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Erasca R&D Day

September 2022

Disclaimer: Forward looking statements and 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, costs, design and conduct of our ongoing and planned preclinical studies and clinical trials for our product candidates, the potential benefits from our current or future arrangements with third parties, the timing and likelihood of success of our plans and objectives, 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 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 are early in our development efforts and have only three product candidates in early clinical development and all of our other development efforts are in the preclinical or development stage; the retrospective analysis of pooled data covers multiple clinical trials with different designs, inclusion criteria, and dosing regimens, which cannot be directly compared, and therefore may not be a reliable indicator of efficacy and safety data; interim results of clinical trials 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 more patient data become available; potential delays in the commencement, enrollment, 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 and acquisitions 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; our ability to fund our operating plans with our current cash, cash equivalents, and investments; our ability to maintain undisrupted business operations due to the COVID-19 pandemic, including delaying or disrupting our clinical trials, manufacturing, and supply chain; unstable market and economic conditions having serious adverse consequences on our business, financial condition and stock price; 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, 2021, 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 R&D Day Agenda

Company Overview	Erasca pipeline and drug development strategy Key scientific hypotheses and supportive preclinical data
ERAS-007 / ERAS-601 Preliminary Results	Retrospective pooled efficacy analysis Preliminary safety and pharmacokinetic assessment Clinical development plan for combinations
Discussant	Dr. David Hong, MD Anderson Cancer Center
Closing Remarks	Future directions Key milestones and clinical trial readouts

Q&A Session

~5.5m lives at stake annually worldwide with RAS/MAPK pathway alterations

Over 70% of unmet needs are “Blue Oceans” with no approved targeted therapies

New cases estimated worldwide per annum (thousands; numbers may not add up due to rounding)

Alterations	GBM	HNSCC	NSCLC	CRC	Melanoma	PDAC	Other solid tumors	AML	US	EU	ROW	Global
EGFR*/FLT3	125	513	184	338	-	-	-	61	82	222	917	1,220
NF1	25	58	98	35	33	1.9	434	3.2	75	159	453	687
KRAS G12C	-	2.8	240	57	-	5.0	45	0.1	36	82	232	350
KRAS G12D	0.2	4.7	68	238	0.5	178	200	1.2	65	171	456	692
Other KRAS	0.4	9.4	183	465	1.2	242	326	3.5	114	299	817	1,230
NRAS	0.5	8.4	11.7	72	71	1.0	116	13.8	42	82	170	295
HRAS	0.2	45	7.8	0.4	3.0	0.2	57	-	11	24	80	114
BRAF V600E/K	2	1.9	23	180	93	1.4	158	0.4	63	127	271	461
BRAF Class 2	0.4	3.8	17.6	6.9	5.3	0.5	58	-	10.8	23.1	58	92
BRAF Class 3	0.1	0.9	11.7	16.8	2.5	-	29	0.2	6.1	14.8	40	61
Other BRAF	-	-	3.9	-	1.9	0.3	0.5	-	0.7	1.0	4.9	6.6
MEK	0.2	1.9	11.7	8.8	4.6	0.2	22	-	5	11	33	50
Co-occurring activating MAPK pathway alterations**	1.4	10.3	62	59	37	7.1	84	3.0	33	69	162	264
US	12	29	93	114	77	51	153	11	542			
EU	34	76	194	398	116	124	324	18		1,285		
Rest of World	109	555	635	964	60	264	1,053	57			3,696	
Global	155	660	923	1,476	253	438	1,530	86				5,522

Blue ocean opportunities Red ocean opportunities

* Post-Osimertinib resistant population shown for EGFRm NSCLC except for SCLC transformation

** Co-occurring activating MAPK pathway alterations exclude EGFR overexpression

Source: SEER database (2020), ECIS database (2020), GLOBOCAN database (2020), The AACR Project GENIE Consortium version 8.1 (2020), TCGA Research Network: https://www.cancer.gov/tcga, Tyner JW et al. (2018) PMID: 30333627, Brenner CW et al (2013) PMID: 24120142, Chen J et al. (2020) PMID: 32015526, and Ostrom QT, et al. (2020) PMID: 33123732

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Erasca's tissue specific and tissue agnostic trials target multiple indications

New cases estimated worldwide per annum (thousands; numbers may not add up due to rounding)

Alterations	GBM	HNSCC	NSCLC	CRC	Melanoma	PDAC	Other solid tumors	AML	US	EU	ROW	Global
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HRAS	0.2	45	7.8	0.4				-				
BRAF V600E/K	2	1.9	23	180				0.4				
BRAF Class 2	0.4	3.8	17.6	6.9	5.3	0.5	58	-	10.8			
BRAF Class 3	0.1	0.9	11.7	16.8	2.5	-	29	0.2	6.1	14.8	40	61
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Global	155	660	923	1,476	253	438	1,530	86				5,522

THUNDERBBOLT-1
801

HERKULES-2
007 + osimertinib

HERKULES-2
007 or 601 + sotorasib

HERKULES-3
007 + palbociclib

HERKULES-3
007 + palbociclib

HERKULES-3
007 + encorafenib & cetuximab (EC)

HERKULES-1 & FLAGSHP-1
ERAS-007 + ERAS-601
(our first MAPKlamp);
601 + cetuximab (triple WT CRC[1] and HPV- HNSCC)

Blue ocean opportunities Red ocean opportunities

* Post-Osimertinib resistant population shown for EGFRm NSCLC except for SCLC transformation
** Co-occurring activating MAPK pathway alterations exclude EGFR overexpression
[1] Triple wildtype CRC is KRASwt, NRASwt, and BRAFwt
Source: SEER database (2020), ECIS database (2020), GLOBOCAN database (2020), The AACR Project GENIE Consortium version 8.1 (2020), TCGA Research Network: https://www.cancer.gov/tcga, Tyner JW et al. (2018) PMID: 30333627, Brenner CW et al (2013) PMID: 24120142, Chen J et al. (2020) PMID: 32015526, and Ostrom QT, et al. (2020) PMID: 33123732

Erasca's clinical development plan generates multiple ways to win for patients

Indication	RAS/MAPK altered solid tumors	EGFRm NSCLC post-osi	KRAS G12C NSCLC	BRAFm CRC EC-naïve	BRAFm CRC EC-treated	KRASm/ NRASm CRC	KRASwt/ NRASwt/ BRAFwt 3L CRC	HPV-negative HNSCC	EGFR altered rGBM
Benchmark[1]	SOC is largely chemo	ORR 29%, mDOR 4.2 mos.	ORR 36%, mDOR 10 mos.	ORR 20%, mDOR 6.1 mos.	ORR ~2%, mDOR NA	ORR ~2%, mDOR NA	ORR 20%, mDOR 5.4 mos.	ORR 13%, mDOR 5.8 mos.	ORR 26%, mDOR 4.2 mos.
Regimen tested	ERAS-007 + ERAS-601 (our first MAPKlamp)	ERAS-007 + osimertinib	ERAS-007 or ERAS-601 + sotorasib	ERAS-007 + encorafenib + cetuximab	ERAS-007 + encorafenib + cetuximab	ERAS-007 + palbociclib	ERAS-601 + cetuximab	ERAS-601 + cetuximab	ERAS-801 monotherapy
	ERAS-007 (alternative schedules)		ERAS-3490						
	ERAS-601 (alternative schedules)								
Erasca trial(s)	HERKULES-1	HERKULES-2 Sub-study 1	HERKULES-2 Sub-study 2	HERKULES-3 Sub-study 1	HERKULES-3 Sub-study 1	HERKULES-3 Sub-study 2	FLAGSHP-1	FLAGSHP-1	THUND-ERBBOLT-1
	FLAGSHP-1		AURORAS-1 (planned)						

100% of CRC

[1] Currently approved standard of care

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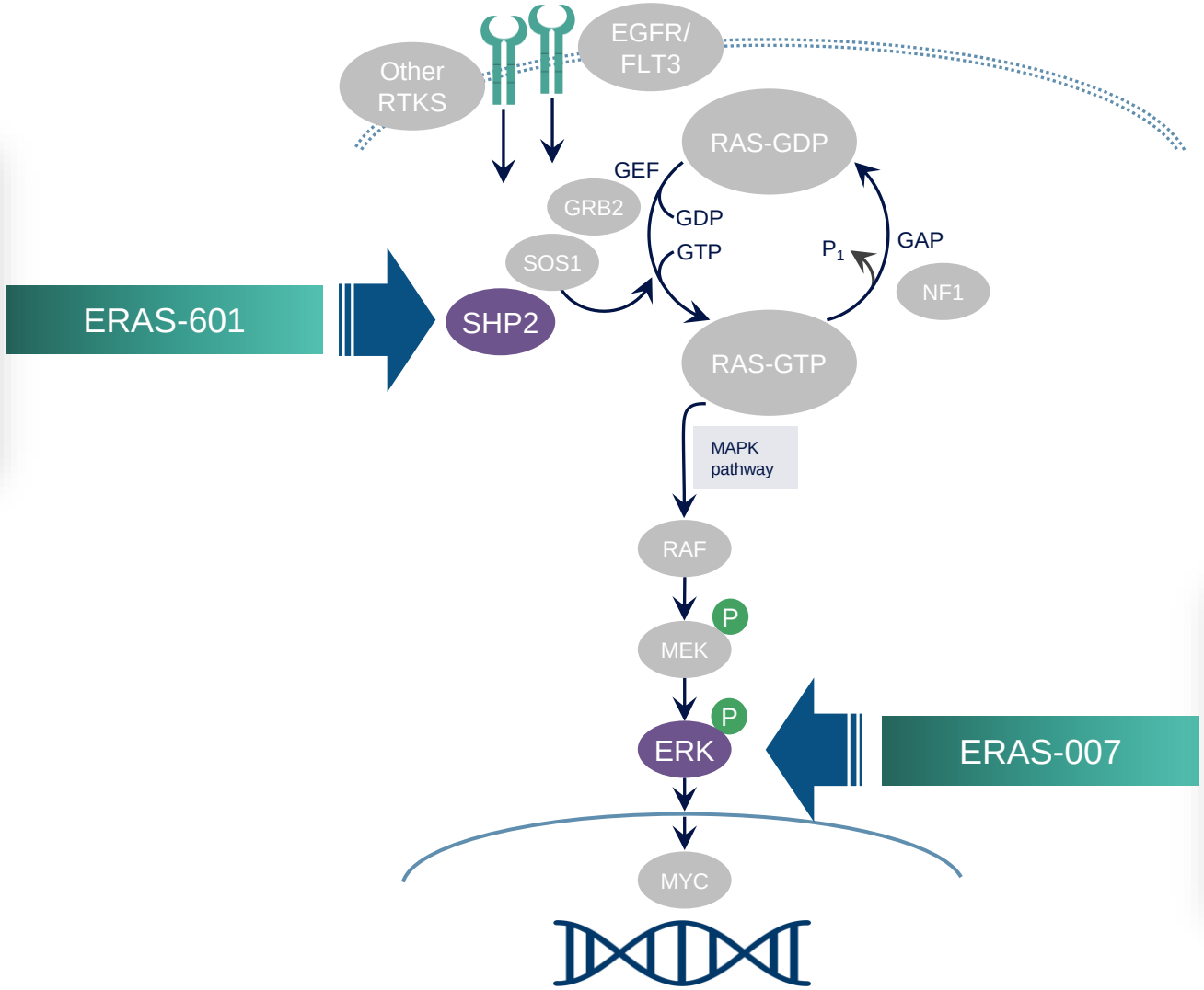
Are there more responsive subsets within the Blue Ocean where promising combination approaches can be particularly effective?

Key scientific hypotheses and supportive preclinical data

Reminder:
ERAS-007 & ERAS-601 target key downstream and upstream RAS/MAPK nodes

Potent, selective, orally bioavailable SHP2 inhibitor in clinical development in FLAGSHP-1 and HERKULES-2



Potent, selective, orally bioavailable ERK inhibitor in clinical development in HERKULES-1, -2, -3 and SU2C

SU2C = Stand Up To Cancer

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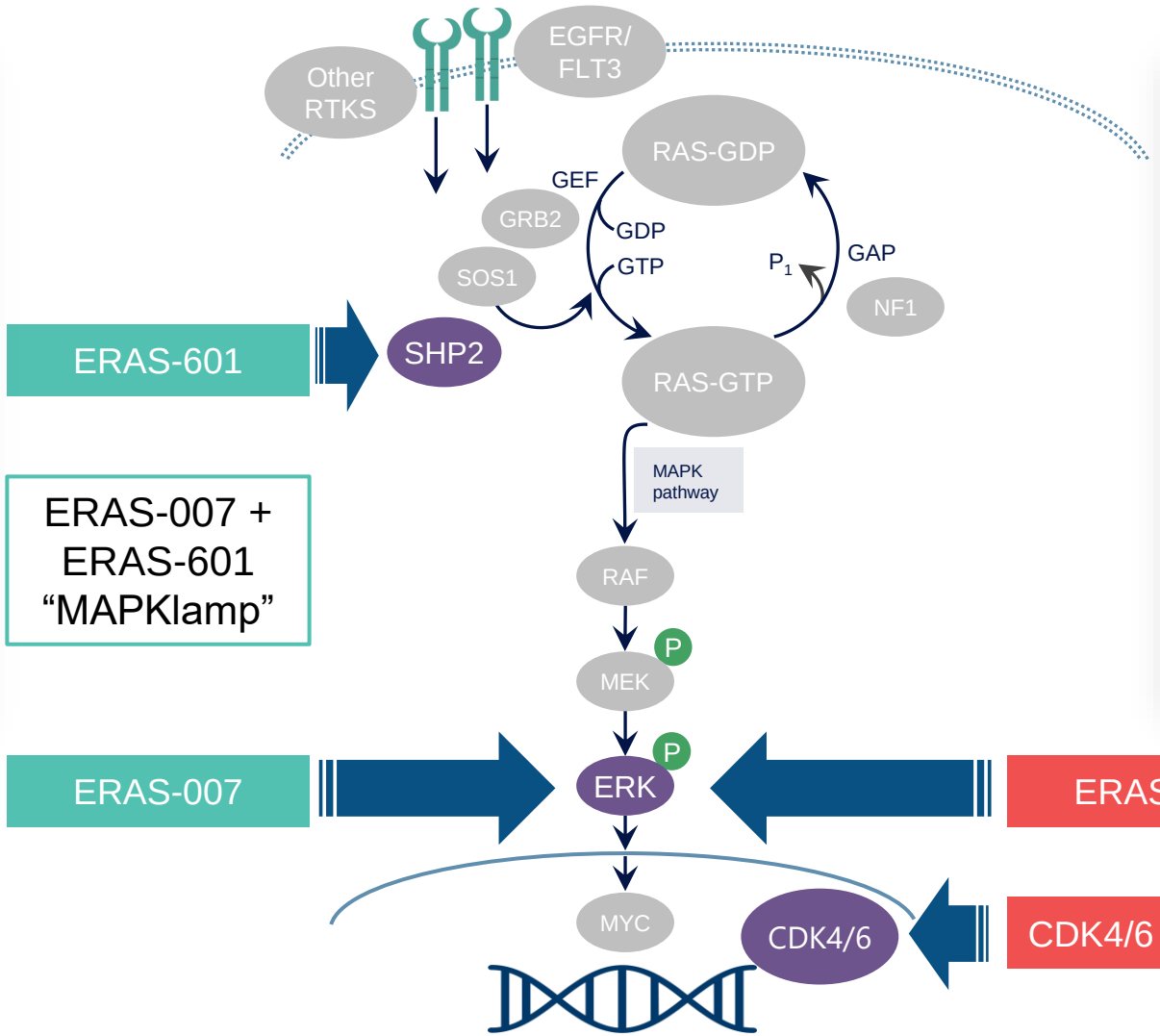
ERK inhibition with CDK4/6 or SHP2 inhibition offers two compelling approaches to target solid tumors with MAPK pathway alterations

Scientific Hypothesis #2:
Simultaneous inhibition of upstream and downstream RAS/MAPK pathway nodes

- Induce stronger pathway suppression and tumor cell killing than either agent alone
- Overcome compensatory reactivation of the RAS/MAPK pathway



Scientific Hypothesis #1:
Dual inhibition of RAS/MAPK and the cell cycle pathways in KRASm/NRASm CRC and KRASm PDAC

- Enhance tumor cell killing due to deeper inhibition of cell cycle progression and cell growth
- Overcome compensatory reactivation of the RAS/MAPK pathway

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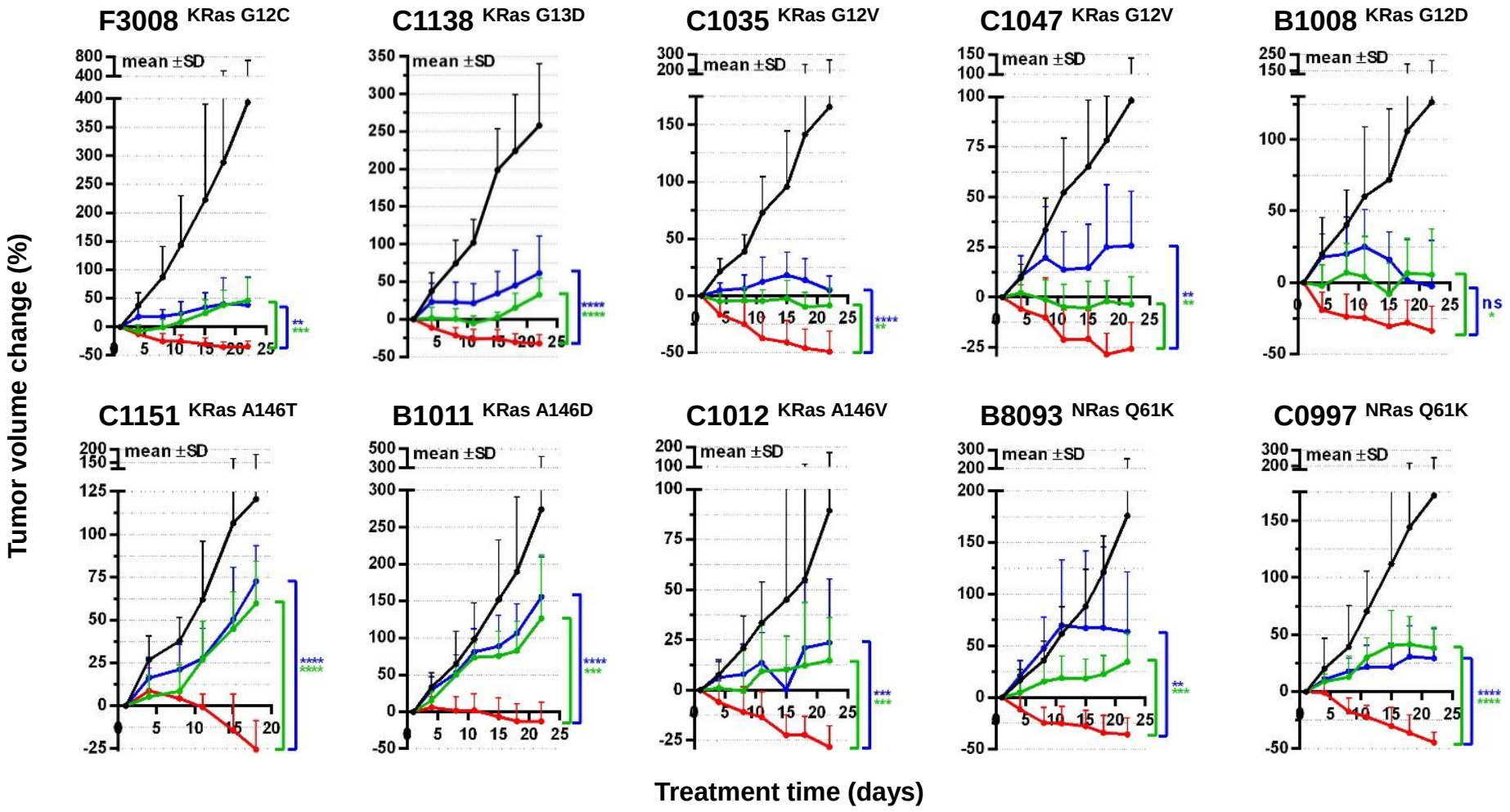
Dual inhibition of cell cycle and MAPK pathways enhanced tumor shrinkage

in PDX models of KRAS/NRAS mutant CRC



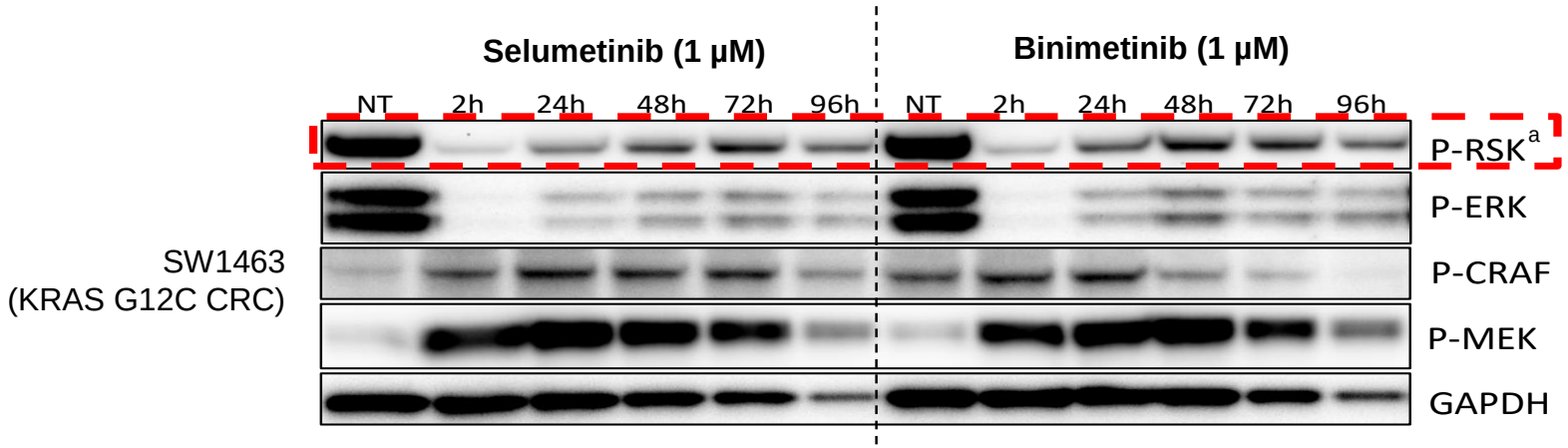
Inhibition of MEK and CDK4/6 represented highly active combination regimen in vivo

