	1.7	5.7	1.5	24.5	7.6	15.2	No Data
	Vd <sub>ss</sub> (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 <sub>0-last</sub> (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 <sub>max</sub> (nM)	745	1,443	1,620	339	472	377	723	No Data
	T <sub>1/2</sub> (h)	6.3	1	6.1	2.5	22.4	7.8	12.3	No Data
	AUC <sub>0-last</sub> (nM*h)	6,786	4,467	15,213	1,427	8,720	2,755	10,004	No Data
	Bioavailability (F%)	48%	33%	38%	14%	22%	21%	17%	No Data

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

# 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**

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

## Preclinical Differentiation

<b>In Vivo Activity</b>	Comparable efficacy at 1/10 <sup>th</sup> -1/5 <sup>th</sup> of RMC-6236 dose in multiple mouse models
<b>PK Properties</b>	<ul style="list-style-type: none"><li>• Improved %F, lower CL, longer T<sub>1/2</sub></li><li>• Preferential tumor distribution with longer residence time</li></ul>
<b>Cellular Potency</b>	Improved potency across RAS-altered cell lines
<b>CYPA Binding</b>	Improved binding affinity (8-21x)

## ERAS-0015 Clinical Data<sup>2</sup>

### 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<sup>3</sup>

### 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**

<sup>1</sup> Data presented are not based on head-to-head studies. See Disclaimer slide regarding Cross-Study Comparisons <sup>2</sup> As of January 7, 2026; <sup>3</sup> Dosing occurred after the January 7, 2026 data cutoff date.

%F = bioavailability; CL = clearance; T<sub>1/2</sub> = half-life; CYPA: cyclophilin A; PR = partial response (cPR = confirmed PR, uPR = unconfirmed PR); QD = once daily; DLT = dose-limiting toxicity; AE = adverse event

# 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

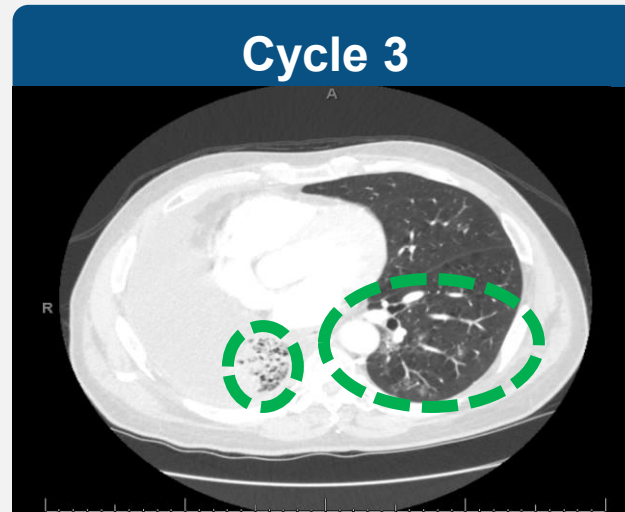
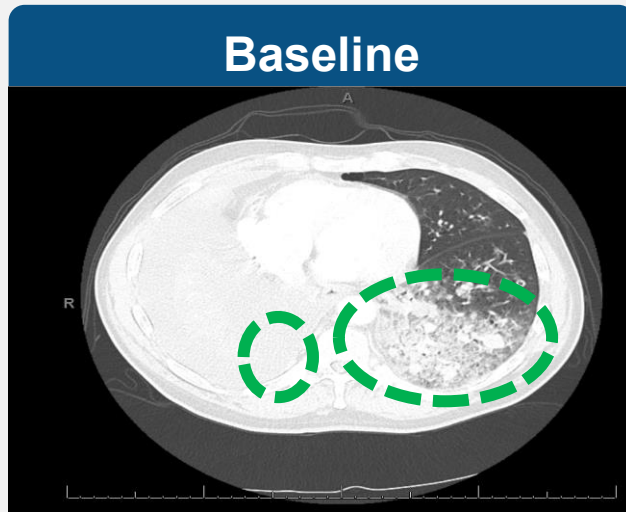
- Stage IV NSCLC; KRAS G12V

