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**UNITED STATES DISTRICT COURT  
SOUTHERN DISTRICT OF CALIFORNIA**

individually and on  
behalf of all others similarly situated,  
  
Plaintiff,  
  
v.  
  
ERASCA, INC., JONATHAN LIM, and  
DAVID CHACKO,  
  
Defendants.

Case No. \_\_\_\_\_

**CLASS ACTION COMPLAINT FOR  
VIOLATION OF THE FEDERAL  
SECURITIES LAWS**

**DEMAND FOR JURY TRIAL**

1 Plaintiff (“Plaintiff”), individually and on behalf of all others similarly  
2 situated, alleges the following upon personal knowledge as to Plaintiff, and upon information and  
3 belief as to all other matters based upon the investigation conducted by and through Plaintiff’s  
4 attorneys, which included, among other things, a review of documents filed by Defendant Erasca, Inc.  
5 (“Erasca” or the “Company”) with the U.S. Securities and Exchange Commission (“SEC”), research  
6 reports issued by securities and financial analysts, press releases issued by Defendants, media and  
7 news reports, and other publicly available information about Defendants. Plaintiff believes that  
8 substantial additional evidentiary support will exist for the allegations set forth herein after a  
9 reasonable opportunity for discovery.

#### 10 NATURE AND SUMMARY OF THE ACTION

11 1. This is a securities fraud class action on behalf of all those who purchased, or otherwise  
12 acquired, Erasca common stock during the period from January 14, 2025 through April 26, 2026,  
13 inclusive (the “Class Period”), who were damaged thereby (the “Class”). This action is brought on  
14 behalf of the Class for violations of Sections 10(b) and 20(a) of the Securities Exchange Act of 1934  
15 (the “Exchange Act”), 15 U.S.C. §§ 78j(b) and 78t(a) and Rule 10b-5 promulgated thereunder by the  
16 SEC, 17 C.F.R. § 240.10b-5.

17 2. Erasca develops oncology therapies for patients with RAS/MAPK pathway-driven  
18 cancers. One of its primary drug candidates is ERAS-0015, a pan-RAS molecular glue targeting solid  
19 tumors.

20 3. Throughout the Class Period, Defendants made false and/or misleading statements,  
21 and failed to disclose material facts, including that: (1) ERAS-0015’s preclinical data was based on  
22 improper comparisons to Revolution Medicines, Inc. (“RevMed”) and placed Erasca at risk of  
23 violating patent and trade secret protections; and (2) based on the foregoing, Defendants lacked a  
24 reasonable basis for their positive statements related to ERAS-0015.

25 4. On April 27, 2026, at 8:31 a.m. EDT before the market opened, Erasca disclosed in a  
26 Form 8-K that it had received a letter from legal counsel for RevMed alleging that Erasca’s ERAS-  
27 0015 infringes a RevMed patent (U.S. Patent No. 12,409,225) and is connected to alleged trade secret  
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1 authorized the publication of the documents, presentations, and materials alleged herein to be  
2 misleading prior to its issuance and had the ability and opportunity to prevent the issuance of these  
3 false statements or to cause them to be corrected. Because of their positions with the Company and  
4 access to material non-public information available to them but not to the public, the Individual  
5 Defendants knew that the adverse facts specified herein had not been disclosed to and were being  
6 concealed from the public and that the positive representations being made were false and misleading.  
7 The Individual Defendants are liable for the false statements pleaded herein.

8 19. Erasca and the Individual Defendants are referred to herein, collectively, as  
9 “Defendants.”

#### 10 **SUBSTANTIVE ALLEGATIONS**

11 20. Erasca is a clinical-stage precision oncology company focused on discovering,  
12 developing, and commercializing therapies for patients with RAS/MAPK pathway-driven cancers.