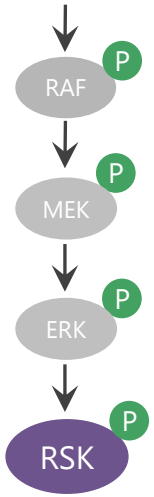
Regimen:

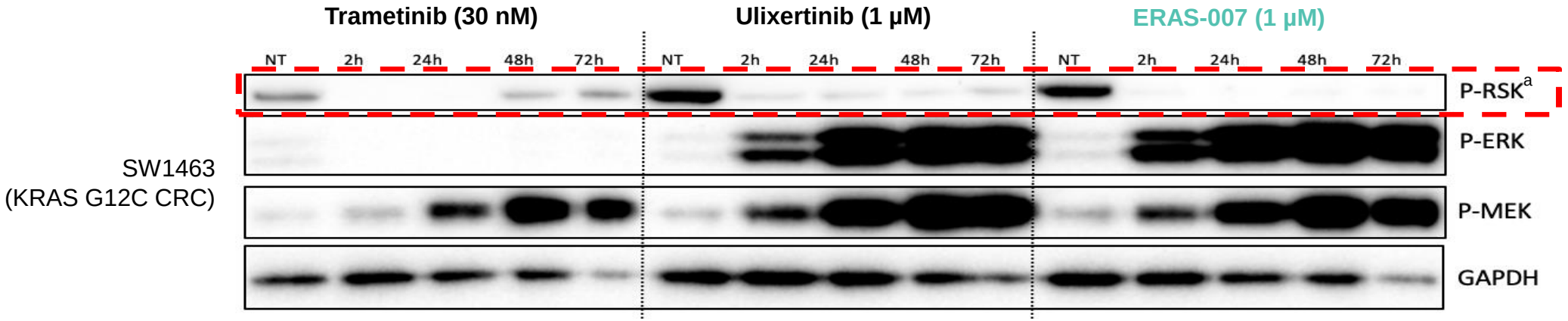
Vehicle	
Palbociclib	**100 mpk QD**
Trametinib	**0.25 mpk QD**
Combo	**100/0.25 mpk QD**

Modified from AACR 2021; Courtesy of Scott Kopetz, MD, PhD (MD Anderson Cancer Center)

ATP-competitive ERK inhibitors were more robust in shutting down RAS/MAPK pathway reactivation than allosteric MEK inhibitors; ERAS-007 was most robust



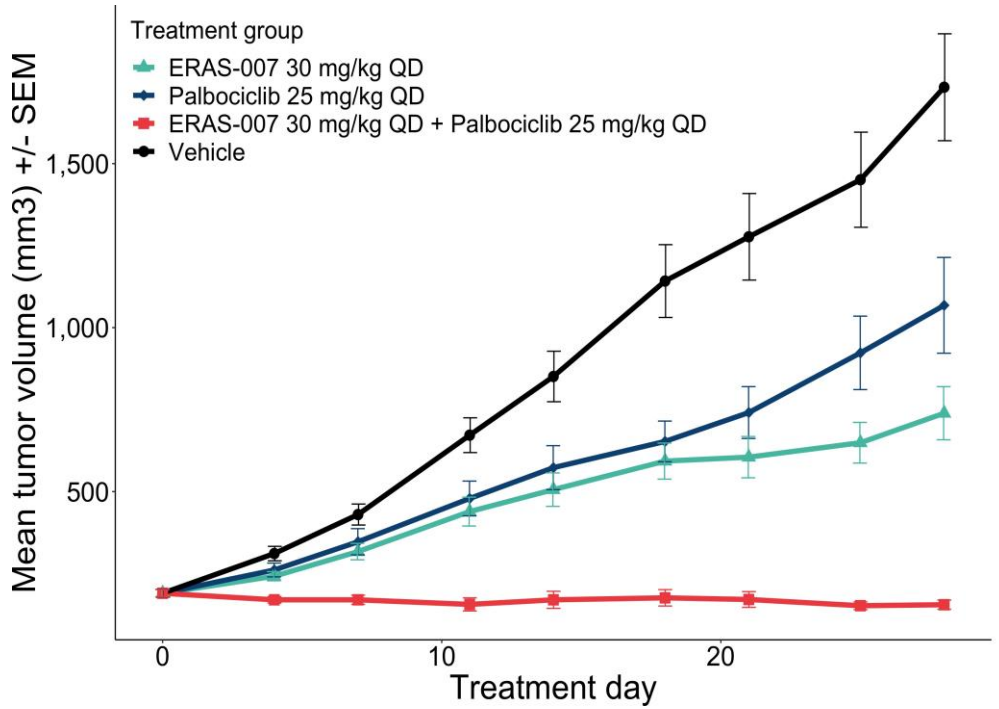




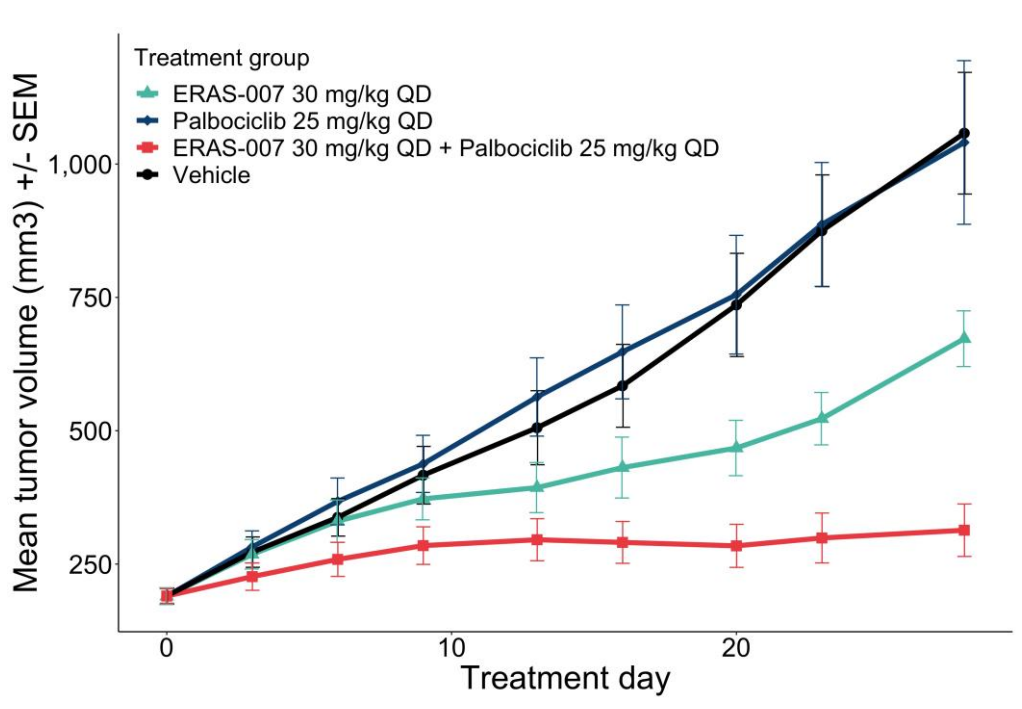
ERK directly phosphorylates the p90 ribosomal S6 kinase protein (RSK) so RSK phosphorylation (P-RSK) serves as a biomarker of ERK and ultimately RAS/MAPK pathway activity
Source: Unpublished data

ERAS-007 + palbociclib enhanced tumor growth inhibition (TGI) in KRASm CRC and PDAC models

TGI in KRAS G13D CRC CDX LoVo



TGI in KRAS G12D PDAC PDX PAN026

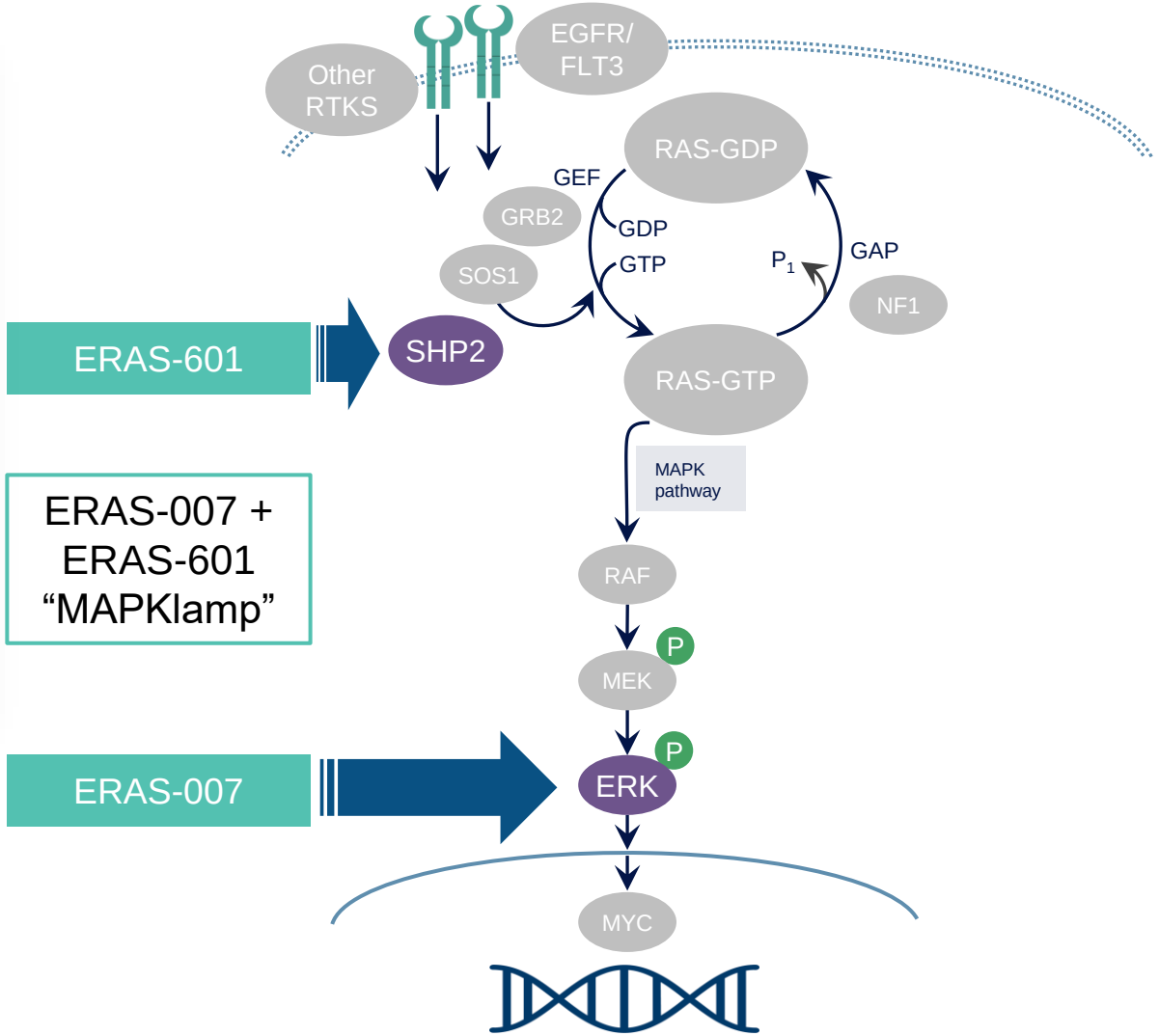


- Combination was tolerated in mice (e.g., no dose holidays, deaths, or euthanizations)
- ERAS-007 and palbociclib were dosed orally and continuously

Scientific Hypothesis #2: “MAPKlamp” inhibition of upstream and downstream RAS/MAPK pathway nodes has potential for deeper, more durable responses

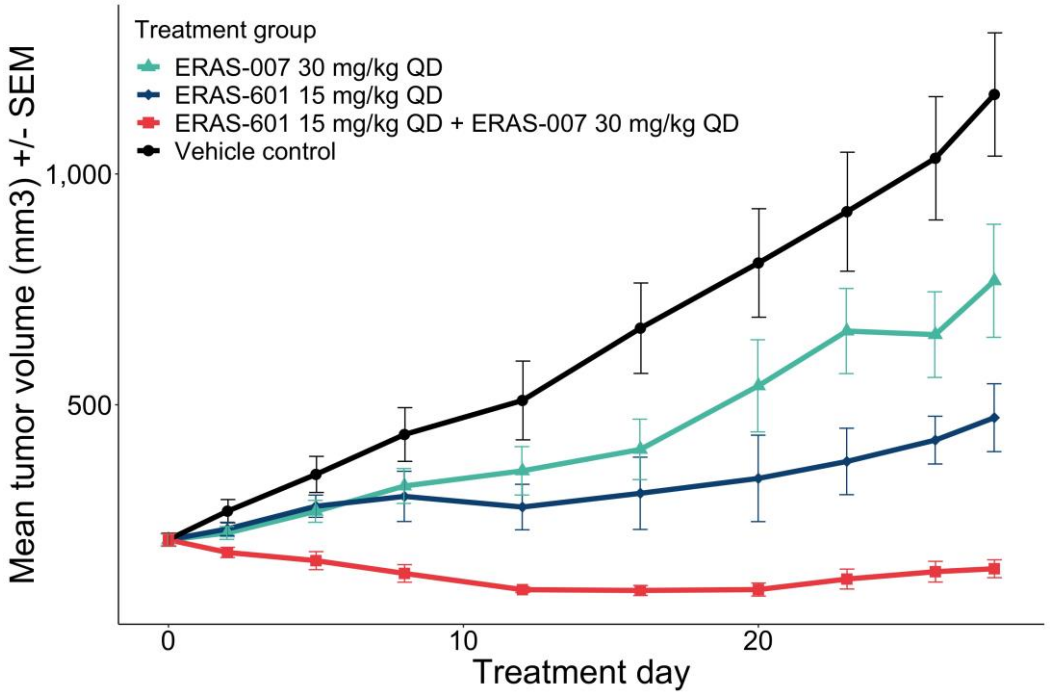
Scientific Hypothesis #2:
Simultaneous inhibition of upstream and downstream RAS/MAPK pathway nodes

- Induce stronger pathway suppression and tumor cell killing than either agent alone
- Overcome compensatory reactivation of the RAS/MAPK pathway

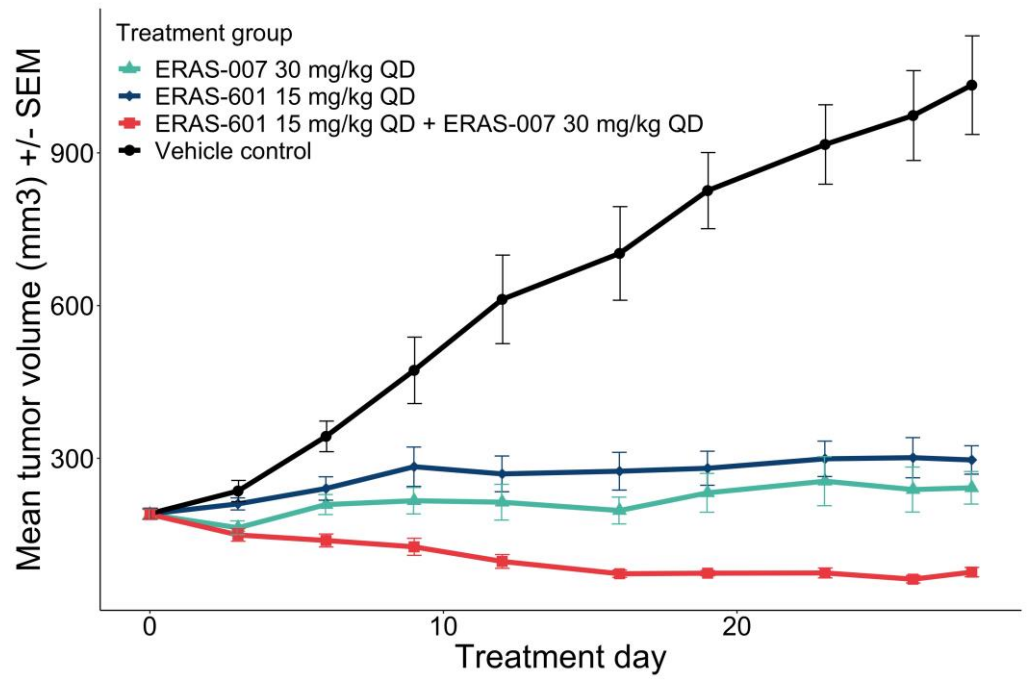


ERAS-007 + ERAS-601 MAPKlamp showed increased *in vivo* tumor growth inhibition in two KRAS mutant NSCLC CDX models