## 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<sup>1</sup>



- 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<sup>1</sup>

<sup>1</sup> 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 <sup>1</sup> FPD (06/22) to DCO (10/23): 16 months			ERAS-0015 1H26 Data FPD to DCO <12 months				
<b>Efficacy</b>	≥10% point increase in ORR in <b>2L+ KRAS<sup>G12X</sup> PDAC</b>	19.6% uORR (5 PR, 4 uPR, N=46)			<p><b>“Differentiated” Scenario</b></p> <p><i>Differentiated Efficacy*</i> ≥ 1 Efficacy Attribute</p> <p><b>OR</b></p> <p><i>Differentiated Safety*</i> ≥ 2 Safety and Tolerability Attributes</p>				
	≥10% point increase in ORR in <b>2L+ KRAS<sup>G12X</sup> NSCLC</b>	37.5% uORR (1 CR, 11 PR, 3 uPR, N=40)							
<b>Safety and Tolerability</b>	Reduced frequency and/or severity of <b>rash<sup>2</sup> TRAEs</b>	All Grades	81%				<p><b>“Paradigm-Shifting” Scenario</b></p> <p><i>Differentiated Efficacy*</i> ≥ 1 Efficacy Attribute</p> <p><b>AND</b></p> <p><i>Differentiated Safety*</i> ≥ 2 Safety and Tolerability Attributes</p>		
		Gr 1	52%						
		Gr 2	23%						
		Gr 3+	6%						
	Reduced frequency and/or severity of <b>GI TRAEs</b>		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 <b>stomatitis/mucositis TRAEs</b>	All Grades	22%						
		Gr 1	12%						
		Gr 2	8%						
		Gr 3+	2%						
	<b>Combinability</b> with SOC agents (ERAS-0015 combo data guided to 2027)	<u>CRC</u> : Anti-EGFR combo not shown to be tolerated <u>PDAC</u> : Dose modification required for chemo			* <b>Compared to RMC-6236 benchmark</b>				

<sup>1</sup> Arbour et al, ESMO 2023; RMC-6236 benchmark pop for safety & tolerability: N=111 patients with NSCLC, PDAC; <sup>2</sup> 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. Note: See Disclaimer slide regarding Cross-Study Comparisons.

# 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<sup>1</sup>
- Favorable ADME properties and PK performance in animals vs. most advanced pan-RAS MG in development<sup>1</sup>
- 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

<sup>1</sup> These data were generated in head-to-head assay and/or in vivo experiments

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

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

SPR-based kinetic biophysical binding characterization of ERAS-4001

Target	KD (nM)	t <sub>1/2</sub> (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

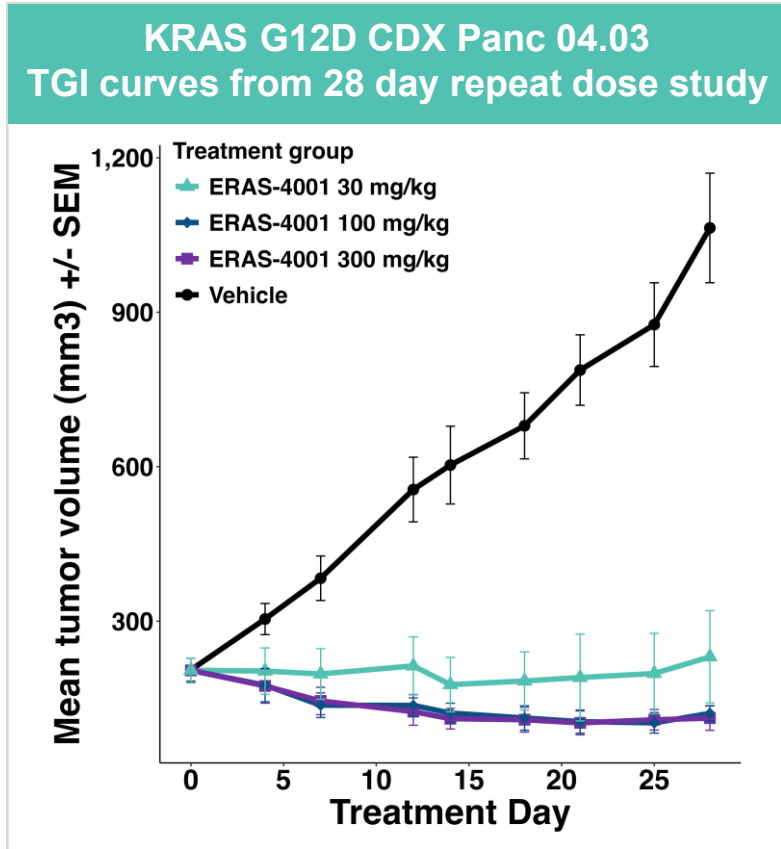
Assay Class	Assay	Target	ERAS-4001 IC50 (nM)
Biochemical Functional	RAS-RAF Binding Assay (RBD)	RBD KRAS G12D GDP	<b>1.6</b>
		RBD KRAS G12D GMPPNP*	<b>6.8</b>

