NASDAQ: MRTX

MIRATI THERAPEUTICS

Targeting the genetic and immunological drivers of cancer

Corporate Overview Presentation

November 2022

Safe Harbor Statement

This presentation contains certain forward-looking statements regarding the business of Mirati Therapeutics, Inc. (“Mirati”). Any statement describing Mirati’s goals, expectations, financial or other projections, intentions or beliefs, development plans and the commercial potential of Mirati’s drug development pipeline, including without limitation adagrasib (MRTX849), sitravatinib, MRTX1133, MRTX1719 and MRTX0902 is a forward-looking statement and should be considered an at-risk statement. Such statements are subject to risks and uncertainties, particularly those challenges inherent in the process of discovering, developing and commercialization of new drug products that are safe and effective for use as human therapeutics, and in the endeavor of building a business around such drugs.

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Our Mission

To discover, design and deliver breakthrough therapies to transform the lives of patients with cancer and their loved ones.

Our Vision

Unified for patients, our vision is to unlock the science behind the promise of a life beyond cancer.

Developing Novel Oncology Therapies, Including Two Registration-Enabling Programs in Large NSCLC Patient Populations

IO Resistance

Sitravatinib Inhibitor of TAM and VEGFR2

KRAS Selective Inhibition

Adagrasib (MRTX849) G12C selective inhibitor

G12D selective (MRTX1133) and other KRAS selective inhibitors

Synthetic Lethality

MRTX1719 MTA Cooperative PRMT5 Inhibitor

Operational and commercial synergies across portfolio, particularly in NSCLC

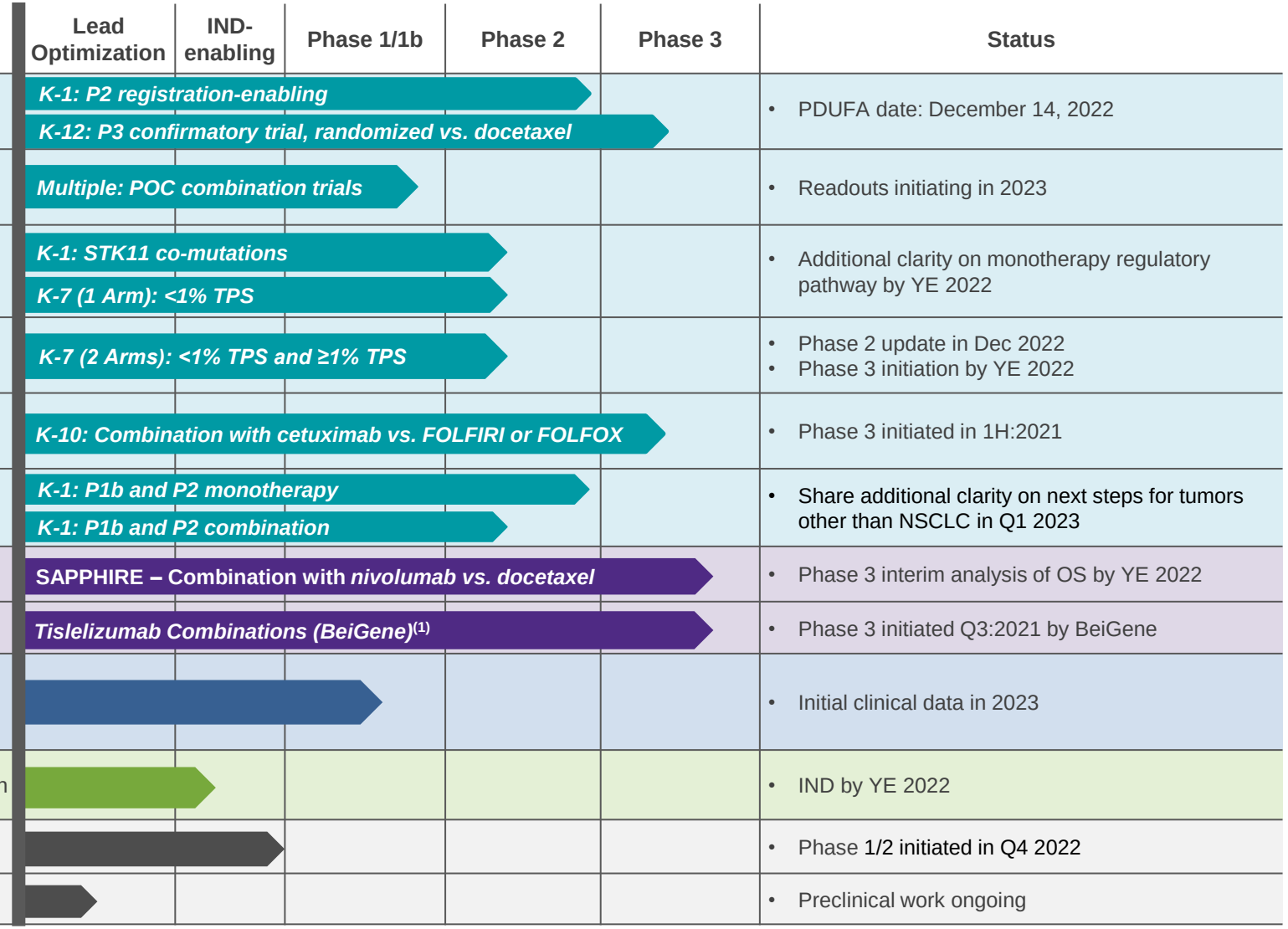
Advancing **targeted novel oncology research platform:** KRAS mutant inhibition and KRAS signaling modifiers (e.g., SOS1)

$1.2B in cash, cash equivalents and short-term investments as of 9/30/22

NSCLC = non-small cell lung cancer; CRC = colorectal cancer; IO = immuno-oncology; IND = investigational new drug; P = phase; MTA = methylthioadenosine; MTAP = methylthioadenosine phosphorylase; TAM = TYRO3, AXL and MER; VEGFR2 = vascular endothelial growth factor receptor 2

Mirati's Pipeline Spans Multiple Novel Targeted Oncology Programs

Compound	Indication	Development Approach	Status
Adagrasib ***KRAS G12C Inhibitor***	2L NSCLC	Monotherapy	• PDUFA date: December 14, 2022
		POC Combo: SHP2, SOS1, CDK4/6, Pan-EGFR, EGFR	• Readouts initiating in 2023
	1L NSCLC	Monotherapy: STK11 co-mutations and TPS <1%	• Additional clarity on monotherapy regulatory pathway by YE 2022
		Combo: Pembrolizumab (PD-1)	• Phase 2 update in Dec 2022 • Phase 3 initiation by YE 2022
	2L CRC	Combo: Cetuximab (EGFR)	• Phase 3 initiated in 1H:2021
	3L+ CRC and Pancreatic	Monotherapy Combo: Cetuximab (EGFR)	• Share additional clarity on next steps for tumors other than NSCLC in Q1 2023
Sitravatinib ***Multi Kinase Inhibitor***	2/3L NS-NSCLC	PD-1	• Phase 3 interim analysis of OS by YE 2022
	2/3L S + NS-NSCLC	PD-1	• Phase 3 initiated Q3:2021 by BeiGene
MRTX1719 ***MTA cooperative PRMT5 Inhibitor***	MTAP-deleted Cancers	Monotherapy	• Initial clinical data in 2023
MRTX1133 ***KRAS G12D Inhibitor***	Pancreatic, CRC, NSCLC	Monotherapy and combination	• IND by YE 2022
Additional KRAS pathway preclinical programs	Solid Tumors	MRTX0902 (SOS1 Inhibitor)	• Phase 1/2 initiated in Q4 2022
	Solid Tumors	Other KRAS mutations	• Preclinical work ongoing

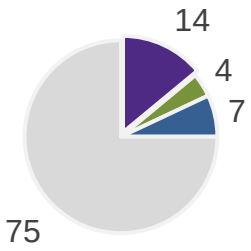


K = KRYSTAL (adagrasib trials); POC = proof of concept; NSCLC = non-small cell lung cancer; CRC = colorectal cancer; OS = overall survival; IND = investigational new drug; NDA = new drug application; TPS = tumor proportion score; ORR = objective response rate; MTAP = methylthioadenosine phosphorylase; CNS = central nervous system. 1. BeiGene is currently conducting certain combination studies of sitravatinib + tislelizumab for solid tumor indications in their territory in Asia (ex-Japan). These trials include a P3 trial in non-squamous and squamous NSCLC randomized vs. docetaxel, as well as proof-of-concept trials in hepatocellular carcinoma, renal cell carcinoma, ovarian cancer and gastric cancers.

Mirati: Deep Commitment to Addressing Cancers With High Unmet Needs

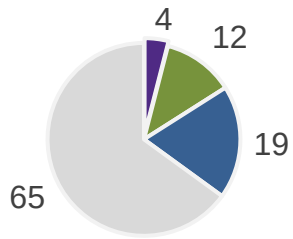
KRAS Prevalence in Tumors With High Unmet Needs[1-3]

NSCLC Adenocarcinoma



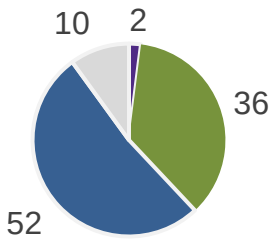
KRAS^{mt} Total	KRAS^{G12C}	KRAS^{G12D}
~25%	~14%	~4%

CRC



KRAS^{mt} Total	KRAS^{G12C}	KRAS^{G12D}
~35%	3-4%	~12%

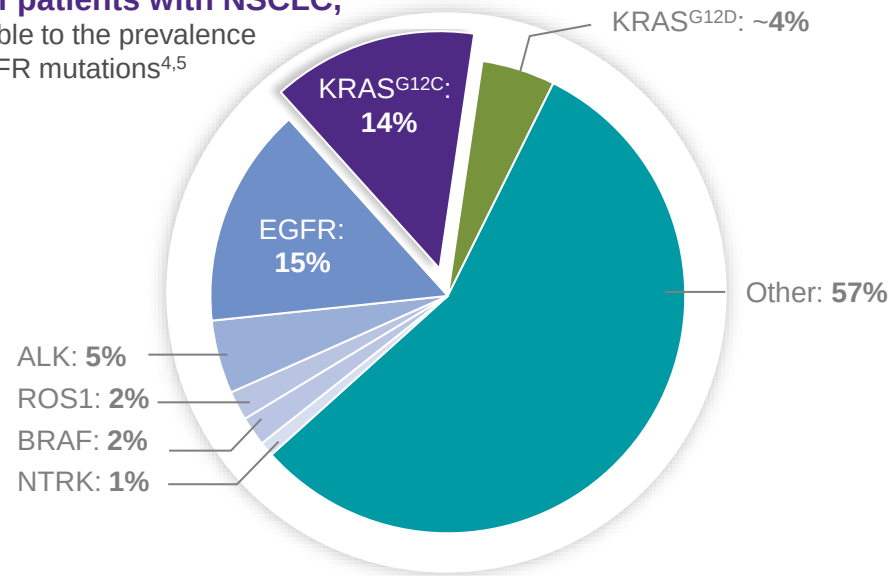
Pancreatic Cancer



KRAS^{mt} Total	KRAS^{G12C}	KRAS^{G12D}
~90%	~2%	~36%

Prevalence of Oncogenic Mutations in Lung Adenocarcinoma[4]

KRAS^{G12C} occurs in **~14% of patients with NSCLC,** comparable to the prevalence of all EGFR mutations[4,5]



Key: KRAS^{G12C} | KRAS^{G12D} | Other KRAS^{mut} | WT KRAS

- KRAS mutations are generally associated with poor prognosis
- The absence of known binding pockets made KRAS historically undruggable; discovery of the switch II binding pocket by Shokat et al has changed this

1. Zehir A, et al. *Nat Med*. 2017;23(6)703-713. 2. Krakstad C, et al. *PLoS One*. 2012;7(12):e52795. 3. NIH TCGA: *The Cancer Genome Atlas*. February 11, 2021. https://www.cbioportal.org. 4. Biernacka A, et al. *Cancer Genet*. 2016;209(5):195-198. 5. Pakkala S, Ramalingam SS. *JCI Insight*. 2018;3(15):e120858.

MIRATI THERAPEUTICS

Adagrasib (MRTX849):

$KRAS^{G12C}$ Selective Inhibitor

Adagrasib: Properties Include Complete Inhibition of KRAS^{G12C} for Full Dosing Interval, Long Half-Life, CNS Penetrance and Dose-Dependent PK

Long Half Life

Human Half Life ~24 hours[1]

Long half-life ensures pathway maximally inhibited throughout entire dosing interval

Comprehensive target coverage combats new KRAS protein synthesis (half-life ~ 24h) and reactivation of signaling[2]

CNS Penetrant

Encouraging CSF/CNS penetration

Encouraging and clinically meaningful adagrasib exposure in patients

Encouraging and durable CNS-specific activity in patients with both active, untreated and treated, stable CNS metastases

Extensive Tissue Distribution

Estimated Human Volume of Distribution (>10 L/Kg[3])

Maximize systemic exposure for duration of dosing

Extensive volume of tissue distribution ensures optimal target coverage throughout dosing interval

PK Profile / dosing

Dose Dependent PK Exposure Response

Dose-dependent PK and emerging exposure-response relationship for adagrasib supports dose modification schema and selected combination strategies

NSCLC = non-small cell lung cancer; CSF = cerebrospinal fluid; CNS = central nervous system; POC = proof of concept; PK = pharmacokinetic

1.Presented at the 32nd EORTC-NCI-AACR Symposium, October 24-25, 2019; 2. Stites and Shaw, CPT Pharmacometrics Syst. Pharmacol. (2018); 3. Estimated from nonclinical data and PBPK modeling;

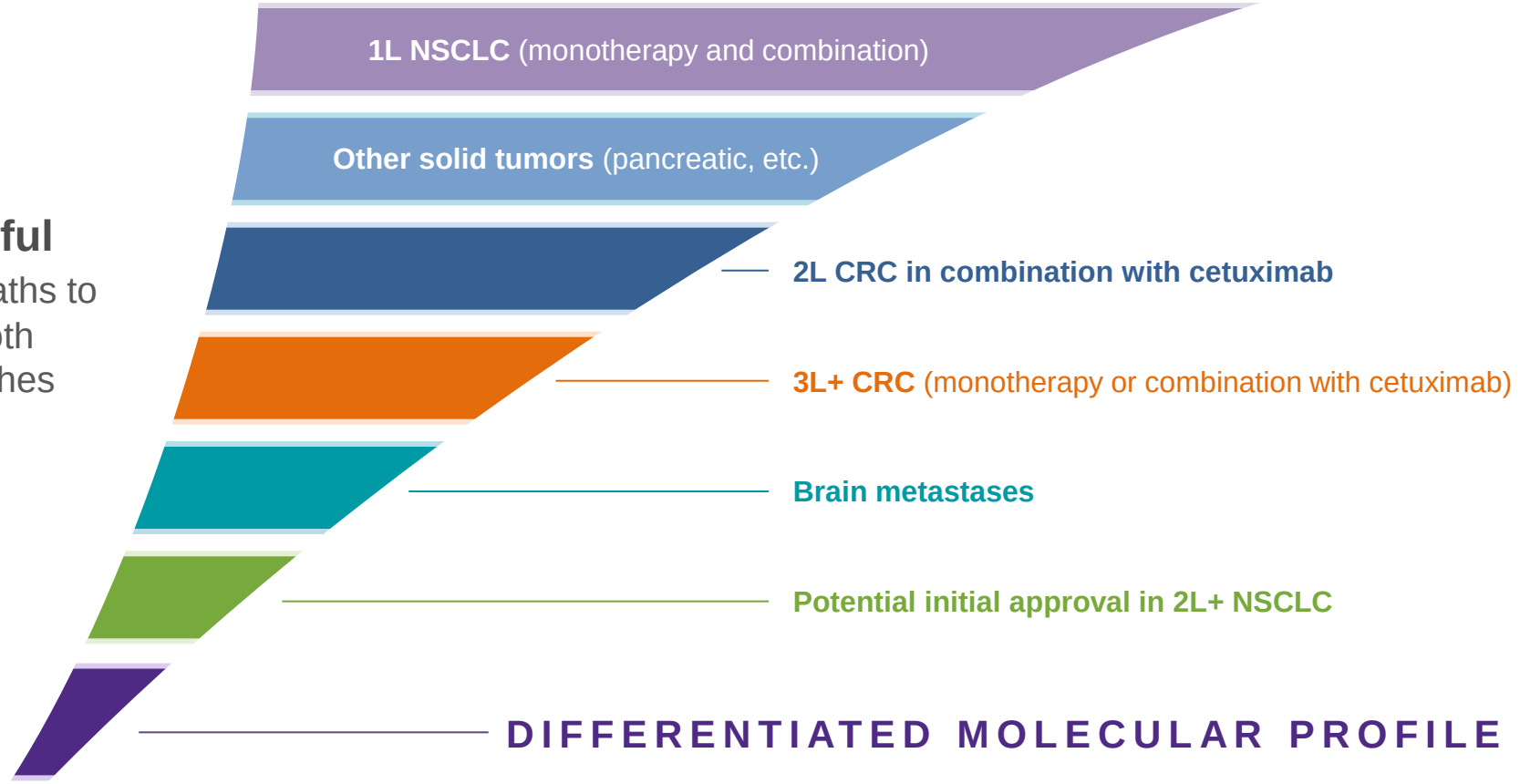
Adagrasib: Potentially Differentiated Therapy in NSCLC, CRC and Other Tumors for Patients with KRAS^{G12C} Mutations

- Molecular profile, including differentiated pharmacokinetic properties, long half-life and CNS penetration
  - Encouraging clinical and preclinical evidence of activity in the brain
- NSCLC: 2nd Line+ NSCLC: clinically meaningful response rate and initial durability in heavily pretreated patients
  - 1st Line NSCLC: preliminary findings support moving forward with 400 mg BID dose of adagrasib in combination with full dose pembrolizumab; Phase 2 KRYSTAL-7 study ongoing
- CRC: 3rd Line+: Response rate and initial durability in heavily pretreated patients both in monotherapy and in combination with cetuximab
  - 2nd Line: Phase 3 randomized trial in combination with cetuximab ongoing
- Other solid tumors
  - Encouraging preliminary results in pancreatic cancer and other solid tumor settings

CNS = central nervous system; NSCLC = Non-Small Cell Lung Cancer; CRC = colorectal cancer

Adagrasib is Poised to be a Leading Brand with a Potential to Positively Impact a Wide Range of Patients with KRAS^{G12C}-Mutated Cancers

Adagrasib's clinically meaningful profile potentially provides multiple paths to long-term value optimization through both monotherapy and combination approaches



Adagrasib: Highly Experienced Commercial Oncology Team Preparing for Successful Launch in 2022

Proven Differentiated Profile

- Clinically meaningful efficacy enabled by a 24-hour half-life that covers the target through the dosing cycle
- Robust early clinical activity in colorectal and pancreatic cancers
- Encouraging early data in patients with brain metastases

Top Biotech and Pharma Talent

- Ability to recruit and retain top talent across biotech and pharma given overwhelming interest in commercialization roles
- Experienced management team with significant oncology launch experience:

TAGRISSO osimertinib | OPDIVO (nivolumab) | IBRANCE palbociclib | XALKORI CRIZOTINIB | SUTENT sunitinib malate | KEYTRUDA (pembrolizumab)