13 21. One of Erasca’s primary drug candidates, ERAS-0015, is a pan-RAS molecular glue  
14 aimed at treating patients with RAS mutant solid tumors. This treatment would have broad  
15 implications in oncology because RAS mutations drive nearly one-fourth of all solid tumors,  
16 including in non-small cell lung cancer, pancreatic ductal adenocarcinoma, and colorectal cancer.

17 22. In Erasca’s Form 10-K filed with the SEC on March 20, 2025 (“2024 10-K”),  
18 Defendants advertised the vast market potential for ERAS-0015, stating it was a “potential best-in-  
19 class” drug that could have “the potential to address unmet medical needs in approximately 2.7  
20 million patients who are diagnosed annually worldwide with RAS-mutant tumors[.]” Defendants also  
21 stated they approved a “reprioritization program” to focus substantial resources to ERAS-0015 and  
22 “deemphasize” certain of its other drug initiatives.

23 23. Erasca’s initial clinical trial for ERAS-0015 is called AURORAS-1. The  
24 investigational new drug application (“IND”) for AURORAS-1 was cleared by the U.S. Food and  
25 Drug Administration (“FDA”) in May 2025. Around the same time, Erasca began touting ERAS-  
26 0015’s potential compared to RevMed’s RMC-6236, including detailed statements comparing  
27 preclinical results in the 2024 10-K.



1 mutations, including wild-type. In vivo, what's particularly impressive is the  
2 incredibly low doses by which tumor regression is observed.

3 So doses as low as 0.3 to 5 milligrams per kilogram PO on a daily basis. The oral  
4 bioavailability is very high across small and large animal species. ***IP is strong with  
5 exclusivity expected through 2043 and no patentability roadblocks identified to date.***

6 27. At the same conference, an analyst from J.P. Morgan asked Lim "to comment a little  
7 bit about ERAS-0015. And what you kind of learned from the evolving competitive landscape from  
8 Revolution Medicines and others." In response, Lim stated:

9 Yes. So I think what's interesting is that the scientific bar for working on a pan-RAS  
10 molecular glue is very high. So it's really hard -- much harder to discover and develop  
11 a pan-RAS molecular glue than it is -- there's many more pan-KRAS small molecules  
12 out there. So I think there's sort of a scarcity issue within this class. ***So Rev Med clearly  
13 is the trailblazer in this space. I think we've learned a lot from their clinical data in  
14 terms of PDAC as well as more recently non-small cell lung cancer.***

15 I think other tumor types will be interesting to see. But being second in an area of high  
16 unmet need across PDAC, lung and other tumor types is a good place to be. There's  
17 one other company in China that has a pan-RAS molecular glue. They are further  
18 behind. ***And also based on the preclinical data we've seen, it looks to have a profile,  
19 not too dissimilar from 6236 at this point, but it's still early days.***

20 ***I think -- yes, so I think we've learned mostly from RMC-6236 in terms of clinical  
21 data for PDAC and lung.*** The PDAC data, in particular, very exciting in terms of the  
22 ability to help patients, and we'll be following them as well as once we start generating  
23 our own data, later this year, I think we'll be right there.

24 28. On March 20, 2025, Erasca filed its Annual Report on Form 10-K with the SEC  
25 reporting on its financial results for its fiscal year ended December 31, 2024 (the "2024 10-K"). In  
26 the 2024 10-K, Defendants stated:

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1 ERAS-0015 is a pan-RAS molecular glue that we believe has the potential to treat  
 2 patients with RAS mutant solid tumors. The molecule forms a tripartite complex with  
 3 Cyclophilin A (CypA) and the active form of RAS to inhibit RAS-dependent  
 4 signaling. In surface plasmon resonance (SPR) and isothermal titration calorimetry  
 5 (ITC) binding assays, *ERAS-0015 has demonstrated 8-21-fold higher binding*  
 6 *affinity to cyclophilin A relative to RMC-6236 (the leading pan-RAS molecular glue*  
 7 *in development), which we believe may enable more potent inhibition.*

Assay	ERAS-0015 (nM)	RMC-6236 (nM)	Binding affinity difference: ERAS-0015/ RMC-6236
SPR K <sub>D</sub>	4.5	92	21x
ITC K <sub>D</sub>	5.3	44.1	8x

13 We performed a tumor PK distribution assessment in TGI studies of ERAS-0015  
 14 versus RMC-6236 in PK-59 and PSN-1 CDX models. PK concentrations of paired  
 15 tumor and blood samples were measured at 4 and 24 hours after the last dose of the  
 16 TGI studies.