TGI in KRAS G12A NSCLC CDX NCI-H2009



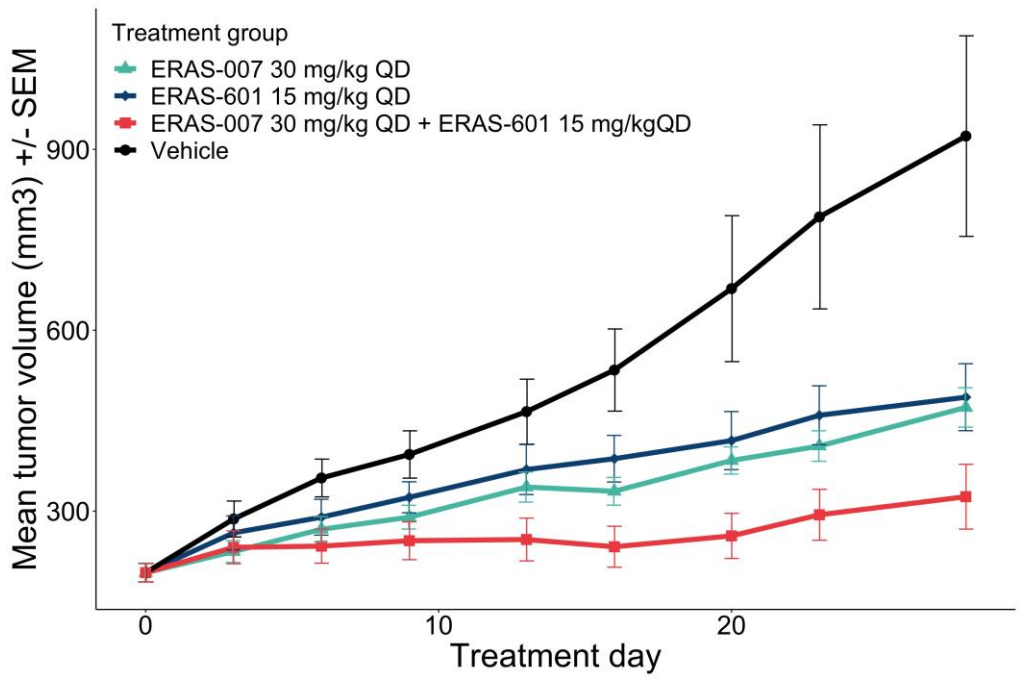
TGI in KRAS G12V NSCLC CDX NCI-H441



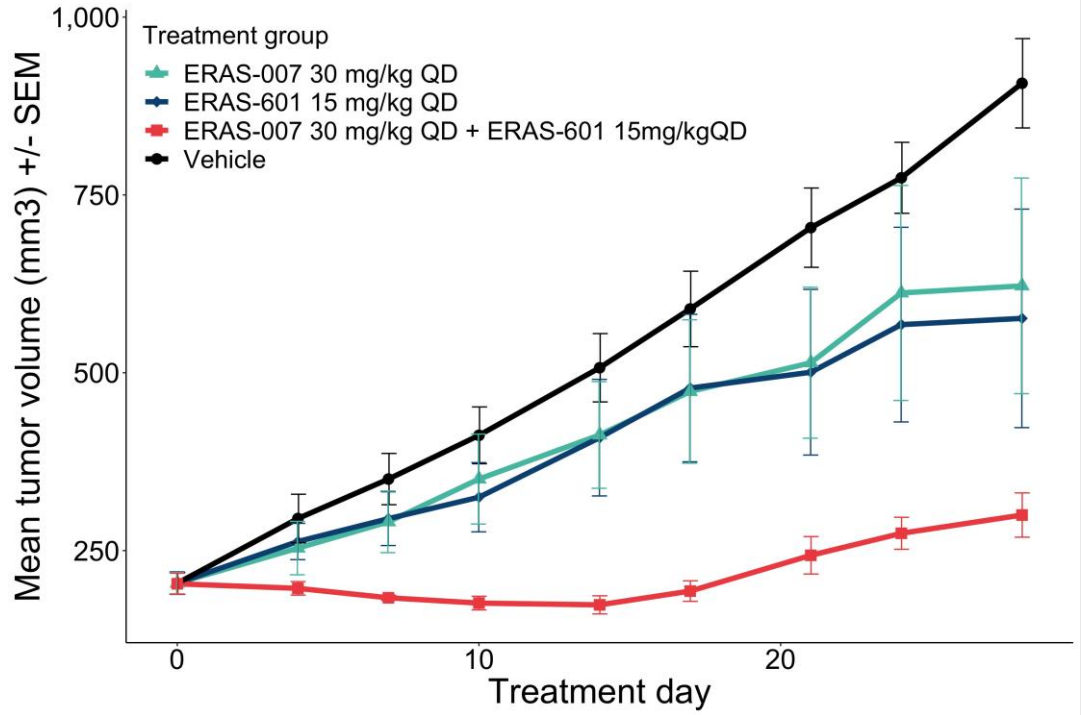
- MAPKlamp combination showed activity in both models and was tolerated in mice (e.g., no dose holidays, deaths, or euthanizations)
- ERAS-007 and ERAS-601 were dosed orally and continuously

ERAS-007 + ERAS-601 MAPKlamp showed increased *in vivo* tumor growth inhibition in two KRAS mutant PDAC PDX models

TGI in KRAS G12D PDAC PDX PAN031



TGI in KRAS G12D PDAC PDX PAN092

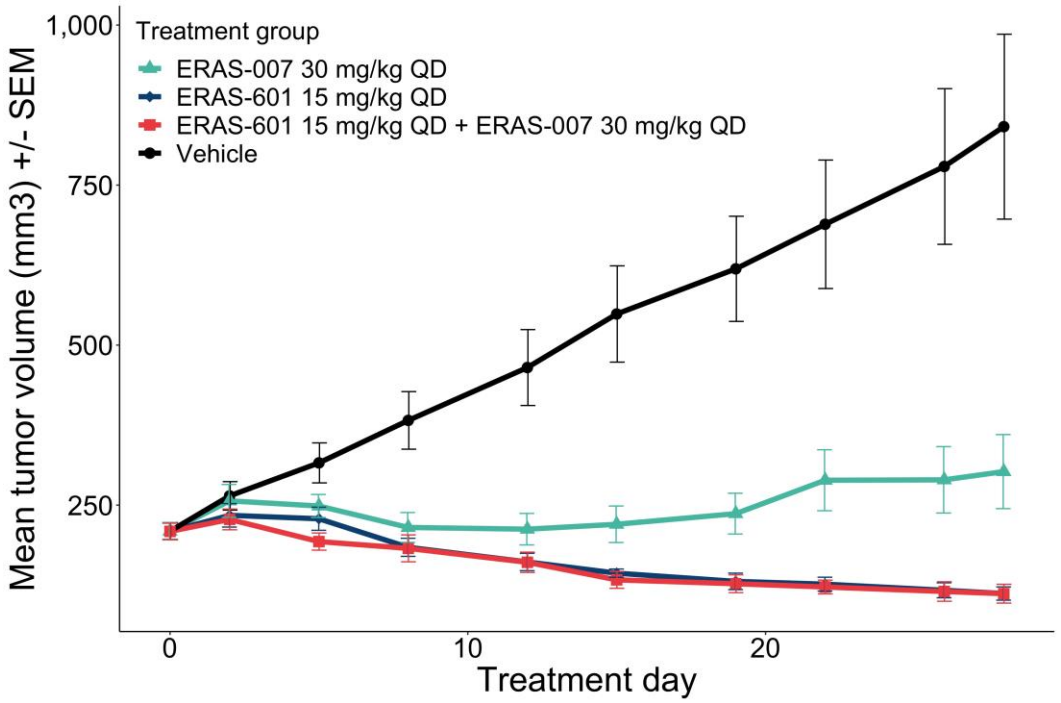


- MAPKlamp combination showed activity in both models and was tolerated in mice (e.g., no dose holidays, deaths, or euthanizations)
- ERAS-007 and ERAS-601 were dosed orally and continuously

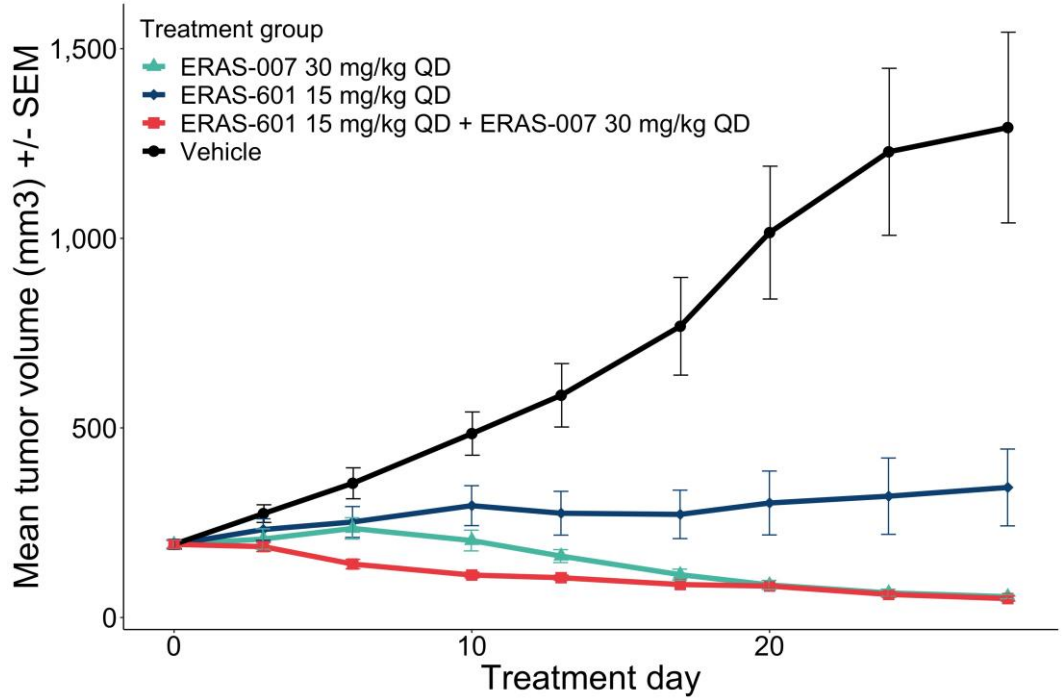
ERAS-007 + ERAS-601 MAPKlamp showed consistent combination activity

in BRAF class 3 models that had differing sensitivities to single agents

TGI in BRAF Class 3 NSCLC CDX NCI-H508



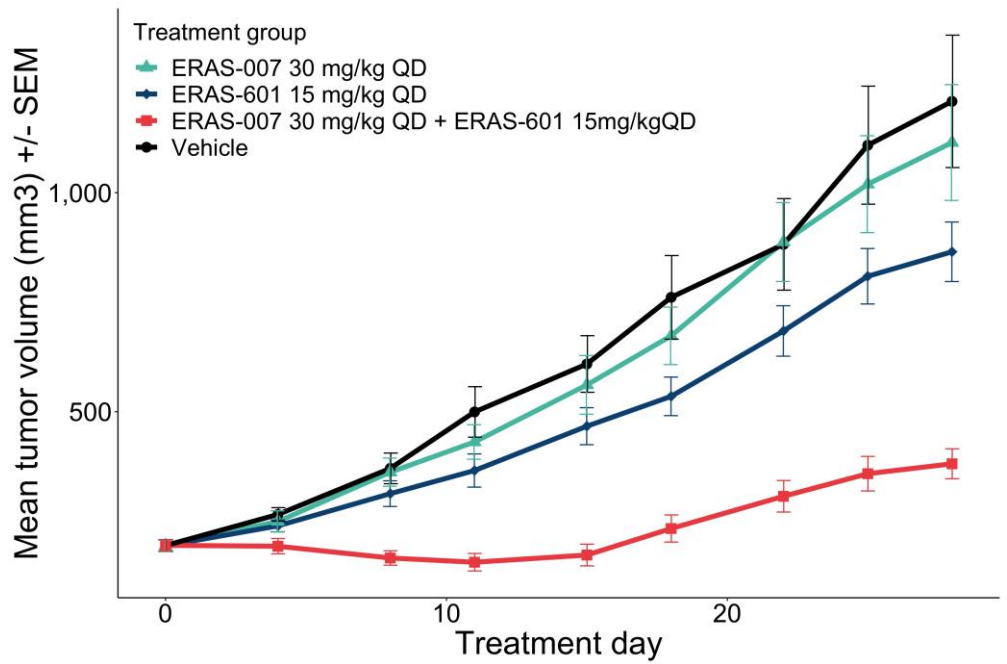
TGI in BRAF Class 3 NSCLC PDX LUN023



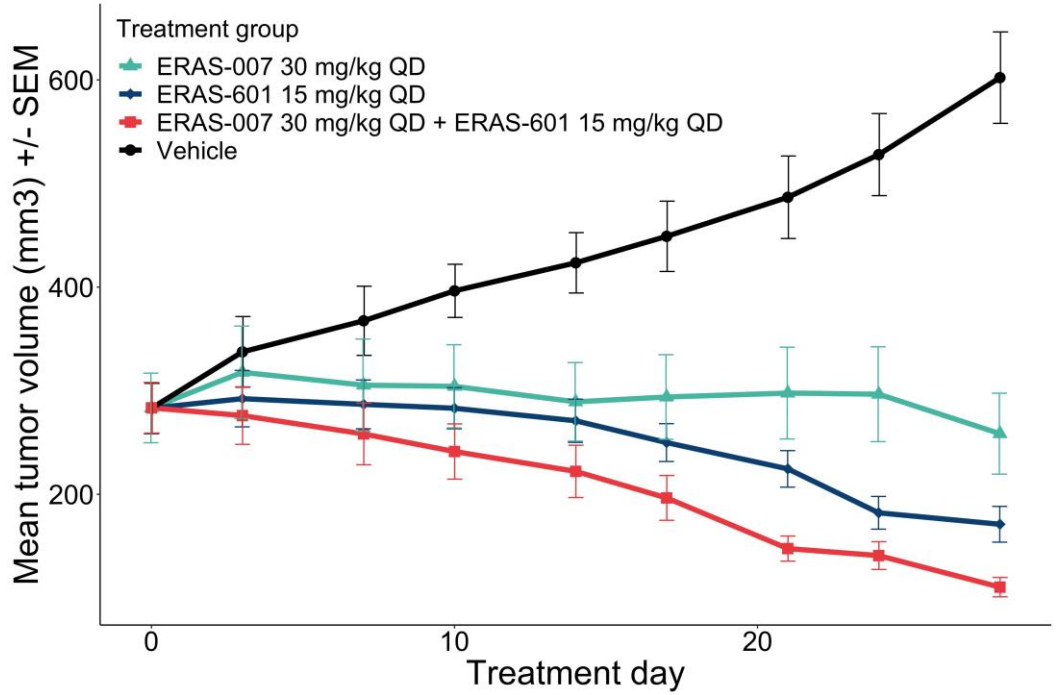
- MAPKlamp combination showed activity in both models and was tolerated in mice (e.g., no dose holidays, deaths, or euthanizations)
- ERAS-601 showed regression as a monotherapy in NCI-H508 while ERAS-007 showed regression as a monotherapy in LUN023
- ERAS-007 and ERAS-601 were dosed orally and continuously

ERAS-007 + ERAS-601 MAPKlamp showed promising combination activity
in two NF1 loss of function (LoF) models

TGI in NF1 LoF melanoma CDX MeWo



TGI in NF1 LoF NSCLC NCI-H1838



- MAPKlamp combination showed activity in both models and was tolerated in mice (e.g., no dose holidays, deaths, or euthanizations)
- ERAS-007 and ERAS-601 were dosed orally and continuously

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Are there more responsive subsets within the Blue Ocean where ERK + cell cycle or ERK + SHP2 inhibition can be particularly effective?

Preliminary clinical data

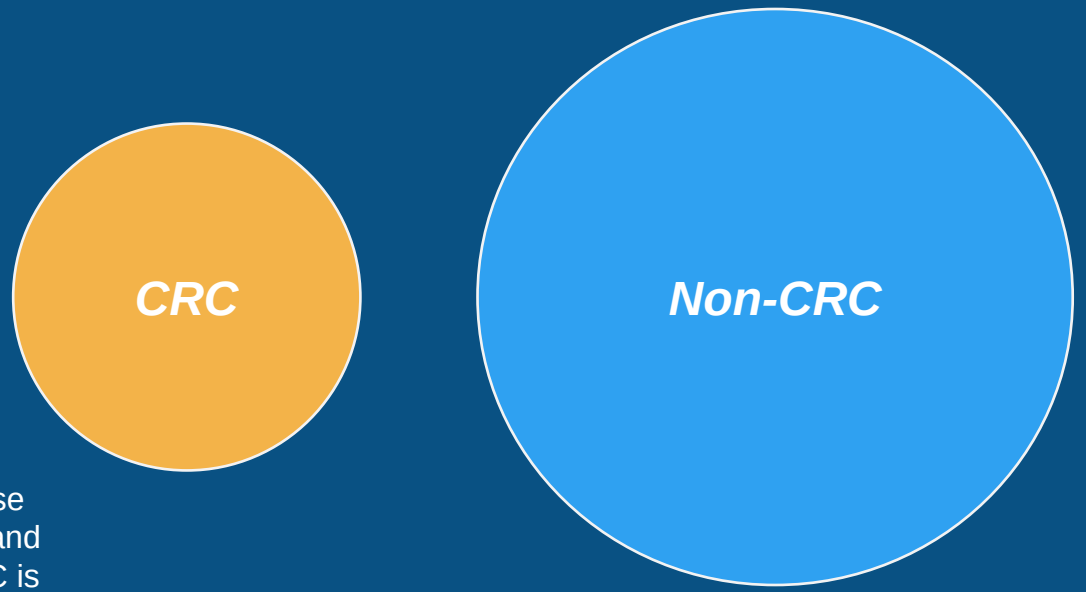
Segmentation framework to identify more responsive subsets within the Blue Ocean

for prioritized combination development

“Blue Ocean indications” of solid tumors with RAS/MAPK alterations and no approved targeted therapies

Less sensitive to monotherapy inhibition due to more RAS/MAPK reactivation and bypass pathway activation

Evaluation of combinations addressing these mechanisms is ongoing in HERKULES-3 (and combination addressing triple wildtype CRC is ongoing in FLAGSHP-1)



Phase 1/1b monotherapy responses used to inform prioritized combination development

Segmentation framework to identify more responsive subsets within the Blue Ocean

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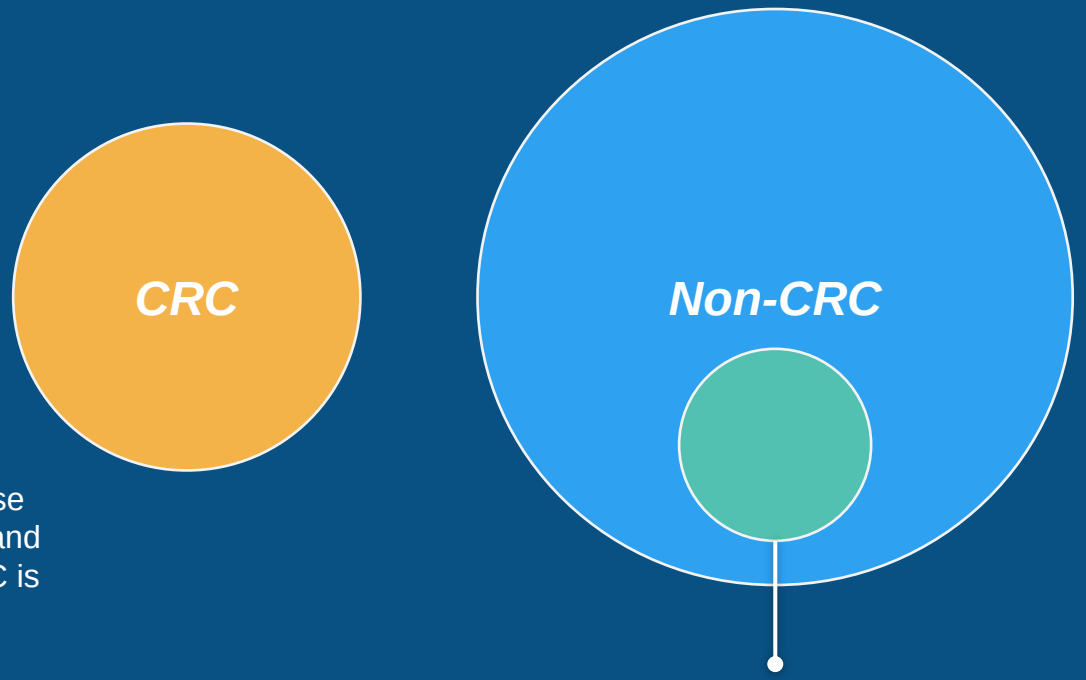

Phase 1/1b monotherapy responses used to inform prioritized combination development

Targetable, more responsive subset of patients prioritized for combination development

Methodology for retrospective pooled efficacy analysis for ERAS-007 and ERAS-601
in solid tumors with RAS/MAPK pathway alterations*

Retrospective Pooled Analysis	All trials assessing ERAS-007 or ERAS-601 as monotherapies: ASN007-101, HERKULES-1, FLAGSHP-1
Dosing Regimens	Biologically relevant regimens above the efficacious dose and at or below the maximum tolerated dose (MTD) for ERAS-007 and at or below the maximum administered dose (MAD) for ERAS-601 ERAS-007: weekly dose intensity between 120mg and 250mg, ERAS-601: daily dose intensity of 40mg
RAS/MAPK Alterations in Solid Tumors	**CRC:** Less sensitive to monotherapy inhibition due to more RAS/MAPK reactivation and bypass pathway activation **Non-CRC:** Less RAS/MAPK reactivation and no targeted therapies with full approval
Efficacy Evaluable Patients	Evaluable tumor assessment at baseline and at least one post dose tumor assessment Evaluated as per RECIST v1.1 by investigator

* The clinical data presented in the following slides are based on a retrospective analysis of pooled data across multiple clinical trials with different designs, inclusion criteria, and dosing regimens. Results across such clinical trials cannot be directly compared.