Integrated Execution and Relentless Mindset

- Cross functional and integrated teams in place, including Medical Affairs, Sales Management, Market Access and R&D
- The Covid-19 pandemic has changed the rules of engagement of prescriber access, leveling the playing field between biotech and big pharma from "repetition" to "relevance"

Well Capitalized

- Available capital that provides sufficient runway to support commercialization and continued development of clinical and preclinical portfolio
- Commercial team progressively built over previous 2+ years

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Adagrasib (MRTX849): *Advanced Non-Small Cell Lung Cancer*

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Adagrasib ($KRAS^{G12C}$ Selective Inhibitor):

Cohort A Phase 2 Registrational Data

Adagrasib in Previously Treated Patients with KRAS^{G12C}-mutated NSCLC: *Tumor Response by BICR*

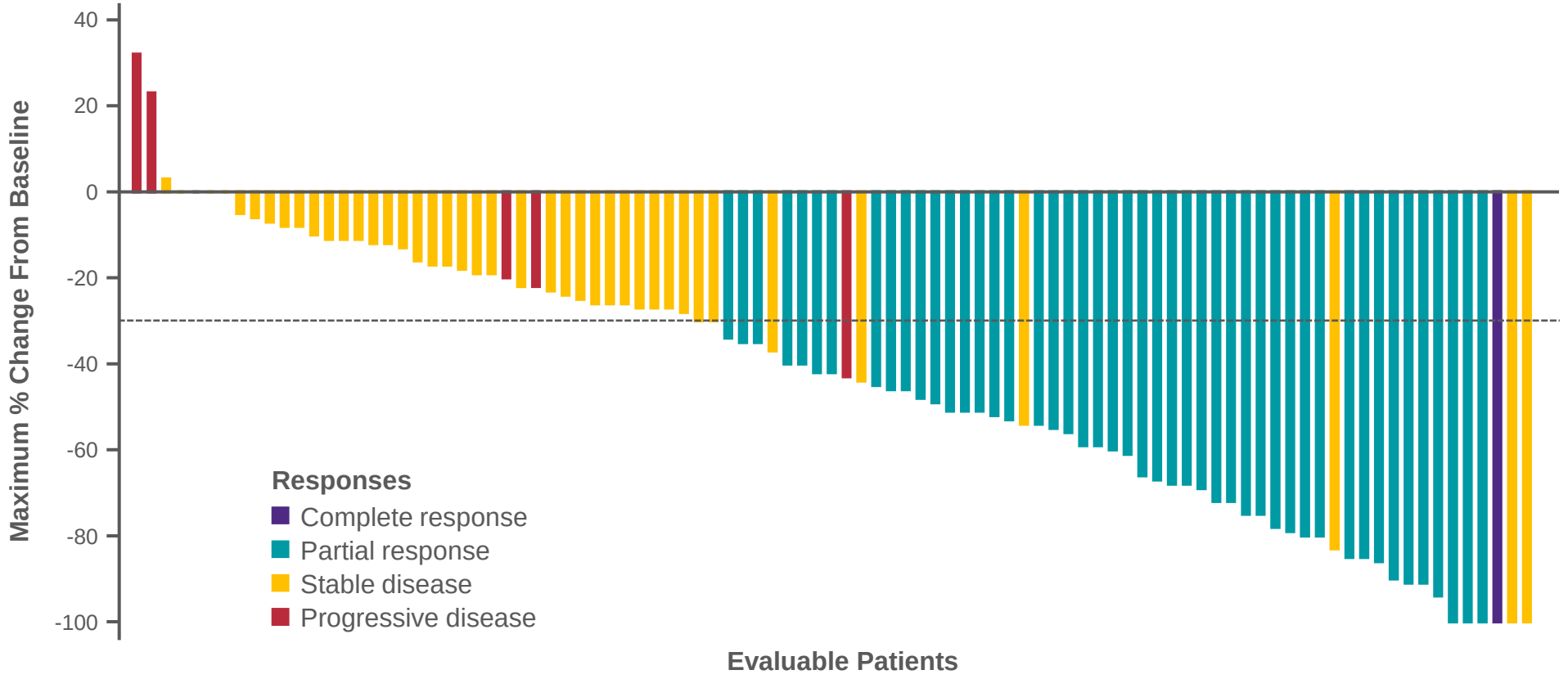
Efficacy Outcome	Adagrasib Monotherapy (n=112)[a]
Objective response rate, n (%)	48 (43%)
Best overall response, n (%)	
Complete response	1 (1%)
Partial response	47 (42%)
Stable disease	41 (37%)
Progressive disease	6 (5%)
Not evaluable	17 (15%)
Disease control rate, n (%)	89 (80%)

- 17 patients were not evaluable due to having received post-baseline scans too early (n=3) or study withdrawal prior to first scheduled assessment (n=14)[b]
- For evaluable patients (on treatment and who had a scan at ~6 weeks[c]), ORR was 51% (48/95)

[a]Full analysis set as per BICR excludes 4 patients who did not have measurable disease at baseline; [b]Due to reasons of: withdrawal by patient (n=5), AEs (n=3; 2 patients experienced AEs not related to treatment, 1 patient experienced a TRAE), global deterioration of health (n=3), death (n=2), non-compliance (n=1); [c]6 weeks ± 10 days

Data as of October 15, 2021 (median follow-up: 12.9 months)

Adagrasib in Previously Treated Patients with KRAS^{G12C}-mutated NSCLC:
Best Tumor Change From Baseline

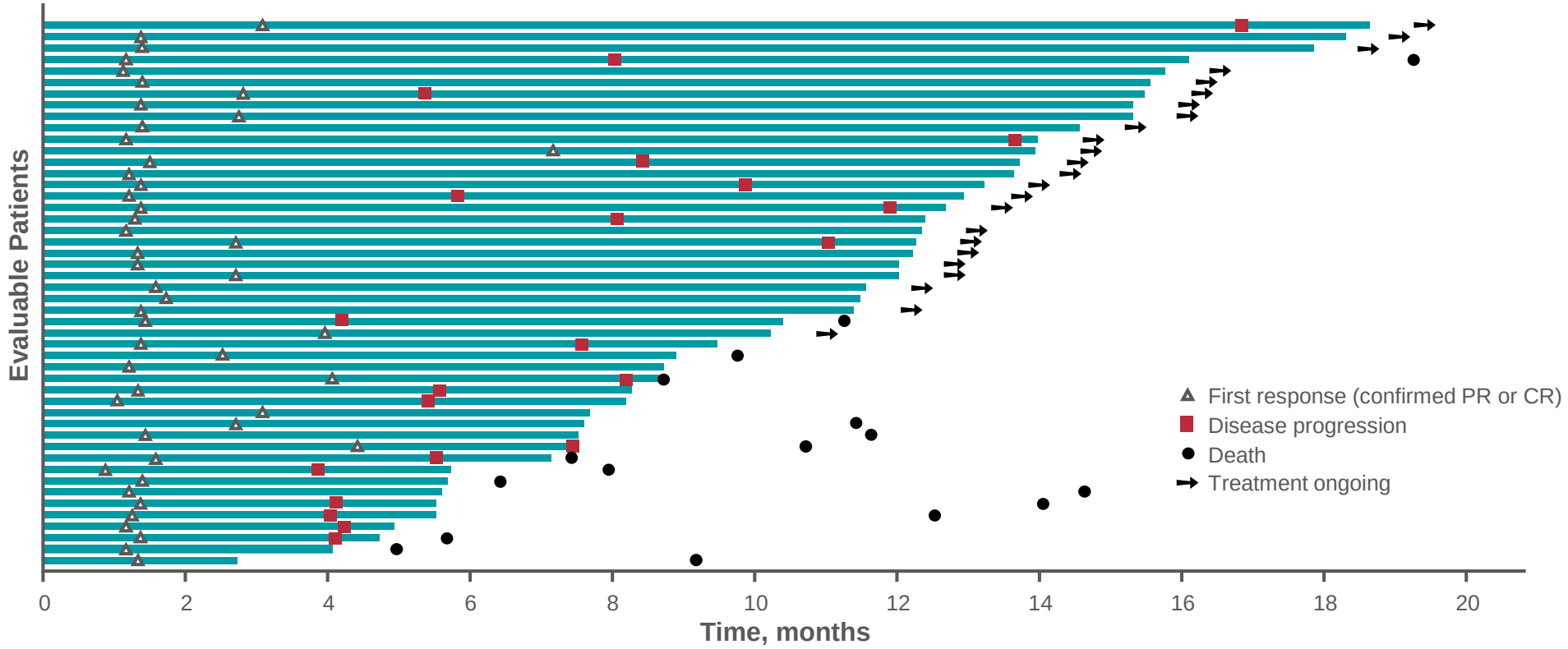


- Objective responses were observed in 43% (95% CI, 33.5–52.6); DCR was 80% (95% CI, 70.8–86.5)
- Responses were deep with 75% of responders achieving >50% tumor reduction

All results are based on BICR. Responses include target lesion tumor regression, as well as non-target lesion assessment

Data as of October 15, 2021 (median follow-up: 12.9 months)

Adagrasib in Previously Treated Patients with KRAS^{G12C}-mutated NSCLC: *Duration of Response*



- Median TTR was 1.4 months (range, 0.9–7.2)
- Median DOR was 8.5 months (95% CI, 6.2–13.8)
- Treatment is ongoing in 50% (24/48) of patients who experienced a response, and 33% (16/48) are still in response

All results are based on BICR. The median duration of treatment was 5.7 months (range, 0.03–19.6)

Data as of October 15, 2021 (median follow-up: 12.9 months)

Treatment-Related Adverse Events

	Adagrasib Monotherapy (N=116) Capsule, Fasted	
TRAEs, n (%)	**Any Grade**	**Grades 3–4**
Any TRAEs	113 (97%)	50 (43%)
Most frequent TRAEs[a], n (%)		
Diarrhea	73 (63%)	1 (<1%)
Nausea	72 (62%)	5 (4%)
Vomiting	55 (47%)	1 (<1%)
Fatigue	47 (41%)	5 (4%)
ALT increase	32 (28%)	5 (4%)
Blood creatinine increase	30 (26%)	1 (<1%)
AST increase	29 (25%)	4 (3%)
Decreased appetite	28 (24%)	4 (3%)

- Grade 1–2 TRAEs occurred in 53% of patients
- There were 2 grade 5 TRAEs (cardiac failure [n=1] and pulmonary hemorrhage [n=1])
- TRAEs led to dose reduction in 60/116 (52%) patients[b] and to dose interruption in 71/116 (61%) patients
- TRAEs led to discontinuation of study drug in 8/116 (7%) patients

[a]Occurring in >20% of patients (any grade), TRAEs occurring in >15% of patients were anemia (21 [18%]), amylase increase (20 [17%]) and QT prolongation (19 [16%]);
[b]Percentage of patients who experienced dose reductions: 400 mg BID (33%), 600 mg QD (11%), 200 mg BID/400 mg QD (14%)

Data as of October 15, 2021 (median follow-up: 12.9 months)

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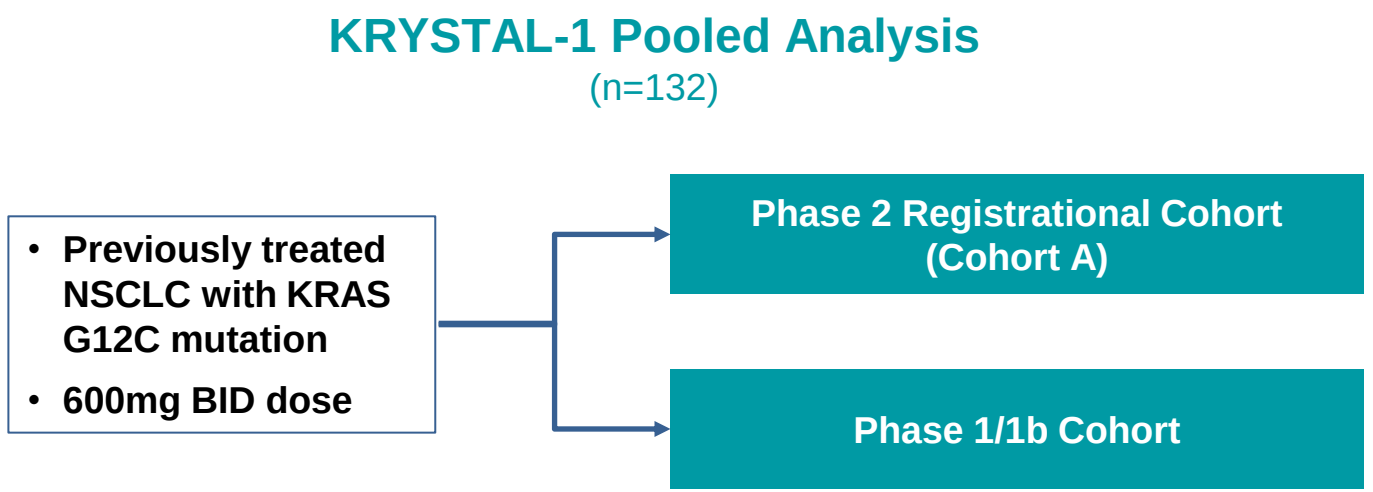
Adagrasib ($KRAS^{G12C}$ Selective Inhibitor):

Pooled Analysis of 600mg BID NSCLC – Phase 1/1b + Phase 2

Pooled Analysis: Registrational Phase 2 and Phase 1/1b NSCLC Cohorts of KRYSTAL-1 Evaluating Adagrasib at 600mg BID Dose

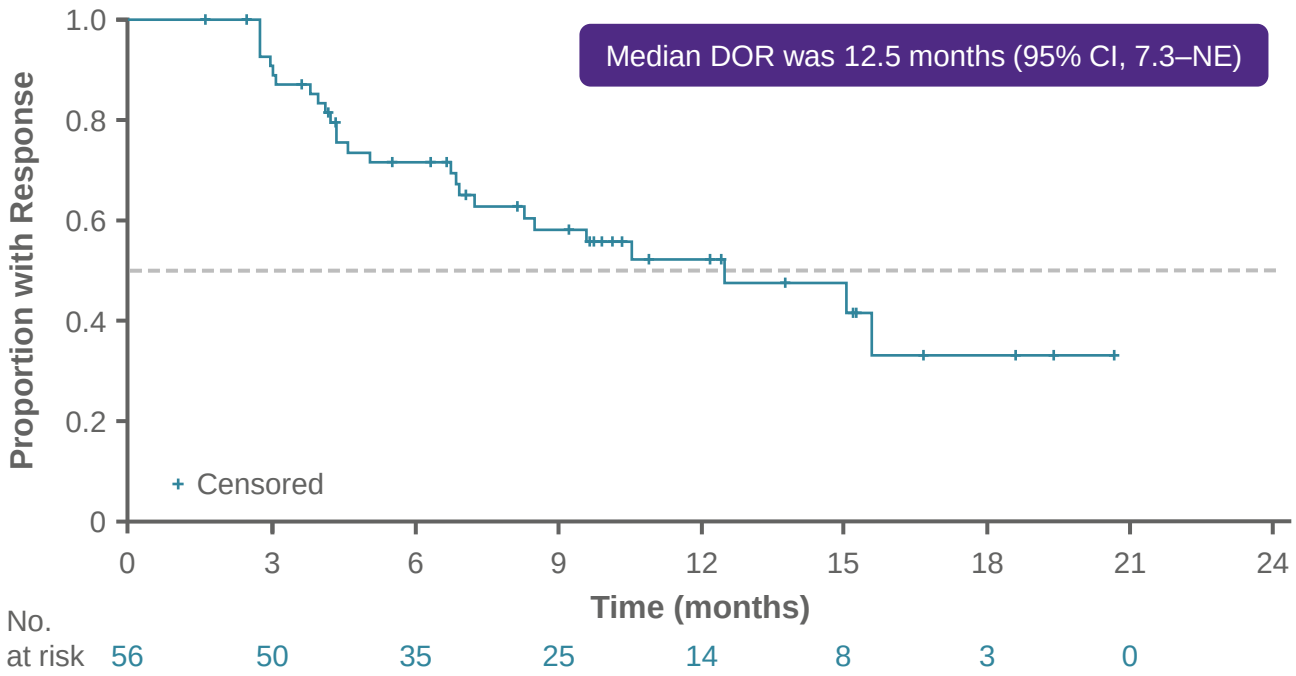


- Baseline characteristics of patients across both cohorts included in pooled analysis were generally consistent
- The safety and tolerability observed in this pooled analysis was consistent with findings reported in the registration-enabling Phase 2 (Cohort A) for adagrasib in patients with advanced NSCLC

Adagrasib Monotherapy in Previously-Treated NSCLC: *Tumor Response*

Pooled dataset: *Phase 1b/2 Patients with NSCLC Enrolled at 600mg BID*

Efficacy Outcome	Adagrasib Monotherapy, 600 mg BID Pooled (N=128)
Objective response rate, n (%)	56 (44%)
Best overall response, n (%)	
Confirmed complete response	3 (2%)
Confirmed partial response	53 (41%)
Stable disease	47 (37%)
Progressive disease	7 (6%)
Not evaluable	18 (14%)
Disease control rate, n (%)	103 (81%)