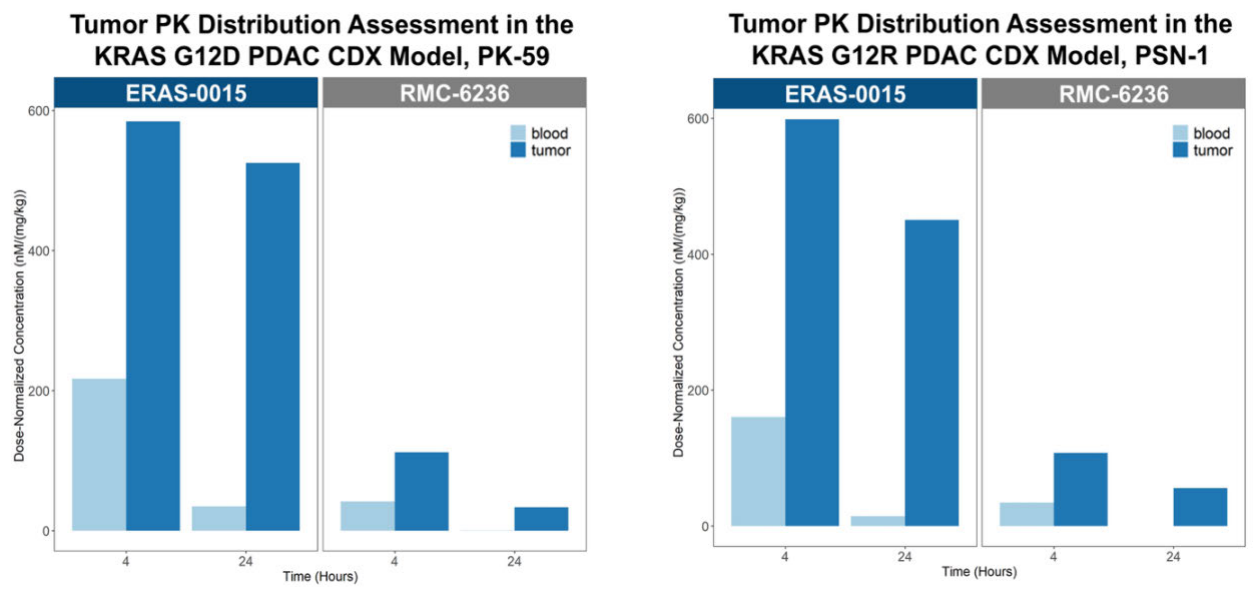
17 *In the PK-59 CDX study, the dose normalized ERAS-0015 (across 3 ERAS-0015*  
 18 *dose levels: 0.1, 0.3, and 1 milligrams per kilograms (mpk)) tumor PK exposures*  
 19 *relative to the corresponding blood concentrations at 4 and 24 hours post-dose were*  
 20 *much higher compared to the same measure for RMC-6236 (dose normalized for 2*  
 21 *RMC-6236 dose levels: 1 and 3 mpk), indicating preferential tumor distribution for*  
 22 *ERAS-0015. In addition, the decrease in ERAS-0015 tumor concentrations from 4*  
 23 *to 24 hours post-dose was much smaller compared to that of RMC-6236, suggesting*  
 24 *longer tumor residence time for ERAS-0015.*

25 Similar findings were also observed in the PSN-1 CDX study as shown in the figure  
 26 on the right. (In the PSN-1 model, ERAS-0015 exposures were dose normalized across  
 27 0.3 and 1 mpk; RMC-6236 exposures were dose normalized across 3 and 10 mpk).

