DARLIFARNIB CLINICAL UPDATE: ASCO 2026

June 3, 2026

Our goal is to develop transformative therapies to extend and improve the lives of patients with cancer

KURA® ONCOLOGY

FORWARD-LOOKING STATEMENTS

This presentation contains forward-looking statements. Such statements include, but are not limited to, statements about our beliefs regarding the Company's value proposition; our research and development activities; the therapeutic potential of our product candidates; the potential of our product candidates to improve patient outcomes and to serve as foundational backbone therapies; darlifarnib's potential to combine with other therapies, induce resensitization, and enhance clinical activity across entire classes of targeted therapy; the potential market opportunity for darlifarnib; potential regulatory pathways for darlifarnib combinations; the development of darlifarnib across multiple targeted therapies and disease settings; potential clinical trials evaluating darlifarnib in combination with third-party therapies; the timing of clinical trials and the availability of clinical data; and our cash runway. The words "believe," "may," "should," "will," "estimate," "plan," "continue," "anticipate," "intend," "expect," "potential," and similar expressions (including the negative thereof) are intended to identify forward-looking statements. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. These risks include, among others: the risk that the Company may not achieve the value proposition we currently anticipate; the risk that our clinical trials may not be successful; the risk that results observed in preclinical studies may not be predictive of results in human clinical trials, and that our product candidates may fail to demonstrate the safety or efficacy suggested by preclinical data; the risk that we may not be successful in entering into or maintaining clinical collaboration and supply arrangements with third-party manufacturers, or in otherwise obtaining access to compounds we seek to evaluate in combination with our product candidates; the risk that the U.S. Food and Drug Administration may not permit our planned studies to proceed on the anticipated timelines, or at all, or may otherwise delay, restrict, or prevent the development, regulatory approval, or commercialization of our product candidates; delays in the initiation, enrollment, completion, or analysis of clinical trials, or in the reporting of data from such clinical trials; challenges in clinical trial design or execution, which could result in increased costs and delays, or limit our ability to obtain regulatory approval; the risk that our product candidates may not demonstrate adequate safety or efficacy, receive regulatory approval, or be successfully commercialized; and our ability to obtain additional financing. Additional risks and uncertainties may emerge from time to time, and it is not possible for the Company's management to predict all such risks and uncertainties.

All forward-looking statements contained in this presentation speak only as of the date on which they were made. Other risks and uncertainties affecting us are described more fully in our filings with the Securities and Exchange Commission. We undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

This presentation may also contain statistical, preclinical and clinical data obtained from and prepared by third parties. The recipient is cautioned not to give undue weight to such data. Neither the Company nor any other person makes any representation as to the accuracy or completeness of such data or undertakes any obligation to update such data after the date of this presentation.

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KURA ONCOLOGY HAS A COMPELLING VALUE PROPOSITION IN 2026

- KOMZIFTI™ (ziftomenib) approved for adult patients with relapsed/refractory *NPM1*-mutated AML
- Robust new patient starts and early launch momentum
- Advancing ziftomenib to address up to 50% of AML patients
- Multiple 2026 readouts expected to support ziftomenib as a broadly combinable AML backbone
- Proof-of-concept data position darlifarnib as a new mechanism of action and foundational backbone therapy in RCC and RAS-driven solid tumors
- Strong Financial Position: $580.8M in cash and investments as of 3/31/26, plus $180M in anticipated payments

PRECISION COMBINATIONS. BETTER PATIENT OUTCOMES.

OUR GOAL: DEVELOP PRECISION COMBINATIONS TO IMPROVE OUTCOMES FOR PEOPLE WITH CANCER

1 Ziftomenib combinations in AML

- Intensive chemotherapy (cytarabine and daunorubicin)
- Non-intensive chemotherapy (venetoclax and azacitidine)
- FLT3 inhibitors (gilteritinib and quizartinib)

2 Ziftomenib combinations in gastrointestinal stromal tumors (GIST)

- KIT inhibitors (imatinib)

3 Darlifarnib combinations in kidney cancer (renal cell carcinomas)

- VEGFR inhibitors (cabozantinib)

4 Darlifarnib combinations in *KRAS*-mutated pancreatic, lung, and colorectal cancers

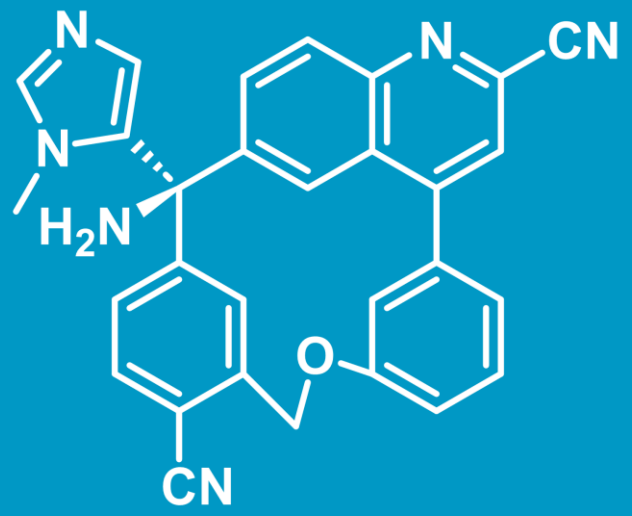
- KRAS G12C inhibitors (adagrasib)

CHALLENGES OF DEVELOPING AN EFFECTIVE COMPANION THERAPEUTIC TO ENHANCE CLINICAL ACTIVITY

Identifying and validating companion targets	Targets must be **druggable**, **selective**, and **broadly relevant** across patient subsets
Toxicity and narrow therapeutic index	**Co-inhibition is frequently poorly tolerated**, requiring dose modifications that compromise efficacy (*e.g.*, SOS1, SHP2 combos)
PK / PD interactions	CYP450/transporter-mediated **drug-drug interactions** can alter drug exposure, increasing toxicity or reducing efficacy

A companion therapeutic to address resistance has significant value

Rational combinations (*e.g.*, BRAF+MEK, EGFR-TKI + chemo) have shown success, but **no broadly applicable solution exists**



DARLIFARNIB

Next-generation farnesyl transferase inhibitor (FTI) optimized for precision combinations

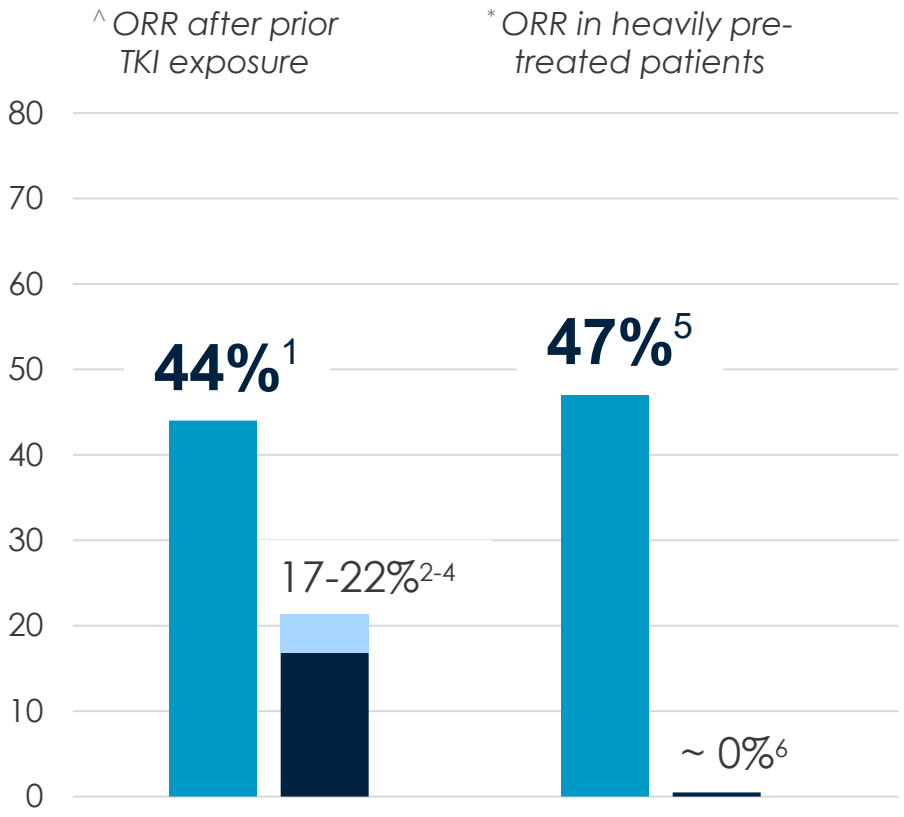
- We're not building another TKI or KRAS inhibitor drug candidate – we have potential to enhance clinical activity across multiple targeted therapies
- FTIs represent a mechanism-driven, targeted therapy-agnostic combination platform approach