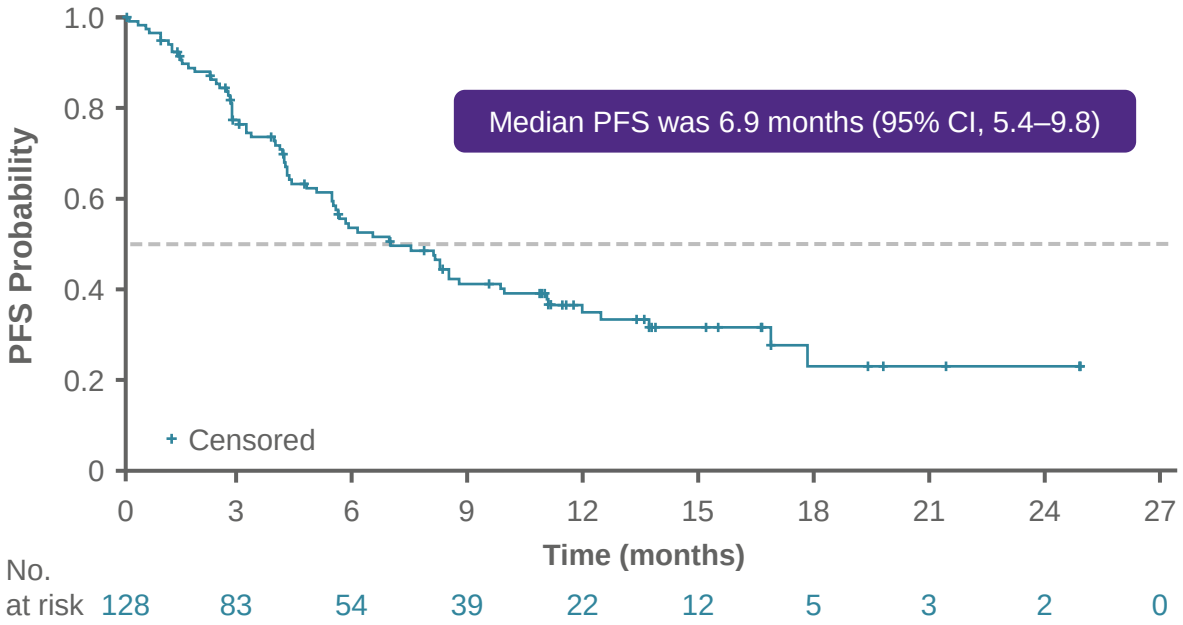


All results are based on BICR
Data as of October 15, 2021

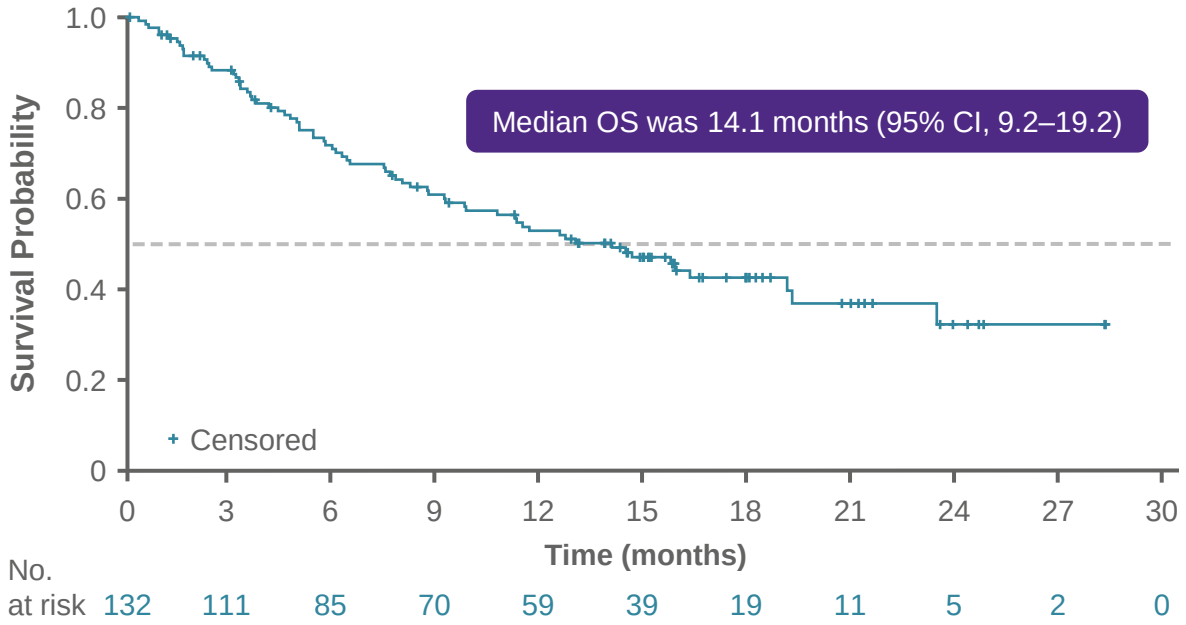
Adagrasib Monotherapy in Previously-Treated NSCLC: *Survival Outcomes*

Pooled dataset: *Phase 1b/2 Patients with NSCLC Enrolled at 600mg BID*

Progression-Free Survival[a]



Overall Survival[b]



PFS results are based on BICR

[a]Data as of October 15, 2021; [b]Data as of January 15, 2022

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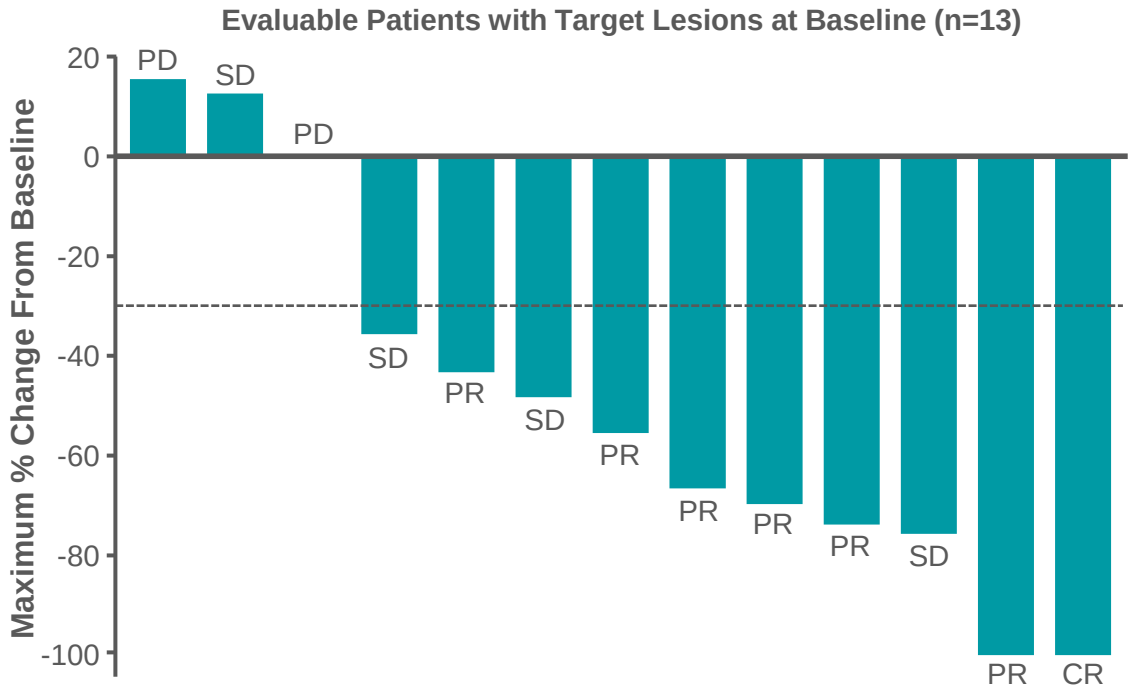
Adagrasib ($KRAS^{G12C}$ Selective Inhibitor):

Cohort A Subset Analysis

Adagrasib in Previously Treated Patients with KRAS^{G12C}-mutated NSCLC:
Intracranial Response in Patients with Treated, Stable CNS Metastases[a]

Best Overall Response	Overall (n=33)[b]	Patients with Non-target Lesions Only (n=19)	Patients with Target Lesions (n=13)[c]
IC ORR, n (%)	**11 (33%)**	**4 (21%)**	**7 (54%)**
Complete response	5 (15%)	4 (21%)	1 (8%)
Partial response	6 (18%)	-	6 (46%)
Stable disease	17 (52%)	13 (68%)	4 (31%)
IC DCR, n (%)	**28 (85%)**	**17 (89%)**	**11 (85%)**



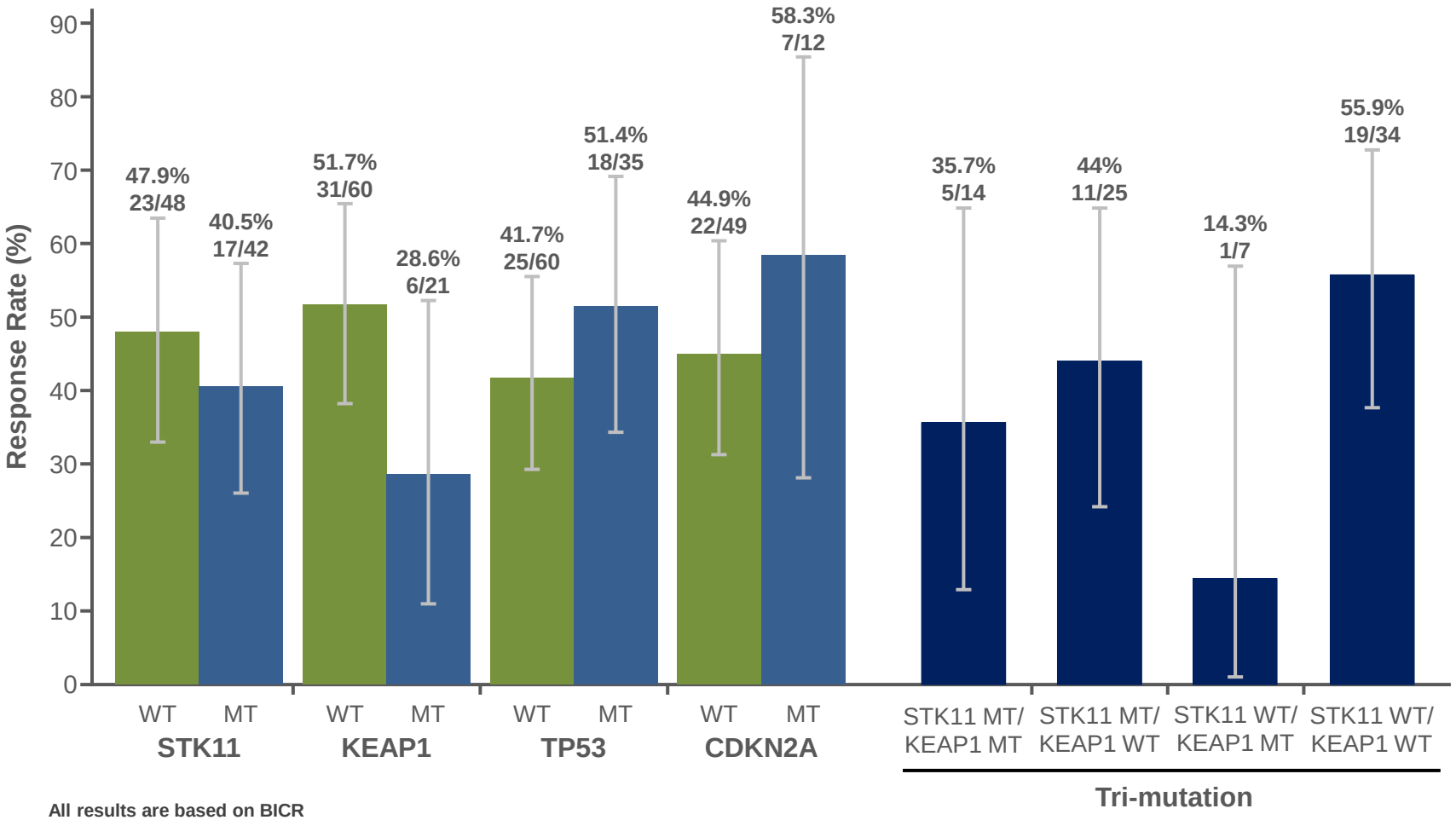
- IC ORR by modified RANO-BM was 33% (95% CI, 18–52); median IC DOR was 11.2 months (95% CI, 3.0–NE)
- IC DCR was 85% (95% CI, 68–95); median IC PFS was 5.4 months (95% CI, 3.3–11.6)

Target lesions: all measurable lesions (size ≥5 mm) with ≤5 lesions in total, and representative of all involved organs; non-target lesions: all non-measurable lesions and measurable lesions not identified as target lesions
[a]Among patients with adequately treated, stable CNS metastases, 33 patients were radiographically evaluable (i.e., had a baseline and on-treatment brain scan for evaluation), of whom 27 (82%) received radiation prior to adagrasib treatment (59% <3 months before study entry and 37% ≥6 months before study entry); [b]One patient with tumor shrinkage of 8% was deemed to be 'not evaluable' as the post-baseline scan was performed too early for evaluation; [c]Patients with target lesions may have also had non-target lesions

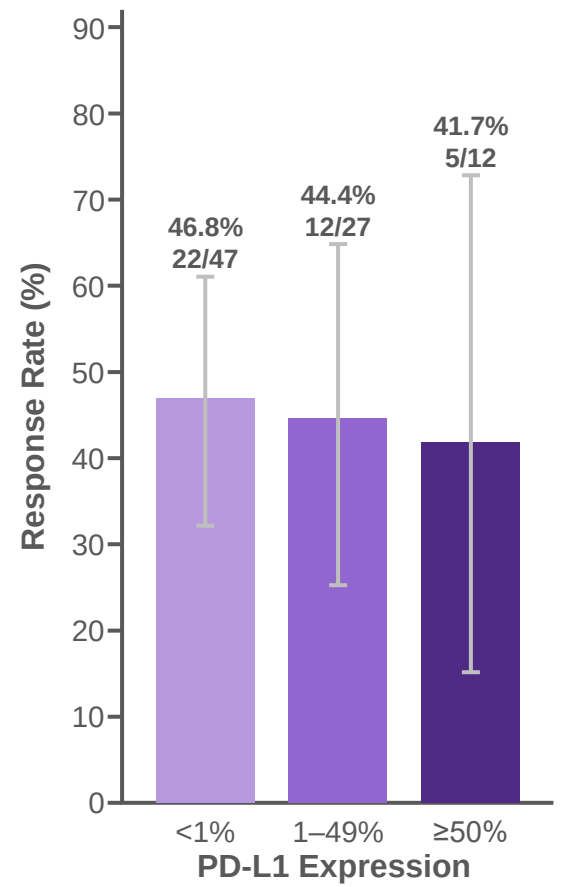
Data as of December 31, 2021 (median follow-up: 15.4 months)

Adagrasib in Previously Treated Patients with KRAS^{G12C}-mutated NSCLC:
Pre-specified Correlative Analyses

ORR in Patients Harboring KRAS^{G12C} Co-mutations



ORR by PD-L1 Subgroups[a]



All results are based on BICR

[a]PD-L1 was centrally tested

Data as of October 15, 2021 (median follow-up: 12.9 months)

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Adagrasib ($KRAS^{G12C}$ Selective Inhibitor):

Active, Untreated CNS Metastases (KRYSTAL-1)

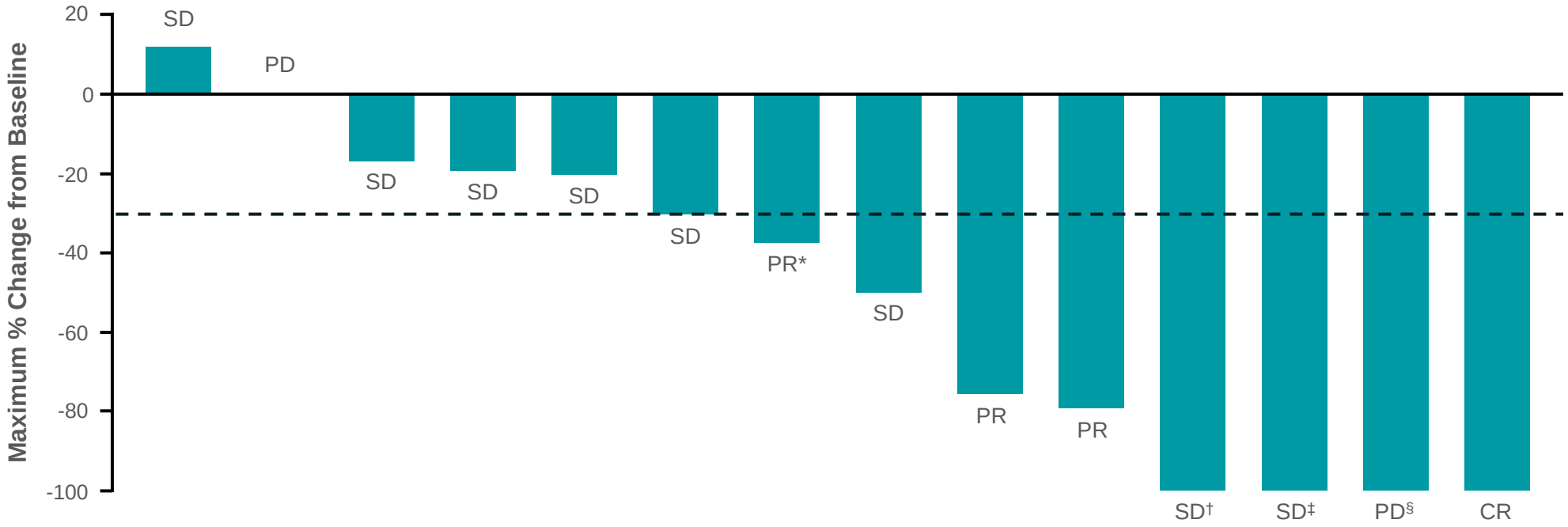
Adagrasib in Patients with Active, Untreated CNS Metastases:
Intracranial Response by BICR

Efficacy Outcome	Patients with Non-target Lesions Only (n=4)	Patients with Target Lesions (n=15)[a]	Overall (n=19)[b]
Objective response rate, n (%)	**2 (50%)**	**4 (27%)**	**6 (32%)**
Best overall response, n (%)			
Complete response (CR)	2 (50%)	1 (7%)	3 (16%)
Partial response (PR)	0	3 (20%)[c]	3 (16%)[c]
Stable disease (SD)	2 (50%)	8 (53%)	10 (53%)
Progressive disease (PD)	0	2 (13%)	2 (11%)
Not evaluable	0	1 (7%)[d]	1 (5%)[d]
Disease control rate, n (%)	**4 (100%)**	**12 (80%)**	**16 (84%)**

All results are based on BICR (mRANO-BM)
[a]Includes patients with target ± non-target lesions; [b]Includes patients in clinically evaluable population with ≥1 post-baseline assessment;
[c]Unconfirmed (n=1), confirmed CR after data cut-off; [d]Not evaluable (n=1) due to scans being too early (100% regression in target lesions)

Data as of December 31, 2021 (median follow-up: 6.6 months)

Adagrasib in Patients with Active, Untreated CNS Metastases:
Intracranial Best Tumor Change From Baseline

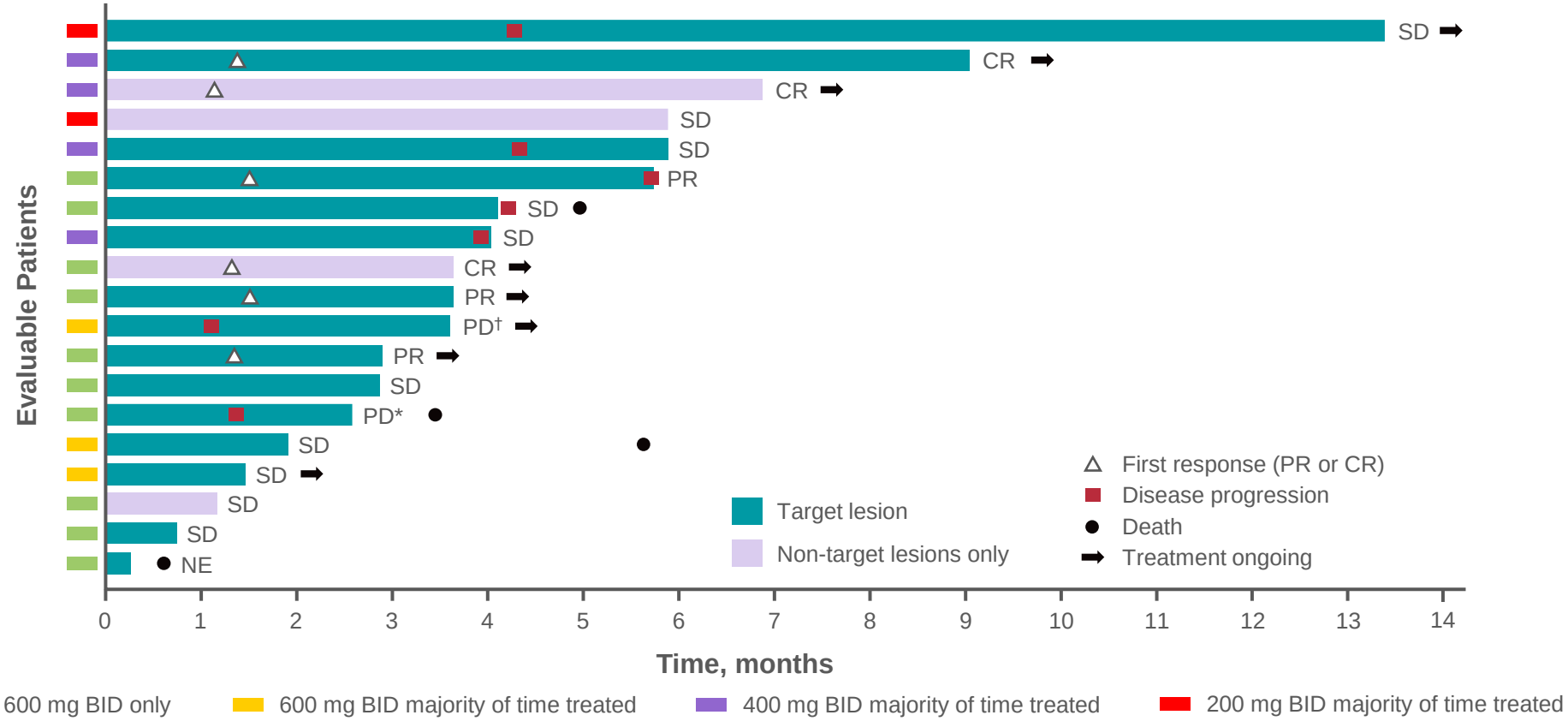


- Objective IC responses were observed in 32% (95% CI, 12.6–56.6)[a]
- IC DCR was 84% (95% CI, 60.4–96.6)