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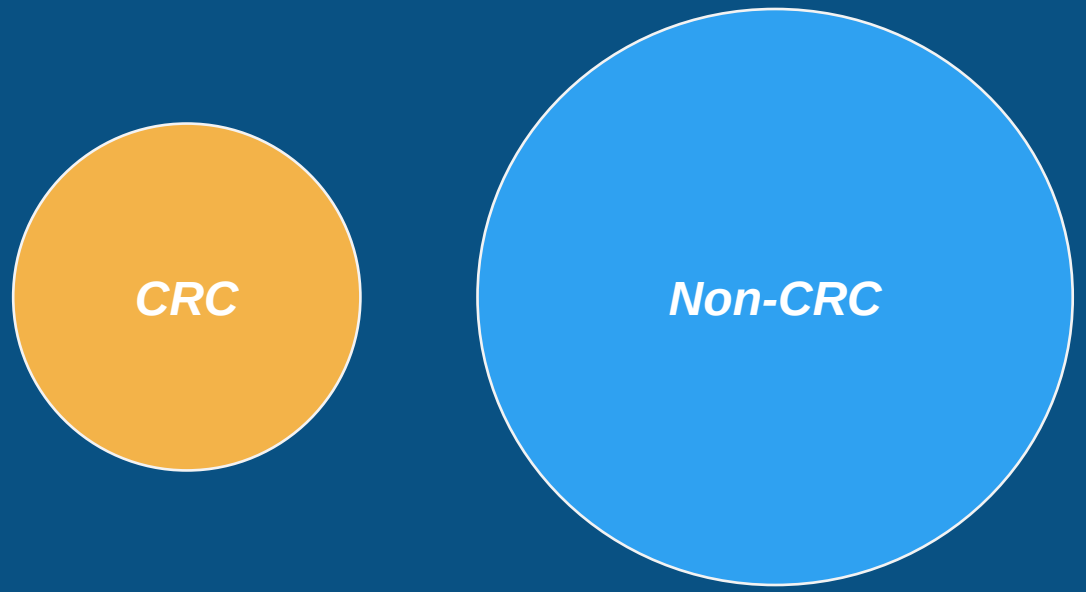
Preliminary responses in solid tumor subsets with RAS/MAPK alterations

“Blue Ocean indications” of solid tumors with RAS/MAPK alterations and no approved targeted therapies

- N = 8
- No single agent responses were observed
- Consistent with outcomes of other targeted therapies



- N = 26
- Single agent responses to either ERAS-007 or ERAS-601 were observed

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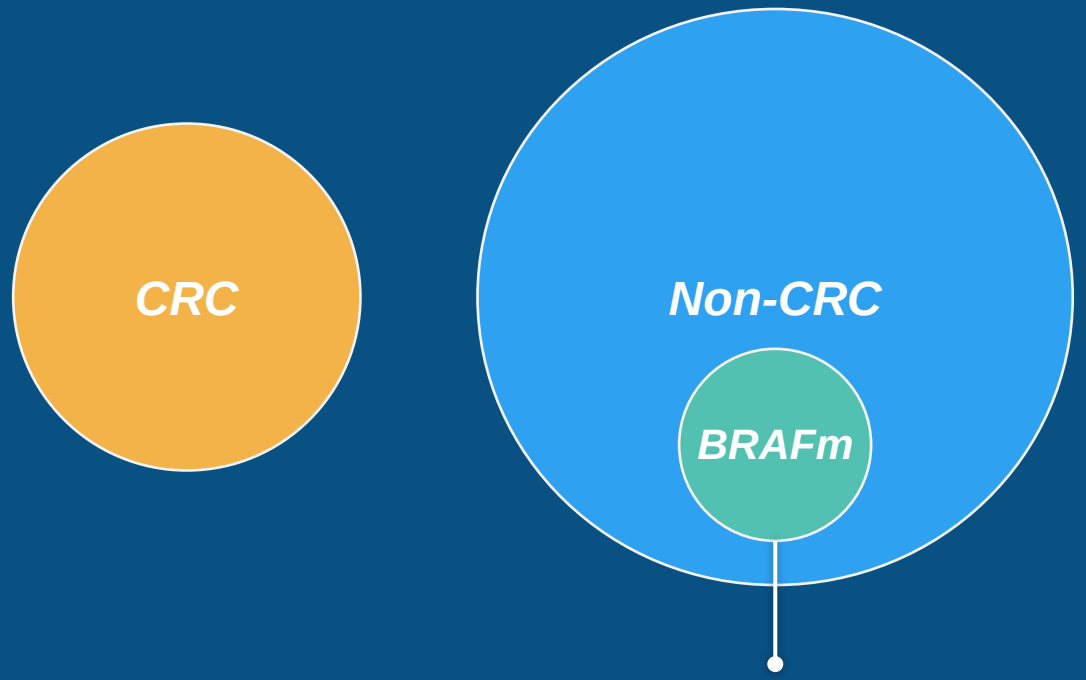
Preliminary responses in solid tumor subsets with RAS/MAPK alterations

“Blue Ocean indications” of solid tumors with RAS/MAPK alterations and no approved targeted therapies

- N = 8
- No single agent responses were observed
- Consistent with outcomes of other targeted therapies



- N = 26
- Single agent responses to either ERAS-007 or ERAS-601 were observed

- N = 9 (subset of the N = 26)
- Higher rate of single agent responses to either agent was observed

26 patients in 15 Blue Ocean Indications treated with biologically relevant doses of ERAS-007 or ERAS-601

Alterations	GBM	HNSCC	NSCLC	CRC	Melanoma	PDAC	Other solid tumors	AML
EGFR*/FLT3	125	513	184	338	-	-	-	61
NF1	25	58	98	35	33	1.9	8 434	3.2
KRAS G12C	-	2.8	240	57	-	5.0	9 45	0.1
KRAS G12D	0.2	4.7	68	238	0.5	6 178	200	1.2
Other KRAS	0.4	9.4	183	465	1.2	7 242	10 326	3.5
NRAS	0.5	8.4	11.7	72	3 71	1.0	11 116	13.8
HRAS	0.2	1 45	7.8	0.4	3.0	0.2	12 57	-
BRAF V600E/K	2	1.9	23	180	93	1.4	13 158	0.4
BRAF Class 2	0.4	3.8	2 17.6	6.9	4 5.3	0.5	14 58	-
BRAF Class 3	0.1	0.9	11.7	16.8	5 2.5	-	15 29	0.2
Other BRAF	-	-	3.9	-	1.9	0.3	0.5	-
MEK	0.2	1.9	11.7	8.8	4.6	0.2	22	-
Co-occurring activating MAPK pathway alterations**	1.4	10.3	62	59	37	7.1	84	3.0

Blue ocean indication		No. of patients
1.	HNSCC, HRAS	1
2.	NSCLC, BRAF Class 2	2
3.	Melanoma, NRAS	2
4.	Melanoma, BRAF Class 2	1
5.	Melanoma, BRAF Class 3	1
6.	PDAC, KRAS G12D	4
7.	PDAC, KRAS G12V	2
8.	Other solid tumors, NF1 LOF	1
9.	Other solid tumors, KRAS G12C	1
10.	Other solid tumors, Other KRAS	3
11.	Other solid tumors, NRAS	1
12.	Other solid tumors, HRAS	2
13.	Other solid tumors, BRAF V600E/K	1
14.	Other solid tumors, BRAF Class 2	3
15.	Other solid tumors, BRAF Class 3	1
		Total = 26

Blue ocean opportunities Red ocean opportunities

NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601 * Post-Osimertinib resistant population shown for EGFRm NSCLC except for SCLC transformation ** Co-occurring activating MAPK pathway alterations exclude EGFR overexpression

Source: SEER database (2020), ECIS database (2020), GLOBOCAN database (2020), The AACR Project GENIE Consortium version 8.1 (2020), TCGA Research Network: https://www.cancer.gov/tcga, Tyner JW et al. (2018) PMID: 30333627, Brenner CW et al (2013) PMID: 24120142, Chen J et al. (2020) PMID: 32015526, and Ostrom QT, et al. (2020) PMID: 33123732

ERASCA

6 confirmed and unconfirmed responses in 6 Blue Ocean Indications were observed with monotherapy ERAS-007 or ERAS-601

Alterations	GBM	HNSCC	NSCLC	CRC	Melanoma	PDAC	Other solid tumors	AML
EGFR*/FLT3	125	513	184	338	-	-	-	61
NF1	25	58	98	35	33	1.9	434	3.2
KRAS G12C	-	2.8	240	57	-	5.0	45	0.1
KRAS G12D	0.2	4.7	68	238	0.5	178	200	1.2
Other KRAS	0.4	9.4	183	465	1.2	7 242	326	3.5
NRAS	0.5	8.4	11.7	72	71	1.0	116	13.8
HRAS	0.2	1 45	7.8	0.4	3.0	0.2	57	-
BRAF V600E/K	2	1.9	23	180	93	1.4	13 158	0.4
BRAF Class 2	0.4	3.8	2 17.6	6.9	4 5.3	0.5	57.5	-
BRAF Class 3	0.1	0.9	11.7	16.8	2.5	-	15 29	0.2
Other BRAF	-	-	3.9	-	1.9	0.3	0.5	-
MEK	0.2	1.9	11.7	8.8	4.6	0.2	22	-
Co-occurring activating MAPK pathway alterations**	1.4	10.3	62	59	37	7.1	84	3.0

Blue ocean indication		No. of responses
1.	**HNSCC, HRAS**	**1 PR**
2.	**NSCLC, BRAF Class 2**	**1 uPR**
3.	Melanoma, NRAS	
4.	**Melanoma, BRAF Class 2**	**1 uPR**
5.	Melanoma, BRAF Class 3	
6.	PDAC, KRAS G12D	
7.	**PDAC, KRAS G12V**	**1 uPR**
8.	Other solid tumors, NF1 LOF	
9.	Other solid tumors, KRAS G12C	
10.	Other solid tumors, Other KRAS	
11.	Other solid tumors, NRAS	
12.	Other solid tumors, HRAS	
13.	**Other solid tumors, BRAF V600E/K**	**1 uPR**
14.	Other solid tumors, BRAF Class 2	
15.	**Other solid tumors, BRAF Class 3**	**1 PR**
		Total = 6 PRs/uPRs

Blue ocean opportunities Red ocean opportunities

NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601

* Post-Osimertinib resistant population shown for EGFRm NSCLC except for SCLC transformation

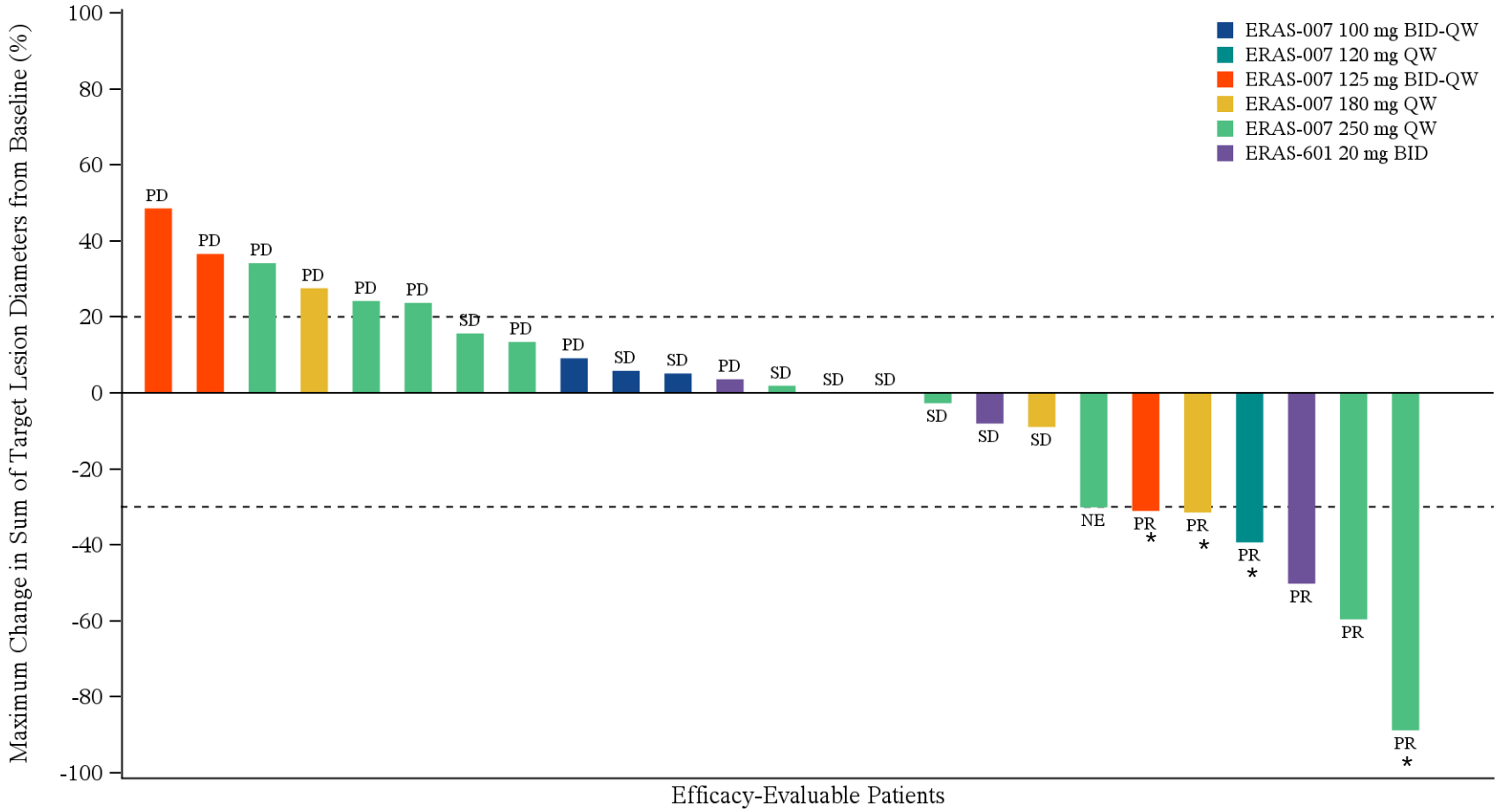
** Co-occurring activating MAPK pathway alterations exclude EGFR overexpression

Source: SEER database (2020), ECIS database (2020), GLOBOCAN database (2020), The AACR Project GENIE Consortium version 8.1 (2020), TCGA Research Network: https://www.cancer.gov/tcga, Tyner JW et al. (2018) PMID: 30333627, Brenner CW et al (2013) PMID: 24120142, Chen J et al. (2020) PMID: 32015526, and Ostrom QT, et al. (2020) PMID: 33123732

ERASCA

Best overall response with ERAS-007 or ERAS-601

in 15 RAS/MAPK-altered Blue Ocean Indications across lines of therapy



23% (6 out of 26) of patients responded (confirmed and unconfirmed PR) to single agent ERAS-007 or ERAS-601

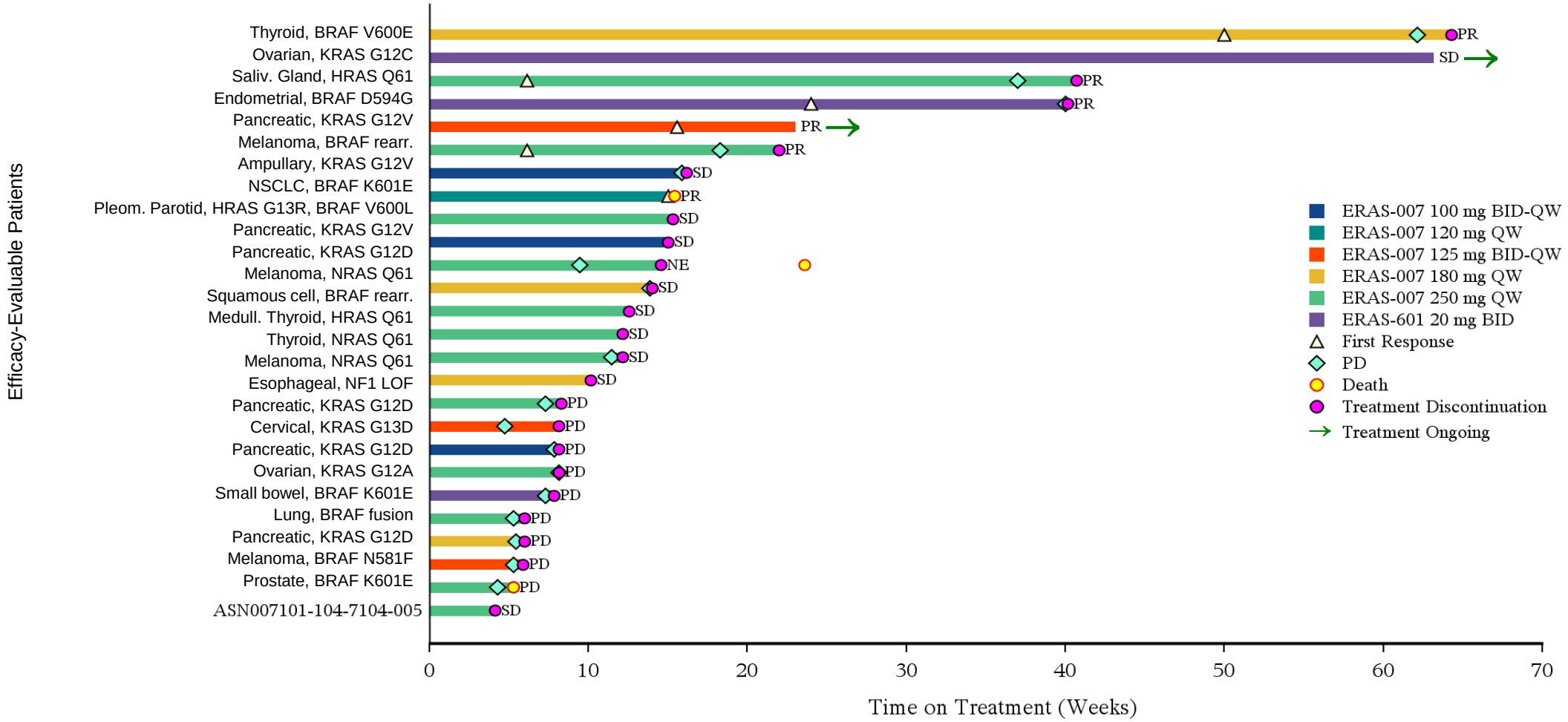
* Unconfirmed partial responses indicated with an asterisk.

NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01. One patient without measurable disease at baseline and at least one post baseline target lesion measurement was excluded from the waterfall plot

ERASCA

Duration of treatment observed with ERAS-007 or ERAS-601

in 15 RAS/MAPK-altered Blue Ocean Indications across lines of therapy



NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01.