COMBINATION WITH KRAS INHIBITOR IS 3RD TEST OF FARNESYL TRANSFERASE INHIBITOR MECHANISM

Clear Cell Renal Cell Cancer

PIK3CA-m Head & Neck Cancer

KRAS-m Solid Tumors

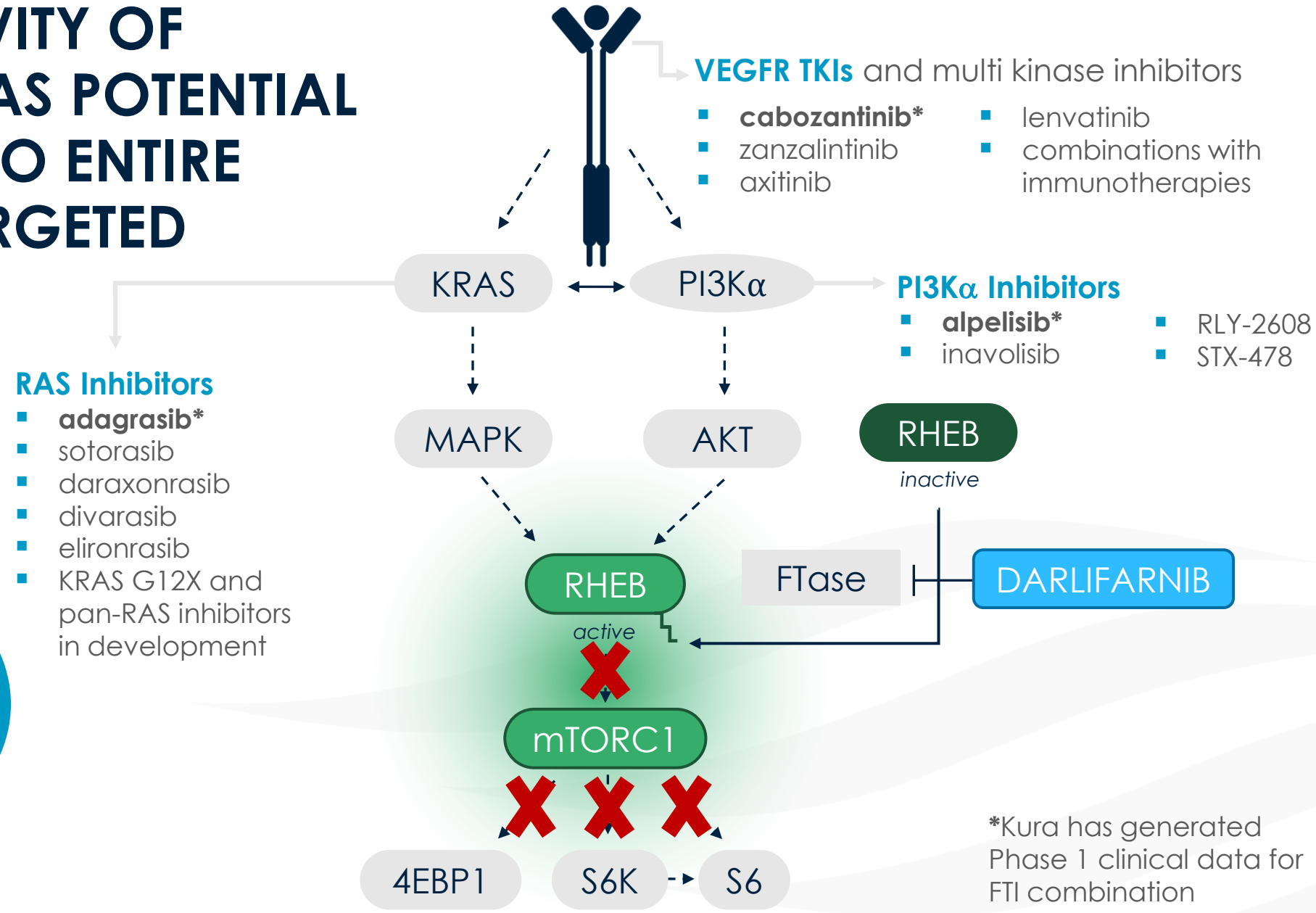


- **Same biology** – new opportunity
- **Same combination strategy** – add-on to targeted therapy
- **Same outcome** – preliminary clinical results demonstrate enhanced clinical efficacy
- **KRAS is next application** for darlifarnib

[1]Zakharia Y *et al.* IKCS: Europe, April 16–18, 2026, Paris, France; [2]Choueiri TK *et al. Lancet Oncol.* 2016;17(7):P917–927; [3]Choueiri TK *et al. N. Engl. J. Med.* 2024;391(8):710–721; [4]Rini BI *et al. Lancet Oncol.* 2020;21:95–104; [5]Hanna GJ *et al.* ESMO Annual Congress, October 17–21, 2025, Berlin, Germany; [6]Juric D *et al. J. Clin. Oncol.* 2018;36:1291-1299. ^darlifarnib, *tipifarnib

CLINICAL ACTIVITY OF DARLIFARNIB HAS POTENTIAL APPLICABILITY TO ENTIRE CLASSES OF TARGETED THERAPY

MULTIPLE OPPORTUNITIES FOR BOTH "GO-IT-ALONE" AND COLLABORATION

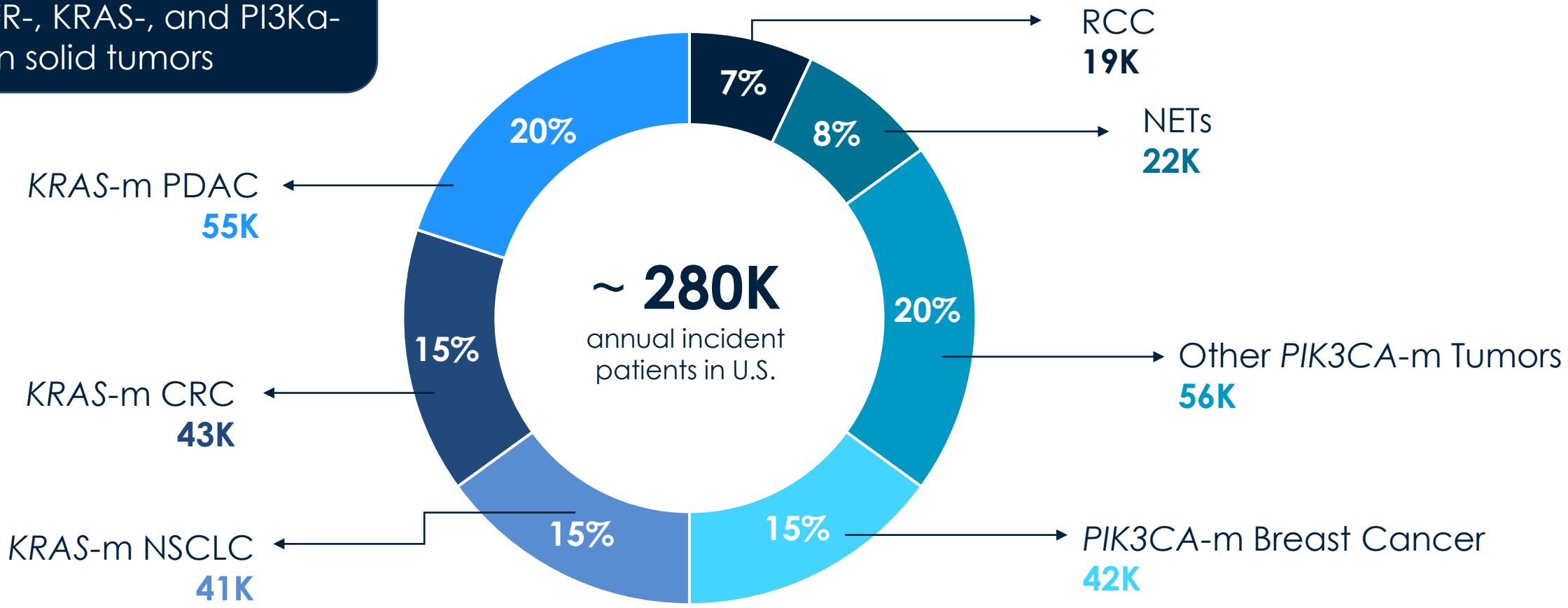


*Kura has generated Phase 1 clinical data for FTI combination

LARGE POTENTIAL OPPORTUNITY FOR DARLIFARNIB

Potential to combine with targeted therapies in VEGFR-, KRAS-, and PI3Kα-driven solid tumors

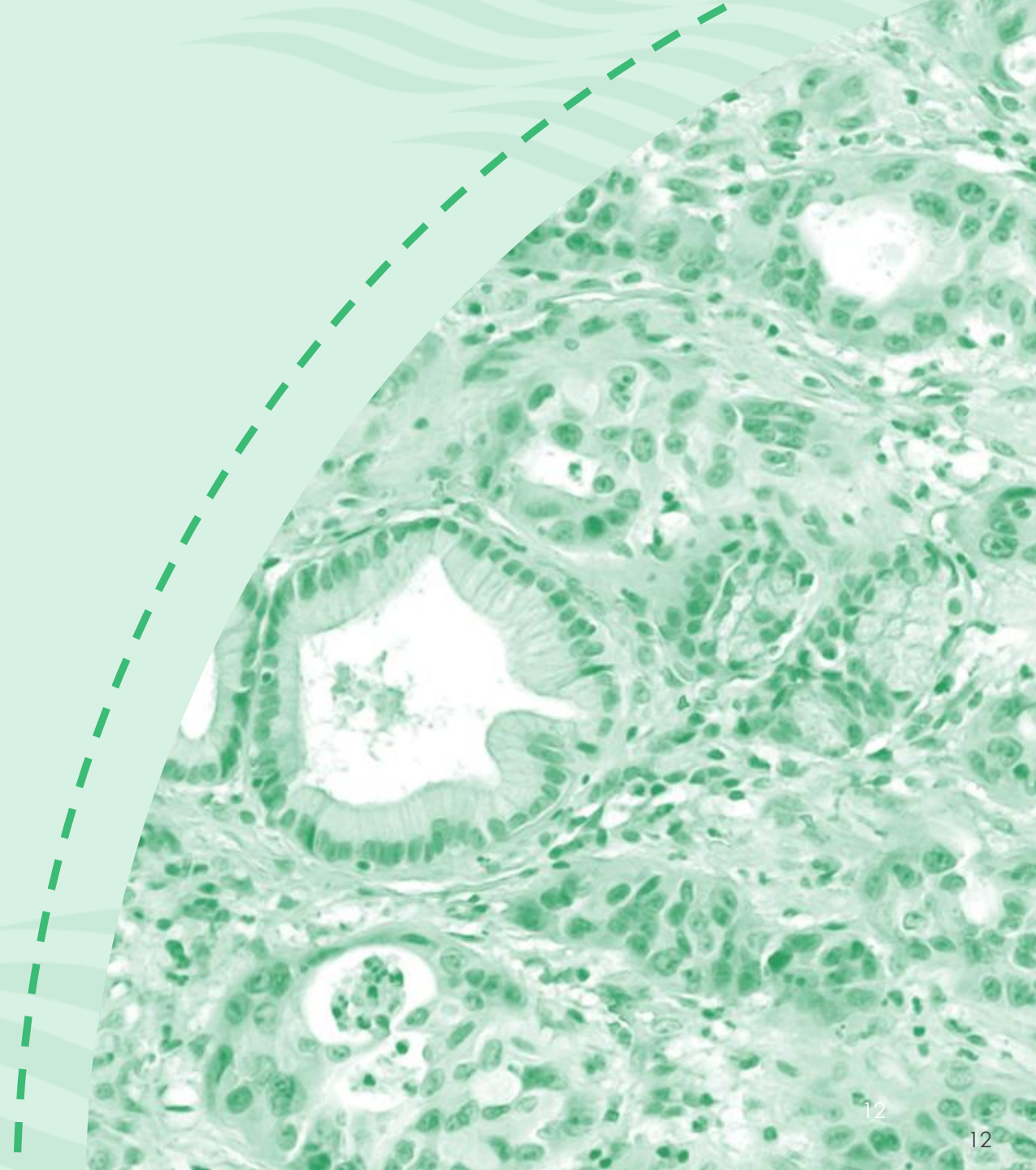
New Patients Per Year (U.S.)



Data sources include Decision Resources Group, Market Forecast Dashboard, 2025; Jalbert JJ *et al. Oncologist* 2020;25(4):e644–e650; Lim TKH *et al. Lung Cancer* 2023;184:107293; Cascetta P *et al. Cancers* 2022;14(21):5430.

DARLIFARNIB + ADAGRASIB IN *KRAS* G12C-MUTATED ADVANCED SOLID TUMORS

David Hong, M.D.