All results are based on BICR (mRANO-BM criteria). Only patients with target lesions and ≥1 post-baseline scans are shown; 1 patient not evaluable for best overall response due to scans being too early (100% regression in target lesions)
*Unconfirmed at data cut-off, confirmed CR after data cut-off; †SD due to non-target lesion progression; ‡Unconfirmed CR due to no subsequent scan; §PD due to new lesions
[a]Includes patients with target and non-target lesions

Data as of December 31, 2021 (median follow-up: 6.6 months)

Adagrasib in Patients with Active, Untreated CNS Metastases:
Duration of Treatment



- Median IC DOR was not reached (95% CI, 4.1–NE)[a]
- Median IC PFS was 4.2 months (95% CI, 3.8–NE)[b]; median OS had not been reached

All results are based on BICR (mRANO-BM criteria)
*IC BOR of PD, systemic BOR of PD; †IC BOR of PD, systemic BOR of SD; [a]Systemic mDOR of confirmed responses was 9.6 months (95% CI, 2.7–9.6); [b]Median systemic PFS was 5.6 months (95% CI, 3.8–11.0)

Data as of December 31, 2021 (median follow-up: 6.6 months)

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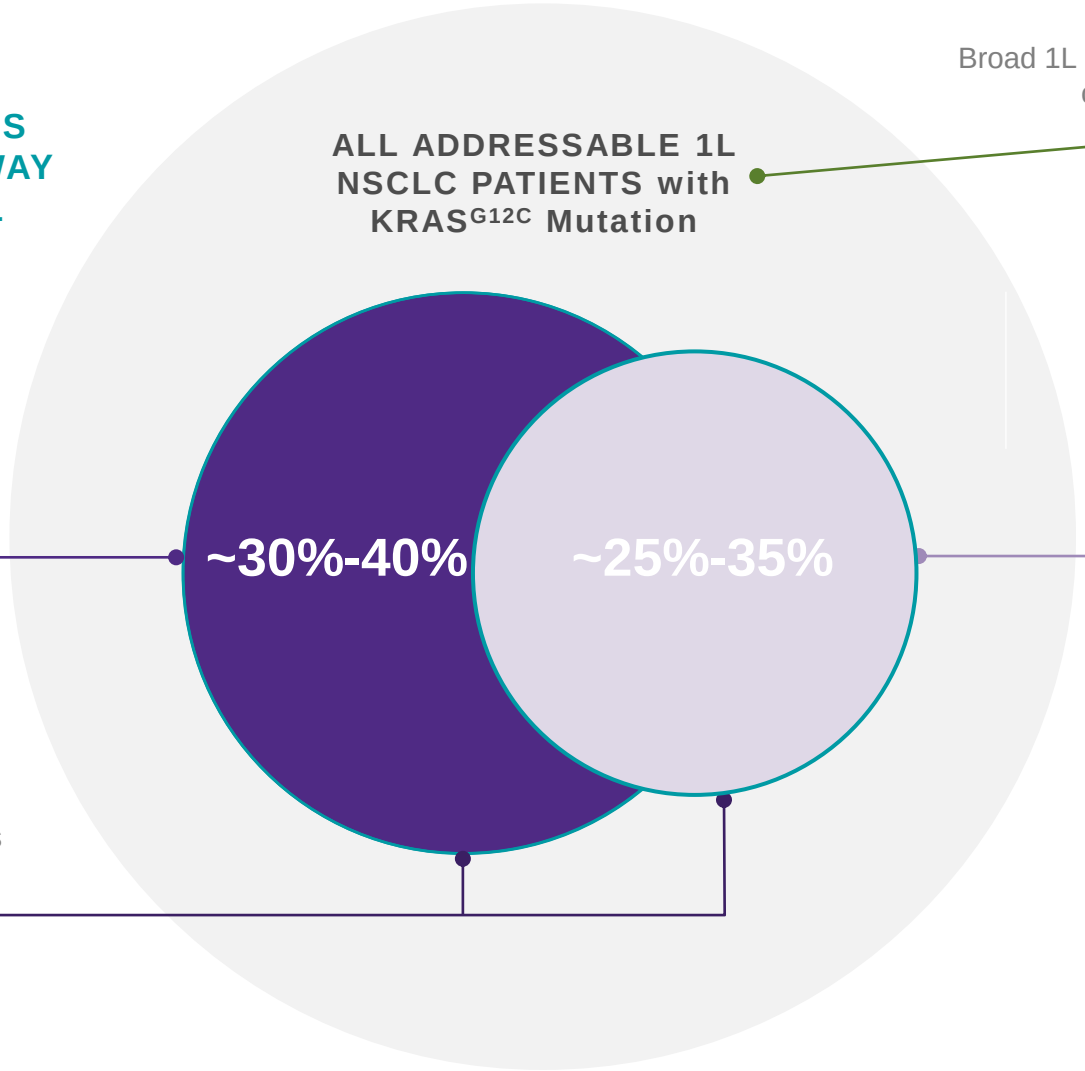
Adagrasib ($KRAS^{G12C}$ Selective Inhibitor):
First-Line NSCLC

Mirati is Pursuing Multiple Approaches to First Line Therapy in NSCLC

POOR PROGNOSIS POPULATIONS REPRESENT A POTENTIAL PATHWAY FOR ACCELERATED APPROVAL

COMBINATION THERAPY

Broad 1L NSCLC patient population addressable through combination development approaches



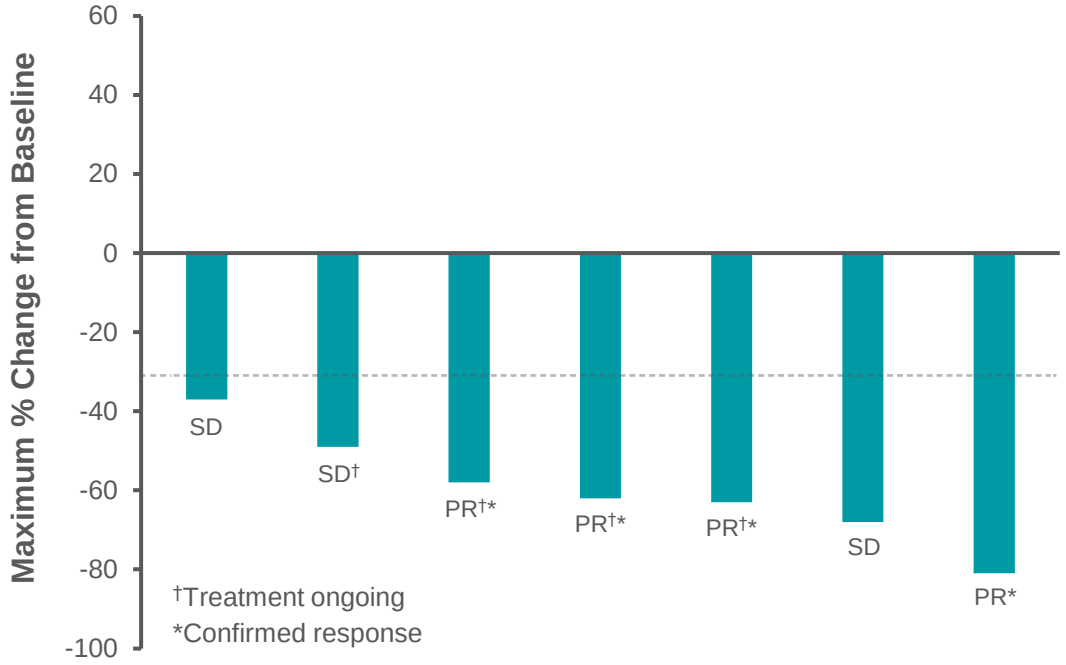
MONOTHERAPY

Patients with TPS scores < 1% or KRAS^{G12C}/STK11 co-mutations have a poor prognosis on SoC and represent a combined **> 40%** of 1L NSCLC patients

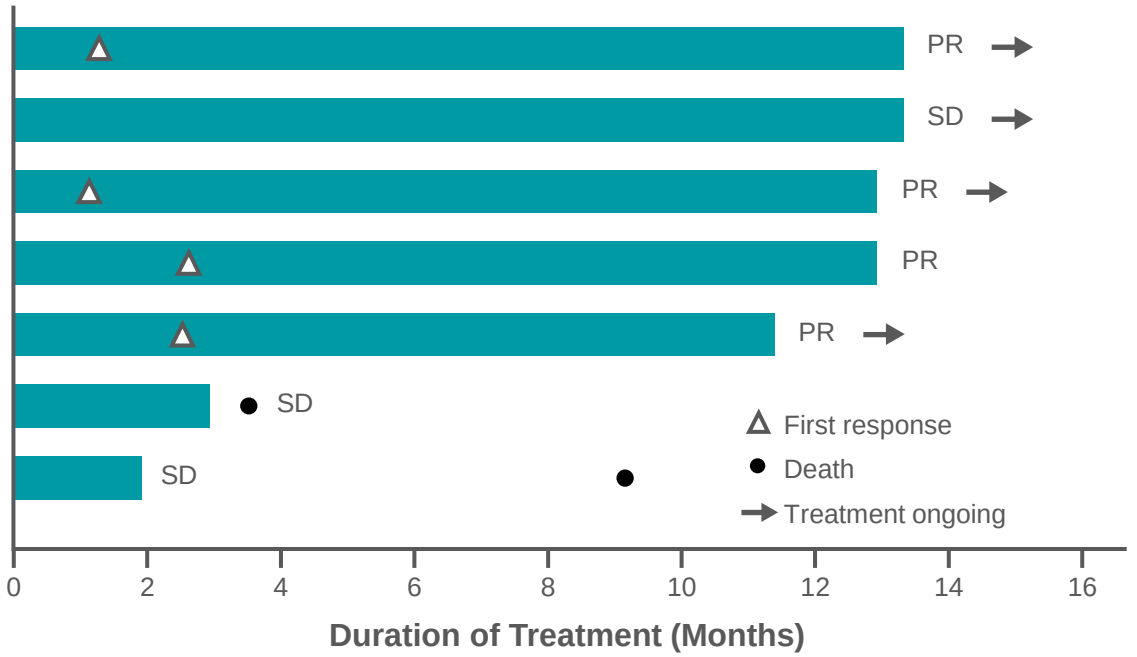
B. Ricciuti et al., "Diminished Efficacy of Programmed Death-(Ligand)1 Inhibition in STK11- and KEAP1-Mutant Lung Adenocarcinoma Is Affected by KRAS Mutation Status", J. Thorac. Oncol., 2021 (in press); H. West et al., Annals Oncol. 31, S817 (2020); TPS = tumor proportion score; NSCLC = non-small cell lung cancer; ORR = overall response rate; PFS = Progression Free Survival; SOC = standard of care

Phase 1b Adagrasib 400 mg BID with Pembrolizumab in Treatment Naïve KRAS^{G12C}-Mutated NSCLC: Efficacy Outcomes in Most Mature Dataset

Best Tumor Change from Baseline (n=7)



Duration of Treatment (n=7)



- 57% (4/7) ORR [a]; Responses were observed in 1/1 patients with PD-L1 TPS ≥50%, 2/3 patients with PD-L1 TPS 1–49%, and 1/2 patients PD-L1 <1%[b]
- Median duration of treatment was 12.9 months (range, 1.9–13.3)
- Median DOR was NR (95% CI, NE–NE), Median PFS was NR (95% CI, 3.6–NE)
- There were no TRAEs leading to dose discontinuation

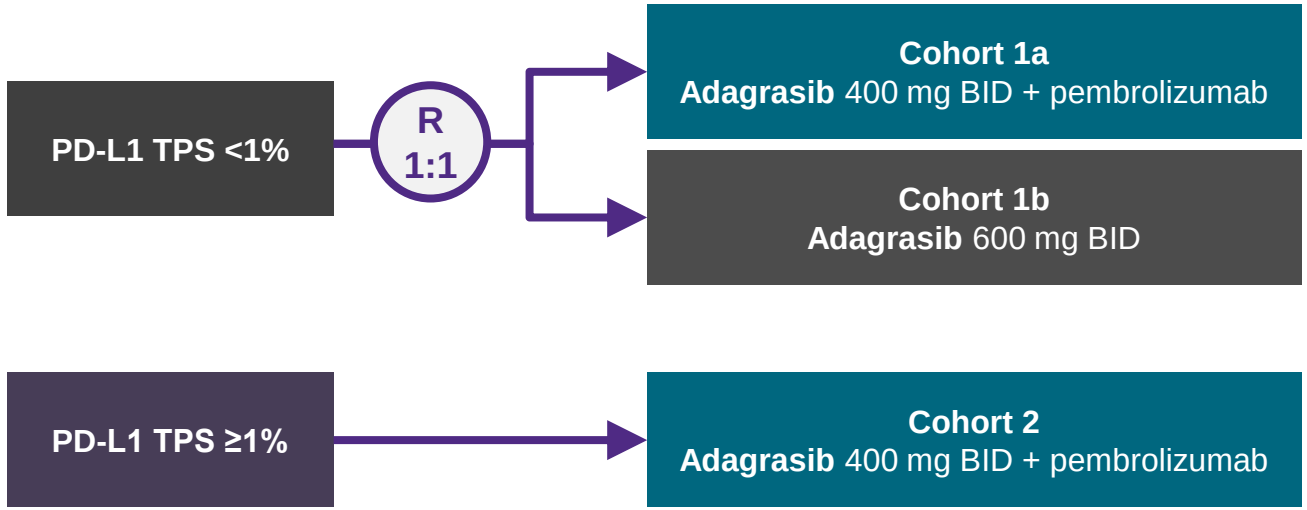
[a] One additional patient experienced a 49% tumor regression, which allowed for tumor resection prior to achieving RECIST-defined confirmed response; [b]n=1 patient PD-L1 status was unknown

 Data as of January 15, 2022

KRYSTAL-7 (849-007): A Phase 2 Trial of Adagrasib, Alone or in Combination With Pembrolizumab, in Patients with Advanced NSCLC with KRAS^{G12C} Mutation

Key Eligibility Criteria

- Advanced, unresectable or metastatic NSCLC with KRAS^{G12C} mutation based on sponsor-approved test
- No prior systemic therapy for locally advanced/metastatic disease
- No active brain metastases



Outcome Measures

Primary: ORR (RECIST 1.1)

Secondary: PFS, DOR, 1-year survival rate, OS, safety, PK

Adagrasib administered PO QD, in 3-weekly cycles; pembrolizumab, 200 mg Q3W

BID, twice-daily; DOR, duration of response; NSCLC, non–small-cell lung cancer; ORR, objective response rate; OS, overall survival; PD-L1, programmed death ligand-1; PFS, progression-free survival; PK, pharmacokinetics; PO, orally; QD, every day; Q3W, every 3 weeks; RECIST, Response Evaluation Criteria In Solid Tumors; TPS, tumor proportion score.

ClinicalTrials.gov. NCT04613596. Accessed May 31, 2022.

1L Adagrasib 400 mg BID with Pembrolizumab in KRAS^{G12C}-Mutated NSCLC: Treatment-Related AEs

	Adagrasib 400 mg BID + Pembrolizumab[a]	
	Grade 1/2 (N=37)	Grade 3/4 (N=37)
Any treatment-related AE[b], n (%)	**12 (32.4%)**	**16 (43.2%)**
Diarrhea	10 (27.0%)	1 (2.7%)
Nausea	8 (21.6%)	4 (10.8%)
Amylase increased	8 (21.6%)	0
Fatigue	7 (18.9%)	1 (2.7%)
ALT increased	**6 (16.2%)**	**2 (5.4%)**
AST increased	**6 (16.2%)**	**2 (5.4%)**
Blood alkaline phosphatase increased	6 (16.2%)	0
Decreased appetite	5 (13.5%)	0
Edema peripheral	4 (10.8%)	0
Vomiting	4 (10.8%)	0
Lipase increased	3 (8.1%)	5 (13.5%)

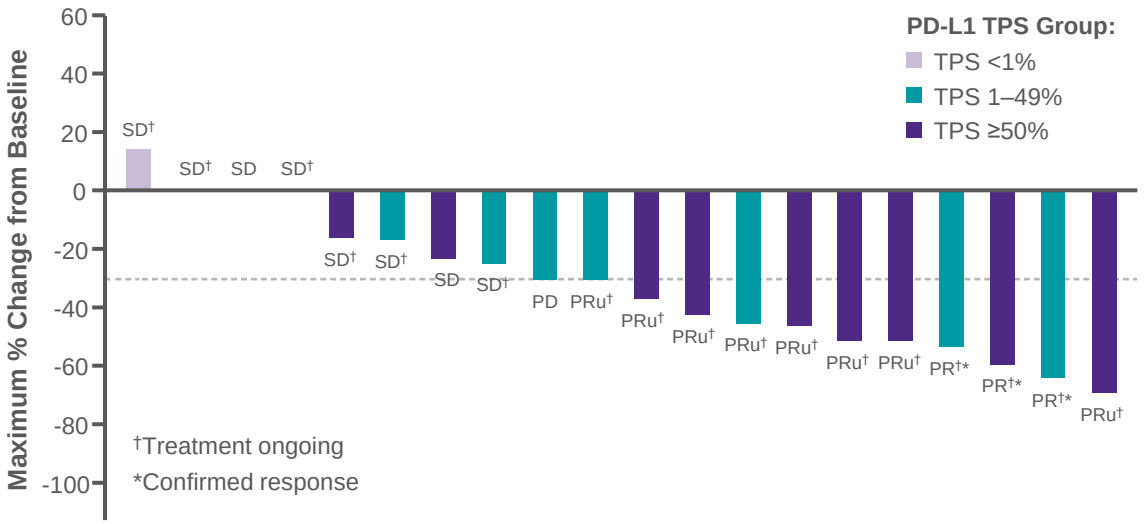
- There were no grade 5 TRAEs
- TRAEs resulted in treatment discontinuations in 1/37 (2.7%) of patients