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*Taken together, these data demonstrated that ERAS-0015 showed preferential tumor distribution and longer residence time relative to RMC-6236, which we believe may help drive antitumor activity.* We believe that the preferential tumor distribution and longer residence time could be related to the higher CypA binding affinity.



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Preclinical potency of ERAS-0015

In multiple in vitro cell lines containing representative mutations in KRAS G12X, Q61R, and G13D as well as KRAS wildtype, *ERAS-0015 showed superior in vitro potency when compared to RMC-6236 with an average of 5 times greater potency.*

The molecule showed subnanomolar to nanomolar potency against KRAS G12X, G13D, and KRAS wildtype and was also active against HRAS and NRAS. ERAS-0015 showed no activity in the BRAF V600E A375 cell line, demonstrating selective inhibition of RAS.

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

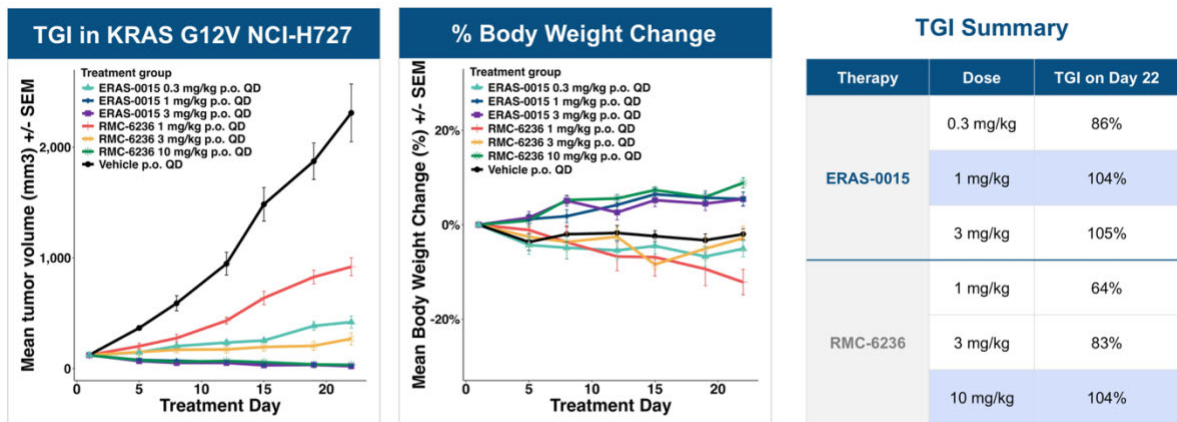
Preclinical efficacy of ERAS-0015

In vivo, ERAS-0015 achieved tumor regression in multiple CDX models across PDAC, CRC, and NSCLC tumor types at doses as low as 0.3 to 0.5 mpk. *Potentially due to its greater binding affinity to CypA than RMC-6236, ERAS-0015 demonstrated comparable to greater in vivo antitumor activity at doses which were approximately one-tenth to one-eighth of the dose of RMC-6236.*

For example, in the PK-59 KRAS G12D PDAC model, the tumor regression that was observed with RMC-6236 at 3 mpk was comparable to the regression that was observed with ERAS-0015 at 0.3 mpk. This difference in in vivo antitumor activity was observed across multiple CDX models including KRAS G12R PDAC CDX PSN-1 and KRAS G12V CRC CDX SW620.