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FARNESYL TRANSFERASE INHIBITOR DARLIFARNIB IN COMBINATION WITH ADAGRASIB IN *KRAS* G12C MUTATED ADVANCED SOLID TUMORS: PRELIMINARY RESULTS FROM FIT-001 PHASE 1 FIRST-IN-HUMAN TRIAL

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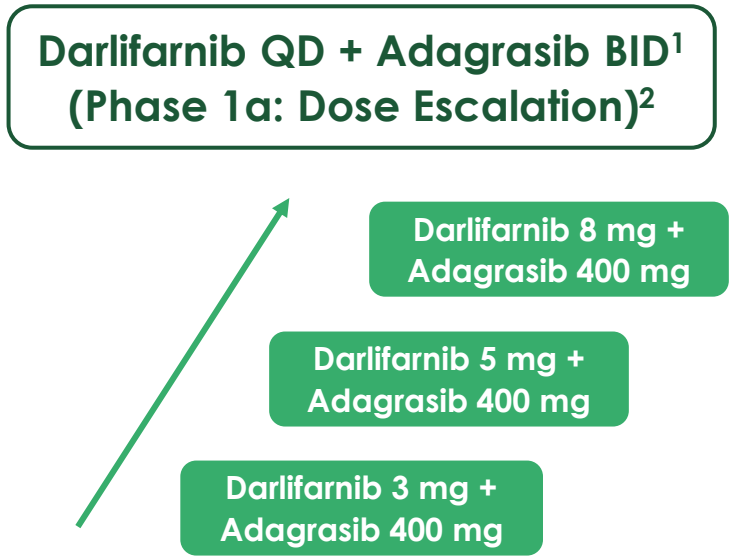
DISCLOSURES

Dr. Hong's disclosures include consulting, speaker or advisory roles with Abbvie, Acuta, Alpha Insights, Amgen, Axiom, Blueprint, Beigene, Boxer Capital, BVF Advisory & Consulting , ClearView Oncology, COR2ed, Cogent Therapeutics, CureBio, EcoR1 Capital, ERASCA, GLG, Guidepoint, HuyaBio, Immunogenesis, Kanaph Therapeutics, Kayak Therapeutics, Kestrel Therapeutics, Medscape, Morgan-Stanley, Nextech Ventures, Pfizer, PharmaResearch, Revolution Medicine, T-Knife, and WebMD.

FIT-001: DARLIFARNIB + ADAGRASIB COMBINATION IN *KRAS* G12C-MUTANT SOLID TUMORS

First-in-human, multicenter, open-label, Phase 1 study evaluating darlifarnib in combination with targeted therapies in patients with advanced solid tumors



- Darlifarnib 3, 5, or 8 mg was administered QD on Days 1–7 and 15–21 of each 28-day cycle in combination with adagrasib 400 mg BID
- Darlifarnib 8 mg + adagrasib 400 mg BID is not being advanced for further evaluation, per protocol

[1]Approved adagrasib monotherapy dose is 600 mg BID. Highlights of Prescribing Information, KRAZATI® (adagrasib) tablets, for oral use, Bristol Myers Squibb, March 2026. [2]Each individual patient received one of the planned dose levels of darlifarnib.

DEMOGRAPHICS AND BASELINE CHARACTERISTICS

As of the Mar 25, 2026, data cutoff date, 30 patients with *KRAS* G12C-mutated NSCLC, PDAC, or CRC received darlifarnib plus adagrasib

n (%)	Darlifarnib 3 mg + adagrasib (n=15)	Darlifarnib 5 mg + adagrasib (n=15)
Median age, years (range)	61 (45–79)	60 (22–74)
Sex		
Male	12 (80)	7 (47)
Female	3 (20)	8 (53)
Race		
White	12 (80)	13 (87)
Non-White[1]	3 (20)	2 (13)
Karnofsky PS		
80–100	14 (93)	14 (93)
50–70	1 (7)	1 (7)
Primary tumor type		
NSCLC	5 (33)	4 (27)
PDAC	3 (20)	3 (20)
CRC	7 (47)	8 (53)

[1]Includes Black or African American, Asian, American Indian or Alaska Native, Other, and Multiple.
Data cutoff: March 25, 2026.

DEMOGRAPHICS AND BASELINE CHARACTERISTICS

n (%)	Darlifarnib 3 mg + adagrasib (n=15)	Darlifarnib 5 mg + adagrasib (n=15)
Prior systemic therapy		
1	5 (33)	5 (33)
2	1 (7)	6 (40)
≥3	9 (60)	4 (27)
Prior KRAS G12Ci (any type)	8 (53)	4 (27)
Prior KRASi type		
Sotorasib	5 (33)	2 (13)
Adagrasib	2 (13)	1 (7)
Daraxonrasib + elironrasib	1 (7)	1 (7)
Divarasib	1 (7)	0

Data cutoff: March 25, 2026.

PRIOR THERAPY BY TUMOR TYPE

n (%)	PDAC (n=6)	NSCLC (n=9)	CRC (n=15)
Prior systemic therapy			
1	4 (67)	5 (56)	2 (13)
2	2 (33)	0	5 (33)
≥3	0	4 (44)	8 (53)
Prior KRAS G12Ci (any type)	0	4 (44)	8 (53)
Prior KRASi type			
Sotorasib	0	4 (44)	3 (20)
Adagrasib	0	0	3 (20)
Daraxonrasib + elironrasib	0	0	2 (13)
Divarasib	0	0	1 (7)

Data cutoff: March 25, 2026.

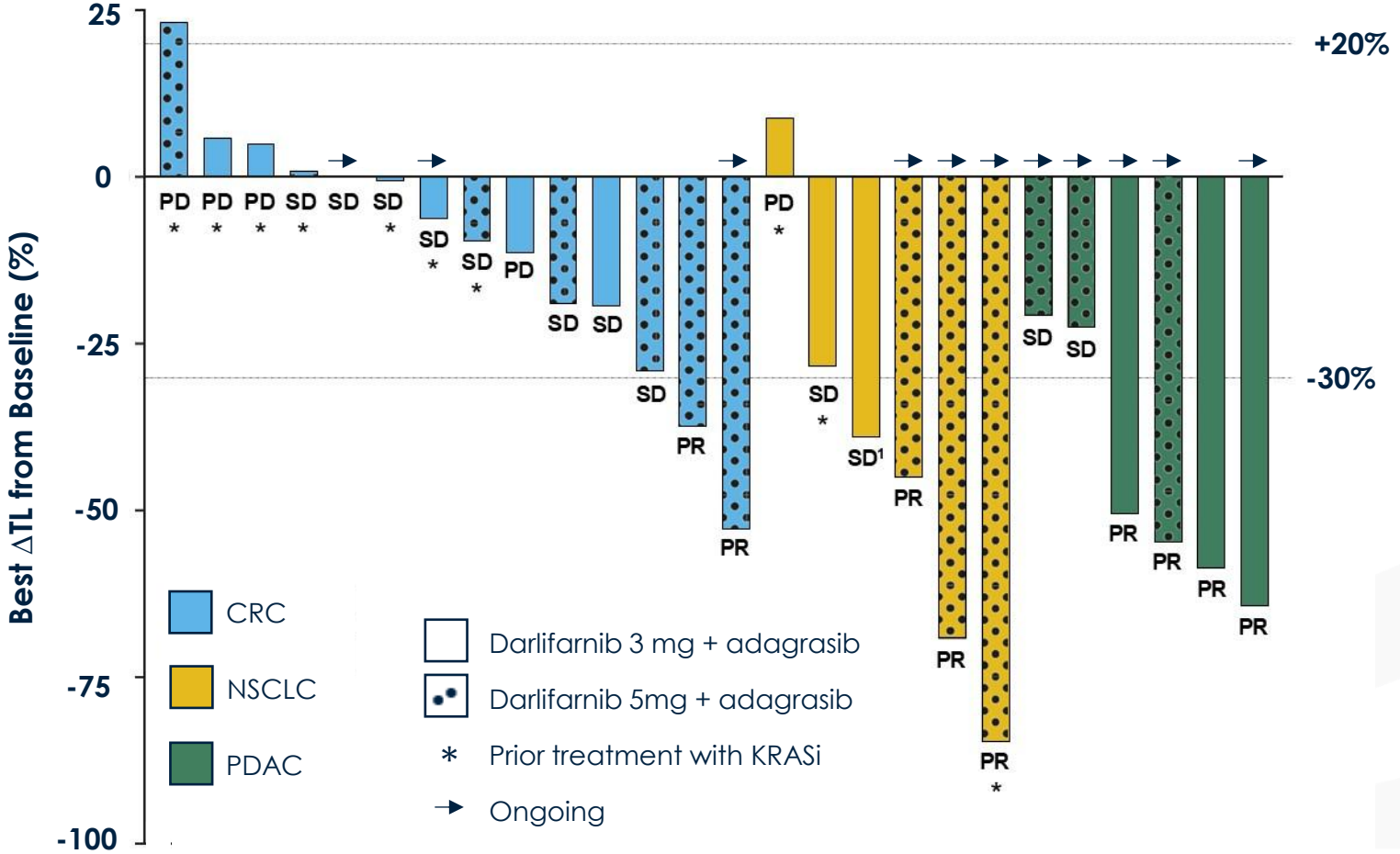
TREATMENT-EMERGENT ADVERSE EVENTS

n (%)	Darlifarnib 3 mg + adagrasib (n=15)	Darlifarnib 5 mg + adagrasib (n=15)
Any-grade TEAEs (≥30% of all patients)	15 (100)	15 (100)
Diarrhea	11 (73)	10 (67)
Nausea	10 (67)	10 (67)
Anemia	4 (27)	12 (80)
Vomiting	9 (60)	6 (40)
Neutropenia	4 (27)	9 (60)
Thrombocytopenia	2 (13)	10 (67)
Asthenia	2 (13)	8 (53)
Grade ≥3 TEAEs (≥15% of all patients)	8 (53)	12 (80)
Neutropenia	3 (20)	7 (47)
Anemia	2 (13)	5 (33)
SAEs	2 (13)	7 (47)
Treatment-related SAEs	1 (7)	2 (13)
TEAEs leading to darlifarnib dose reduction	0	3 (20)
TRAEs leading to darlifarnib dose reduction	0	3 (20)
TEAEs leading to darlifarnib dose interruption	7 (47)	14 (93)
TRAEs leading to darlifarnib dose interruption	5 (33)	13 (87)
TEAEs leading to darlifarnib drug withdrawal	1 (7)	1 (7)
TRAEs leading to darlifarnib drug withdrawal	0	0

Data cutoff: March 25, 2026.