[a]Pooled data from Cohorts 1a and 2; [b]Occuring in >10% of patients

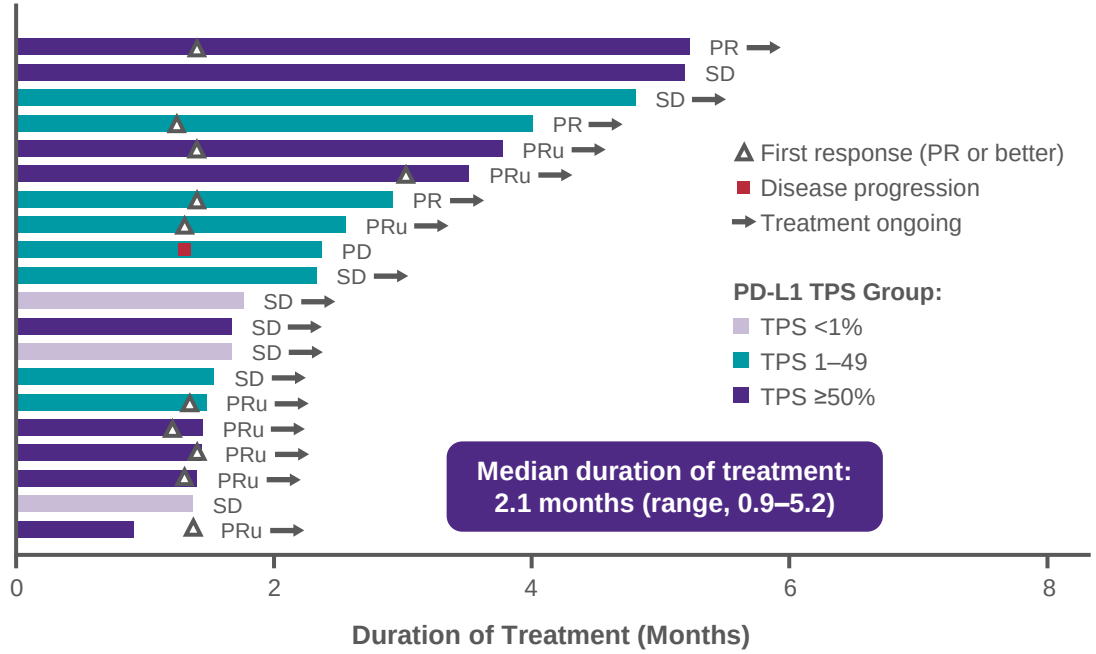
Data as of April 1, 2022

1L Adagrasib 400 mg BID with Pembrolizumab in KRAS^{G12C}-Mutated NSCLC: Efficacy Outcomes

Best Tumor Change from Baseline[a]



Duration of Treatment[a]



- ORR was 77% (7/9) in patients with PD-L1 TPS ≥50%, and 50% (4/8) in patients with PD-L1 TPS 1–49%

[a]n=20; one additional patient with a TPS score of <1% did not have post baseline scan at time of data cutoff

 Data as of April 1, 2022

MIRATI THERAPEUTICS

Adagrasib (MRTX849): Heavily Pretreated *Colorectal Cancer*

Prognosis on Standard of Care in CRC with KRAS^{G12C} Mutations Have Historically Been Worse Than the Broader CRC Population

Population	Historical Efficacy Outcomes 3rd Line and Beyond
KRAS-agnostic	• Regorafenib[1] or Trifluridine/Tipiracil[2,3]: – ORR: 1-2% – mPFS: 1.9-2.0 months – mOS: 6.4-8.0 months
KRAS-mutant	• Trifluridine/Tipiracil[3]: – KRAS-mut mOS = 6.5 months

- Patient outcomes in CRC have historically been poor and progressively worse in later lines of therapy
- KRAS-mutant CRC patients tend to have worse outcomes than the broader CRC patient population

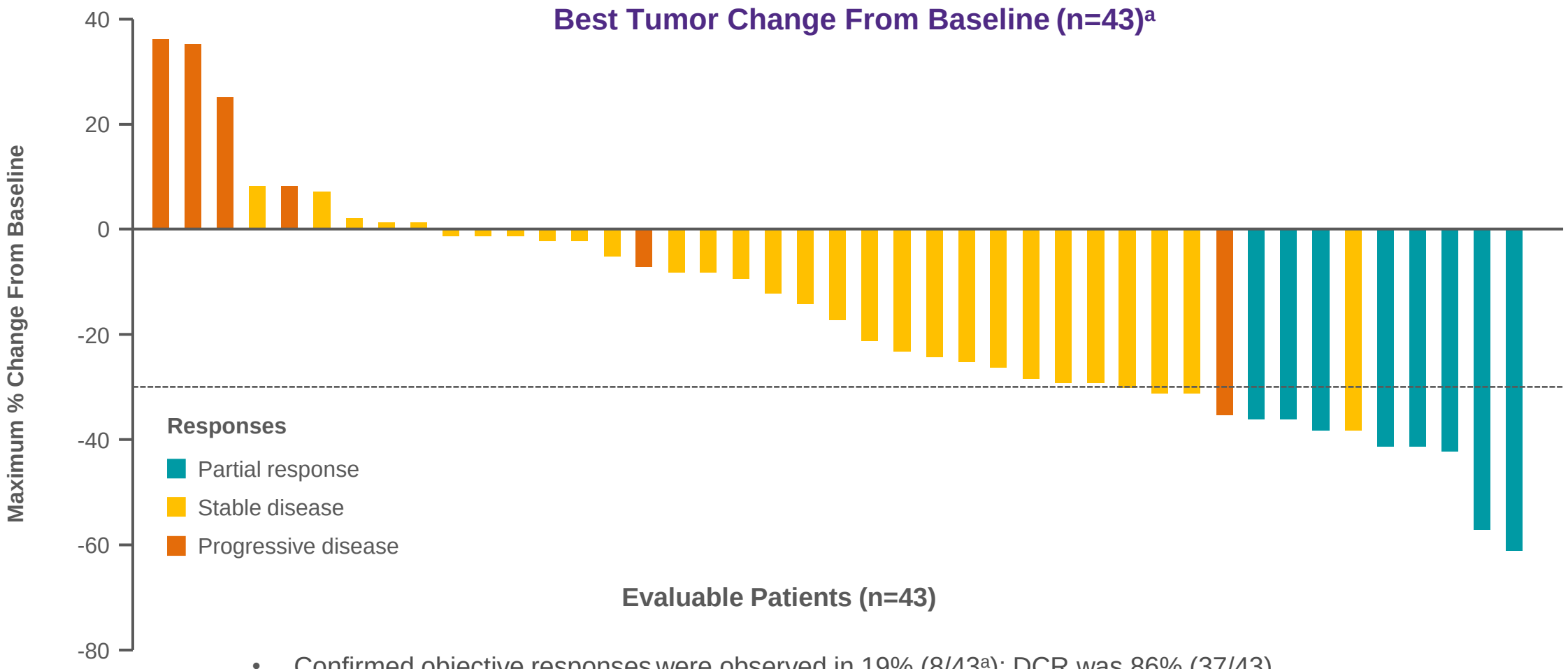
[1] Obermannová R, et al. Ann Oncol. 2016;27(11):2082-2090. [2] Grothey A, et al. *Lancet*. 2013;381(9863):303-312. [3] Mayer RJ, et al. *N Engl J Med*. 2015;372(20):1909-1919. [3]Van Cutsem E, et al. *Eur J Cancer*. 2018;90:63-72.; CRC = colorectal cancer;

Studied CRC patients Were Heavily Pretreated; 90% of Patients in Combination Treatment Were 3rd Line or Beyond

	Adagrasib Monotherapy[a] (n=44)	Adagrasib + Cetuximab[b] (n=32)
Median age, y (range)	59 (29–79)	60 (41–74)
Female, n (%)	22 (50%)	17 (53%)
Race, n (%)		
White	33 (75%)	26 (81%)
Black	6 (14%)	4 (13%)
Asian	3 (7%)	2 (6%)
Other	2 (5%)	0 (0%)
ECOG PS, n (%)		
0	23 (52%)	14 (44%)
1	21 (48%)	18 (56%)
Prior lines of systemic anticancer therapy, median (range)	3 (1–9)	3 (1–8)
Prior lines of systemic anticancer therapy, %		
1 / 2 / 3 / ≥4	18% / 21% / 25% / 36%	9% / 25% / 34% / 31%
Prior systemic anticancer therapy, %		
Fluoropyrimidine / oxaliplatin / irinotecan	100% / 98% / 80%	100% / 100% / 88%
Anti-VEGF	82%	88%
Anti-EGFR biological therapy	2%	0%
Regorafenib and/or trifluridine/tipiracil	23%	19%

[a]Adagrasib monotherapy was administered at a dose of 600 mg BID. [b]Adagrasib was administered at a dose of 600 mg BID. Cetuximab was administered IV at a dose of 400 mg/m^2 followed by 250 mg/m^2 QW, or 500 mg/m^2 Q2W (Phase 1b); Data as of June 16, 2022 (median follow-up adagrasib monotherapy, 20.1 months; adagrasib + cetuximab, 17.5 months); CRC = colorectal cancer

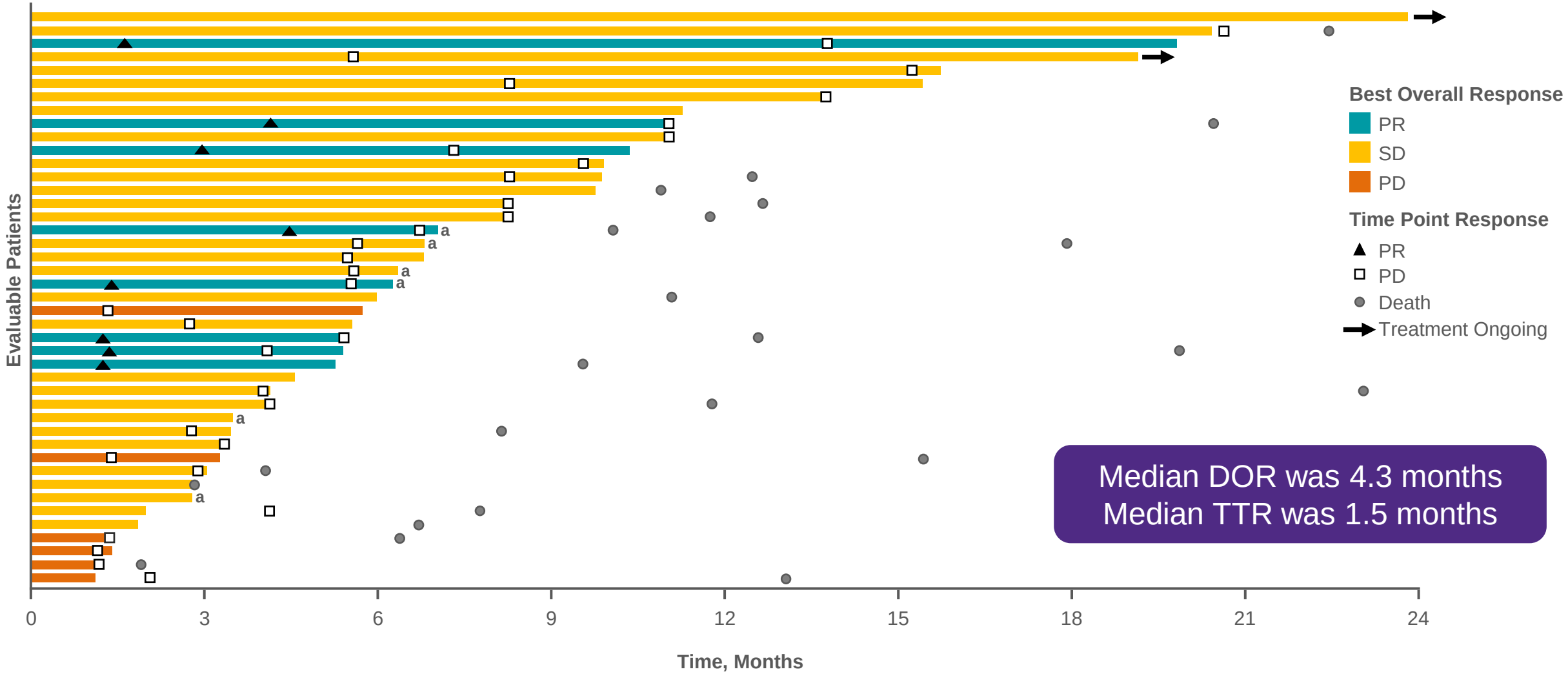
Adagrasib Monotherapy in Patients With Advanced CRC: Best Overall Response



- Confirmed objective responses were observed in 19% (8/43[a]); DCR was 86% (37/43)
- Tumor shrinkage of any magnitude occurred in 79% of patients

[a]Response per investigator assessment (n=43; one patient withdrew consent prior to the first scan); Data as of June 16, 2022 (median follow-up, 20.1 months)
CRC = colorectal cancer; DCR = disease control rate

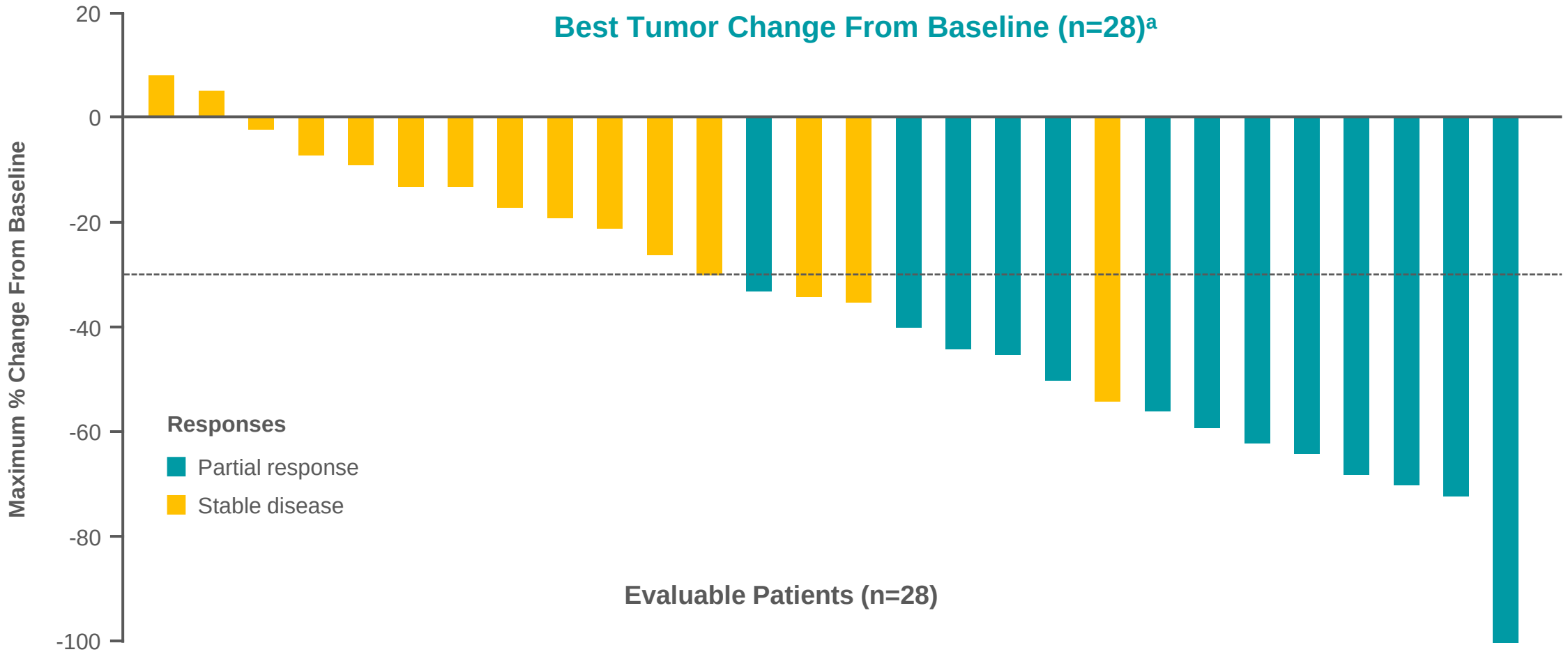
Adagrasib Monotherapy in Previously Treated Patients with KRAS^{G12C}-Mutated CRC: Duration of Treatment



[a]Patients who crossed over to receive adagrasib + cetuximab; data are summarized prior to crossover; Response outcomes per investigator assessment

Data as of June 16, 2022 (median follow-up, 20.1 months)

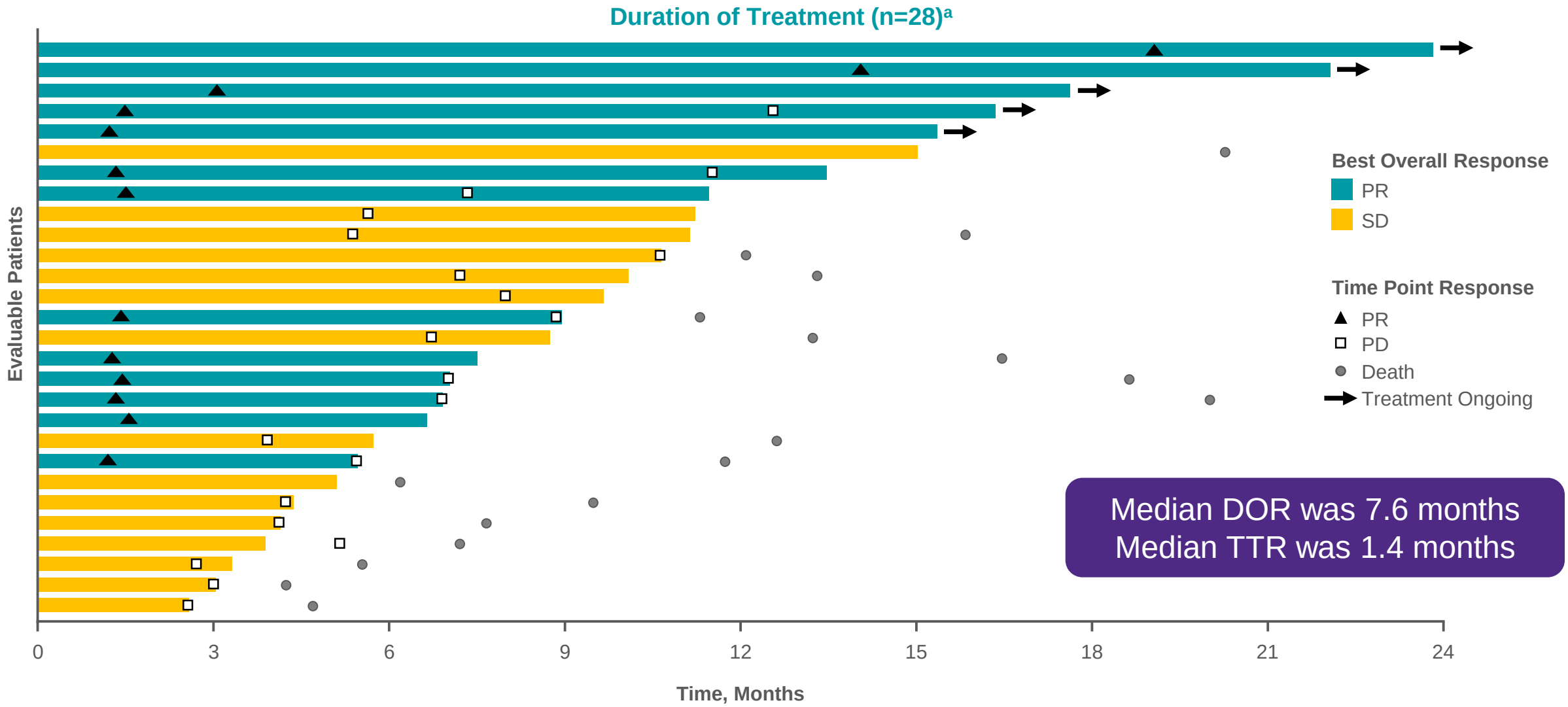
Adagrasib + Cetuximab in Patients with Advanced CRC: Best Overall Response