\* 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

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



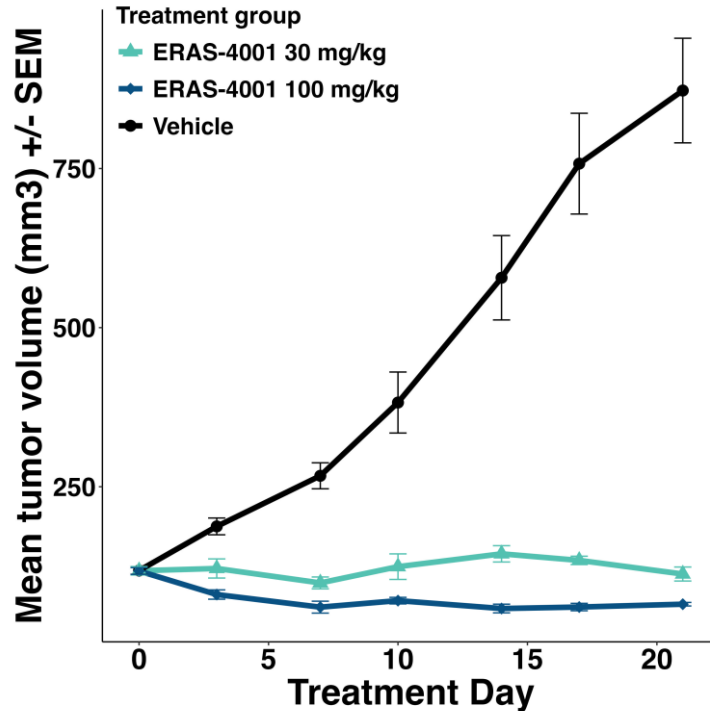
TGI, PD (pERK) and PK (AUC<sub>0-last</sub>) Summary

Therapy	Dose	TGI on Day 28	pERK Inhibition at 8 hr	AUC <sub>0-last</sub> (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

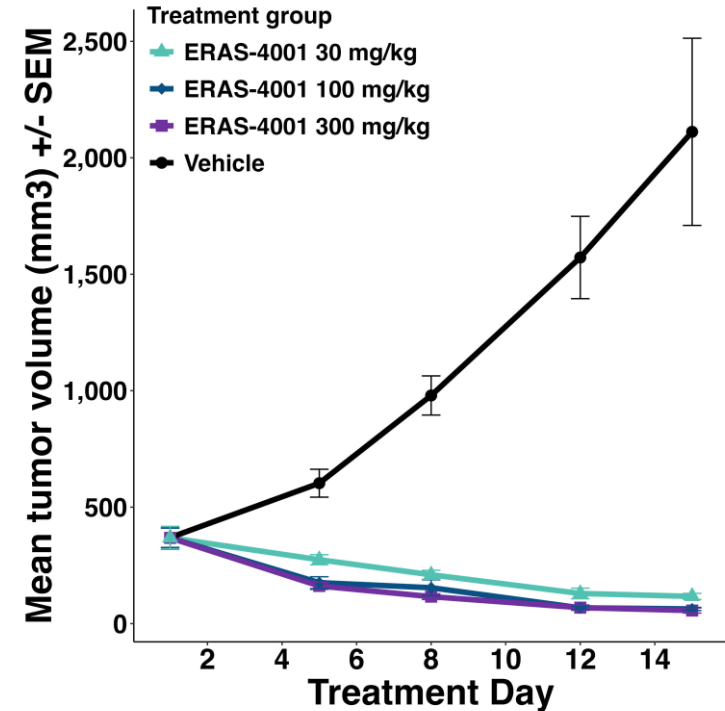
- ERAS-4001 was well tolerated at doses up to 300 mg/kg BID for 28 days (i.e., no dose reductions or holidays; no body weight loss or significant health observations)