ENCOURAGING ANTI-TUMOR ACTIVITY

Best percent change from baseline in target lesion size



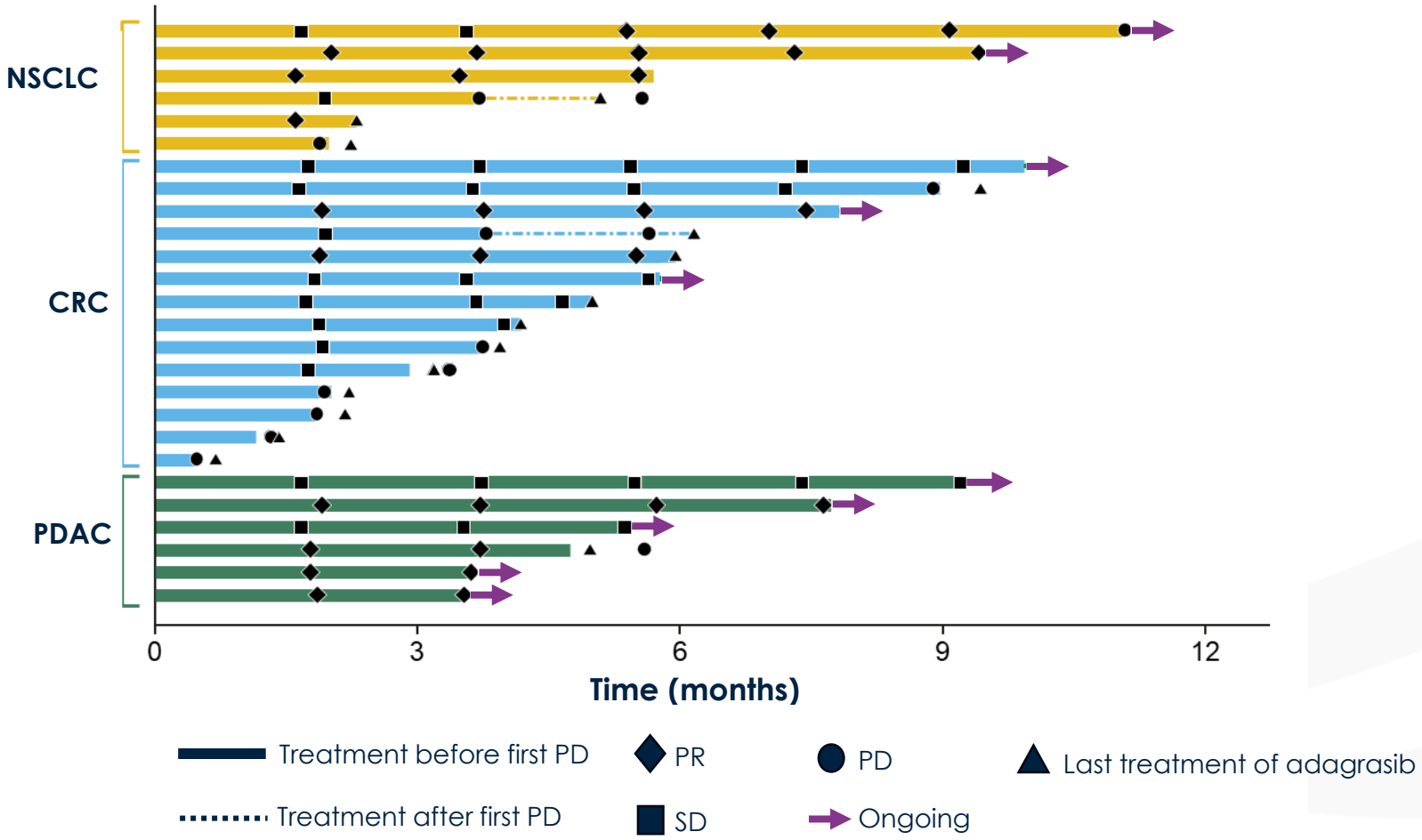
Tumor shrinkage observed:

- At both 3 mg and 5 mg darlifarnib doses
- In 77% (20/26) of all response-evaluable patients
- In 94% (15/16) of all response-evaluable KRASi-naïve patients
- In KRASi-pretreated patients

Response-evaluable patients had ≥1 darlifarnib dose and post-baseline scan. Adagrasib 400 mg BID.
Data cutoff: March 25, 2026.

ENCOURAGING CLINICAL ACTIVITY

Time on treatment and response in evaluable[1] patients



Median follow-up time in months (range):

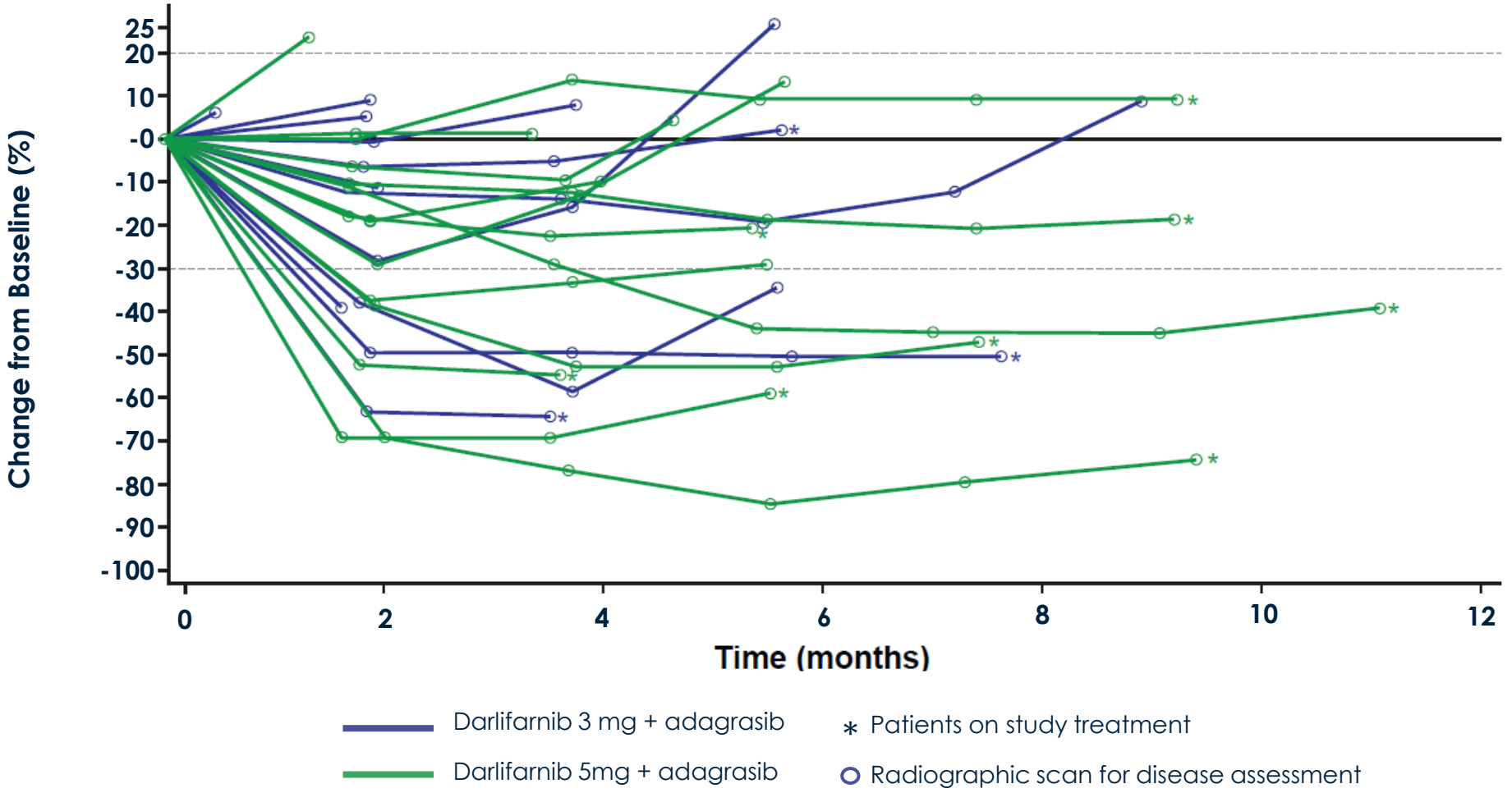
- NSCLC 6.9 (3.2–11.8)
- CRC 8.9 (1.2–13.2)
- PDAC 6.7 (4.0–10.4)

Responses observed across **all tumor types** at **both doses** of darlifarnib + adagrasib

[1]Response-evaluable patients had ≥1 darlifarnib dose and post-baseline scan.
Data cutoff: March 25, 2026.

ENCOURAGING CLINICAL ACTIVITY

Change in target lesion sum of diameters over time in evaluable[1] patients



[1]Response-evaluable patients had ≥1 darlifarnib dose and post-baseline scan.
Data cutoff: March 25, 2026.

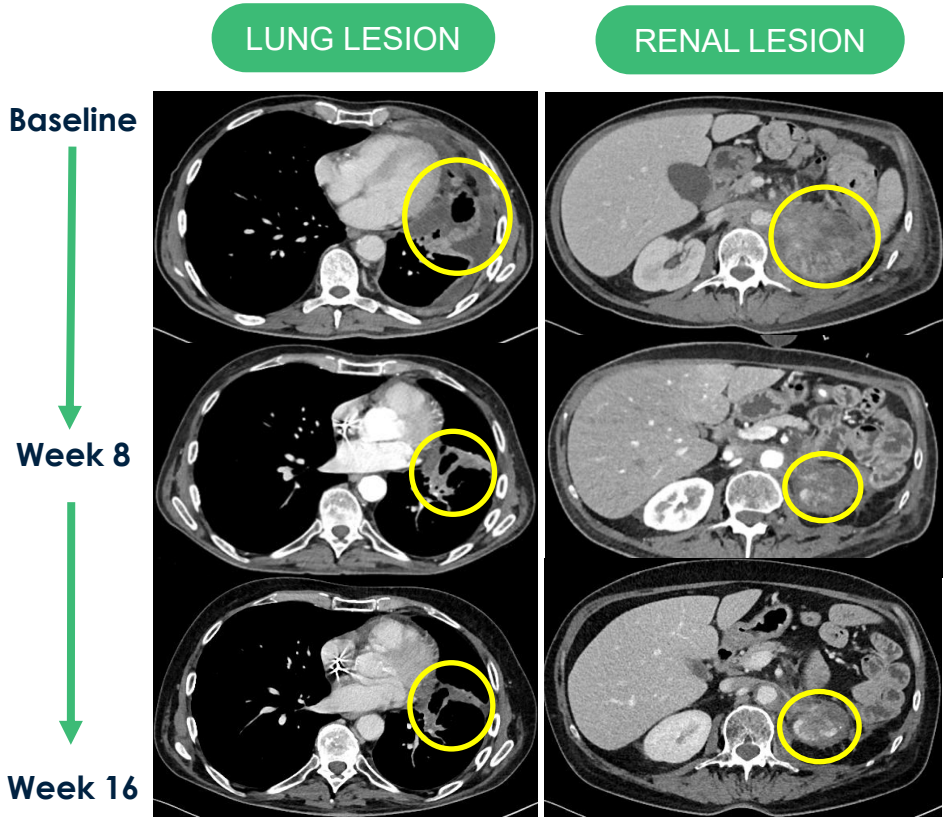
ENCOURAGING ANTI-TUMOR ACTIVITY

% (95% CI)	n	ORR	CBR[1]	mDOR, mos
Tumor type				
PDAC	6	**67** (22–96)	100 (54–100)	NE (3.8–NE)
NSCLC	6	**50** (12–88)	50 (12–88)	NE (5.7–NE)
KRASi-naïve NSCLC	3	67 (9-99)	67 (9-99)	5.7 (NE–NE)
CRC	14	14 (2–43)	43 (18–71)	NE (4.4–NE)
KRASi-naïve CRC	7	**29** (3–60)	43 (8–70)	NE (4.4–NE)
Dose level				
Darlifarnib 3 mg + adagrasib	12	25 (6–57)	42 (15–72)	NE (3.8–NE)
Darlifarnib 5 mg + adagrasib	14	43 (18–71)	71 (42–92)	5.7 (4.4–NE)

- Among 26 response-evaluable[2] patients, confirmed responses and clinical benefit were observed in each tumor type and at each dose level