- Confirmed objective responses were observed in 46% (13/28[a]); DCR was 100% (28/28)
- Tumor shrinkage of any magnitude occurred in 93% of patients

[a]Response per investigator assessment (n=28; four patients are not included due to no post-baseline assessment of target lesions); Data as of June 16, 2022 (median follow-up, 17.5 months)
CRC = colorectal cancer; DCR = disease control rate

Adagrasib + Cetuximab in Patients with Advanced CRC: Duration of Treatment



Median DOR was 7.6 months
Median TTR was 1.4 months

Response outcomes per investigator assessment (n=28; four patients are not included due to no post-baseline assessment of target lesions); Data as of June 16, 2022 (median follow-up, 17.5 months)
CRC = colorectal cancer; DOR = duration of response; TTR = time to response; SD = stable disease; PR = partial response

Adagrasib ± Cetuximab in Previously Treated Patients With KRAS^{G12C}-Mutated CRC: Treatment-Related Adverse Events

Most Frequent TRAEs	Adagrasib Monotherapy[a] (n=44)		Adagrasib + Cetuximab[b] (n=32)	
TRAEs, %	**Any Grade**	**Grade 3–4**	**Any Grade**	**Grade 3–4**
Any TRAEs[c]	93%	34%	100%	16%
Most frequent TRAEs,[d] %				
Diarrhea	66%	7%	56%	3%
Nausea	57%	0	63%	0
Fatigue	46%	5%	47%	0
Vomiting	46%	0	53%	0
Decreased appetite	18%	0	16%	0
Anemia	16%	9%	9%	0
QT prolongation	16%	5%	16%	3%
Peripheral edema	16%	0	19%	0
Headache	5%	0	31%	0
Dizziness	5%	2%	25%	0
Dry skin	2%	0	41%	0
Rash maculopapular	2%	0	25%	0
Stomatitis	2%	0	22%	3%
Dermatitis acneiform	–	–	47%	3%

Adagrasib Monotherapy

- No Grade 5 TRAEs
- No TRAEs led to discontinuation

Adagrasib + Cetuximab

- No Grade 5 TRAEs
- No TRAEs led to discontinuation of adagrasib
- 16% of TRAEs led to discontinuation of cetuximab[e]

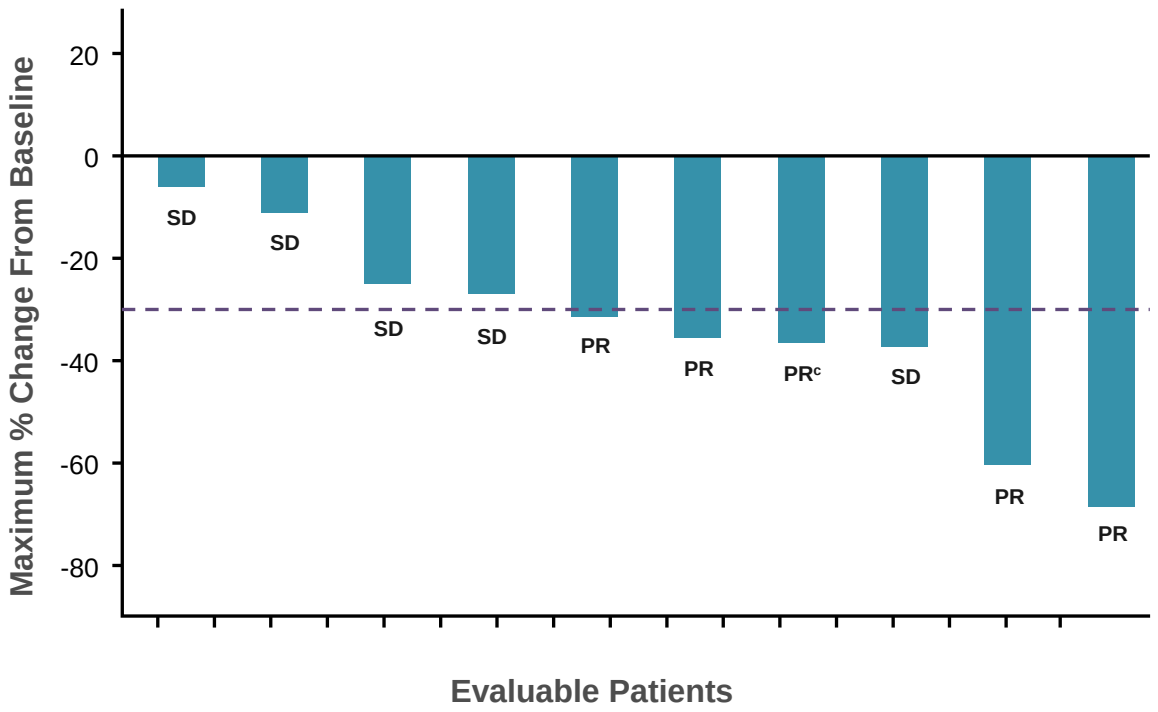
[a]Adagrasib 600 mg BID (capsule, fasted). [b]Adagrasib 600 mg BID (capsule, fasted) and cetuximab 400 mg/m^2 followed by 250 mg/m^2 QW, or 500 mg/m^2 Q2W
[c]By maximum grade. [d]Occurring in >15% of patients (any grade) in the adagrasib monotherapy cohort, or >20% of patients in the adagrasib + cetuximab cohort
[e]TRAEs leading to cetuximab discontinuation were cetuximab-related infusion-related reaction (n=3), malaise (n=1), and vascular flushing (n=1)
Data as of June 16, 2022 (median follow-up, 20.1 months for adagrasib monotherapy; 17.5 months for adagrasib + cetuximab)

MIRATI THERAPEUTICS

Adagrasib (MRTX849): Pancreatic Ductal Adenocarcinoma Cancer and Other Gastro-Intestinal Tumors

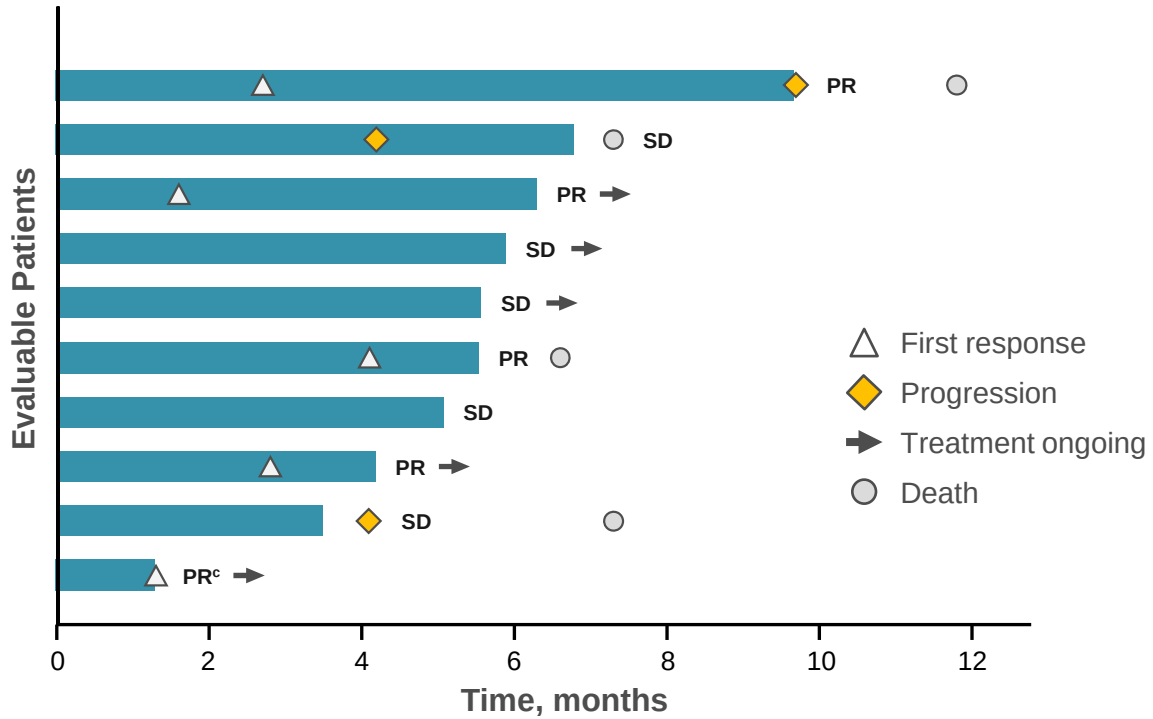
Adagrasib in Patients With Unresectable or Metastatic PDAC: Best Tumor Change From Baseline and Duration of Treatment

Best Tumor Change From Baseline (n=10)[a,b]



- Response rate: 50% (5/10), including 1 unconfirmed PR
- SD: 50% (5/10 patients)
- DCR: 100% (10/10 patients)

Duration of Treatment (n=10)[a,b]

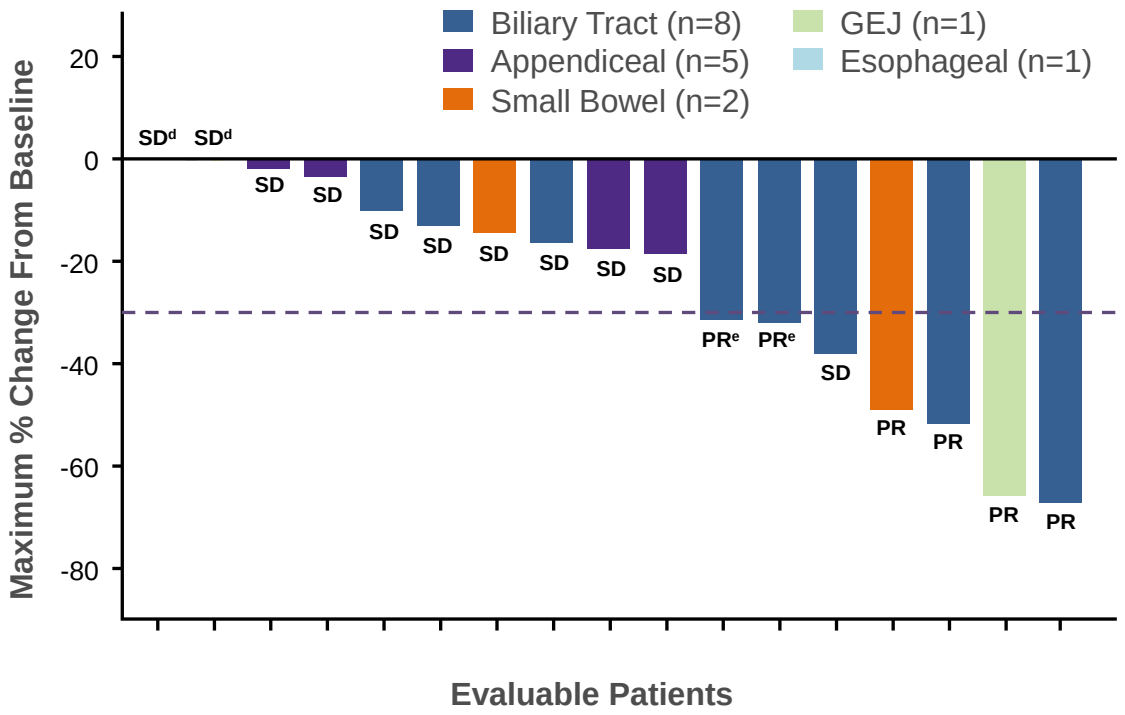


- Median TTR: 2.8 months
- Median DOR: 6.97 months
- Median PFS: 6.6 months (95% CI 1.0–9.7)
- Treatment ongoing in 50% (5/10) of patients

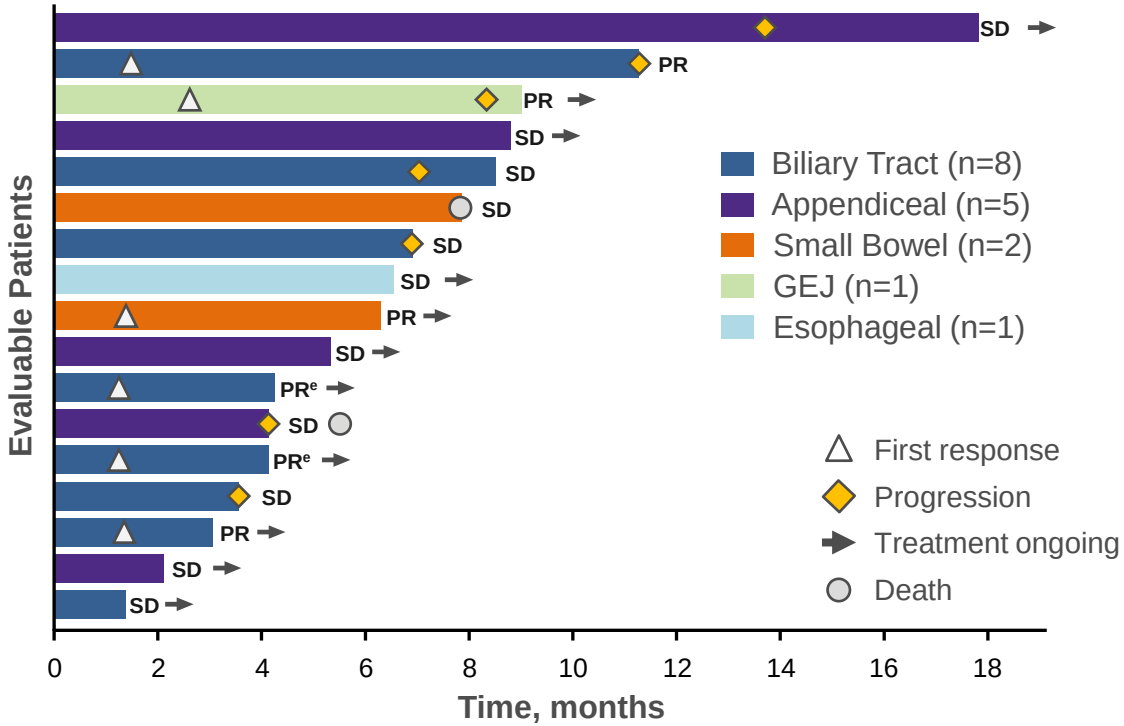
DCR, disease control rate; DOR, duration of response; PR, partial response; SD, stable disease; TTR, time to response.
[a]Evaluable population (n=10) excludes 2 patients who had discontinued treatment prior to first scan due to unrelated adverse events and were not evaluable for clinical activity; [b]All results are based on investigator assessments;
[c]At data cut-off, 1 patient had unconfirmed PR.
Data as of 10 Sept 2021 (median follow-up: 8.1 months).

Adagrasib in Patients With Other GI Tumors:[a] Best Tumor Change From Baseline and Duration of Treatment

Best Tumor Change From Baseline (n=17)[b,c]



Duration of Treatment (n=17)[b,c]



- Response rate:
  - Biliary tract cancer: 50% (4/8), including 2 unconfirmed PRs
  - GEJ and small bowel cancer: 1 PR each
- DCR: 100% (17/17 patients)

- Median TTR: 1.3 months
- Median DOR: 7.85 months
- Median PFS: 7.85 months (95% CI 6.90–11.30)
- Treatment ongoing in 65% (11/17) of patients

DCR, disease control rate; DOR, duration of response; GEJ, gastro-esophageal junction; PR, partial response; SD, stable disease; TTR, time to response.
[a]Excluding CRC and PDAC; [b]Evaluable population (n=17) excludes 1 patient who withdrew consent prior to the first scan; [c]All results are based on investigator assessments; [d]1 patient with appendiceal cancer and 1 patient with esophageal cancer had maximum % change from baseline of 0; [e]At data cut-off, 2 patients had unconfirmed PR.
Data as of 10 Sept 2021 (median follow-up: 6.3 months).

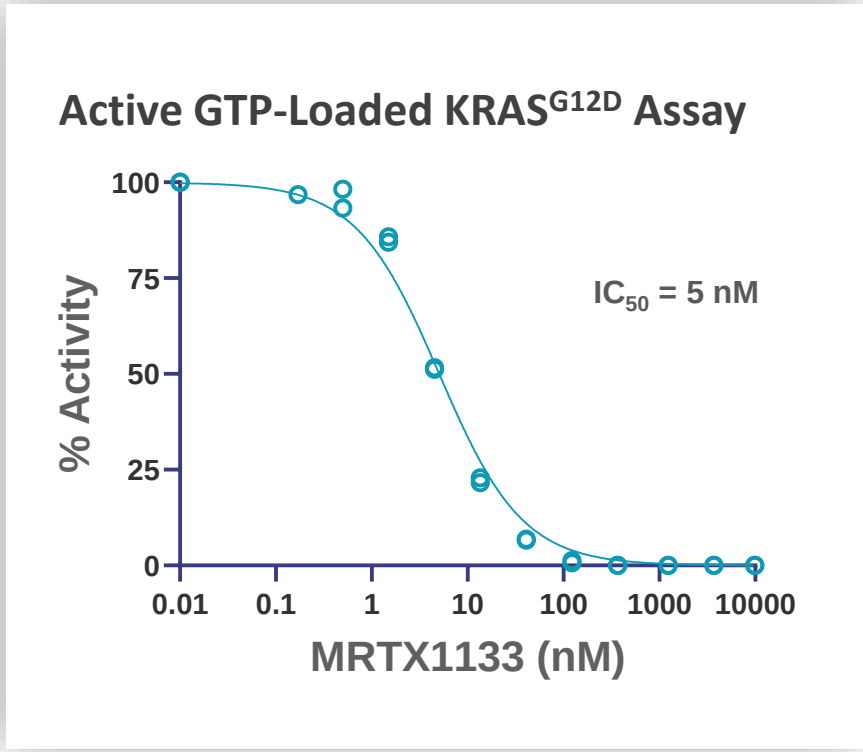
MIRATI THERAPEUTICS

MRTX1133:

$KRAS^{G12D}$ Selective Inhibitor

MRTX1133: Potential First-in-Class KRAS^{G12D} Selective Inhibitor

Assay	Criteria	MRTX1133
KRAS^{G12D} cell activity	<10nM	**~5 nM**
Selectivity over KRAS^{WT}	>100-fold	**>1,000-fold**
Predicted human half-life	>24 hours	**~50 hours**
Low risk for hERG/off-target pharmacology	>10μM	✓
Drug-drug interaction (CYPs)	Low risk	✓



- MRTX1133 is a small molecule that selectively & reversibly binds to & inhibits KRAS^{G12D} in both active & inactive states
- MRTX1133 demonstrates selective inhibition of cell viability of KRAS^{G12D} mutant, but not KRAS wild-type, tumor cells

MRTX1133: Clinical Development Path and Design Principles

PATH TO CLINICAL DEVELOPMENT

- Optimizing target coverage throughout the dosing interval is important for maximizing antitumor activity in KRAS mutated cancers
- We have emphasized the development of formulation strategies designed to enhance oral absorption and increase systemic drug exposure
- In parallel, we have also worked to develop long-acting injectable IV formulations, including liposomal strategies
- IND filing for the oral formulation planned for YE 2022, and plan to bring liposomal IV formulation into clinic later in 2023

CLINICAL TRIAL DESIGN PRINCIPLES

- Multi-cohort Phase 1 monotherapy trial comparable to adagrasib
  - Rapid dose escalation strategies to define a tolerated and active dose
- Multiple expansion cohorts for pancreatic, colorectal, lung and other G12D patients
- Rational combination approaches are similar to G12C and enabled in first-in-human clinical trials