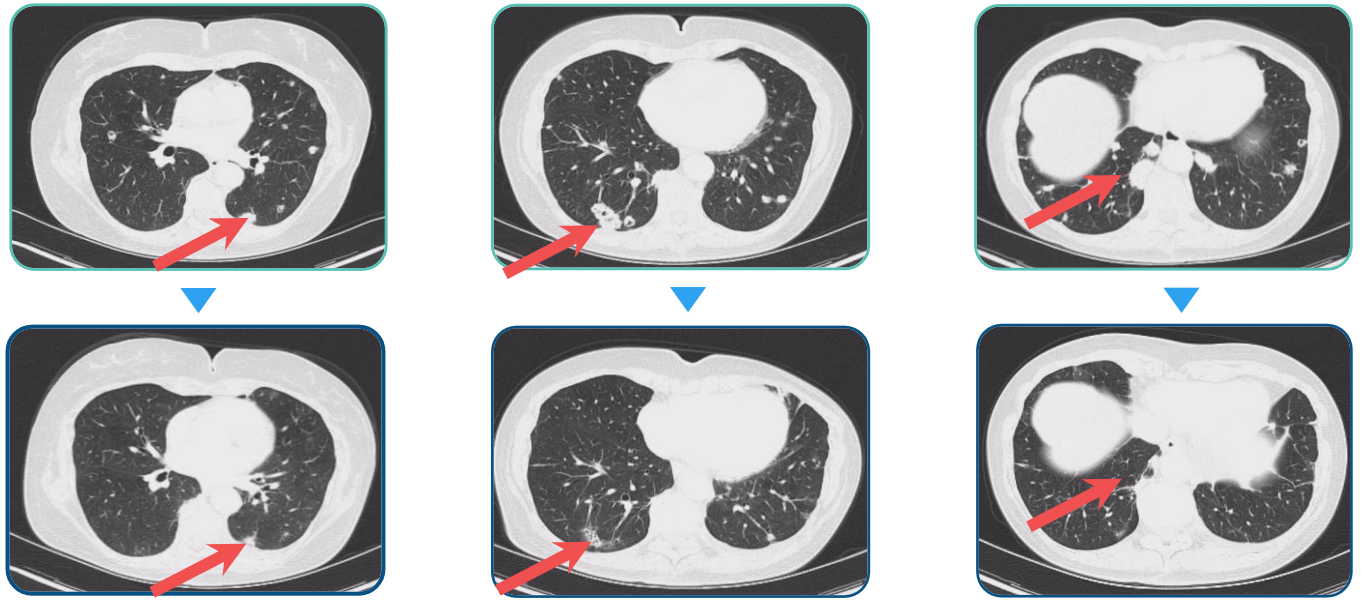
HERKULES-1 Case Study: Single agent ERAS-007 response

70-year-old female (Patient 0033) with KRAS G12V metastatic pancreatic cancer

Diagnosis	Stage II pancreatic cancer, metastatic disease, KRAS G12V, initially diagnosed in January 2018
Sites of Metastases	Lung, lymph nodes
Prior Therapy	Surgery, adjuvant radiation, gemcitabine/ capecitabine (#1); 5FU/oxaliplatin/irinotecan (#2); gemcitabine/abraxane (#3); 5FU/liposomal irinotecan (#4); alomfilimab (ICOS-targeted antibody)/atezolizumab (#5); MVT-5873 (anti-CA 19-9 antibody) (#6)
Dosing	ERAS-007 125 mg BID-QW

Baseline

16 Weeks



31% reduction
in tumor size

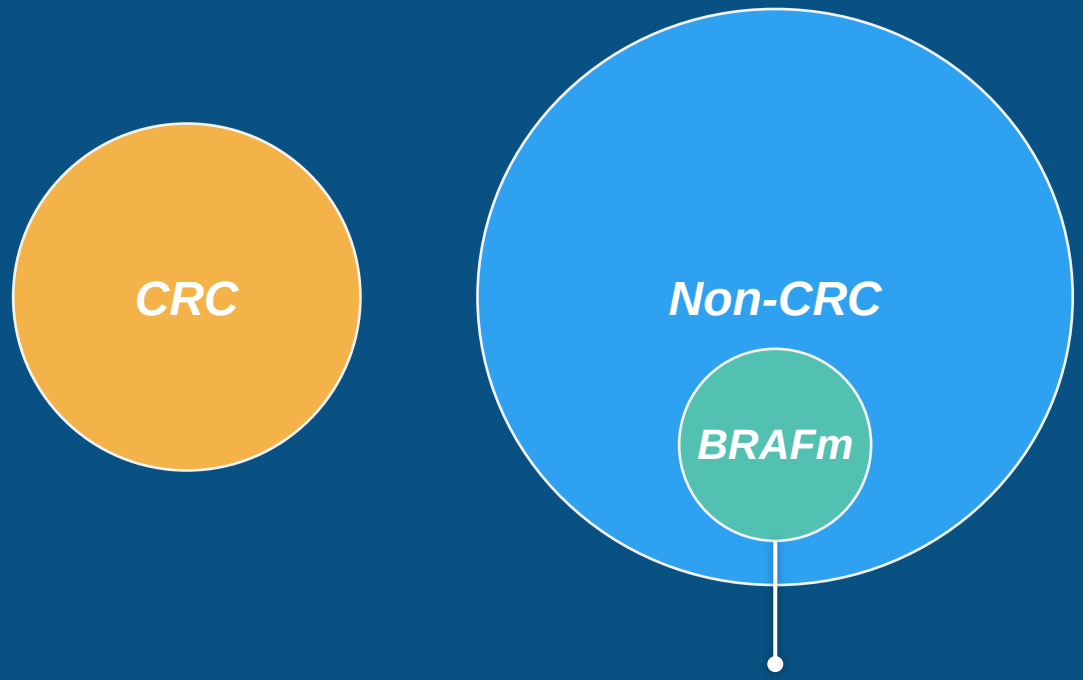
Per RECIST 1.1: ≥30% = objective response

Patient progressed with new lesion at subsequent assessment

ERASCA

Targetable, more responsive subset of patients with RAS/MAPK alterations

“Blue Ocean indications” of solid tumors with RAS/MAPK alterations and no approved targeted therapies

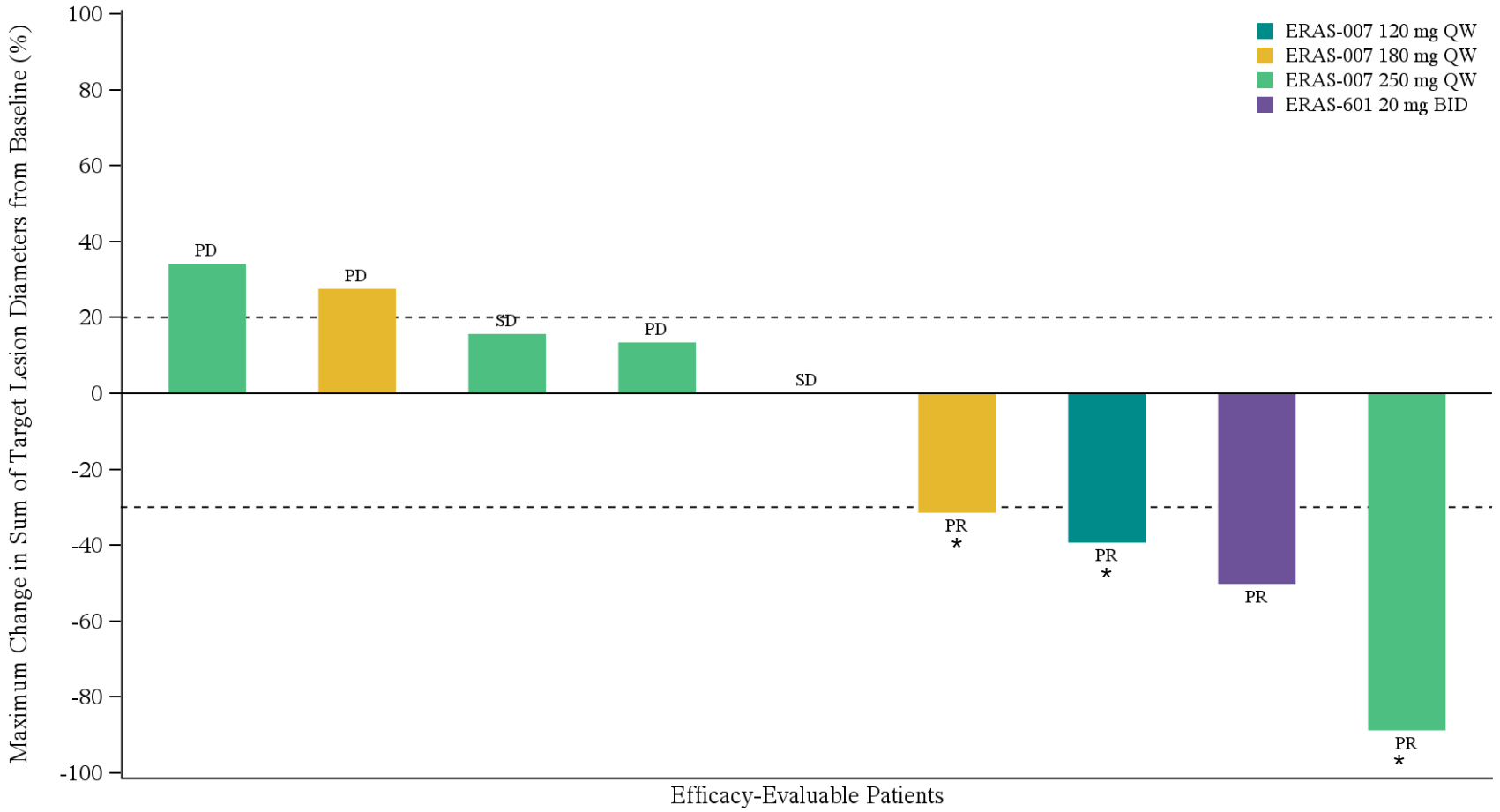


- N = 9 (subset of the N = 26)
- Higher rate of single agent responses to either agent was observed

ERASCA

Best Overall Response Observed with ERAS-007 or ERAS-601

in BRAF-driven Blue Ocean Indications across lines of therapy



44% (4 out of 9) of patients responded (confirmed and unconfirmed PR) to single agent ERAS-007 or ERAS-601

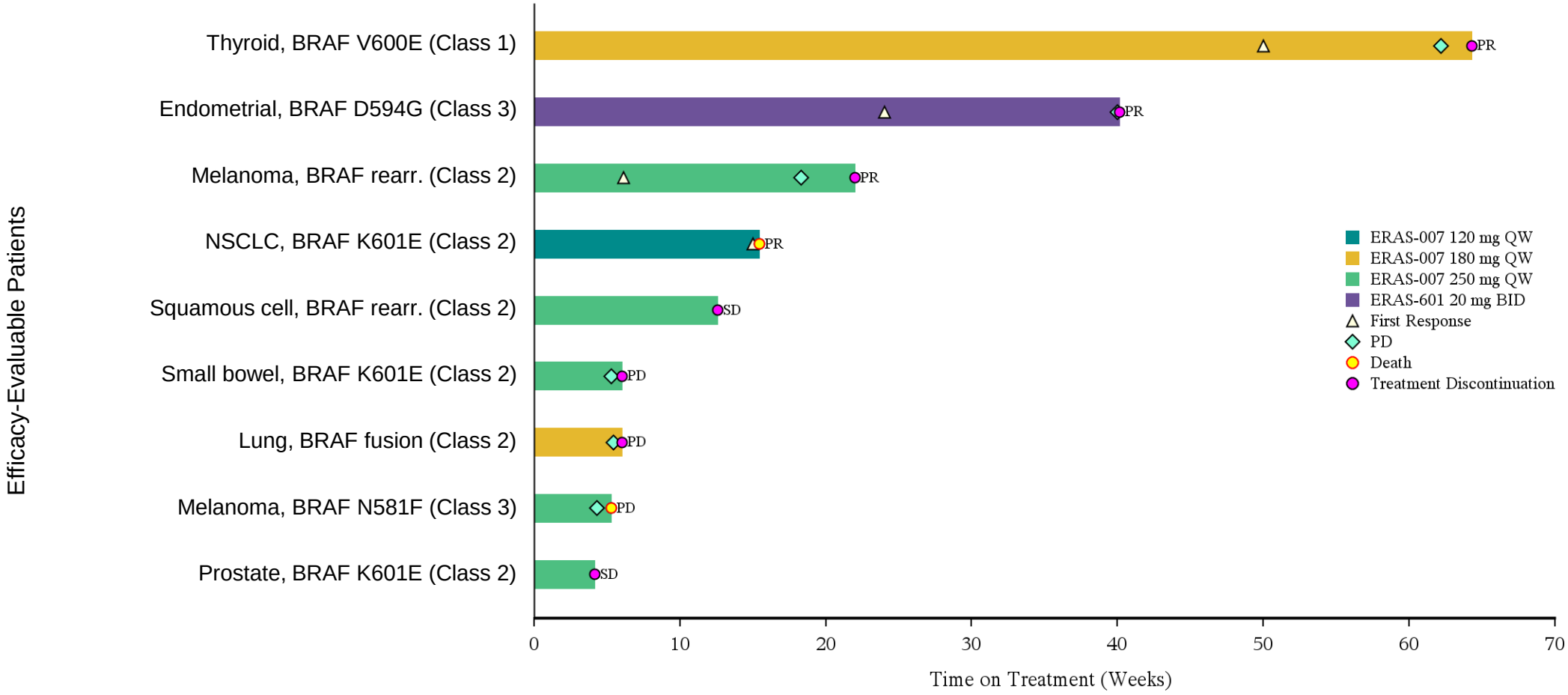
* Unconfirmed partial responses indicated with an asterisk

NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01.

ERASCA

Duration of Treatment Observed with ERAS-007 or ERAS-601

in BRAF-driven Blue Ocean Indications

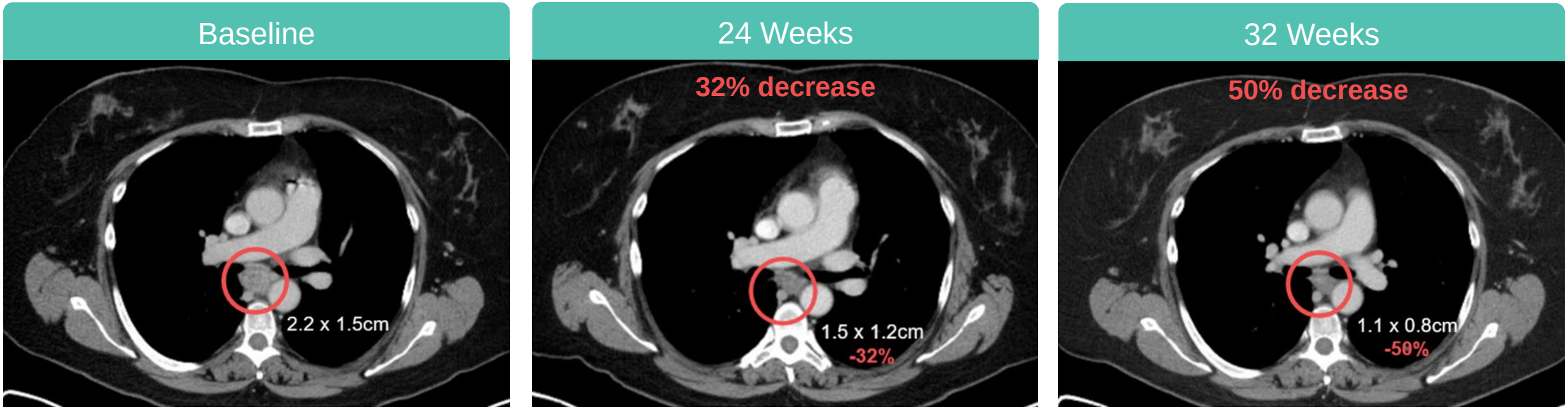


NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01.

FLAGSHP-1 Case Study: Single agent ERAS-601 response

63-year-old female (Patient 0009) with BRAF Class 3 metastatic endometrial cancer

Diagnosis	Stage III/IV endometrial cancer, metastatic disease, BRAF Class 3, initially diagnosed in September 2018
Sites of Metastases	Lung, lymph nodes
Prior Therapy	Surgery, chemotherapy, pembrolizumab
Dosing	ERAS-601 20 mg BID



Per RECIST 1.1: ≥30% = objective response

Tumor assessment (5) (Jan 4, 2022): patient had radiologic progressive disease (PD) due to a new lesion Peri-Esophageal lesion, shrinkage in non-target lesions also noted (not shown)

ERASCA

Are there more responsive subsets within the Blue Ocean where ERK + cell cycle or ERK + SHP2 inhibition can be particularly effective?

Non-CRC Solid Tumors

RAS/MAPK alterations

BRAF-driven RAS/MAPK alterations

- Single agent activity of ERAS-007 and ERAS-601 indicates responsiveness in certain tumor types and molecular alterations
- Combination approaches have potential to deepen responses and improve durability

Unmet needs in the 6 Blue Ocean Indications where single agent responses were observed comprise ~half a million patients worldwide per year

Alterations	GBM	HNSCC	NSCLC	CRC	Melanoma	PDAC	Other solid tumors	AML	US	EU	ROW	Global
EGFR*/FLT3	125	513	184	338	-	-	-	61	82	222	917	1,220
NF1	25	58	98	35	33	1.9	434	3.2	75	159	453	687
KRAS G12C	-	2.8	240	57	-	5.0	45	0.1	36	82	232	350
KRAS G12D	0.2	4.7	68	238	0.5	178	200	1.2	65	171	456	692
Other KRAS	0.4	9.4	183	465	1.2	7 242	326	3.5	114	299	817	1,230
NRAS	0.5	8.4	11.7	72	71	1.0	116	13.8	42	82	170	295
HRAS	0.2	1 45	7.8	0.4	3.0	0.2	57	-	11	24	80	114
BRAF V600E/K	2	1.9	23	180	93	1.4	13 158	0.4	63	127	271	461
BRAF Class 2	0.4	3.8	2 17.6	6.9	4 5.3	0.5	57.5	-	10.8	23.1	58	92
BRAF Class 3	0.1	0.9	11.7	16.8	2.5	-	15 29	0.2	6.1	14.8	40	61
Other BRAF	-	-	3.9	-	1.9	0.3	0.5	-	0.7	1.0	4.9	6.6
MEK	0.2	1.9	11.7	8.8	4.6	0.2	22	-	5	11	33	50
Co-occurring activating MAPK pathway alterations**	1.4	10.3	62	59	37	7.1	84	3.0	33	69	162	264

NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601

* Post-Osimertinib resistant population shown for EGFRm NSCLC except for SCLC transformation

** Co-occurring activating MAPK pathway alterations exclude EGFR overexpression

Blue ocean opportunities Red ocean opportunities

Source: SEER database (2020), ECIS database (2020), GLOBOCAN database (2020), The AACR Project GENIE Consortium version 8.1 (2020), TCGA Research Network: https://www.cancer.gov/tcga, Tyner JW et al. (2018) PMID: 30333627, Brenner CW et al (2013) PMID: 24120142, Chen J et al. (2020) PMID: 32015526, and Ostrom QT, et al. (2020) PMID: 33123732

ERASCA

Preliminary data support existing and new combination trials to help address majority of unmet needs in these Blue Ocean Indications

Alterations	GBM	HNSCC	NSCLC	CRC	Melanoma	PDAC	Other solid tumors	AML
EGFR*/FLT3	125	513	184	338	-	-	-	61
NF1	25	58	98	35	33	1.9	434	3.2
KRAS G12C	-	2.8	240	57	-	5.0	45	0.1
KRAS G12D	0.2	4.7	68	238	0.5	178	200	1.2
Other KRAS	0.4	9.4	183	465	1.2	7 242		
NRAS	0.5	8.4	11.7	72	71	1.0	116	13.8
HRAS	0.2	1 45	7.8	0.4	3.0	0.2	57	-
BRAF V600E/K	2	1.9	23	180	93	1.4	13 158	0.4
BRAF Class 2	0.4	3.8	2 17.6	6.9	4 5.3	0.5	57.5	-
BRAF Class 3	0.1	0.9	11.7	16.8	2.5	-	15 29	0.2
Other BRAF	-	-	3.9	-	1.9	0.3	0.5	-
MEK	0.2	1.9	11.7	8.8	4.6	0.2	22	-
Co-occurring activating MAPK pathway alterations**	1.4	10.3	62	59	37	7.1	84	3.0

HERKULES-3
007 + palbociclib

MAPKlamp trial:
Can combination of ERAS-007 + ERAS-601 achieve compelling ORR and duration of response to support tissue agnostic BRAF indication?