Model and TGI Measurement Day	ERAS-0015 Dose (mg/kg QD)	ERAS-0015 TGI	RMC-6236 Dose (mg/kg QD)	RMC-6236 TGI	TGI Ratio
PK-59 Day 23 (KRAS G12D PDAC)	0.3	106%	3	105%	~10
PSN-1 Day 14 (KRAS G12R PDAC)	1	97%	10	95%	~10
SW620 Day 26 (KRAS G12V CRC)	3	102%	25	103%	~8
NCI-H727 Day 22 (KRAS G12V NSCLC)	1	104%	10	104%	~10

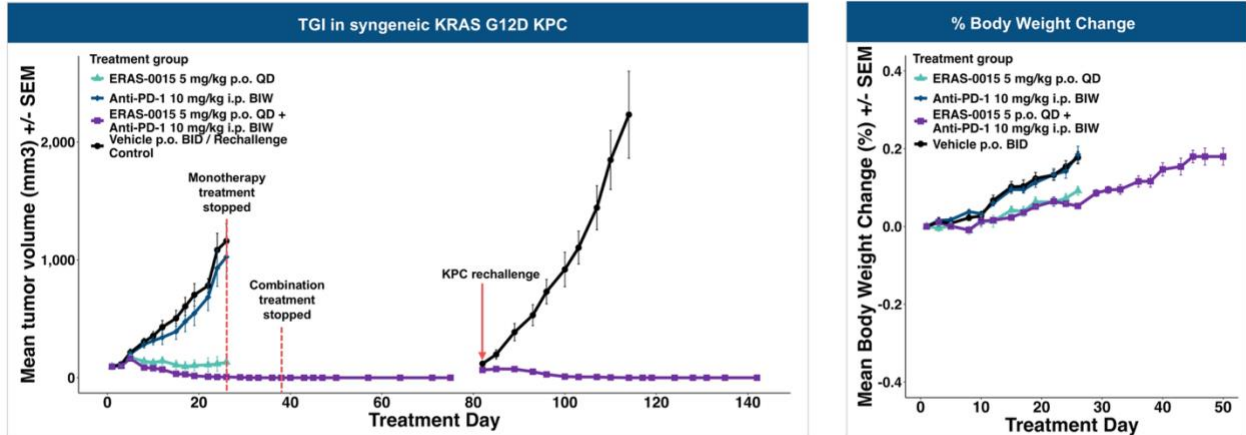
This difference in in vivo antitumor activity was also observed in the NCI-H727 KRAS G12V NSCLC CDX model. *ERAS-0015 was able to achieve tumor regression at 1 mpk relative to RMC-6236 at 10 mpk.*



- ERAS-0015 achieved comparable tumor regression to RMC-6236 in this model at 1/10<sup>th</sup> the dose at 1 mg/kg p.o. QD
- ERAS-0015 was well tolerated at all doses

Activity was also observed with the combination of ERAS-0015 and anti-PD-1 therapy in a KPC KRAS G12D CDX model where tumor regression was maintained even after treatment was stopped on Day 31. Furthermore, tumors did not form after a rechallenge with tumor cells, which involved injecting the tumor cells in the

1 contralateral side of the animal without administering any drug therapy. These  
2 rechallenge data suggest that the combination of ERAS-0015 and anti-PD-1 can  
3 stimulate immunologic antitumor memory in the rechallenged mouse. Doses  
4 administered were tolerable as measured by body weight change.



- ERAS-0015 in combination with anti-PD-1 therapy resulted in a sustained complete response in 7 out of 7 treated mice starting on day 31
  - ERAS-0015 as a monotherapy and in combination with an anti-PD-1 was well tolerated
  - Monotherapy treatments stopped on study day 26 and combination treatment stopped on study day 38
  - Tumor formation was not observed up to 60 days after KPC rechallenge (KPC tumor cells were reinoculated on day 79)
- p.o.: orally administered; BIW: twice a week; BID: twice a day; QD: once daily; CDX: cell line-derived xenograft; TGI: tumor growth inhibition

## 15 Pharmacokinetics and ADME of ERAS-0015

16 ERAS-0015 has shown encouraging PK results in multiple species, including mouse,  
17 rat, dog, and monkey. ***In a head-to-head comparison of ERAS-0015 and RMC-6236,***  
18 ***ERAS-0015 outperformed RMC-6236 on three key metrics (specifically, lower***  
19 ***clearance, longer half-life, and higher bioavailability demonstrated across all***  
20 ***species tested) that we believe may provide ERAS-0015 a clinical advantage in the***  
21 ***clinic over RMC-6236.***