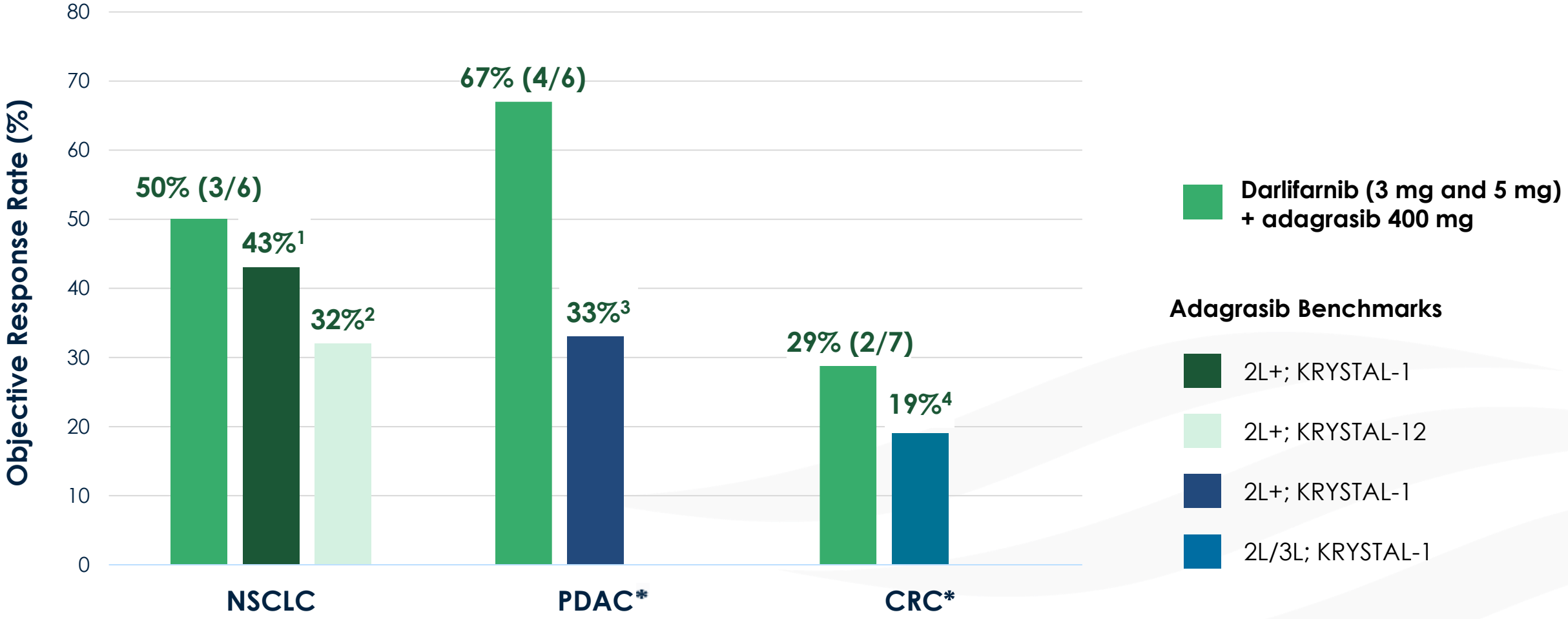
[1]CBR is the percentage of patients who achieved confirmed CR, confirmed PR, or ≥12 weeks SD duration.[2]Response-evaluable patients had ≥1 darlifarnib dose and post-baseline scan.

ENCOURAGING ACTIVITY OF COMBINATION IN 5TH LINE NSCLC PATIENT WHO HAD RECEIVED PRIOR KRAS G12C INHIBITOR



- 51-year-old male, former smoker with stage IV *KRAS* G12C-mutated NSCLC
- **5th line treatment**; prior therapies:
  - Induction/consolidation: Cisplatin + gemcitabine, durvalumab
  - 1L: Docetaxel + nintedanib
  - 2L: **Sotorasib** (BOR PR, discontinued due to PD)
  - 3L: Cisplatin + pemetrexed
  - 4L: Vinorelbine
- Enrolled to darlifarnib 5 mg + adagrasib
- Response:
  - Best overall response: **PR**
  - Best change from baseline: **−84.6%**
  - **PR achieved at week 8 and maintained through week 40, the last disease assessment before data cutoff**

ENCOURAGING ANTI-TUMOR ACTIVITY RELATIVE TO ADAGRASIB MONOTHERAPY BENCHMARKS



*In KRASi-naïve patients. [1]KRYSTAL-1, NSCLC (N=112), Jänne PA *et al. N. Engl. J. Med.* 2022;387:120-131; [2]KRYSTAL-12, NSCLC (N=301), Mok TSK *et al. J Clin. Oncol.* 2024;42(17 Suppl.):LBA8509; [3]KRYSTAL-1, PDAC (N=21), Bekaii-Saab TS *et al. J. Clin. Oncol.* 2023;41(25):4097-4106; [4]KRYSTAL-1, CRC (N=43), Yaeger R *et al. N. Engl. J. Med.* 2023;388:44-54. Data cutoff: March 25, 2026.

COMBINATION DEMONSTRATES ENCOURAGING ACTIVITY IN *KRAS* G12C-MUTATED SOLID TUMORS

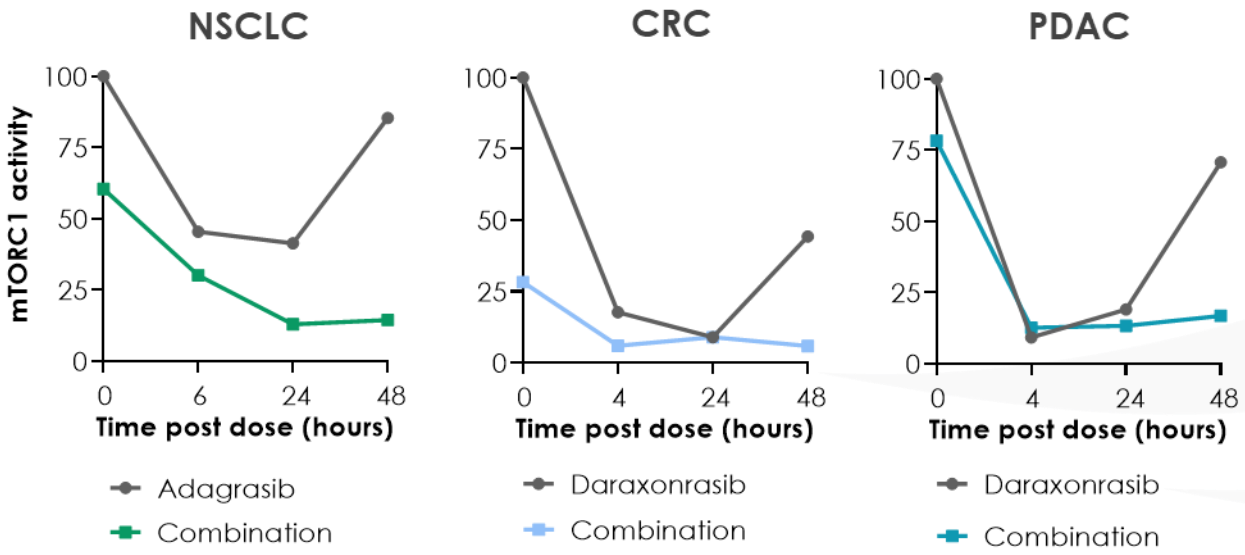
- Darlifarnib + adagrasib was well tolerated with a **manageable safety profile**
- **Encouraging antitumor activity** observed in **heavily pretreated patients**
- ORR of combination **compares favorably to historical benchmarks** for adagrasib monotherapy
  - PDAC: 67%
  - NSCLC: 50%
  - KRASi-naïve CRC: 29%
- Clinical activity observed in patients with **prior KRAS inhibitor exposure**
- Data support **further evaluation** of darlifarnib in combination with mutant-selective and pan-RAS inhibitors

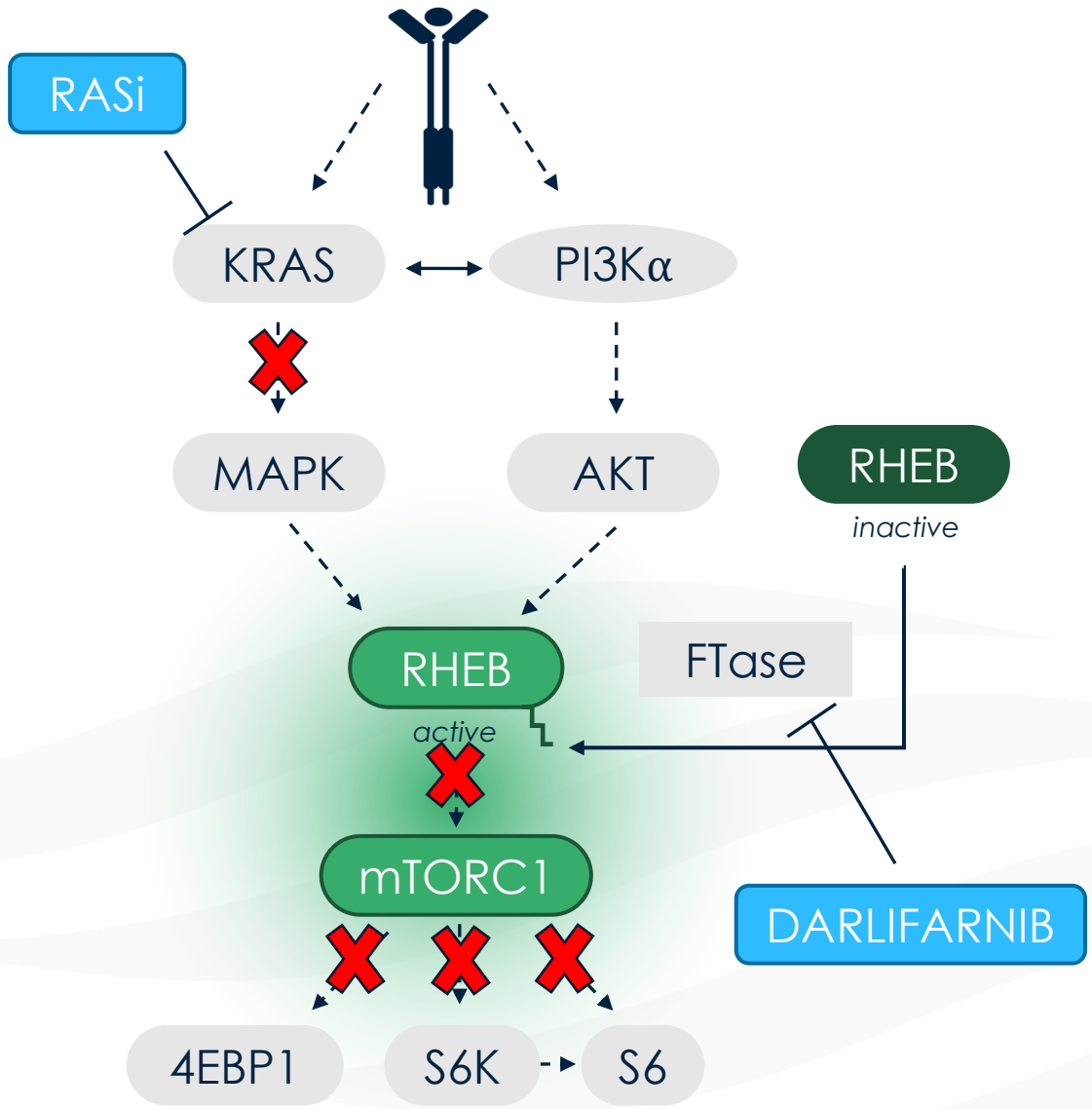
TRANSLATIONAL EVALUATION OF DARLIFARNIB WITH KRAS INHIBITORS

Francis Burrows, Ph.D.