Blue ocean opportunities Red ocean opportunities

NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601
* Post-Osimertinib resistant population shown for EGFRm NSCLC except for SCLC transformation
** Co-occurring activating MAPK pathway alterations exclude EGFR overexpression
Source: SEER database (2020), ECIS database (2020), GLOBOCAN database (2020), The AACR Project GENIE Consortium version 8.1 (2020), TCGA Research Network: https://www.cancer.gov/tcga, Tyner JW et al. (2018) PMID: 30333627, Brenner CW et al (2013) PMID: 24120142, Chen J et al. (2020) PMID: 32015526, and Ostrom QT, et al. (2020) PMID: 33123732

ERASCA

Combinations of ERK inhibition with CDK4/6 or SHP2 inhibition offer two compelling approaches to targeting solid tumors with MAPK pathway alterations

ERAS-007 + ERAS-601 “MAPKlamp”

ERAS-007 + palbociclib

Opportunities	Challenges
Target alterations with no available direct inhibitors	Potential for increased overlapping toxicity
Achieve deeper pathway suppression than either agent alone	May not cover all mechanisms of resistance
May overcome some resistance mechanisms against individual agents	

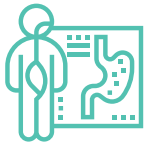
ERASCA

Can ERAS-007 be combined safely with palbociclib or ERAS-601 in patients?

Expected TRAEs associated with ERK and CDK4/6 inhibitors have been limited

Common ERKi TRAEs

Skin



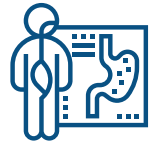
Gastrointestinal

Eye

General (fatigue)

Common CDK4/6i TRAEs

Hematologic



Gastrointestinal

Infections

General (fatigue)

TRAE: treatment-related adverse event

ERAS-007 combination with palbociclib is enrolling and is currently at a biologically relevant dose level

HERKULES-3

GI Cancer

Regimen	Indication	Status
ERAS-007 + palbociclib	KRAS- or NRAS-mutant CRC; KRAS-mutant PDAC	Dose escalation ongoing Currently dosing ERAS-007 100 mg BID-QW + 100 mg QD palbociclib

BID-QW: twice a day on a single day each week. QD: once daily.

Expected TRAEs associated with SHP2 and ERK inhibitors have been limited

Common SHP2i TRAEs

Hematologic

Cardiovascular

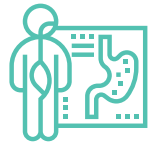
Hepatic

Gastrointestinal

General
(peripheral edema)

Common ERKi TRAEs

Skin



Gastrointestinal

Eye

General (fatigue)

TRAE: treatment-related adverse event

TRAEs of ERAS-601 and ERAS-007 have been largely non-overlapping

ERAS-601 and ERAS-007 by common SHP2i TRAEs

Treatment-related AEs in Preferred Terms	ERAS-601 20 and 40 mg BID (N=13)		ERAS-007 50-125mg BID-QW (N=23)	
	All Grade	Gr ≥ 3	All Grade	Gr ≥ 3
HEMATOLOGIC				
Thrombocytopenia*	3 (23.1%)	2 (15.4%)	0	0
Anemia	3 (23.1%)	1 (7.7%)	1 (4.3%)	1 (4.3%)
CARDIOVASCULAR				
Hypertension	3 (23.1%)	1 (7.7%)	0	0
Hypertensive encephalopathy	1 (7.7%)	1 (7.7%)	0	0
HEPATIC				
AST increase	2 (15.4%)	1 (7.7%)	0	0
ALT increase	2 (15.4%)	0	0	0
Blood bilirubin increased	0	0	1 (4.3%)	1 (4.3%)
GENERAL				
Peripheral edema	4 (30.8%)	0	1 (4.3%)	0

Gr 4 AEs:
ERAS-601: anemia, hypertensive encephalopathy
ERAS-007: none

- Data cut off for FLAGSHP-1: 11JUL2022 & for HERKULES-1: 23May2022
- In this table is reported the number of patients who experienced the reported AE at the highest grade.
- TRAEs included in this table met at least one of the following criteria: (1) experienced by ≥ 2 patients in either the 20 and 40 mg BID treatment group for ERAS-601 **OR** the 50-125 mg BID-QW column for ERAS-007; (2) experienced by at least 1 patient and Grade ≥3.
- *includes platelets count decrease

ERAS-601 and ERAS-007 by common ERKi TRAEs

Treatment-related AEs in Preferred Terms	ERAS-601 20 and 40 mg BID (N=13)		ERAS-007 50-125mg BID-QW (N=23)	
	All Grade	Gr ≥ 3	All Grade	Gr ≥ 3
SKIN				
Maculopapular rash	0	0	2 (8.7%)	0
Dermatitis acneiform	2 (15.4%)	0	8 (34.8%)	0
EYE DISORDERS				
Blurred vision	2 (15.4%)	0	5 (21.7%)	1 (4.3%)
Retinopathy	0	0	6 (26.1%)	0
Retinal Detachment	0	0	1 (4.3%)	1 (4.3%)
Vision Impairment	0	0	1 (4.3%)	1 (4.3%)
GASTROINTESTINAL				
Nausea	0	0	12 (52.2%)	0
Vomiting	0	0	7 (30.4%)	0
Diarrhea	5 (38.5%)	1 (7.7%)	5 (21.7%)	0
Constipation	0	0	2 (8.7%)	0
Dyspepsia			2 (8.7%)	0
GENERAL				
Fatigue	1 (7.7%)	0	9 (39.1%)	2 (8.7%)
Dehydration	0	0	4 (17.4%)	0
Dizziness	0	0	2 (8.7%)	0

Potential overlapping tox; can be managed proactively

Likely recommended dose of ERAS-007 for combinations was well tolerated

Treatment-related Adverse Events Occurring in ≥ 20% and ≥ 2 Patients at Any Dose

(arranged by descending frequency in the 250mg QW any grade column)

System Organ Class/ Preferred Term	50 mg BID-QW (n=4)		100 mg BID-QW (n=11)		125 mg BID-QW (n=8)		250 mg QW (n=29)	
	Any Grade	Grade ≥ 3	Any Grade	Grade ≥ 3	Any Grade	Grade ≥ 3	Any Grade	Grade ≥ 3
At least one TRAE	4 (100.0%)	1 (25.0%)	9 (81.8%)	2 (18.2%)	8 (100.0%)	3 (37.5%)	27 (93.1%)	10 (34.5%)
Eye Disorders*	1 (25.0%)	0	6 (54.5%)	1 (9.1%)	5 (62.5%)	2 (25.0%)	16 (55.2%)	5 (17.2%)
Diarrhea	0	0	2 (18.2%)	0	3 (37.5%)	0	16 (55.2%)	1 (3.4%)
Nausea	2 (50.0%)	0	5 (45.5%)	0	5 (62.5%)	0	14 (48.3%)	0
Vomiting	1 (25.0%)	0	3 (27.3%)	0	3 (37.5%)	0	9 (31.0%)	2 (6.9%)
Dermatitis acneiform	1 (25.0%)	0	4 (36.4%)	0	3 (37.5%)	0	6 (20.7%)	0
Rash maculopapular	0	0	1 (9.1%)	0	1 (12.5%)	0	6 (20.7%)	1 (3.4%)
Dehydration	2 (50.0%)	0	1 (9.1%)	0	1 (12.5%)	0	4 (13.8%)	0
Fatigue	1 (25.0%)	1 (25.0%)	4 (36.4%)	0	4 (50.0%)	1 (12.5%)	5 (17.2%)	1 (3.4%)

Likely recommended dose between 50 – 100mg BID-QW for combinations was well tolerated

*includes uniocular blindness (one patient in 250mg QW cohort), chorioretinopathy, papilloedema, retinal detachment, retinal oedema, retinopathy, serous retinal detachment, subretinal fluid, vision blurred, visual impairment, and vitreous floaters. Data extraction for ASN-007-101 was on 6 Nov. 2020; data cutoff for HERKULES-1 was 23 May 2022

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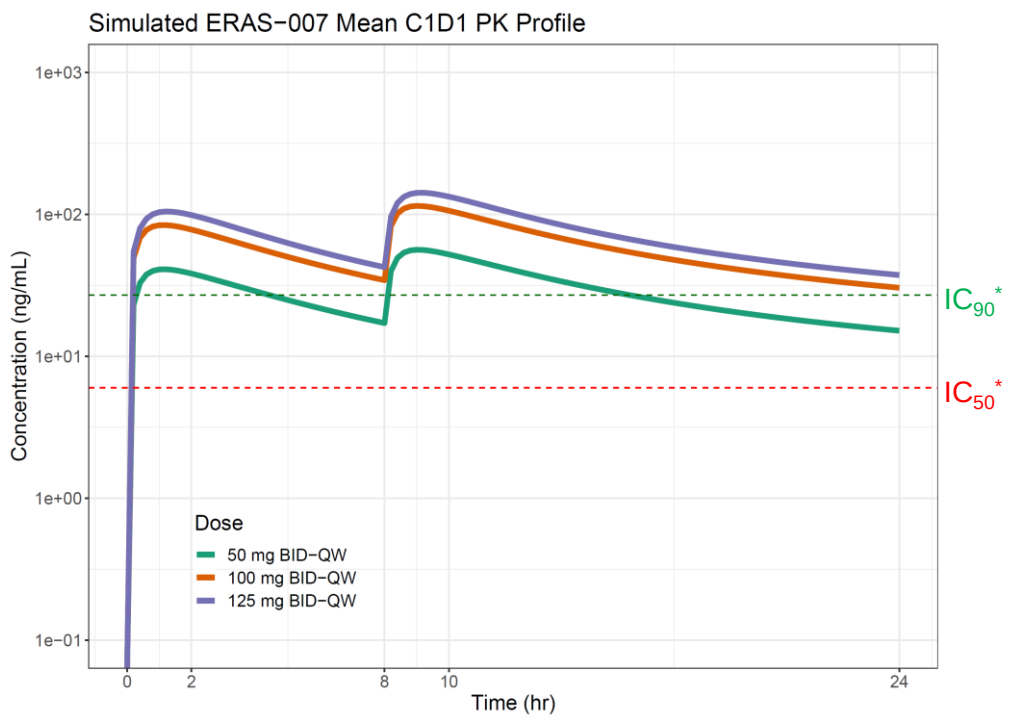
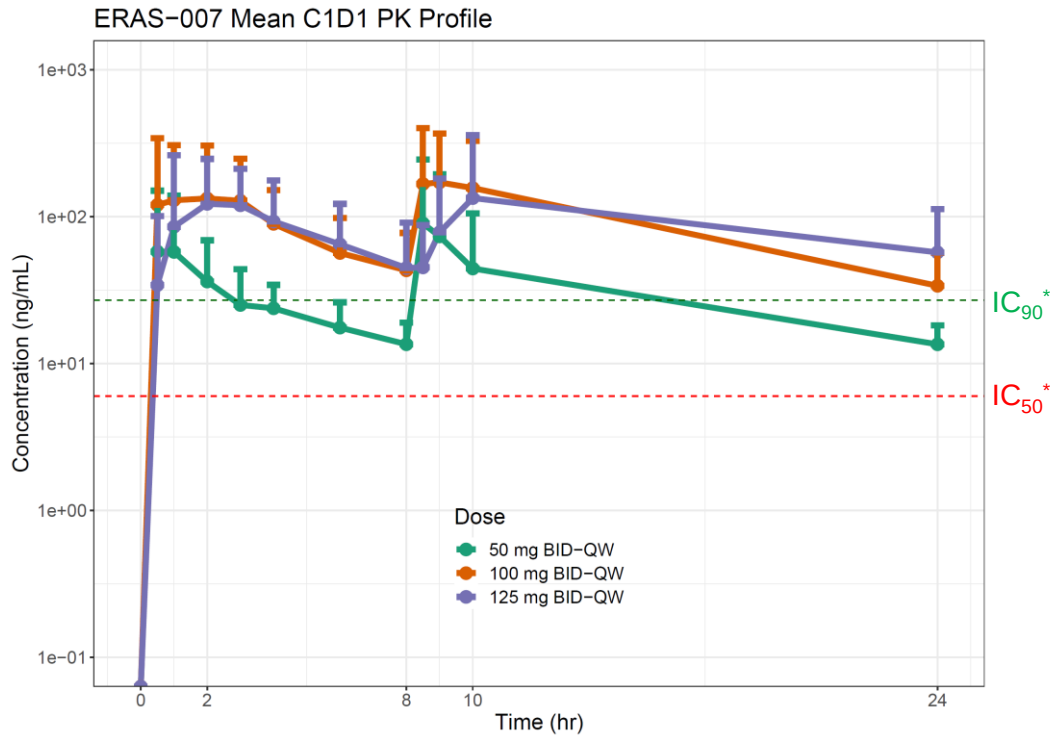
Will ERAS-601 and ERAS-007 provide adequate target inhibition at the combination dose and schedule?

HERKULES-1: Preliminary observed PK characteristics of ERAS-007 have been generally aligned with model prediction

Model-Based Simulated PK Profile



Observed PK Profile

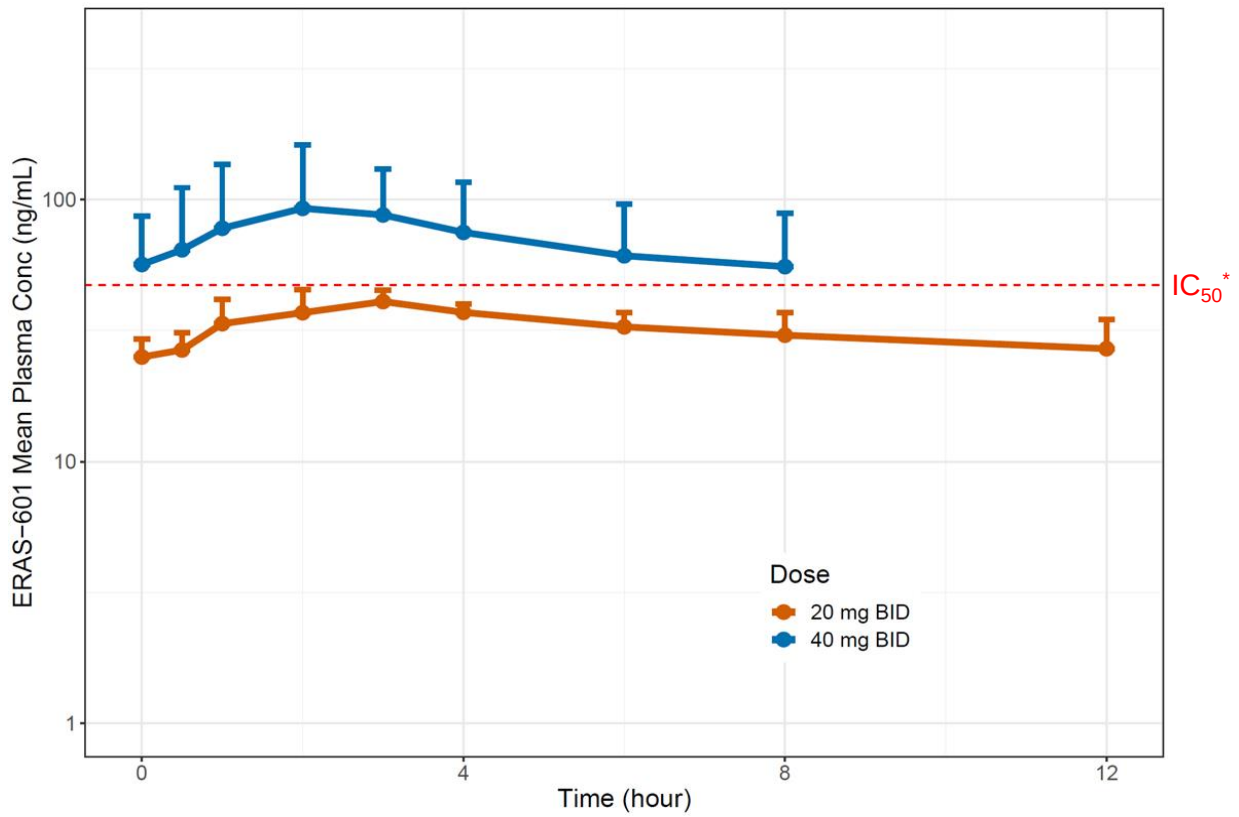


- In general, ERAS-007 PK exposure at 100mg and 125mg BID-QW had comparable IC90 coverage compared to that of 250mg QW, but with lower Cmax

*HCT 116 anti-proliferation assay

FLAGSHP-1: ERAS-601 showed well behaved PK characteristics

ERAS-601 Steady-State Mean PK Profiles



Rapid absorption (median T_{max} < 4h post dose)

Estimated terminal half-life: ~15-22h

PK exposure increased in a dose-dependent manner

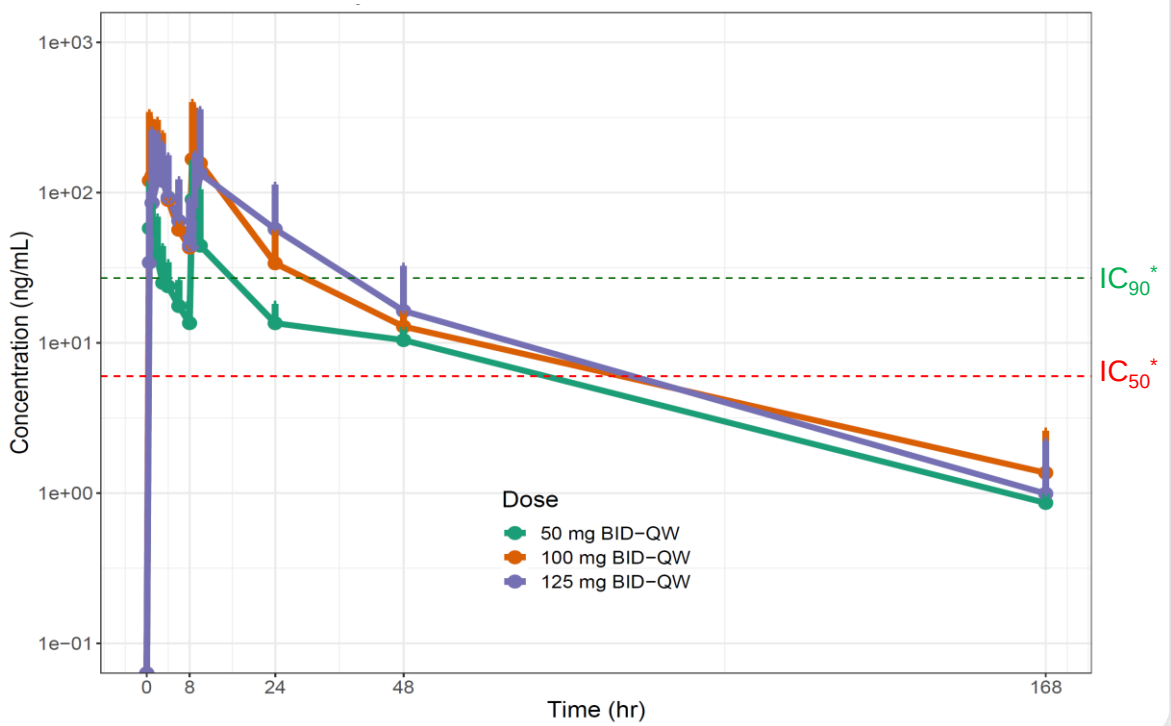
At 40mg BID, steady-state mean exposure exceeded pERK IC_{50} (NCI-H358) throughout the dosing interval