		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>	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

ERAS-0015 has shown favorable overall ADME properties in vitro and encouraging PK characteristics in in vivo animal studies that we believe support development in clinic. ***Based on the differentiated potency and PK/ADME results, we predict that ERAS-0015 will be efficacious at lower doses than the leading pan-RAS molecular glue in development, which may lower the risk of solubility-limited absorption issues and enable linear PK exposure relative to the leading pan-RAS molecular glue in development. In addition, the hERG IC50 was greater than 10 micromolar which suggests potentially lower concern for cardiovascular risk.***

29. On January 12, 2026, Erasca provided an update regarding the initial clinical progress of ERAS-0015 in a presentation filed on Form 8-K with the SEC. In the presentation, Erasca included multiple slides touting the superior potential of ERAS-0015 compared to RMC-6236, with slides highlighting preclinical data titled, ***“ERAS-0015’s higher CYP4 binding affinity may be a differentiator from RMC-6236, demonstrating potential best-in-class profile[,]” “ERAS-0015 demonstrated significantly more potent inhibition of cell growth across KRAS mutant cell lines vs. RMC-6236[,]” “ERAS-0015 demonstrated comparable antitumor activity to RMC-6236 at 1/10th of the dose in a sensitive KRAS G12D PDAC CDX model[,]” “ERAS-0015 demonstrated comparable antitumor activity to RMC-6236 at 1/10th of the dose in an insensitive KRAS G12V***

1 *NSCLC CDX model[,]” “ERAS-0015 demonstrated preferential tumor distribution and longer*  
2 *residence time vs. RMC-6236 in vivo[,]” and “ERAS-0015 showed promising PK in mouse, rat, dog,*  
3 *and monkey”* in comparison to RMC-6236.

4 30. On January 13, 2026, Erasca again presented at the Annual J.P. Morgan Healthcare  
5 Conference, which was attended by Lim and Chacko. At the conference, Lim stated:

6 So I’m going to start with a deep dive on ERAS-0015. *This is the potential best-in-*  
7 *class pan-RAS molecular glue*, and I’ll also update you on the clinical update that we  
8 shared earlier this week. *So ERAS-0015’s potential differentiation really stems from*  
9 *the high binding affinity to cyclophilin A or CypA. And so this works by a similar*  
10 *molecular glue mechanism where this has about 8 to 21-fold higher binding affinity*  
11 *to CypA versus RMC-6236.* And as a result of that, you just get many more of these  
12 bipartite moieties.

13 So if you have 0015 with CypA, that bipartite moiety, we call it a Pacman molecule  
14 goes hunting for RAS to form a tripartite or ternary complex that then takes RAS out  
15 of circulation. And so by having the higher binding affinity, you just have a lot more  
16 -- this is a nod to all of you who grew up in the ‘80s, but pan-RAS Pacman molecules  
17 that just take RAS out of circulation. Now what does that mean? *Well, it results in*  
18 *better potency across multiple cell lines. You can see various degrees of full potency*  
19 *versus 6236 against different mutations of interest across the spectrum of KRAS and*  
20 *other drivers.*

21 And you could see sparing of RTK-altered cell lines. *And importantly, in vivo, we’re*  
22 *seeing ERAS-0015 demonstrating comparable antitumor activity to RMC-6236 at*  
23 *just 1/10 of the dose.* And so I’m going to show you on this slide, a sensitive KRAS  
24 G12D pancreatic model, where you could see it’s called PK59. *6236 was able to*  
25 *achieve very good tumor regression with 3 mpk. And what’s really impressive is that*  
26 *ERAS-0015 was able to achieve the same degree of tumor regression with 0.3 mpk*  
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1 *or 1/10 of the dose. So that just gives you a sense of how potent both of these*  
2 *molecules are, but 6236 in this comparison requires 10x the dose.*