# ERAS-4001 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

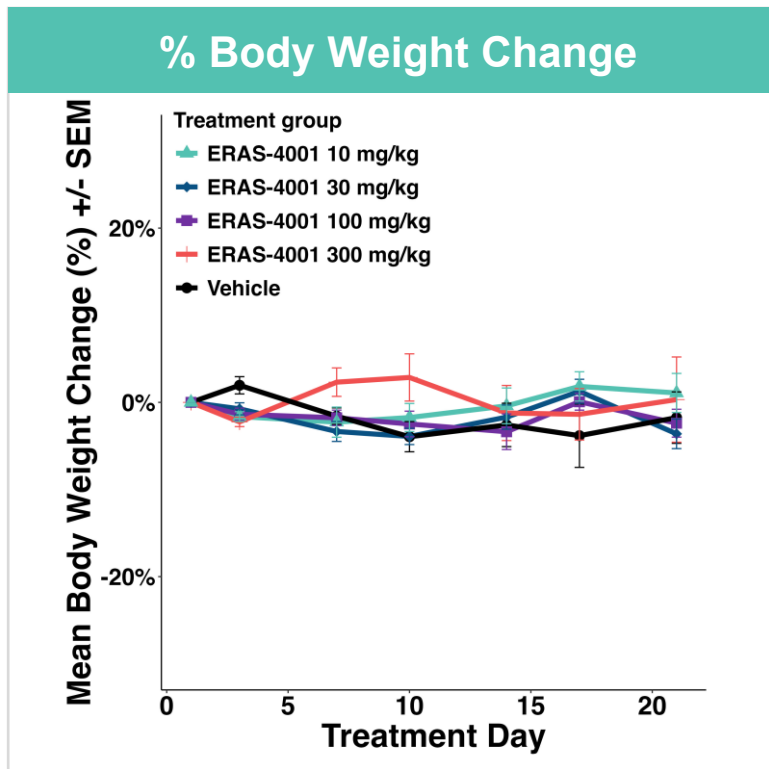
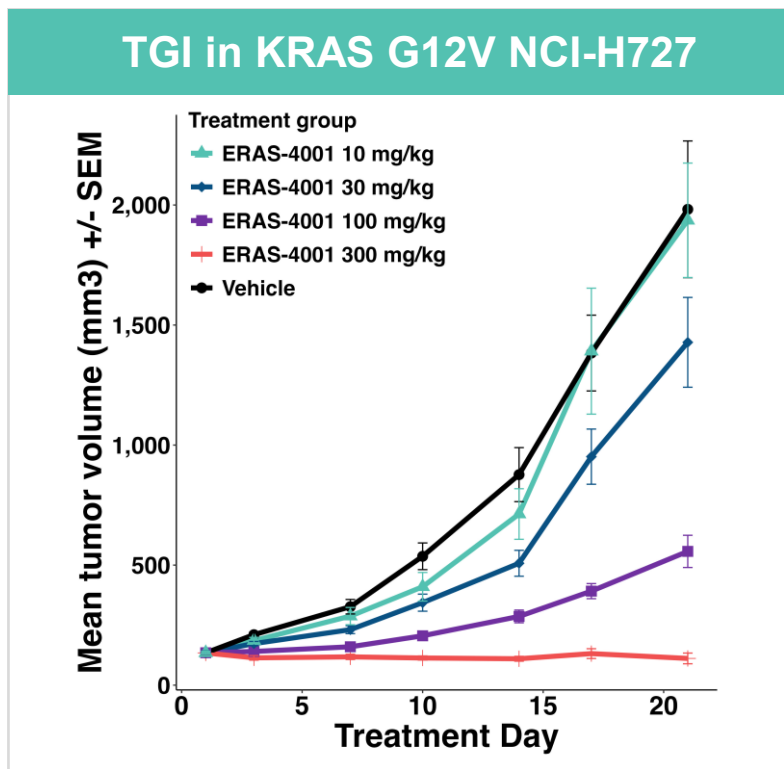


## TGI in KRAS G12V Ovarian CDX RKN



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



### 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 <sub>0</sub> (nM)	1,722	1,083	1,669
	T <sub>1/2</sub> (h)	1.9	3	5.8
	V <sub>d</sub> (L/kg)	5.16	10.1	14.1
	CL (mL/kg/min)	45.5	70.9	53.1
	AUC <sub>0-last</sub> (nM·h)	938	615	827
Oral	Dose (mpk)	30.3	30.9	15.3
	C <sub>max</sub> (nM)	2,090	584	323
	T <sub>max</sub> (h)	1.5	4	0.5
	T <sub>1/2</sub> (h)	1.5	2.3	5.4
	AUC <sub>0-last</sub> (nM·h)	4,498	2,562	962
	Bioavailability (F %)	27	27	16