*pERK in NCI-H358

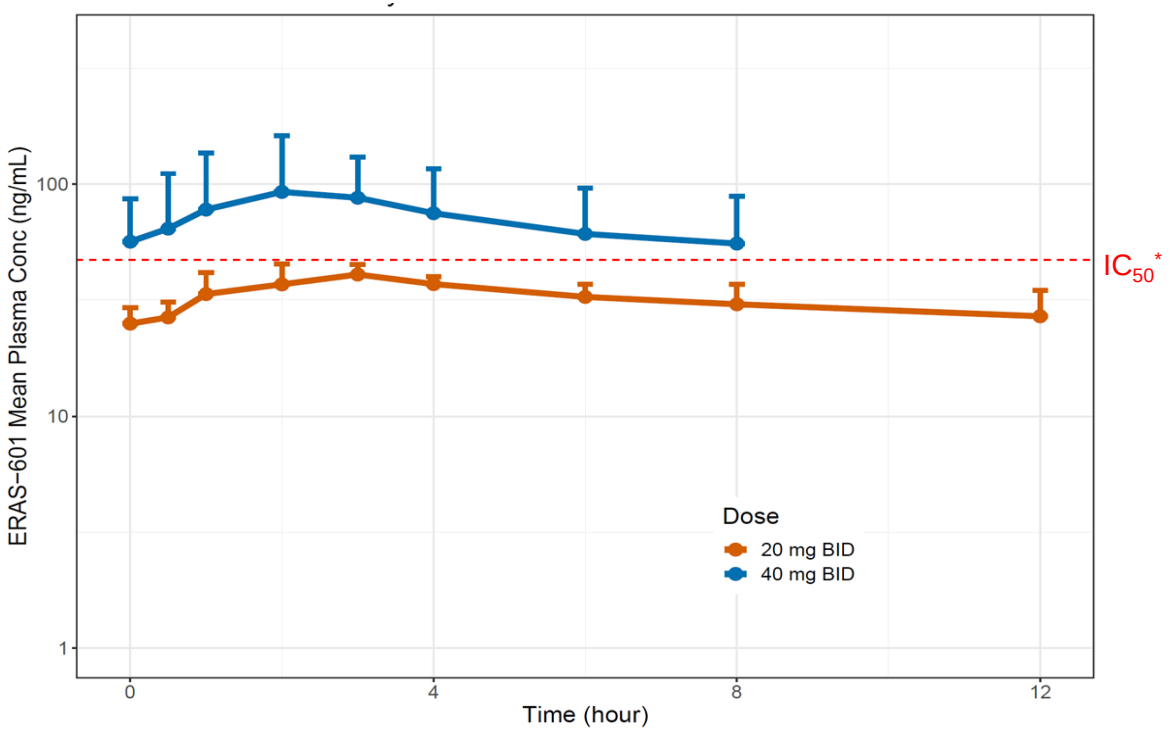
Source: FLAGSHP-1 trial

ERAS-007 and ERAS-601 use different target coverage strategies that seek to achieve optimal efficacy and safety

ERAS-007 Mean Cycle 1 PK Profile



ERAS-601 Mean Steady State PK Profile

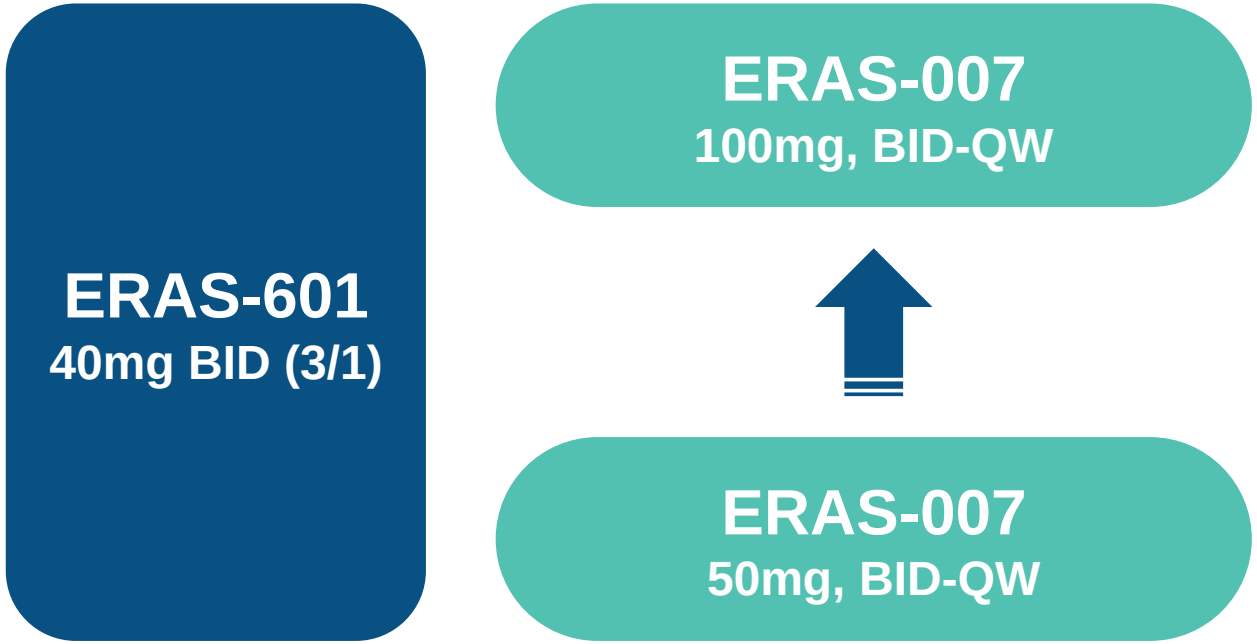


ERAS-007: 50-125mg BID-QW dosing provided high target coverage ($C>IC_{90}$) for maximum activity, followed by lower PK coverage ($C<IC_{50}$) for MAPK pathway recovery to alleviate target driven toxicity

ERAS-601: 40mg BID dosing provided sustained target coverage ($C>IC_{50}$) throughout the dosing interval

*HCT 116 anti-proliferation assay for ERAS-007; pERK in NCI-358 for ERAS-601

Proposed MAPKlamp dose escalation in HERKULES-1 is designed to maximize target inhibition and optimize risk/benefit for patients



BID (3/1): twice a day, three weeks on, one week off
BID-QW: twice a day on a single day each week

Erasca's tissue specific and tissue agnostic trials target multiple indications

Pooled analysis has sharpened the focus of MAPKlamp trial on BRAF-driven solid tumors

Alterations	GBM	HNSCC	NSCLC	CRC	Melanoma	PDAC	Other solid tumors	AML
EGF	125	513	184			-	-	61
NF1	25	58	98			1.9	434	3.2
KRAS G12C	-	2.8	240			5.0	45	0.1
KRAS G12D	0.2			238	0.5	178		
Other KRAS	0.4			465	1.2	242		
NRAS	0.5	8.4	11.7	72	71	1.0	116	13.8
HRAS	0.2	45	7.8	0.4				-
BRAF V600E/K	2	1.9	23	180				0.4
BRAF Class 2	0.4	3.8	17.6	6.9	5.3	0.5	58	-
BRAF Class 3	0.1	0.9	11.7	16.8	2.5	-	29	0.2
Other BRAF	-	-	3.9	-	1.9	0.3	0.5	-
MEK	0.2	1.9	11.7	8.8	4.6	0.2	22	-
Co-occurring activating MAPK pathway alterations**	1.4	10.3	62	59	37	7.1	84	3.0

THUNDERBBOLT-1
801

HERKULES-2
007 + osimertinib

HERKULES-2
007 or 601 + sotorasib

HERKULES-3
007 + palbociclib

HERKULES-3
007 + palbociclib

HERKULES-3
007 + encorafenib & cetuximab (EC)

FLAGSHP-1
601 + cetuximab (triple WT CRC[1] and HPV- HNSCC)

HERKULES-1 (dose escalation) and MAPKlamp trial (dose expansion):
ERAS-007 + ERAS-601 combination

Blue ocean opportunities Red ocean opportunities

NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601

[1] Triple wildtype CRC is KRASwt, NRASwt, and BRAFwt

* Post-Osimertinib resistant population shown for EGFRm NSCLC except for SCLC transformation

** Co-occurring activating MAPK pathway alterations exclude EGFR overexpression

Source: SEER database (2020), ECIS database (2020), GLOBOCAN database (2020), The AACR Project GENIE Consortium version 8.1 (2020), TCGA Research Network: https://www.cancer.gov/tcga, Tyner JW et al. (2018) PMID: 30333627, Brennel CW et al. (2013) PMID: 24120142, Chen J et al. (2020) PMID: 32015526, and Ostrem JM et al. (2020) PMID: 33128732

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Discussant:

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THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History®

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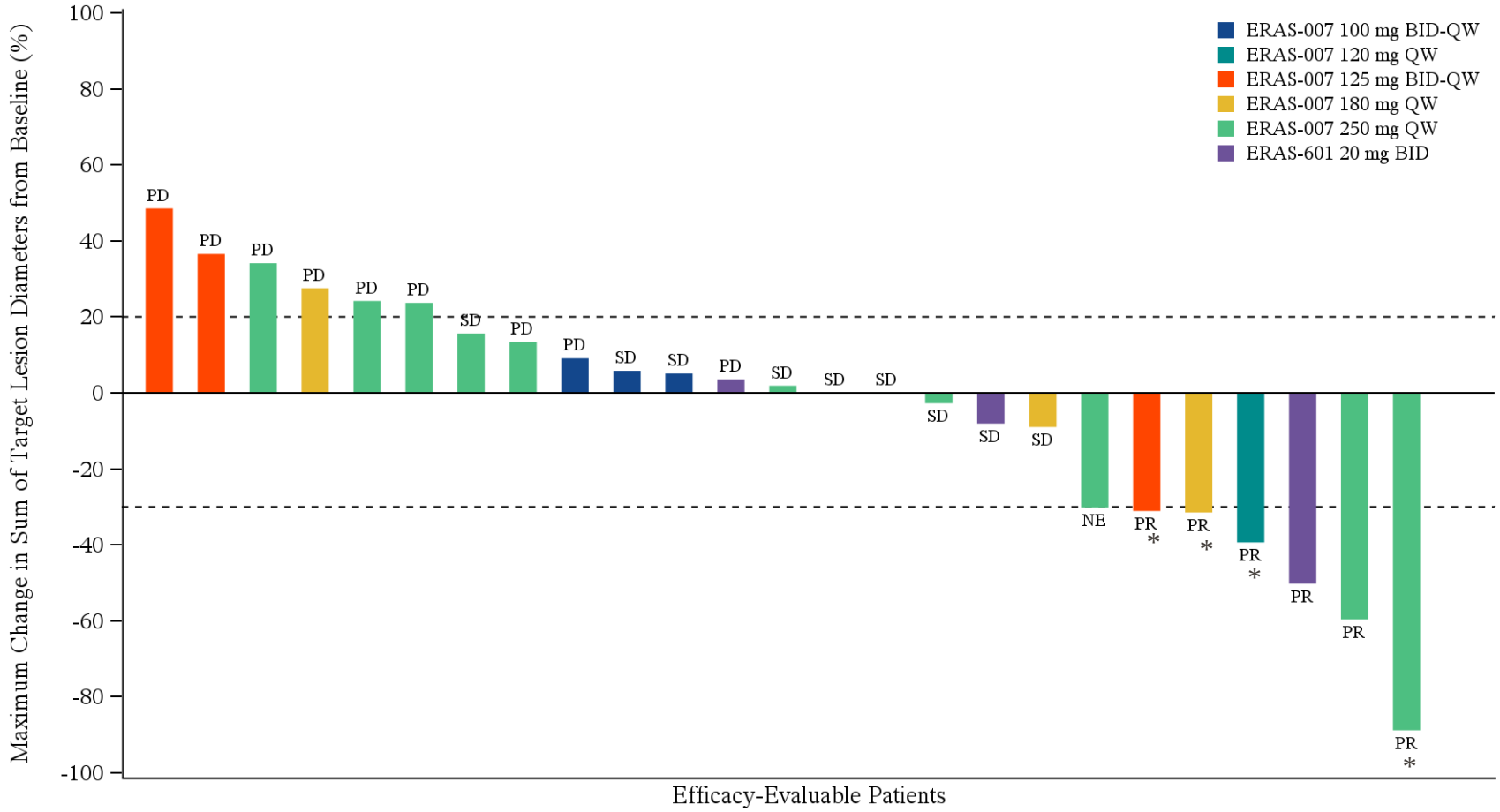
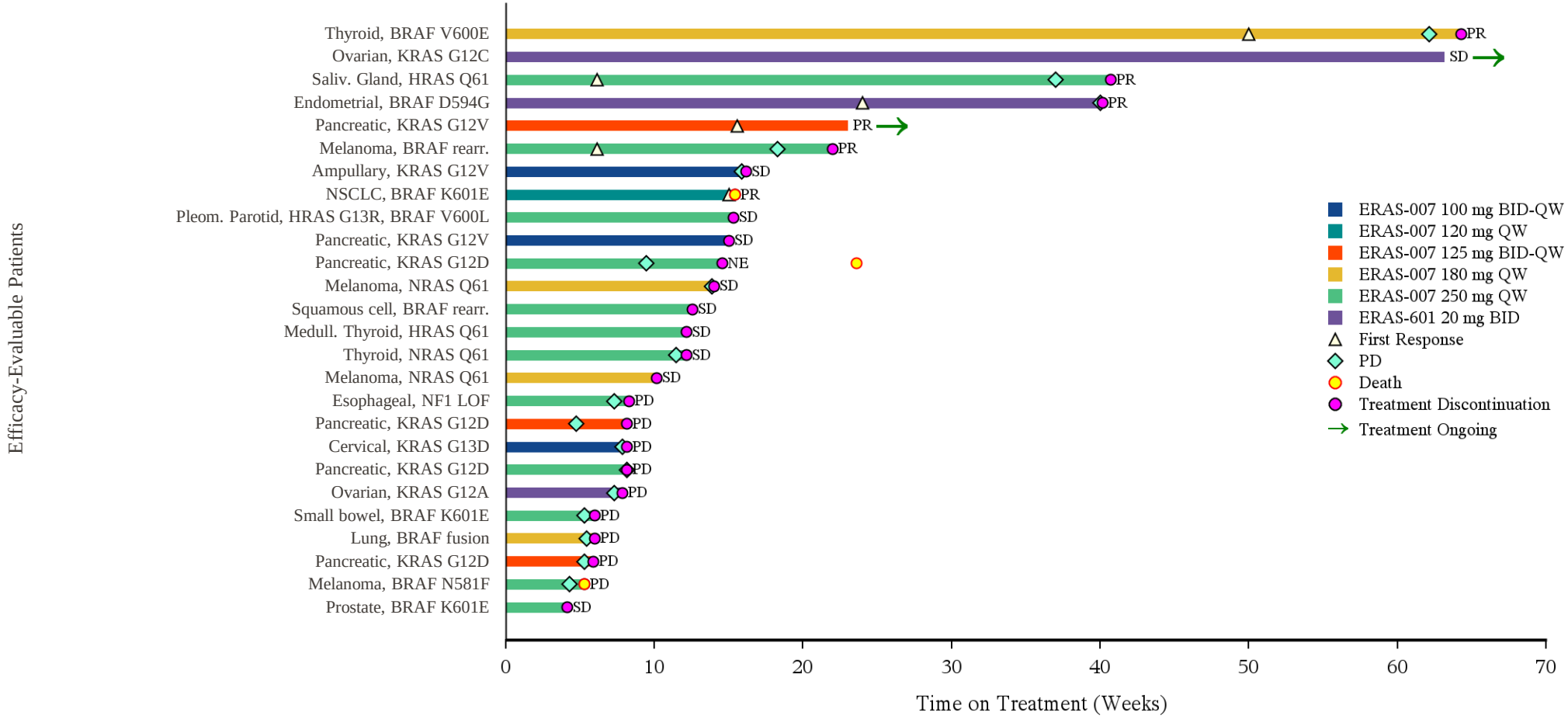
Best overall response with ERAS-007 or ERAS-601 in 15 RAS/MAPK-altered Blue Ocean Indications across lines of therapy



23% (6 out of 26) of patients responded (2 confirmed and 4 unconfirmed PR) to single agent ERAS-007 or ERAS-601

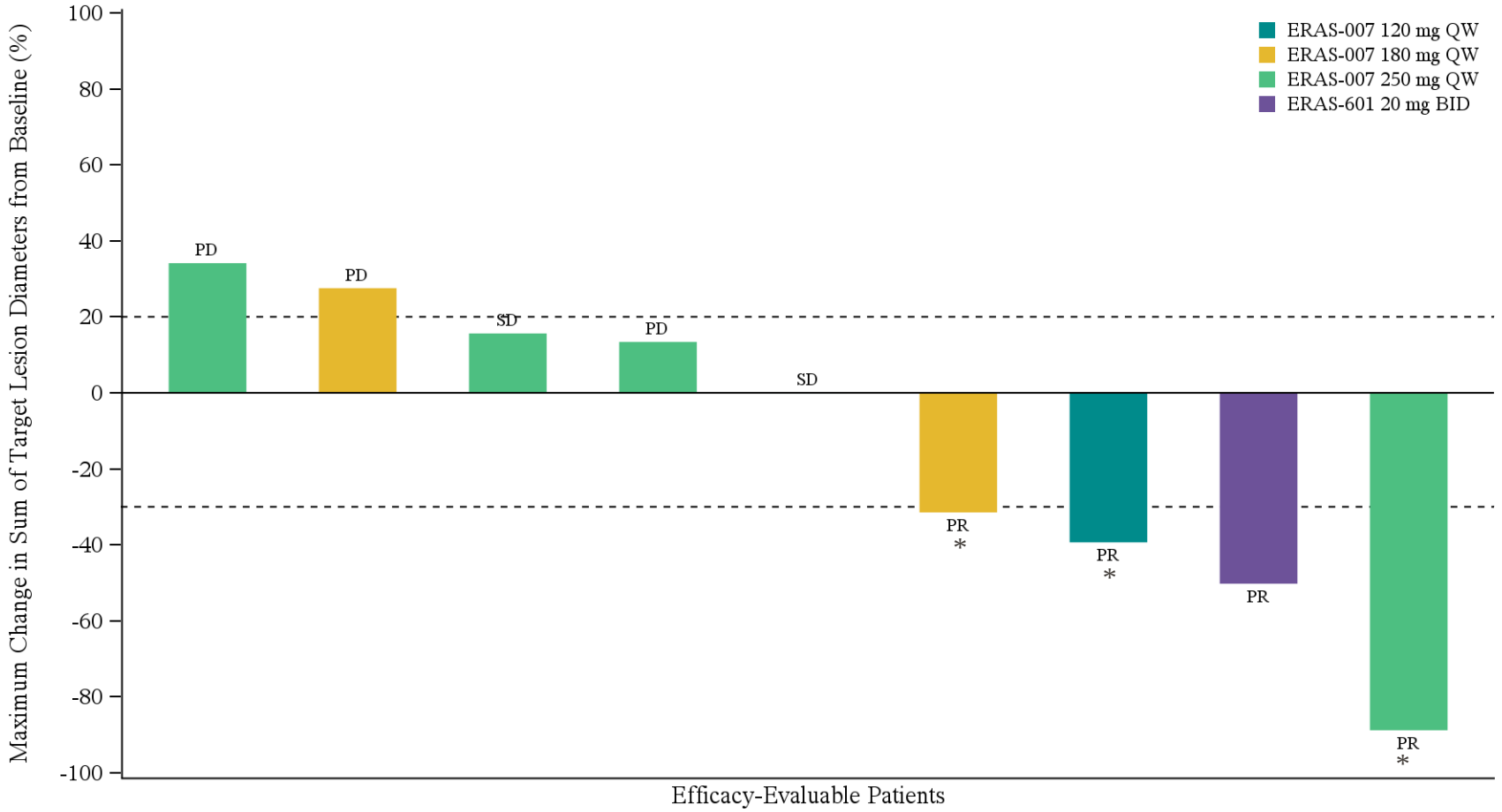
* Unconfirmed partial responses indicated with an asterisk.
NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01. One patient without measurable disease at baseline and at least one post baseline target lesion measurement was excluded from the waterfall plot

Duration of treatment observed with ERAS-007 or ERAS-601 in 15 RAS/MAPK-altered Blue Ocean Indications across lines of therapy



NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01.