FTIs ENHANCE ACTIVITY OF RAS INHIBITORS BY TARGETING THE mTORC1 NODE

- **Partial inhibition and/or rebound of mTOR signaling limits single agent efficacy of (K)RAS inhibitors**
- Darlifarnib inhibits RHEB farnesylation leading to a **sustained blockade of mTORC1 signaling** and enhancing the anti-tumor activity of RAS inhibitors across the class



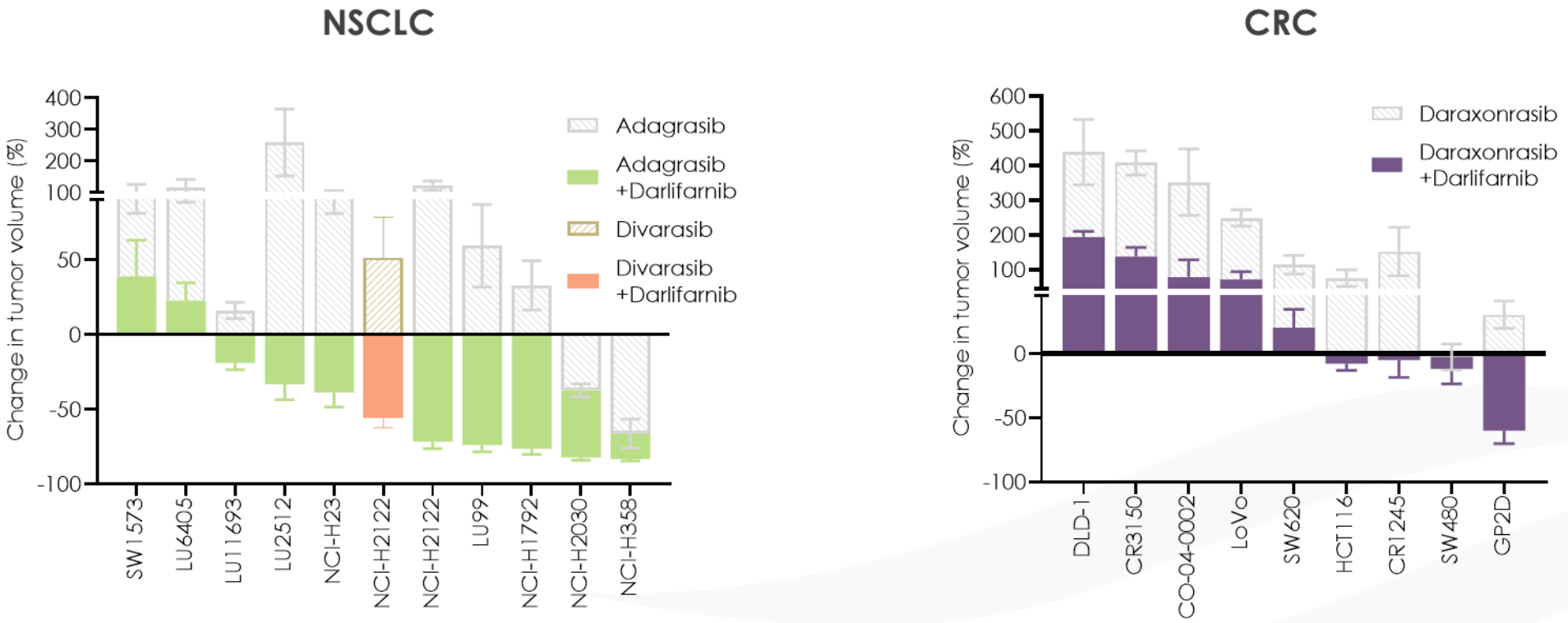


Patel HV *et al. Cancer Res.* 2026;86(9):2273-2285.

EVOLVING KRAS LANDSCAPE EXPANDS OPPORTUNITY FOR THERAPEUTIC COMBINATIONS

	DEVELOPMENT	COMMERCIAL
KRAS G12C	Revolution Medicines: Elironrasib MERCK: Calderasib Genentech / Roche: Divarasib Jacobio: Glecirasib Lilly: Olomorasib	AMGEN: (LUMAKRAS®) Sotorasib Bristol Myers Squibb: (KRAZATI®) Adagrasib
KRAS G12D	VERASTEM ONCOLOGY: VS-7375 Lilly: LY3962673 Incyte: INCB161734 Astellas: Setidegrasib Revolution Medicines: Zoldonrasib	
Pan-RAS / Pan-KRAS	BeOne: BGB-53038 BBOT BridgeBio Oncology Therapeutics: BBO-11818 Lilly: LY4066434 Revolution Medicines: Daraxonrasib AMGEN: AMG-410 ERASCA: ERAS-4001 Boehringer Ingelheim: BI-3706674	

DARLIFARNIB ENHANCES ANTI-TUMOR ACTIVITY OF RAS INHIBITORS IN NSCLC AND CRC PRECLINICAL MODELS

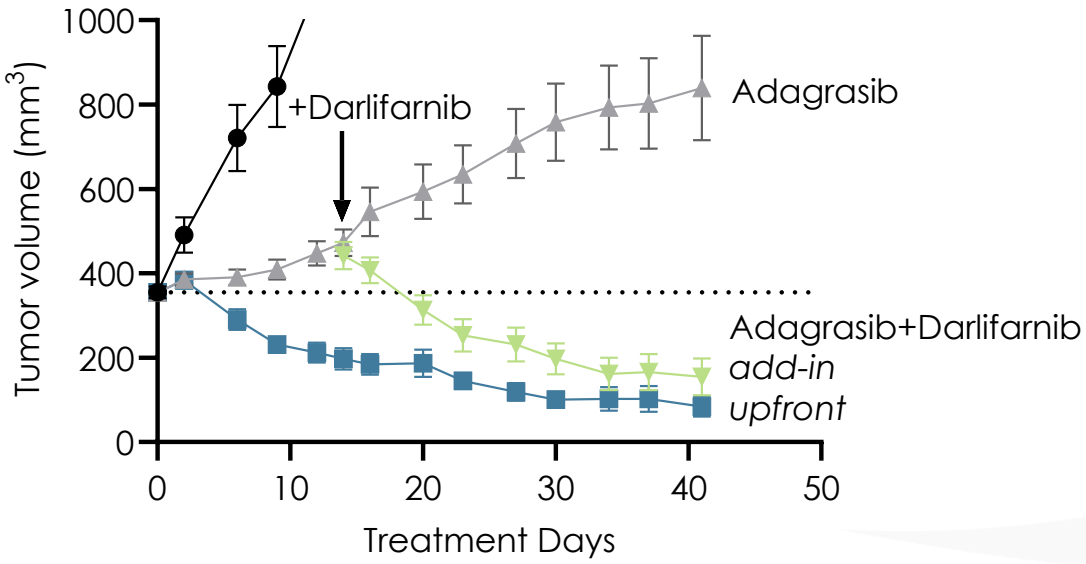


Darlifarnib enhances activity of **all classes of RAS inhibitors across their full activity range** in both NSCLC and CRC CDX and PDX models

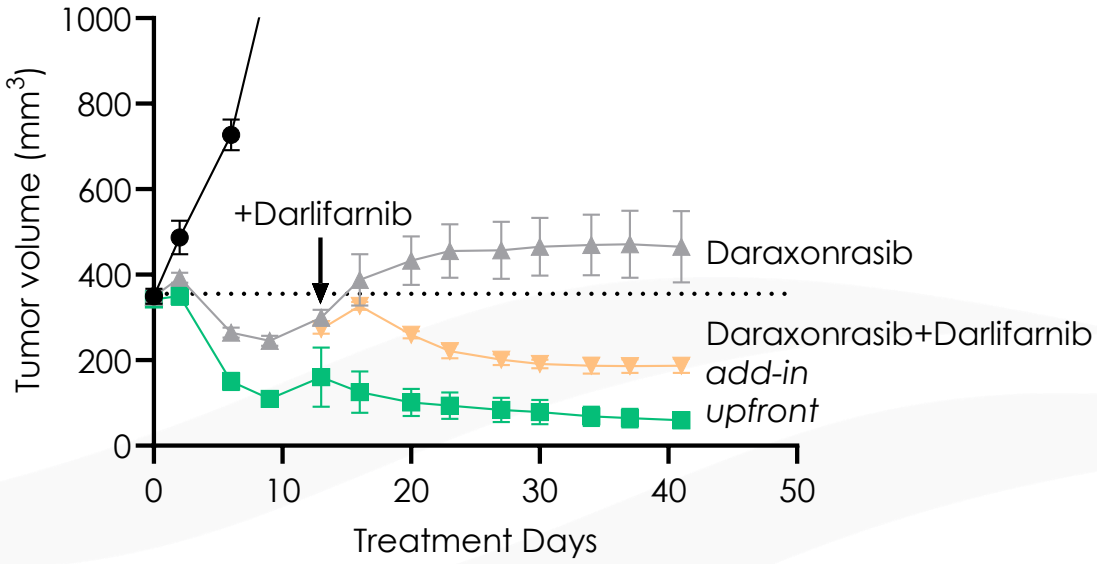
Patel HV *et al. Cancer Res.* 2026;86(9):2273-2285.