3 On this insensitive model, KRAS G12V non-small cell lung cancer, this is a model  
4 called NCI-H727. In the industry, amongst academia, especially, this is widely viewed  
5 as a bellwether model of sorts because it's where you have – it's very difficult to see  
6 tumor regression with pan-KRAS molecules in this type of model, but you could see  
7 that 6236 was able to achieve good tumor regression with 10 mpk. *And also*  
8 *impressively, ERAS-0015 was able to achieve that with just 1 mpk. So again, the*  
9 *1/10 of the dose in an insensitive model.*

10 So we have a lot of other models that we've shared. They are in the public domain, so  
11 I encourage you to view our prior presentation. So we won't walk you through the half  
12 dozen or so other models. *But importantly, from a PK perspective, the kinetics of*  
13 *ERAS-0015 also look very promising. So if you look at the tumor distribution and*  
14 *residence time, 15 does look like it has preferential tumor distribution and longer*  
15 *residence time.* So if you focus on the graphs on the left, this is the same PK59 model,  
16 that sensitive PDAC model that I just mentioned a couple of slides prior.

17 So if you look at the light blue bars for ERAS-0015 versus RMC-6236, you could see  
18 that the levels of both drugs go down pretty substantially from 4 to 24 hours. *But if*  
19 *you look at the medium to dark blue bars, you could see that 6236 diminishes or*  
20 *decreases over that 24-hour period, but ERAS-0015 levels are sustainably high.*

21 *And so what that tells us is that -- well, the reason for that is that there is a CypA*  
22 *that's overexpressed in multiple solid tumor types. And because of the higher*  
23 *binding affinity of ERAS-0015 to CypA, it's just lingering in the tumor and*  
24 *surrounding tissues a little longer. And so that tumor distribution and residence*  
25 *time, if that translates -- well, certainly, the PK kinetics look preferential in that*  
26 *case. But if that translates into a safety or efficacy advantage, then that will be really*

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1            *cool to see. And then that's not just one model, but it's also seen in another model*  
2            *called PSN1 on the right.*

3            From a PK perspective, ERAS-0015 showed promising PK across multiple small and  
4            large animal species. So I'll draw your attention to the boxed areas. Clearance-wise,  
5            *ERAS-0015 had lower clearance across the board relative to 6236, longer half-life*  
6            *and a higher oral bioavailability* as expressed by F percent.

7            31.        During the same conference, Lim also stated:

8            So I know that Anupam is going to ask me about all the different doses that were  
9            evaluated in this trial. *We will disclose all of that in the first half of this year. So top*  
10           *line safety, tolerability, PK and initial efficacy data for dozens of patients are*  
11           *planned for this first half. But in the meantime, based on the early signs of activity*  
12           *as well as safety and tolerability and PK, we think 15 has the potential to become a*  
13           *preferred RAS targeting backbone for combinations.*

14           32.        On March 12, 2026, Erasca filed its Annual Report on Form 10-K with the SEC  
15           reporting on its financial results for its fiscal year ended December 31, 2025 (the "2025 10-K"). In  
16           the 2025 10-K, Defendants repeated the same preclinical data alleged herein in ¶28, comparing  
17           ERAS-0015 to RMC-6236.

18           33.        In the 2025 10-K, Defendants also stated, "On January 12, 2026, we provided an  
19           update regarding the initial clinical progress of ERAS-0015. The update consisted of the following,  
20           as of a data cutoff of January 7, 2026...*Favorable safety and tolerability results, with no dose-*  
21           *limiting toxicities and predominantly low-grade adverse events observed at all dose levels*  
22           *evaluated[.]"*

23           34.        The statements referenced above in ¶¶26-33 were materially false and/or misleading  
24           when made because they failed to disclose the following adverse facts pertaining to the Company's  
25           business, which were known to Defendants or recklessly disregarded by them as follows:

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1 a) ERAS-0015's preclinical data was based on improper comparisons to  
2 Revolution Medicines ("RevMed") and placed Erasca at risk of violating patent and trade  
3 secret protections; and

4 b) that, based on the foregoing, Defendants lacked a reasonable basis for their  
5 positive statements about the Company's business, operations, and prospects related to  
6 ERAS-0015.