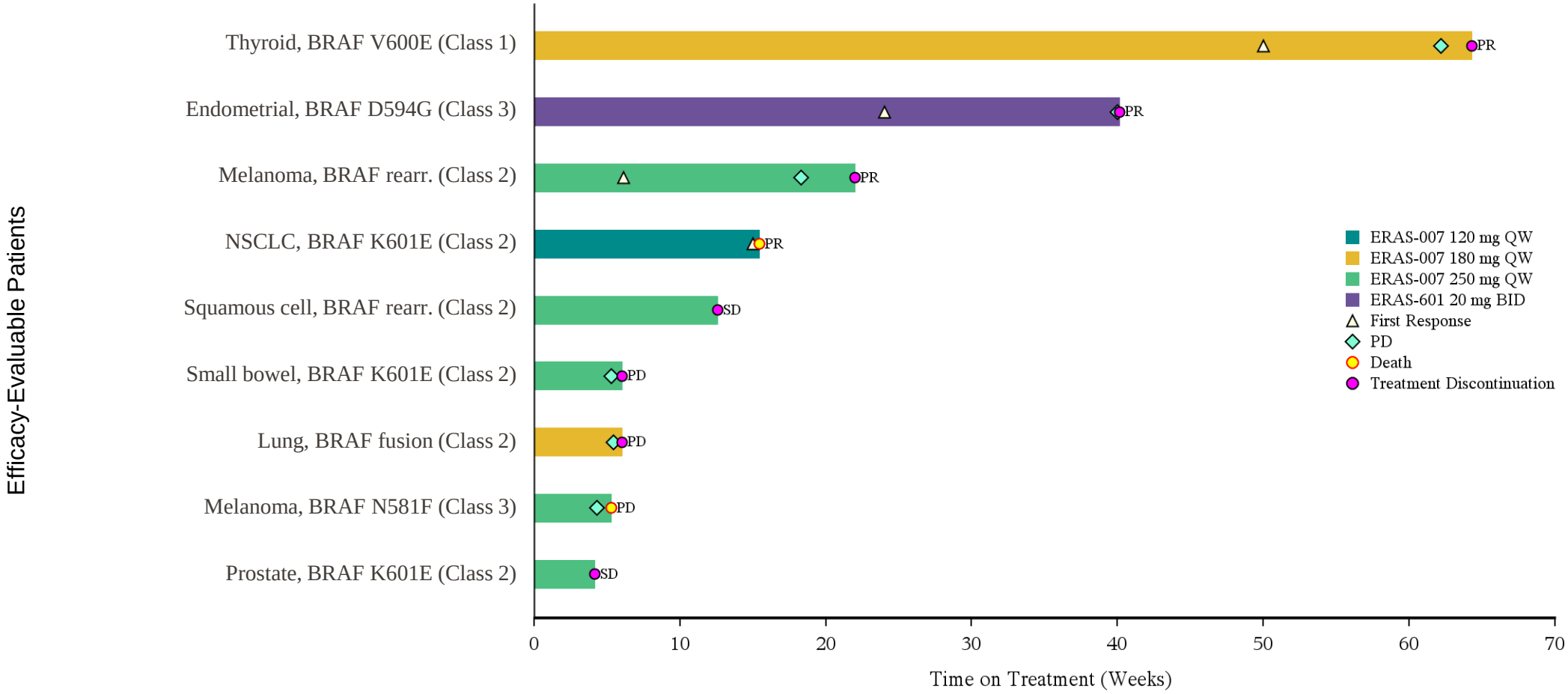
Best Overall Response Observed with ERAS-007 or ERAS-601 in BRAF-driven Blue Ocean Indications across lines of therapy



44% (4 out of 9) of patients responded (1 confirmed and 3 unconfirmed PR) to single agent ERAS-007 or ERAS-601

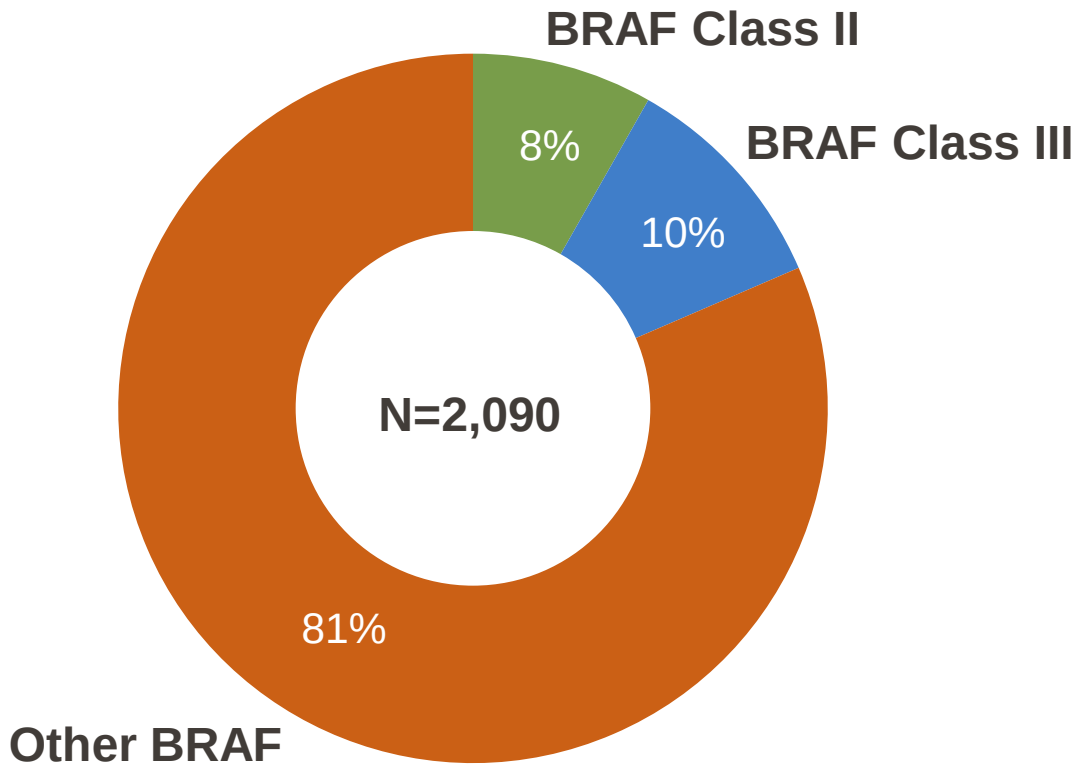
* Unconfirmed partial responses indicated with an asterisk
NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01.

Duration of Treatment Observed with ERAS-007 or ERAS-601 in BRAF-driven Blue Ocean Indications



NOTE: Blue Ocean Indications defined as populations of unmet need with relevant tumor types and RAS/MAPK alterations where no targeted therapies are approved; Biologically relevant dose defined as weekly monotherapy dose intensity between 120mg and 250mg for ERAS-007 and daily monotherapy dose intensity of 40mg (40mg QD or 20mg BID) for ERAS-601; data extraction date: 6NOV2020 for ASN007-101, 11JUL2022 for ERAS-601-01, 16May2022 for ERAS-007-01.

At MD Anderson, ~20% of BRAF patients have Class II and III mutations



BRAF Class II

- Most common tumor types include:
  - Genitourinary
  - Lung
  - Head and neck

BRAF Class III

- Most common tumor types include:
  - Genitourinary
  - Head and neck
  - Gastrointestinal

Ample number of BRAF Class II and III patients, and they can be identified

Source: MD Anderson Institute for Personal Cancer Therapy database, 2019-2022
Other BRAF includes Class I and unannotated mutations

Conclusions

- While Erasca data set is small, encouraging monotherapy activity has been observed with both ERAS-007 and ERAS-601 in patients with tumors driven by an activated RAS/MAPK pathway.
    - 6 of 26 patients with RAS/MAPK alterations experienced a confirmed or unconfirmed PR independent of tumor type. Half of the patients that responded were heavily pre-treated.
    - 4 of 9 patients with BRAF-driven solid tumors experienced a confirmed or unconfirmed PR. Three of the 4 responding patients with BRAF mutations had BRAF Class II/III alterations, for which there are no approved targeted therapies.
- Since ERAS-007 and ERAS-601 target two critical convergent nodes in the RAS/MAPK, complete pathway inhibition may be easier to obtain using the combination versus each drug in isolation.
- The clinical data Erasca has generated to date on both of their investigational molecules (ERAS-007 and ERAS-601) support further exploration of the combination and indicate that the team has good insight into the potential overlapping toxicities. The initial data suggest that the AEs are largely non-overlapping, monitorable, and manageable for ERAS-007 and ERAS-601.
- MD Anderson plans to participate in Erasca’s MAPKlamp (ERAS-007 + ERAS-601) trial.

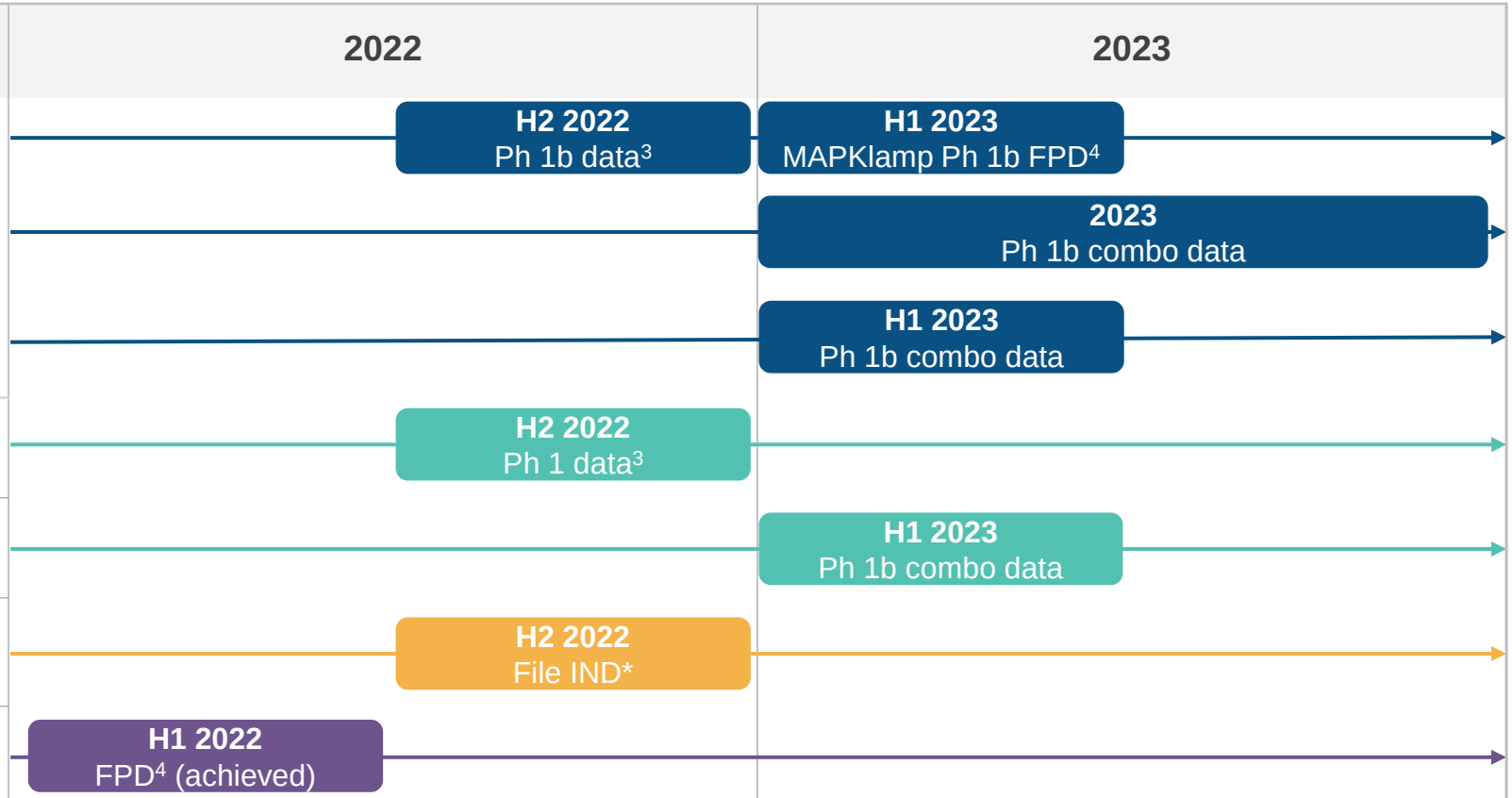
Future Directions and Key Milestones

Future directions

- ERAS-007 and ERAS-601 are being developed as **foundational combination agents** for targeting solid tumors with RAS/MAPK alterations. These agents are currently being explored:
  - **In combination with multiple other agents**, including inhibitors of BRAF (e.g., encorafenib), EGFR (e.g., cetuximab), and CDK4/6 (e.g., palbociclib)
  - **Across several tumor types** (e.g., BRAF V600E CRC, KRASm/NRASm CRC, KRASm PDAC, KRAS G12C NSCLC, EGFRm NSCLC)
- **Promising monotherapy antitumor activities** of ERAS-007 and ERAS-601 **heighten our conviction for combos** in the HERKULES and FLAGSHP trials, including the new MAPKlamp (ERAS-007 + ERAS-601) combo
  - The **MAPKlamp dose escalation** is expected to begin by **H1 2023**

Anticipated key milestones and clinical trial readouts

Program *Mechanism*	Trial Name *Indication*
ERAS-007 and/or ERAS-601 (MAPKlamp[1]) *ERK1/2 inhibitor and/or SHP2 inhibitor*	**HERKULES-1** *Advanced Solid Tumors*
	HERKULES-2 *Lung Cancers*
	HERKULES-3 *GI Cancers*
ERAS-601 *SHP2 inhibitor*	**FLAGSHP-1** *Advanced Solid Tumors*
	FLAGSHP-1 *Triple WT CRC[2]*
ERAS-3490 *CNS-penetrant KRAS G12C inhibitor*	**AURORAS-1** *KRAS G12Cm NSCLC*
ERAS-801 *CNS-penetrant EGFR inhibitor*	**THUNDERBBOLT-1** *Glioblastoma Multiforme*



* Note: ERAS-3490 has completed the preclinical assessment we believe necessary to support IND submission, including clearing GLP toxicology studies

[1] ERAS-007 (oral ERK1/2 inhibitor) and ERAS-601 (oral SHP2 inhibitor) together comprise our first innovative MAPKlamp
[2] Triple wildtype CRC is KRASwt, NRASwt, and BRAFwt
[3] Data to include preliminary monotherapy safety and pharmacokinetics to support dose selection for combinations
[4] FPD = first patient dosed

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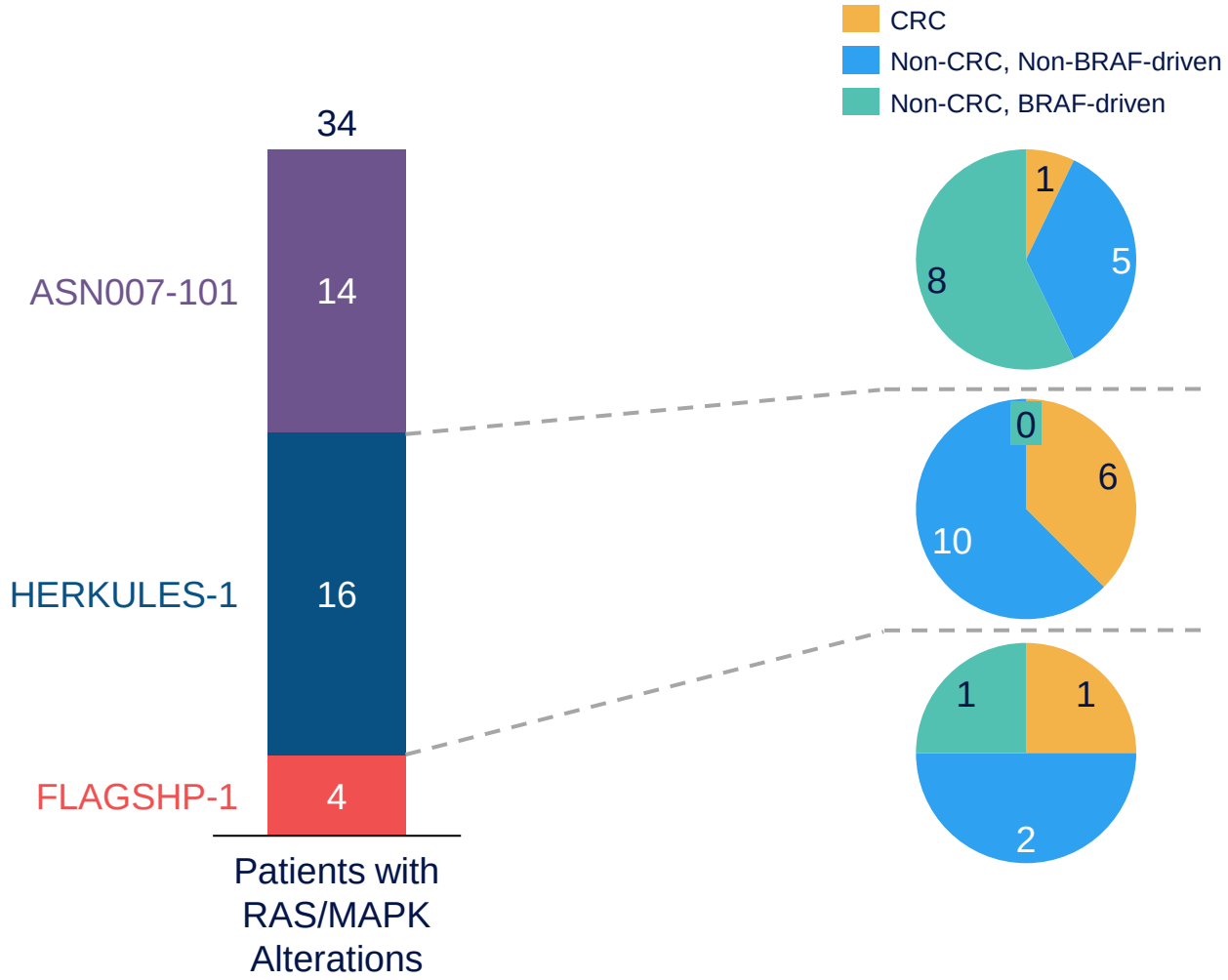
Thank You!

Breakdown of tumor types and molecular drivers by trial

Number of patients in ERAS-007 and ERAS-601 trials



ASN007-101 trial[1]:

- ~93% of patients had non-CRC tumors
- Of these, ~62% were BRAF-driven

HERKULES-1 trial[1]:

- ~63% of patients had non-CRC tumors
- Of these, none were solely BRAF-driven[2]

FLAGSHP-1 trial[1]:

- 75% of patients had non-CRC tumors
- Of these, ~33% were BRAF-driven

1 ASN007-101 trial was sponsored by Asana BioSciences; HERKULES-1 and FLAGSHP-1 trials are sponsored by Erasca
2 One patient had HRAS G13R and BRAF V600L, so BRAF was not the sole oncogene driver

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Other solid tumors

Other solid tumor type and alteration	Blue Ocean Indication
Small bowel adenocarcinoma, BRAF Class 2	14
Prostate cancer, BRAF Class 2	14
Thyroid cancer, NRAS Q61	11
Squamous cell carcinoma, BRAF rearrangement	14
Thyroid cancer, BRAF V600E	13
Medullary thyroid carcinoma, HRAS Q61	12
Ex-pleomorphic adenoma of right parotid, BRAF Class 1	13
Cervical cancer, KRAS G13D	10
Ampullary cancer, KRAS G12V	10
Esophageal cancer, NF1 LOF	8
Endometrial cancer, BRAF Class 3	15
Ovarian cancer, KRAS G12C	9
Ovarian cancer, KRAS G12A	10