DARLIFARNIB ENHANCES ACTIVITY OF MUTANT-SELECTIVE OR PAN-RAS INHIBITION IN NSCLC MODEL

NCI-H2122
($KRAS^{G12C}$ NSCLC)



Mutant-selective inhibitor

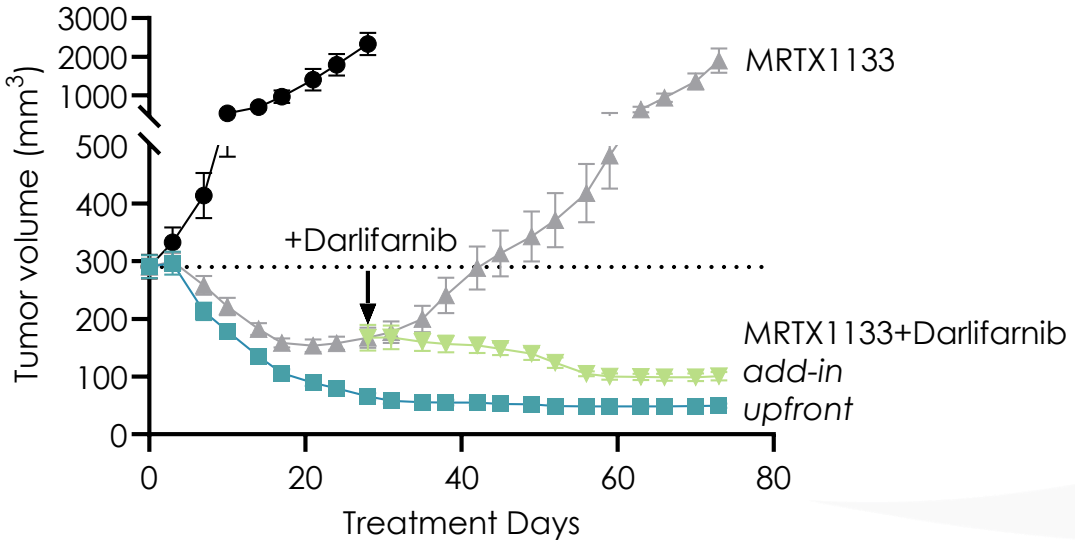


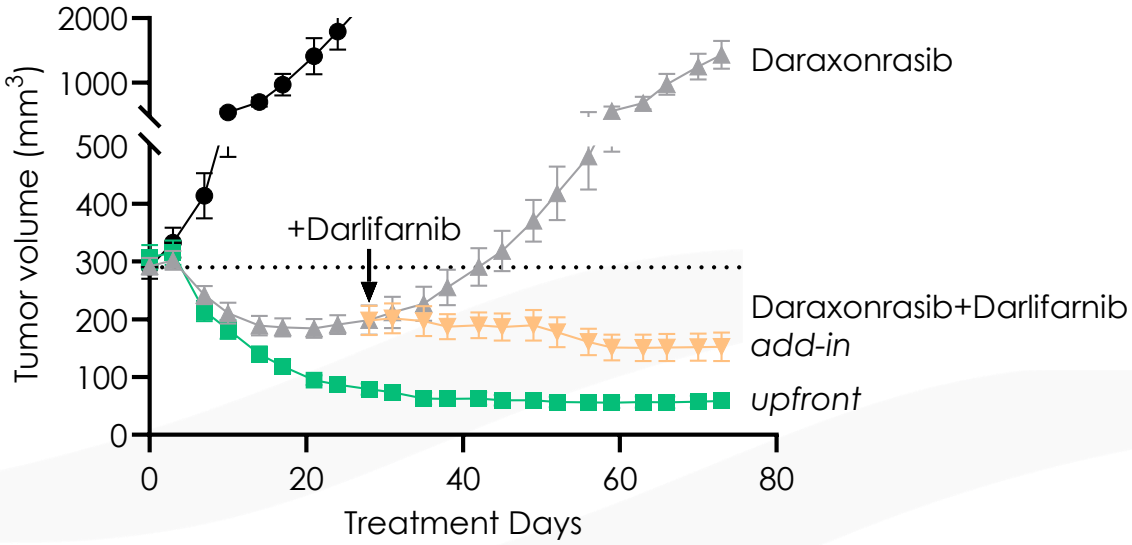
Pan-RAS inhibitor

Patel HV *et al. Cancer Res.* 2026;86(9):2273-2285.

DARLIFARNIB ENHANCES ACTIVITY OF RAS INHIBITION IN COLORECTAL CANCER PRECLINICAL MODEL

GP2D
(KRAS^{G12D}, PIK3CA^{H1047L} CRC)





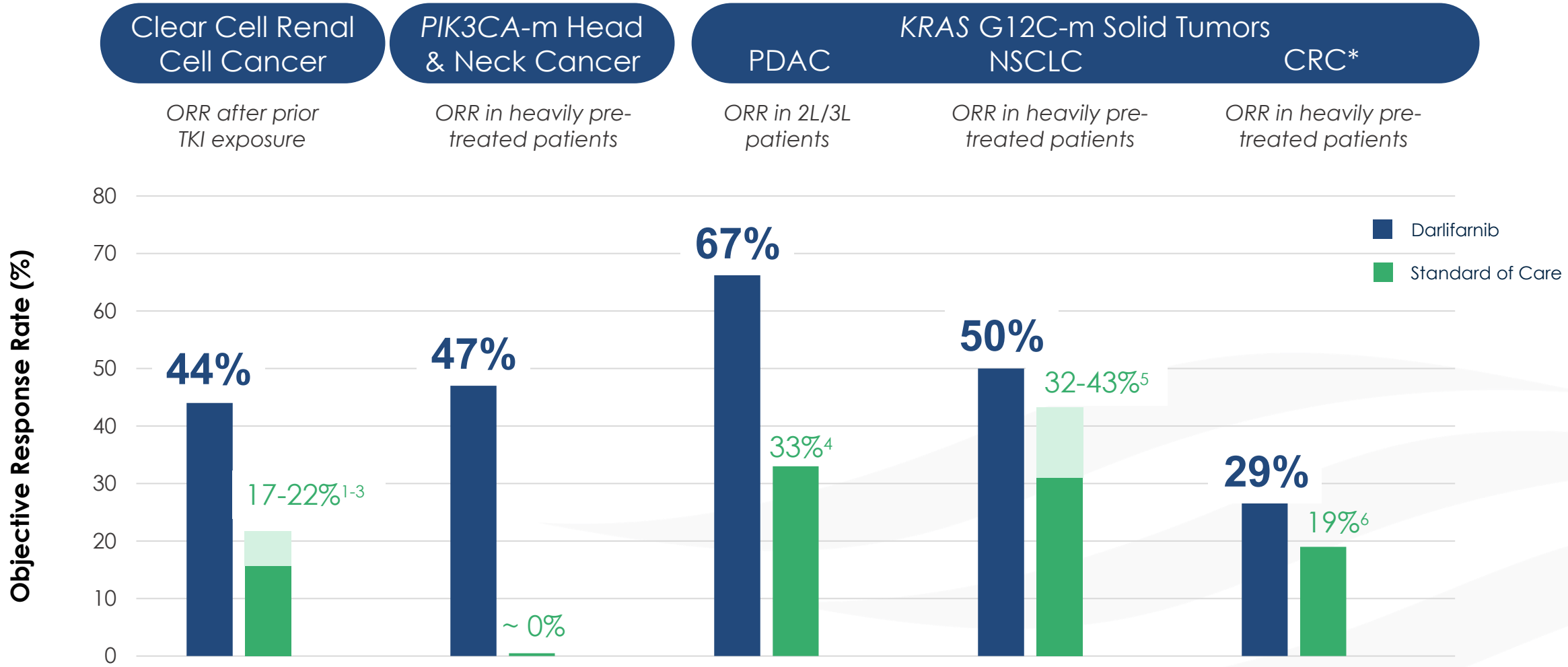
PRECLINICAL DATA SUPPORTS BROAD APPLICABILITY OF DARLIFARNIB TO RAS INHIBITORS

- Darlifarnib enhances activity of **all classes of RAS inhibitors** across their full activity range in **multiple preclinical models**
- KRAS inhibitor/darlifarnib combination therapy **induces regressions** in preclinical tumors **previously exposed** to mutant-selective or pan-RAS inhibitor monotherapy
- Preclinical data supports evaluating darlifarnib with both **pan-RAS and mutant-selective inhibitors**
- Darlifarnib represents a mechanism-driven, **RAS inhibitor-agnostic combination platform**

COMBINATION LANDSCAPE AND ANTICIPATED NEXT STEPS

Mollie Leoni, M.D.

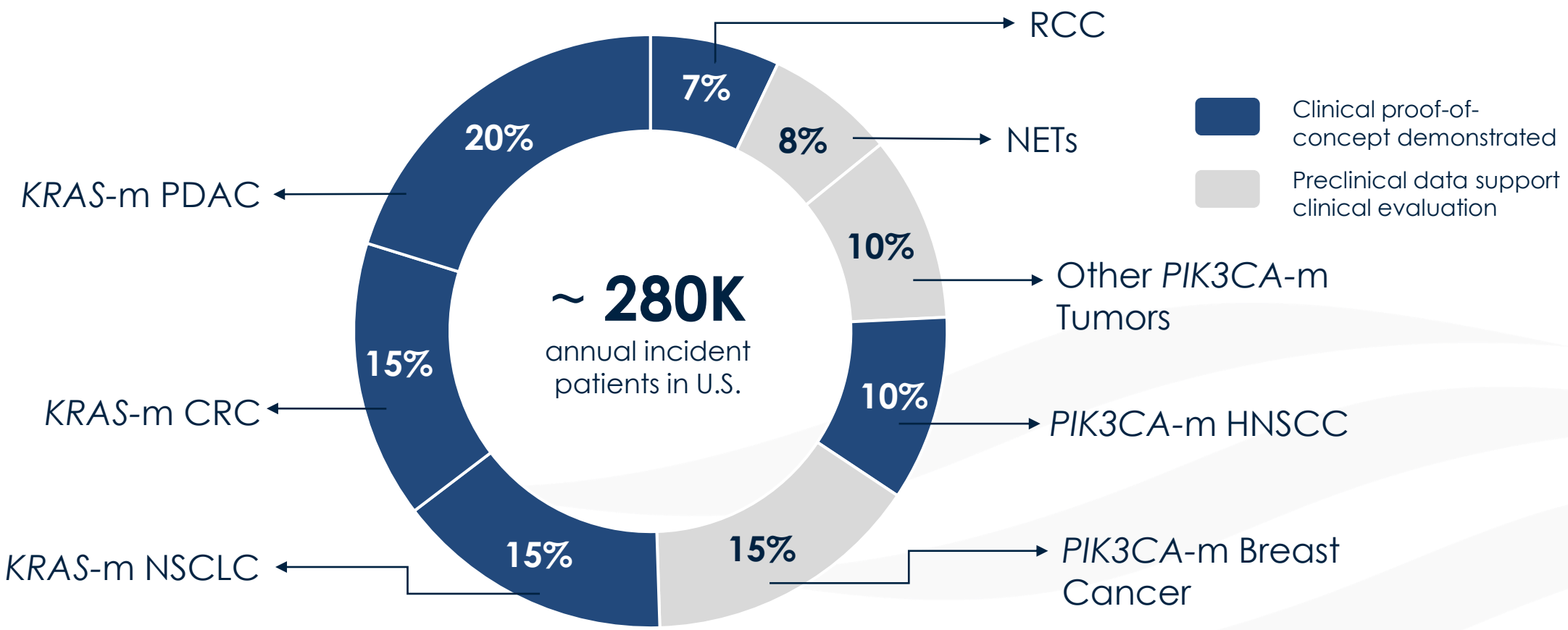
MEANINGFUL CLINICAL ACTIVITY DEMONSTRATED WITH FTI COMBINATIONS IN 3 OF 3 CLINICAL SETTINGS



*In KRASi-naïve patients. [1]Choueiri TK *et al. Lancet Oncol.* 2016;17(7):P917–927; [2]Choueiri TK *et al. N. Engl. J. Med.* 2024;391(8):710–721; [3]Rini BI *et al. Lancet Oncol.* 2020;21:95–104; [4]Bekaii-Saab TS *et al. J. Clin. Oncol.* 2023;41(25):4097-4106; [5]Jänne PA *et al. N. Engl. J. Med.* 2022;387:120-131, Mok TSK *et al. J Clin. Oncol.* 2024;42(17 Suppl.):LBA8509; [6]Yaeger R *et al. N. Engl. J. Med.* 2023;388:44-54.