# Anticipated key milestones in 2026-2027

<b>Program</b> <i>Mechanism</i>	<b>Trial Name</b> <i>Indication</i>	<b>Anticipated Milestone</b>
<b>ERAS-0015</b> <i>Pan-RAS molecular glue</i>	<b>AURORAS-1</b> <i>RASm solid tumors</i>	<ul style="list-style-type: none"> <li>• <b>H1 2026:</b> Ph 1 monotherapy data<sup>1</sup></li> <li>• <b>H2 2026:</b> <ul style="list-style-type: none"> <li>- Initiate monotherapy expansion cohorts</li> <li>- Initiate combination dose escalation cohorts</li> </ul> </li> <li>• <b>2027:</b> <ul style="list-style-type: none"> <li>- Monotherapy expansion data</li> <li>- Combination dose escalation data</li> </ul> </li> </ul>
<b>ERAS-4001</b> <i>Pan-KRAS inhibitor</i>	<b>BOREALIS-1</b> <i>KRASm solid tumors</i>	<ul style="list-style-type: none"> <li>• <b>H2 2026:</b> Ph 1 monotherapy data</li> <li>• <b>2027:</b> <ul style="list-style-type: none"> <li>- Initiate monotherapy expansion cohorts</li> <li>- Initiate combination dose escalation cohorts</li> </ul> </li> </ul>

<sup>1</sup> 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-Viciano, UCL; Cichowski, HMS; Blacklow, HMS; Corcoran, MGH), precision oncology (Demetri, DFCI; Bernards, NCI), 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**



**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	 Alterome THERAPEUTICS ALTA-3263
Mutant Selective <sup>1</sup>	 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 competitors shown based on public disclosures; list is not intended to be exhaustive; updated as of March 2026

<sup>1</sup> Mutant selective beyond KRAS G12C inhibitors

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

	Preclinical (in vitro and in vivo) Activity <sup>1</sup>	OBA <sup>2</sup>	IP <sup>3</sup>
<b>ERAS-0015</b> Pan-RAS Molecular Glue	<p>KRAS G12D: 0.2 – 13.3 nM</p> <p>KRAS G12V: 0.4 – 2.5 nM</p> <p>KRAS G12C: 0.8 – 1.4 nM</p> <p>KRAS G12X: 4.1 – 7.4 nM</p> <p>KRAS G13D: 2.8 – 5.5 nM</p> <p>KRAS WT: 4.1 – 13.8 nM</p> <p>H/NRAS: Active</p> <p><b>KRAS G12D:</b> Tumor regression in PK-59 CDX model at <u>0.3 mpk PO QD</u></p> <p><b>KRAS G12V:</b> Tumor regression in NCI-H727 CDX model at <u>1 mpk PO QD</u></p> <p><b>KRAS G12R:</b> Tumor regression in PSN1 CDX model at <u>5 mpk PO QD</u></p>	<p>Mouse: 48%</p> <p>Rat: 38%</p> <p>Dog: 22%</p> <p>Monkey: 17%</p>	<ul style="list-style-type: none"> <li>IP exclusivity expected through <b>2043</b></li> <li>US patent covering composition of matter issued in Oct. 2025</li> </ul>
<b>ERAS-4001</b> Pan-KRAS Inhibitor	<p>KRAS G12D: 1.0 – 2.6 nM</p> <p>KRAS G12V: 0.7 – 9.1 nM</p> <p>KRAS G12C: 1.1 – 4.5 nM</p> <p>KRAS G12X: 6.5 – 37.7 nM</p> <p>KRAS G13D: 5.8 – 56.0 nM</p> <p>KRAS WT: 3.6 – 10.8 nM</p> <p>H/NRAS WT: No activity</p> <p><b>KRAS G12D:</b> Tumor regression in Panc04.03, PK-59, and LU-01-1381 CDX/PDX models at 30 – 100 mpk PO BID; <u>combo with anti-PD-1 achieved complete disappearance of tumors in all mice (7/7) on D31</u> at 100 mpk PO BID</p> <p><b>KRAS G12V:</b> Tumor regression in RKN and NCI-H727 CDX models at 30 – 300 mpk PO BID</p>	<p>Mouse: 27%</p> <p>Rat: 5 – 27% (variable PK in rat)</p> <p>Dog: 16%</p>	<ul style="list-style-type: none"> <li>IP exclusivity expected through <b>2043</b></li> <li>US patent covering composition of matter issued in Feb. 2026</li> </ul>