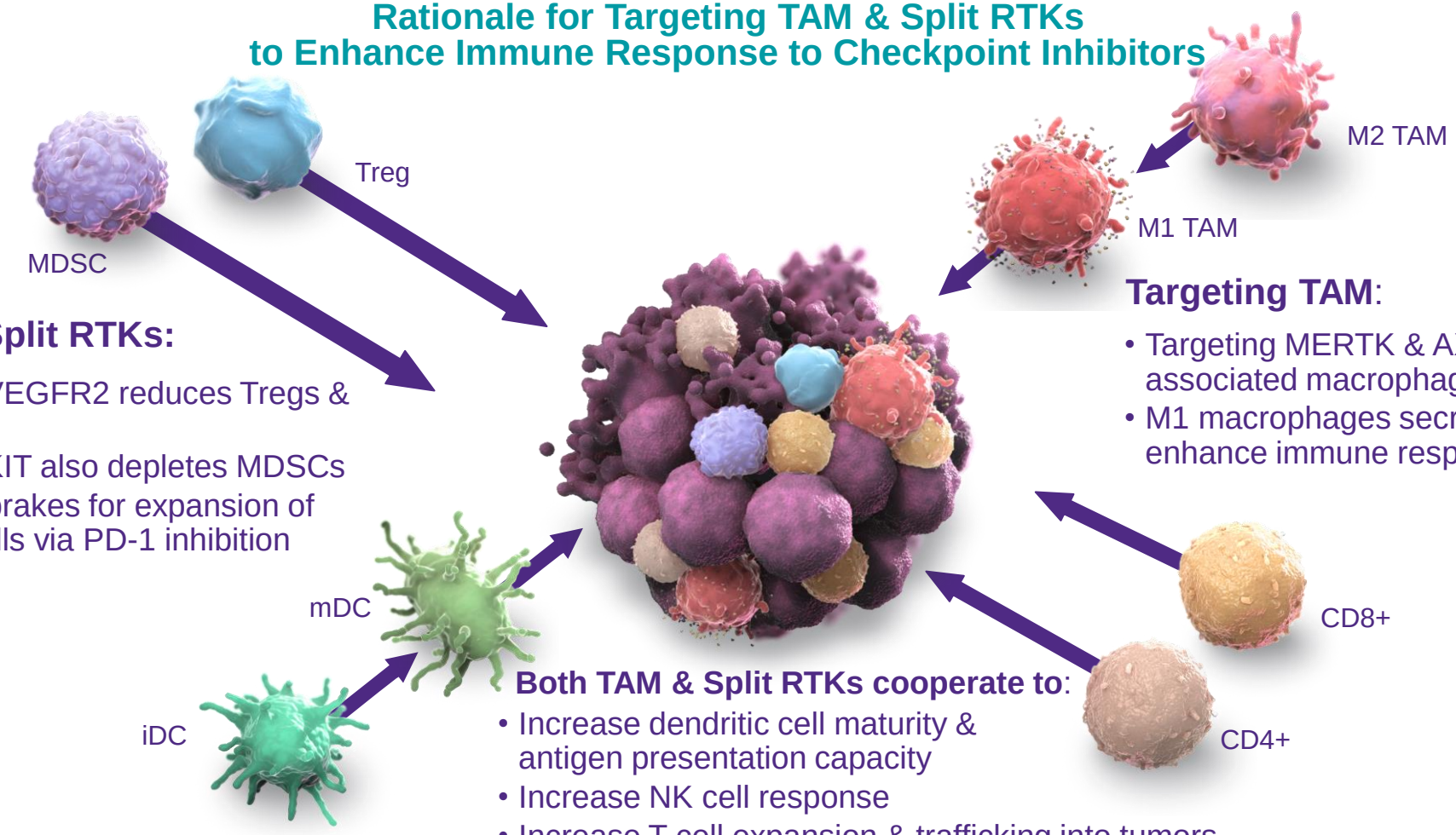
IND: investigational new drug; IV = intravenous

MIRATI THERAPEUTICS

Sitravatinib + Checkpoint Inhibitors

Sitravatinib Inhibits TAM (TYRO3, AXL and MER), VEGFR2, and KIT Receptors and May Restore Immune Response

Rationale for Targeting TAM & Split RTKs to Enhance Immune Response to Checkpoint Inhibitors



Targeting Split RTKs:

- Targeting VEGFR2 reduces Tregs & MDSCs
- Targeting KIT also depletes MDSCs
- Releases brakes for expansion of CD8+ T cells via PD-1 inhibition

Targeting TAM:

- Targeting MERTK & AXL shifts tumor associated macrophage (TAM) type to M1
- M1 macrophages secrete cytokines that enhance immune response (IL-12, TNF)

Both TAM & Split RTKs cooperate to:

- Increase dendritic cell maturity & antigen presentation capacity
- Increase NK cell response
- Increase T cell expansion & trafficking into tumors

Pircher et al., Synergies of Targeting Tumor Angiogenesis and Immune Checkpoints. Int J Mol Sci, 2017. 18(11).
Garton et al., Anti-KIT Monoclonal Antibody Treatment Enhances the Antitumor Activity of Immune Checkpoint Inhibitors by Reversing Tumor-Induced Immunosuppression. Mol Cancer Ther, 2017. 16(4)
Akalu, Y.T., C.V. Rothlin, and S. Ghosh, TAM receptor tyrosine kinases as emerging targets of innate immune checkpoint blockade for cancer therapy. Immunol Rev, 2017. 276(1)
Graham, D.K., D. DeRyckere, K.D. Davies, and H.S. Earp, The TAM family. Nat Rev Cancer, 2014. 14(12)
Du, W., Huang, H., Sorrelle, N., & Brekken, R. A. (2018). Sitravatinib potentiates immune checkpoint blockade in refractory cancer models. JCI Insight, 3(21).

MRTX-500: Phase 2, Open-Label Study of Sitravatinib + Nivolumab in Patients with Non-squamous NSCLC with Prior Clinical Benefit from Checkpoint Inhibitor Therapy

Key Eligibility Criteria
(n=68)

- Advanced/metastatic nonsquamous NSCLC[a]
- No actionable driver mutations
- Anti–PD-1/L1 must be the most recent line of therapy
- Prior Clinical Benefit (PCB) to CPI: CR, PR, or SD ≥12 weeks from prior CPI therapy
- No uncontrolled brain metastases
- ECOG PS 0-2

Sitravatinib 120 mg QD + nivolumab

Primary Endpoint:

- Objective Response Rate[b] (ORR), as defined by RECIST 1.1

Secondary Endpoints:

- Safety and tolerability
- DOR
- CBR
- PFS
- OS
- 1-year survival rate

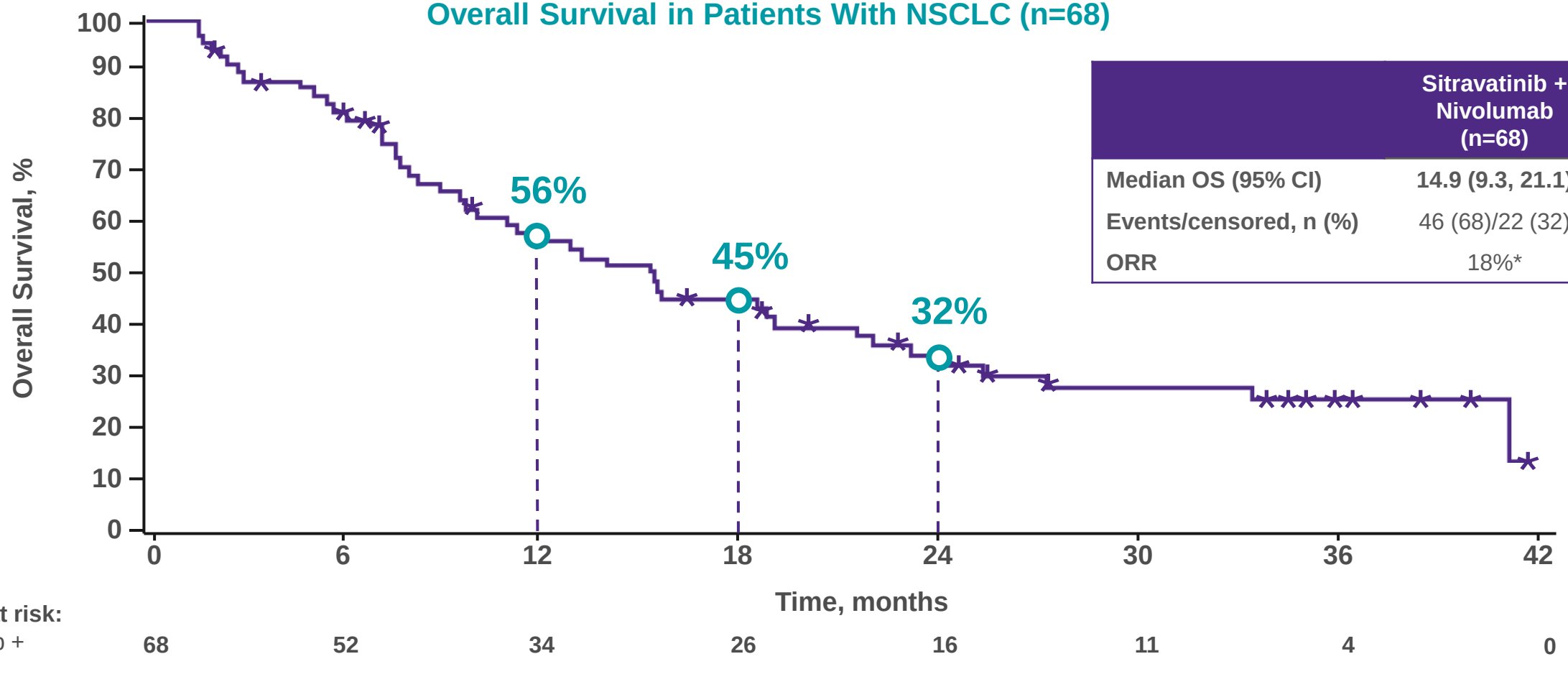
Data as of 1 June 2021

[a] Additional cohorts included a CPI-experienced cohort that did not receive prior clinical benefit from CPI therapy (radiographic progression of disease ≤12 weeks after initiation of treatment with CPI) and a CPI-naive cohort in patients that were previously treated with platinum-based chemotherapy. [b]ORR based on investigator assessment. Dosing: sitravatinib free base formulation; nivolumab, 240 mg Q2W or 480 mg Q4W. Treatment discontinuation could be due to (but is not limited to) disease progression, global health deterioration, AEs, protocol violation, lost to follow-up, refusal of further treatment, study termination, or death. NSCLC = non-small cell lung cancer; DOR = duration of response; CR = complete response; PR = partial response; SD = stable disease; PFS = progression free survival; OS = overall survival; CPI = checkpoint inhibitor; QD = once a day dosing

Presented at the European Society for Medical Oncology (ESMO) Congress, 20 September 2021

Overall Survival with Sitravatinib + Nivolumab in Patients with Non-squamous NSCLC With Prior Clinical Benefit From CPI Therapy

Overall Survival in Patients With NSCLC (n=68)



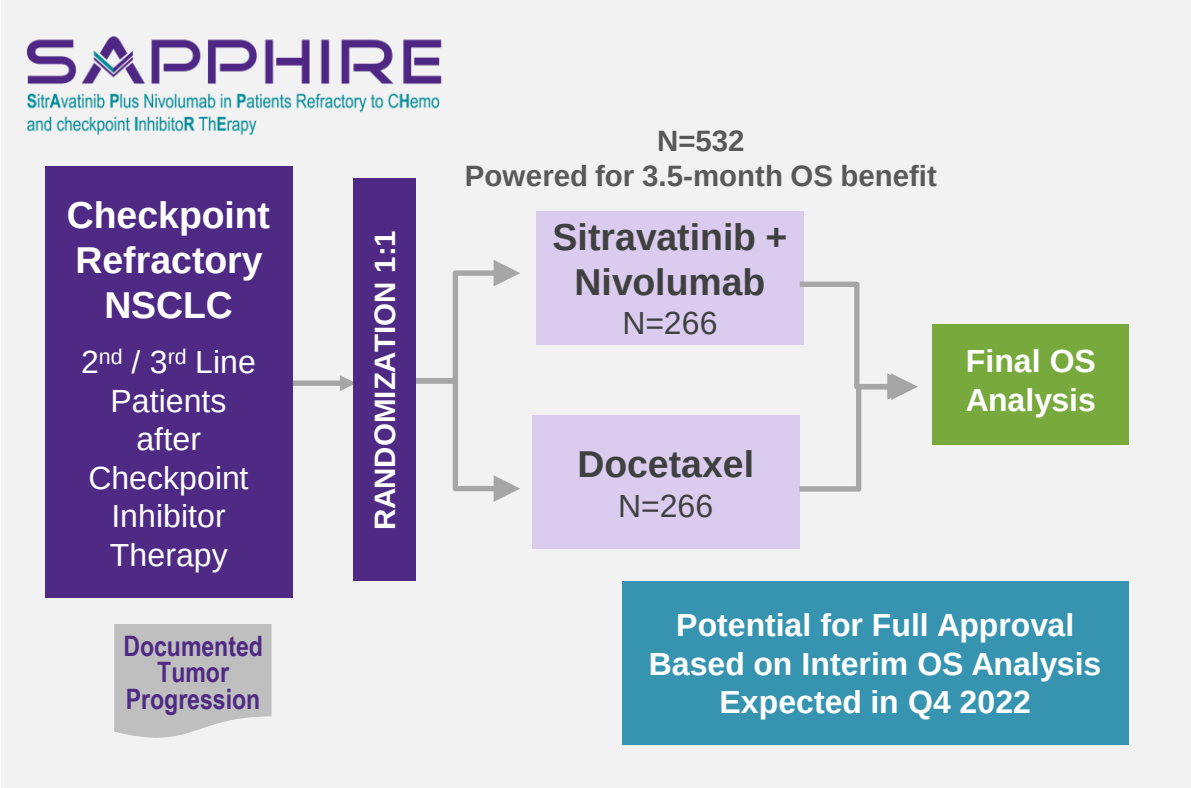
	Sitravatinib + Nivolumab (n=68)
Median OS (95% CI)	14.9 (9.3, 21.1)
Events/censored, n (%)	46 (68)/22 (32)
ORR	18%*

Median follow-up in PCB cohort: 33.6 months; Data as of 1 June 2021.
* ORR of 18% included 2 complete responses (3%) and 10 partial responses (15%); ORR = overall response rate; OS = overall survival; CPI = checkpoint inhibitor; NSCLC = non-small cell lung cancer

Presented at the European Society for Medical Oncology (ESMO) Congress, September 2021

Compelling Phase 2 Results Support and Inform SAPPHIRE Phase 3 Trial in 2nd / 3rd Line Non-Squamous NSCLC

- **Encouraging Overall Survival (OS)** data from Phase 2 trial [1]
  - Median OS of 14.9 months[1] in 2nd Line or 3rd Line patients with Prior Clinical Benefit (PCB) on a prior checkpoint inhibitor (CPI) and subsequent disease progression (n=68)
    - 56% and 32% of patients alive at 1- and 2-years, respectively
- Phase 3 SAPPHIRE clinical trial inclusion criteria in PCB patients who received the combination as either 2nd or 3rd line therapy after progressing on treatment with checkpoint inhibitor
- **Potential to establish sitravatinib + nivolumab as new standard of care after checkpoint inhibitor failure**
  - >2nd line NSCLC U.S. & EU Populations (circa 2020): over 100,000 patients with ~70,000 being non-squamous



1. MRTX-500 Phase 2 trial: full Prior Benefit Cohort (PCB) (n=68), data cut-off of June 1, 2021, and presented at European Society for Medical Oncology (ESMO) Congress on September 18, 2021. Patients with PCB on a checkpoint inhibitor as part of their last treatment regimen prior to enrollment. PCB is defined as either complete response, partial response or stable disease for ≥12 weeks. PCB patients who received the combination as either 2nd or 3rd line of therapy after progressing on treatment with a checkpoint inhibitor. [a]10 (14.7%) patients were not evaluable for ORR: 8 patients without post-baseline scan, 1 patient without measurable disease at baseline, and 1 patient for whom all post-baseline scans were NE. Median follow-up in the PCB cohort was 33.6 months.
2. Data represented are from the CheckMate 057,KEYNOTE 010 and OAK studies and do not reflect results that might have been obtained from head-to-head studies. Results from Mirati's on-going Phase 3 SAPPHIRE trial comparing sitravatinib + nivolumab to docetaxel may differ materially from prior studies presented.
3. Borghaei H, et al. *New England Journal of Medicine* 2015;373:1627-1639, Herbst RS, et al. *Lancet*. 2016;387:1540-1550, Rittmeyer A, et al. *Lancet*. 2017;389:255-265.