LARGE POTENTIAL OPPORTUNITY FOR DARLIFARNIB

New Patients Per Year (U.S.)



Data sources include Decision Resources Group, Market Forecast Dashboard, 2025; Jalbert JJ *et al. Oncologist* 2020;25(4):e644–e650; Lim TKH *et al. Lung Cancer* 2023;184:107293; Cascetta P *et al. Cancers* 2022;14(21):5430.

POTENTIAL REGISTRATIONAL PATH IN RCC

Darlifarnib + Cabozantinib in Clear Cell Renal Cell Carcinomas

PRESENT STATUS	FIT-001 KURA KO-2806-001	Ph1b: darlifarnib + cabozantinib vs cabozantinib *(actively enrolling)*	**Data anticipated in 2027**
POTENTIAL INITIAL REGISTRATIONAL PATH	**2L/3L**	Phase 3 registrational study of darlifarnib + cabozantinib in refractory ccRCC	**Initiation anticipated in 2028**
EXPANSION POTENTIAL	**1L/2L**	Darlifarnib + TKI in triplet regimens with immuno-oncology (IO) agents and/or HIF2-a inhibitors in earlier line settings to enhance depth and durability	**Expansion anticipated in 2028+**

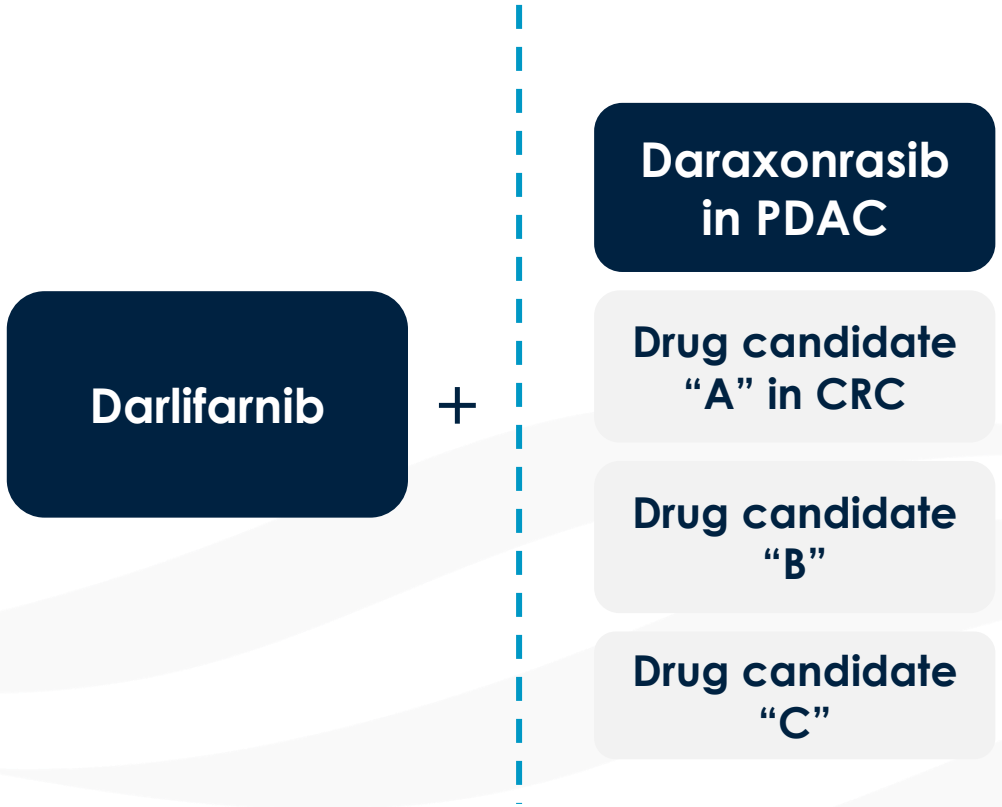
POTENTIAL REGISTRATIONAL PATH IN *KRAS*-MUTANT CANCERS

Darlifarnib + Mutant-Selective/pan-RAS/pan-KRAS Inhibitors

PRESENT STATUS	**New Platform Study**	First combination: Phase 1a/b with darlifarnib + daraxonrasib	**Initiation anticipated early 2027**
POTENTIAL INITIAL REGISTRATIONAL PATH	**2L+**	Build on SOC and/or investigational agents in *KRAS*-mutant solid tumors	**Initiation anticipated as early as 2028**
EXPANSION POTENTIAL	**1L**	Expand into earlier line settings to enhance depth and durability	**TBD**

PLATFORM STUDY ENABLES DEVELOPMENT ACROSS MULTIPLE TARGETED THERAPIES AND DISEASE SETTINGS

- Flexible design allows combinations with both approved therapies and investigational, targeted therapies
- Multiple combinations and indications can be evaluated in same study
- New combinations can be added
- Successful combinations "graduate" to dedicated registrational studies



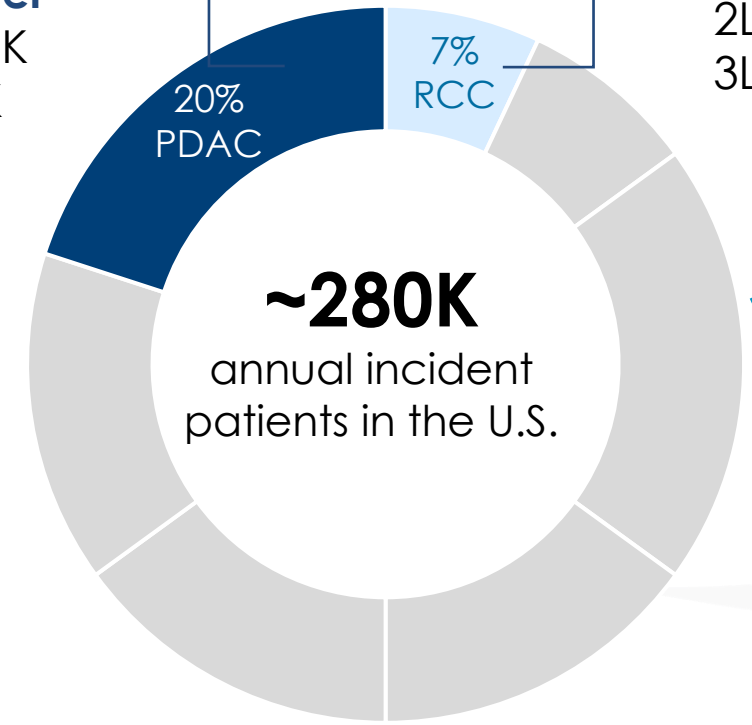
CLOSING REMARKS

Troy Wilson, Ph.D., J.D.

POTENTIAL ADDRESSABLE MARKET FOR DARLIFARNIB IN 2L/3L RCC AND PDAC

***KRAS*-m Pancreatic Cancer**
2L: 11K
3L: 3K

Renal Cell Carcinoma
2L: 12K
3L: 9K



Market Opportunity

RCC[1] $8B/yr
$6B in 2L
$2B in 3L+

PDAC[2] $6B/yr
$5B in 2L
$1B in 3L+

[1]RCC Assumptions: Overall RCC epidemiology based on DRG RCC Forecast Report, 2025; 2L: 12K eligible patients; 3L: 5K eligible patients annually; DOT 2L: 15 months, 3L 8 months; $40K/month per patient; [2]PDAC Assumptions: Overall PDAC epidemiology based on Pant *et al.*, ascopubs.org, 2026; 2L+ treatment assumptions based on CancerMPact 2025 TA US Pancreatic Cancer Report, 2025; DOT 2L: 11 months, 3L: 8 months; $40k/month per patient

KURA ONCOLOGY HAS A COMPELLING VALUE PROPOSITION IN 2026 AND BEYOND

Ziftomenib

- **FDA Approved KOMZIFTI™** for adult R/R *NPM1*-mutated AML patients
- Robust new patient starts and **early launch momentum**
- Advancing ziftomenib to address up to **50% of AML patients**
- **Multiple 2026 readouts** expected to support ziftomenib as a **broadly combinable AML backbone**

Darlifarnib

- Mechanism-driven, targeted therapy-agnostic **combination platform**
- Phase 1b expansion with **cabozantinib in RCC** underway
- Phase 1a escalation with **daraxonrasib in 2L+ PDAC** planned
- Precision combination(s) platform study has potential to provide **additional optionality and value creation**

Strong Financial Position: $580.8M in cash and investments as of 3/31/26, plus $180M in anticipated payments

ABBREVIATIONS

1l: first line
2L: second line
3L: third line
4L: fourth line
AKT: protein kinase B
AML: acute myeloid leukemia
BID: twice daily
BRAF: B-Raf proto-oncogene
ccRCC: clear cell renal carcinoma
CDX: caudal gene
CRC: colorectal cancer
EGFR: epidermal growth factor receptor
FLT3: fms-like tyrosine kinase 3
Ftase: farnesyltransferase
FTI: farnesyl transferase inhibitor
GILT: gilteritinib
GIST: gastrointestinal stromal tumor
HIF2-a: hypoxia-inducible factor 2-alpha
HNSCC: head and neck squamous cell carcinoma
IO: immuno-oncology therapy
KIT: targeted cancer therapies and treatments that block the activity of the KIT receptor tyrosine kinase
KRAS: Kirsten rat sarcoma viral oncogene homolog
KRASi: Kirsten rat sarcoma viral oncogene homolog inhibitor
MAPK: mitogen-activated protein kinase
mDOR: median duration of response
MEK: mitogen-activated protein kinase enzyme
MOS: median overall survival
MTOR: mammalian target of rapamycin
mTORC1: mechanistic target or rapamycin complex 1
NE: non estimable
NET: neuroendocrine tumor
NPM1: Nucleophosmin 1
NSCLC: non-small cell lung cancer
ORR: objective response rate
PD: progressive disease or pharmacodynamics
PDAC: pancreatic ductal adenocarcinoma
PDX: patient-derived xenograft
PIK3a: phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit alpha
PK: pharmacokinetics
PR: partial response
PS: performance status
RAS: rat sarcoma
RCC: renal cell carcinoma
RHEB: Ras homolog enriched in brain
R/R: relapsed/refractory
SAE: serious adverse events
SD: stable disease
SOC: standard of care
SoD: sum of diameters
TAM: total addressable market
TEAEs: treatment emergent adverse events
TKI: tyrosine kinase inhibitor
TL: target lesion
TORC1: target of rapamycin complex 1
TRAEs: treatment-related adverse events
QD: once daily
Quiz: quizartinib
VEGF: vascular endothelial growth factor
Ven/aza: venetoclax + azacitidine
-i: inhibitor
-m: mutated
-r: rearranged

QUESTIONS & ANSWERS

THANK YOU

Our goal is to develop transformative therapies to extend and improve the lives of patients with cancer