### 7 **ADDITIONAL SCIENTER ALLEGATIONS**

8 35. As alleged herein, Defendants acted with scienter in that they knew the public  
9 documents and statements issued or disseminated in the name of the Company were materially false  
10 and misleading; knew that such statements or documents would be issued or disseminated to the  
11 investing public; and knowingly and substantially participated or acquiesced in the issuance or  
12 dissemination of such statements or documents and in actions intended to manipulate the market price  
13 of Erasca's common stock as primary violations of the federal securities laws. As set forth elsewhere  
14 herein in detail, Defendants, by virtue of their receipt of information reflecting the true facts regarding  
15 Erasca, their control over, and/or receipt or modification of, the Company's allegedly materially  
16 misleading misstatements, and/or their associations with the Company that made them privy to  
17 confidential proprietary information concerning Erasca, participated in the fraudulent scheme alleged  
18 herein. The adverse events at issue also involved one of the Company's main drug programs.

19 36. Defendants capitalized on Erasca's artificially inflated stock price to conduct a  
20 common stock offering that closed on January 23, 2026, pursuant to a shelf registration statement on  
21 Form S-3, including a base prospectus, that was previously filed with the SEC and declared effective  
22 on August 22, 2025. Erasca represented that its gross proceeds from the offering, before deducting  
23 the underwriting discounts and commissions and other offering expenses, were approximately \$258.8  
24 million. Defendants were motivated to artificially inflate Erasca's stock price for this purpose, and  
25 the timing of the offering in relation to Defendants' misstatements and omissions about ERAS-0015  
26 further support a strong inference of scienter.









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**COUNT II**  
**For Violation of §20(a) of the Exchange Act**  
**(Against the Individual Defendants)**

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57. Plaintiff repeats and re-alleges each and every allegation contained above as if fully set forth herein.

58. The Individual Defendants acted as controlling persons of the Company within the meaning of §20(a) of the Exchange Act as alleged herein. By virtue of their high-level positions at the Company, the Individual Defendants had the power and authority to cause or prevent the Company from engaging in the wrongful conduct complained of herein. The Individual Defendants were provided with or had unlimited access to the documents where false or misleading statements were made and other statements alleged by Plaintiff to be false or misleading both prior to and immediately after their publication, and had the ability to prevent the issuance of those materials or to cause them to be corrected so as not to be misleading. By reason of such conduct, the Individual Defendants are liable pursuant to §20(a) of the Exchange Act.

**PRAYER FOR RELIEF**

WHEREFORE, Plaintiff prays for relief and judgment, as follows:

A. Determining that this action is a proper class action pursuant to Rule 23(a) and 23(b)(3) of the Federal Rules of Civil Procedure on behalf of the Class as defined herein, and a certification of Plaintiff as class representative pursuant to Rule 23 of the Federal Rules of Civil Procedure and appointment of Plaintiff's counsel as Lead Counsel;

B. Awarding compensatory damages in favor of Plaintiff and the other class members against all Defendants, jointly and severally, for all damages sustained as a result of Defendants' wrongdoing, in an amount to be proven at trial, including pre-judgment and post-judgment interest thereon.

C. Awarding Plaintiff and other members of the Class their reasonable costs and expenses in this litigation, including attorneys' fees, experts' fees and other reasonable costs and disbursements; and

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D. Awarding Plaintiff and the other Class members such other relief as this Court may deem just and proper.

**DEMAND FOR JURY TRIAL**

Plaintiff hereby demands a trial by jury.