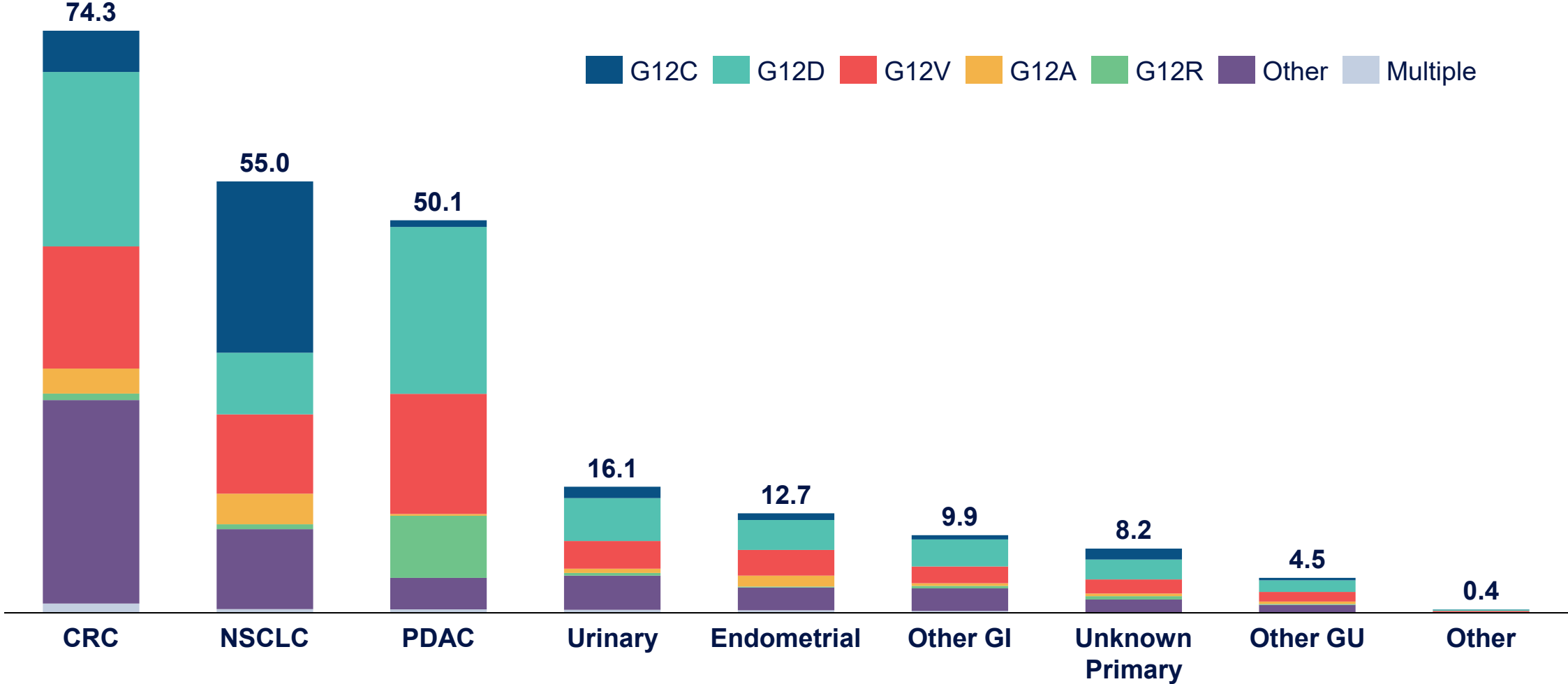
Potential BIC **Pan-RAS MG** for RASm solid tumors, which showed ~5x – 10x greater antitumor activity and favorable ADME properties and PK performance in animal species (vs. most advanced Pan-RAS MG in development)

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; <sup>1</sup> in vitro potency assessed by CTG 2D and 3D-cell proliferation assay IC50s; <sup>2</sup> OBA assessed by %F; <sup>3</sup> IP includes composition of matter, methods of use, and methods of making licensed compounds; date is absent any patent term adjustments or extensions

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

Estimated number of patients affected by KRAS mutant tumors in the US (thousands)



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