OS: overall survival; NSCLC: non-small cell lung cancer;

MIRATI THERAPEUTICS

MRTX1719:

Novel PRMT5 Inhibitor in MTAP-deleted Cancers

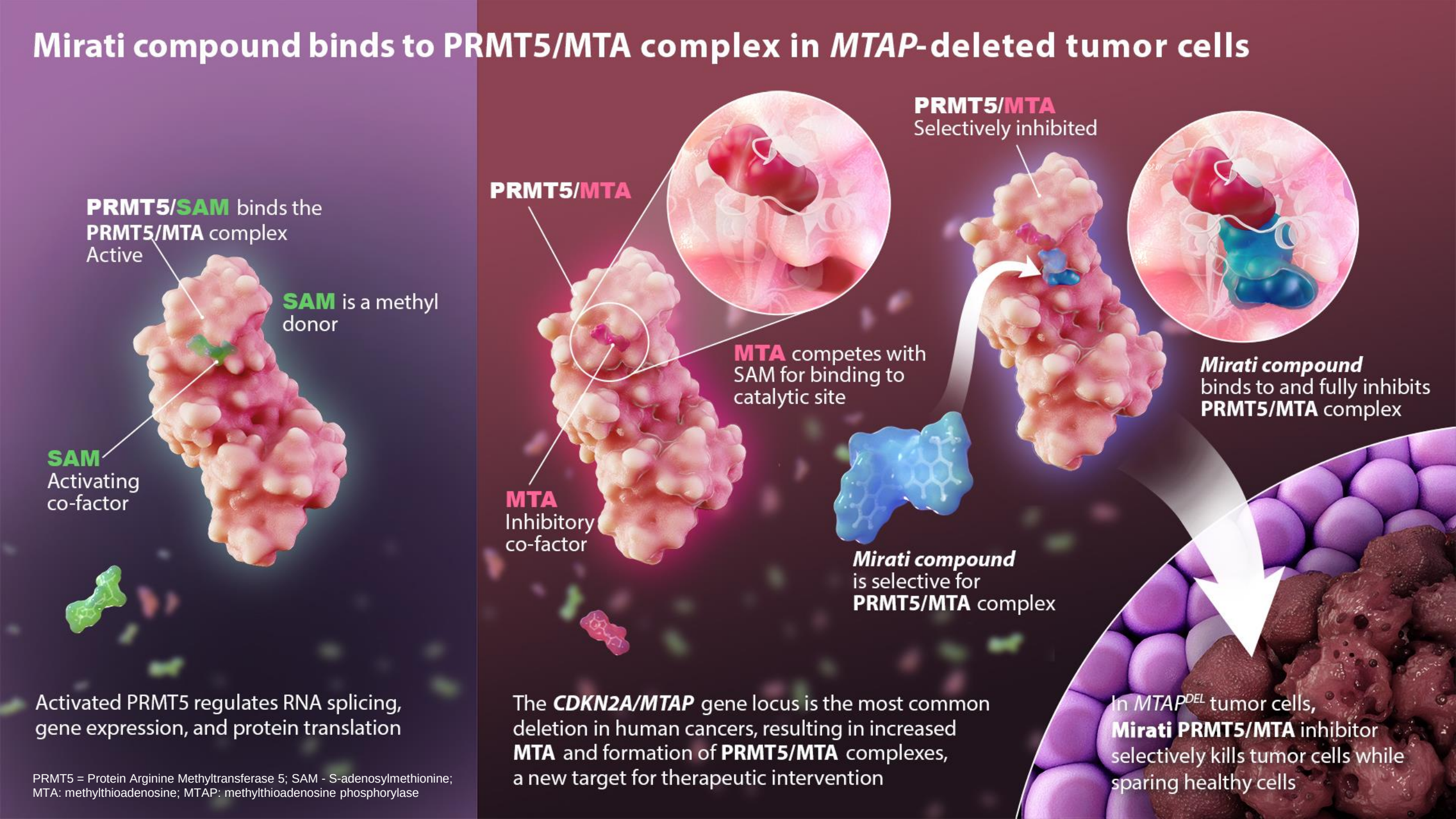
MRTX1719: Novel PRMT5 Inhibitor Selective for MTAP-deleted Cancers

- *MTAP* deletions occur in approximately 10%[1] of all human cancers including lung, pancreatic ductal adenocarcinoma and mesthelioma
  - Patients have a poor prognosis, representing a significant unmet medical need
- Internally discovered MTA-cooperative PRMT5 inhibitor represent a potential precision medicine for *MTAP*-deleted cancers
  - Program leverages a synthetic lethal approach and selectively targets the PRMT5/MTA complex in *MTAP*-deleted cancer cells
  - Designed to spare normal human cells and demonstrates improved therapeutic index in preclinical studies relative to first generation approaches
- Phase 1/2 clinical trial initiated in Q1:2022 and granted fast-track designation in Q3:2022

[1] cBioPortal

PRMT5 = Protein Arginine Methyltransferase 5; SAM - S-adenosylmethionine; MTA: methylthioadenosine; MTAP: methylthioadenosine phosphorylase; IND: investigational new drug

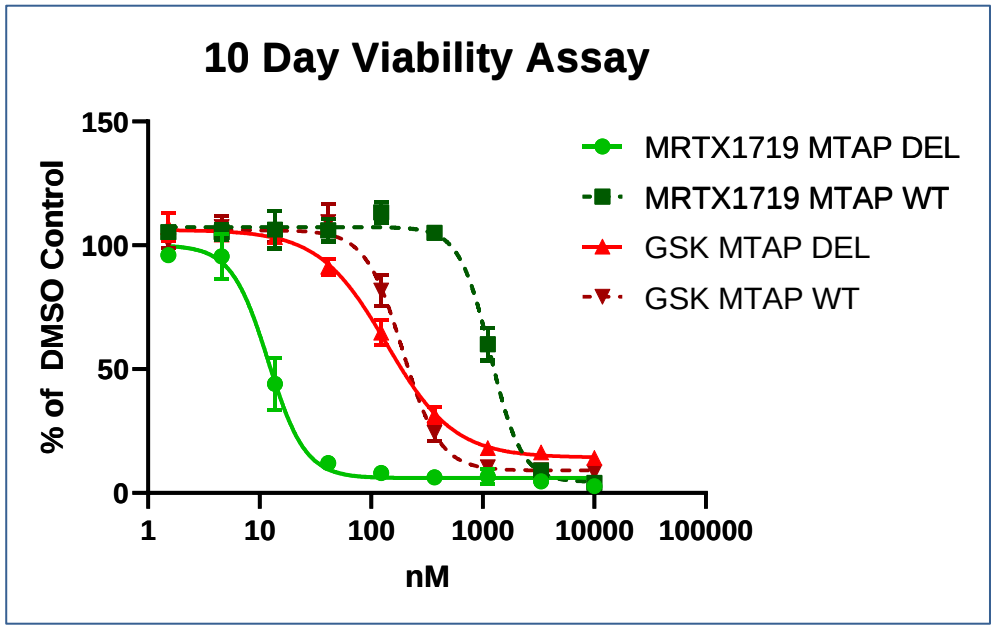
Mirati compound binds to PRMT5/MTA complex in *MTAP*-deleted tumor cells



PRMT5 = Protein Arginine Methyltransferase 5; SAM - S-adenosylmethionine; MTA: methylthioadenosine; MTAP: methylthioadenosine phosphorylase

MRTX1719: Potential First-in-class Selective Inhibitor of the PRMT5/MTA Complex

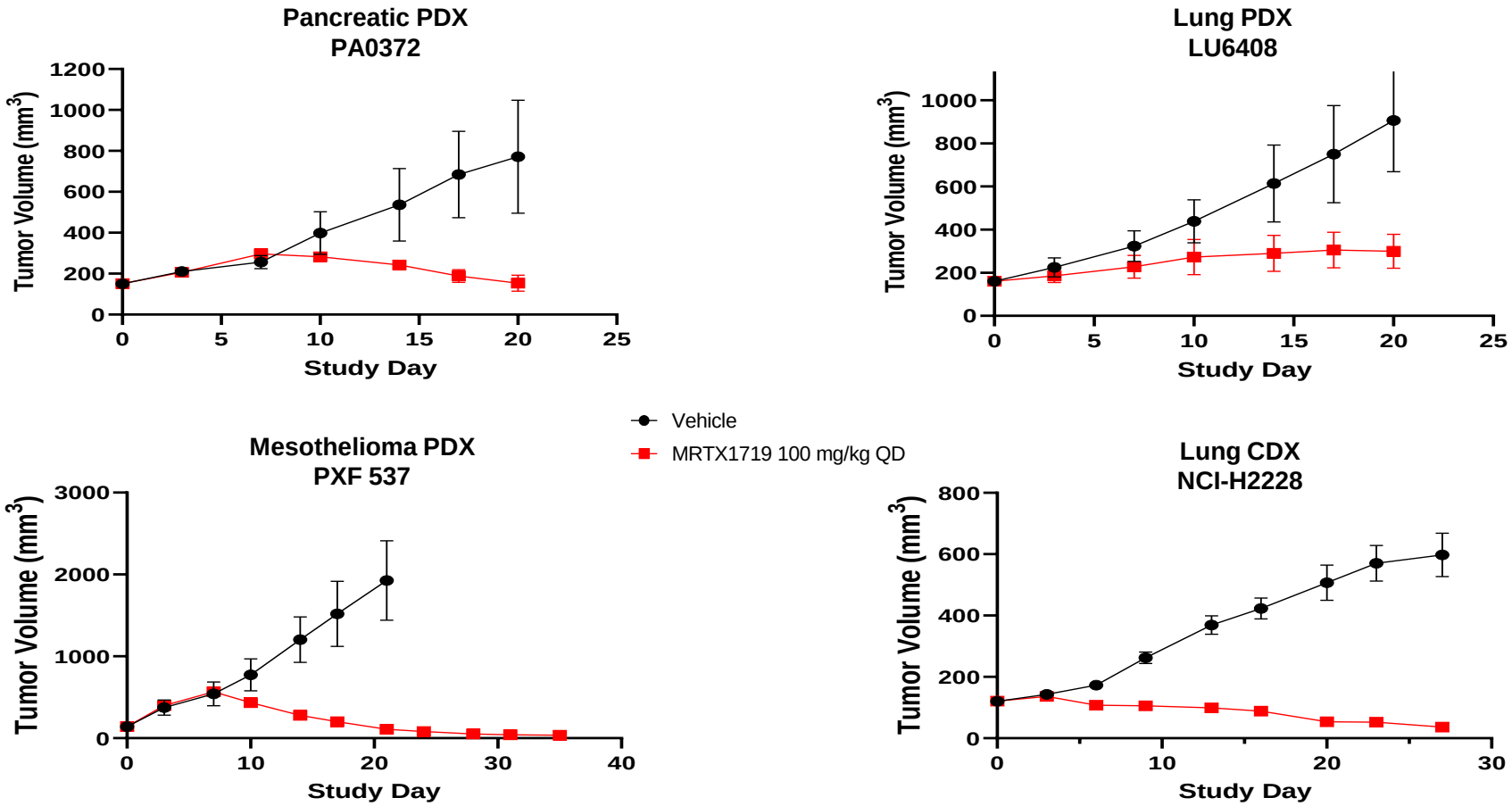
Assay	Criteria	MRTX1719
PRMT5/MTA *MTAP*^{DEL} SDMA cell activity	<15nM	**<10 nM**
Selectivity for *MTAP*^{WT} cells (SDMA)	>20-fold	**>70-fold**
Drug-drug interaction (CYPs)	Low risk	✓
Favorable bioavailability	Low risk ADME	✓



- MRTX1719 selectively inhibits the PRMT5/MTA complex with a very slow off rate and tight binding leads to prolonged PD effects in preclinical models
- Greater inhibition of PRMT5 in *MTAP*-deleted (tumor) cells suggest the potential for an increased therapeutic index with fewer adverse events (e.g., bone marrow suppression) compared to non-PRMT5/MTA selective inhibitors

WT = wild type; ADME: absorption, distribution, metabolism, and excretion; PD = pharmacodynamic; SDMA = symmetric dimethylarginine; PRMT5 = Protein Arginine Methyltransferase 5; MTAP: methylthioadenosine phosphorylase

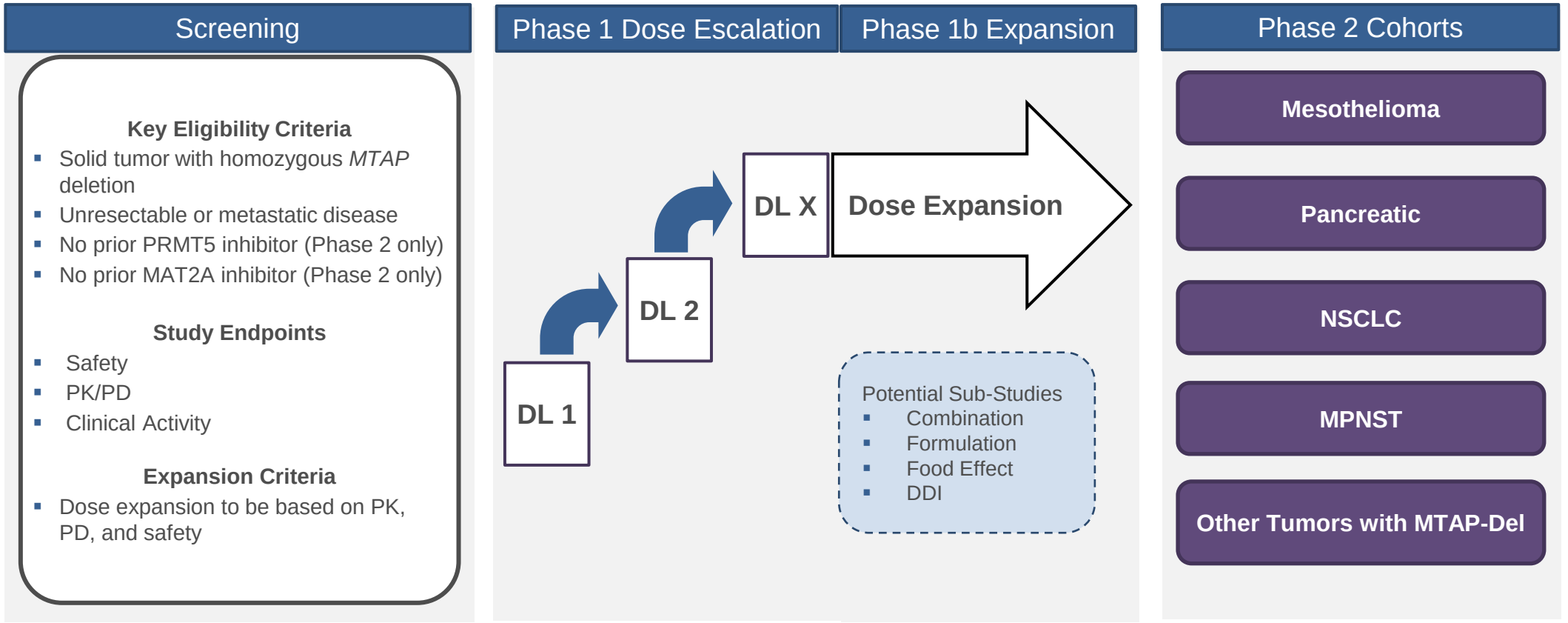
MRTX1719 Demonstrates Selective Activity *in vitro* and *in vivo* and Induces Regression in a Subset of Cell Line- and Patient-derived Xenograft Models



- MRTX1719 demonstrates strong antitumor activity in numerous tumor models, including lung, pancreatic ductal adenocarcinoma and mesothelioma

Presented at the AACR-NCI-EORTC Meeting in October 2021

MRTX1719 Profile and Preclinical Results Shape Clinical Development Strategy in MTAP-deleted Cancers



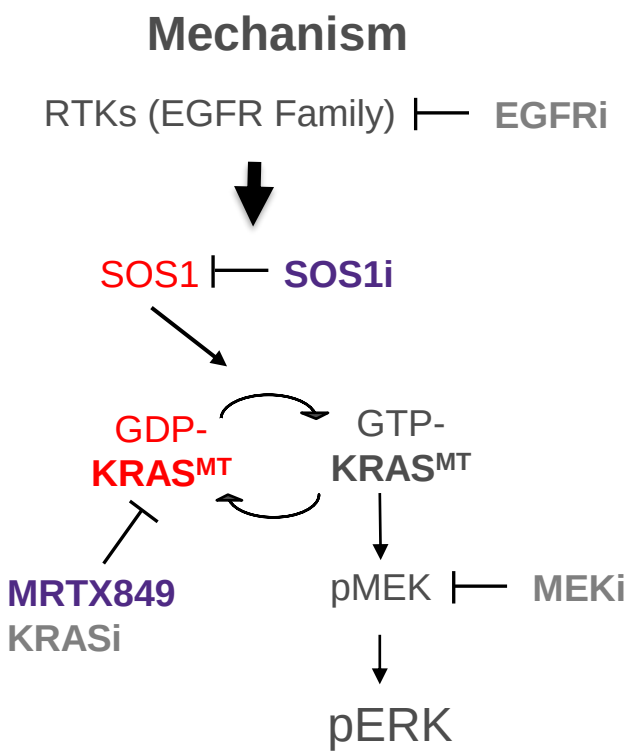
MRTX1719 clinical development will include broad range of MTAP-deleted cancers as both single agent and in combination

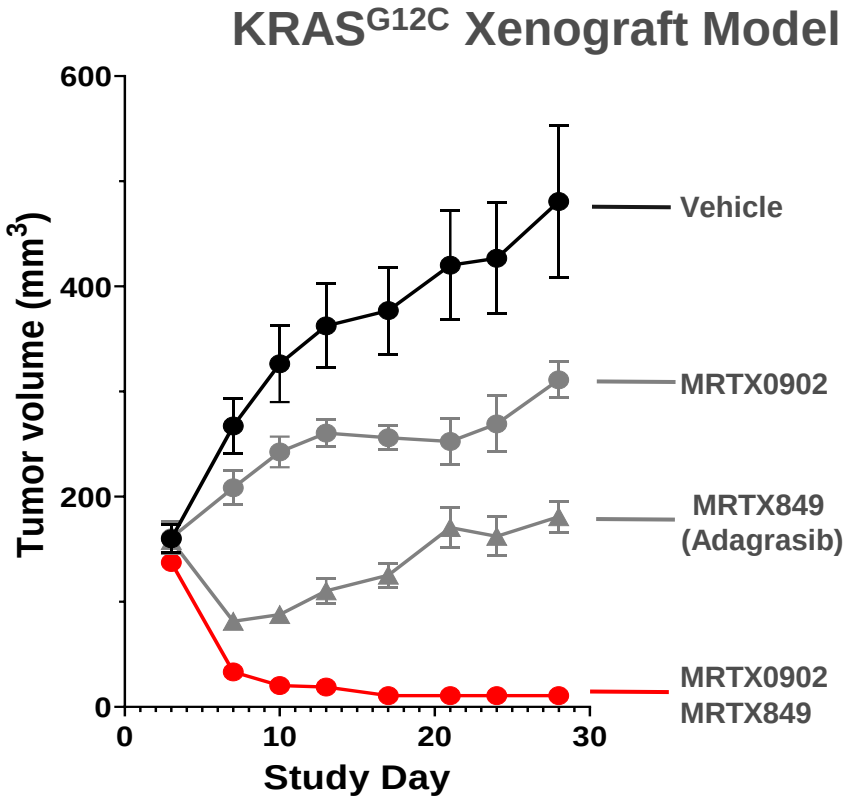
DL: dose level; NSCLC: non-small cell lung cancer; MPNST: malignant peripheral nerve sheet tumor

MIRATI THERAPEUTICS

MRTX0902: SOS1 Inhibitor

MRTX0902 (SOS1 Inhibitor) Improves Anti-tumor Efficacy in Combination with Targeted MAPK-pathway inhibitors





- SOS1 facilitates nucleotide exchange and KRAS activation
- MRTX0902 exhibits favorable potency, selectivity, and oral exposure characteristics and shows improved anti-tumor efficacy in combination with inhibitors of KRAS, MEK, and EGFR in pre-clinical models
- IND filed in Q3 2022; Phase 1/2 clinical trial initiated in Q4 2022

MIRATI THERAPEUTICS

Differentiated Discovery Capabilities Enhance Potential for Long-Term Growth and Sustainability

Highly Productive Discovery Capability Advancing Additional Best-in-Class / First-in-Class Opportunities

- Mirati has built a highly productive in-house discovery and preclinical development capability
  - Our research has resulted in 30 published patent applications for 8 portfolio projects across both clinical or preclinical compounds, including:
    - Adagrasib ($KRAS^{G12C}$), MRTX1133 ($KRAS^{G12D}$), MRTX1719 (MTA-cooperative PRMT5), ORIC-944 (EED)*, and MRTX0902 (SOS1)
- Discovery efforts are focused on advancement of novel targeted cancer therapies that:
  - Further complement existing pipeline
  - Offer potential practice-changing opportunities for cancer patients
- Near-term focus on advancing MRTX1133 and next-generation KRAS programs

*In August 2020, Mirati and Oric entered into an exclusive worldwide license for development and commercialization rights for PRC2 inhibitor, ORIC-944.

Growing our Leadership Position in the Development of Next Generation KRAS Therapies

- Demonstrated initial preclinical proof-of-concept data, including the meaningful tumor regression, targeting other oncogenic forms of KRAS with spectrum-selective mutant KRAS inhibitors
- Our next generation KRAS targeting strategy represents a potential platform approach
  - Potential to yield development candidates targeting various KRAS mutations with distinct selectivity profiles
- Spectrum-selective KRAS inhibitor programs are in the lead optimization stage
  - Specific compounds and multiple lead series under evaluation in various stages of preclinical development

MIRATI THERAPEUTICS

Financial Update

Select Company Financials

NASDAQ	**MRTX**
Cash as of September 30, 2022*	$1.2B
Shares outstanding as of September 30, 2022**	65.2M
Q3 2022: Operating Expenses	$191.9M
Q3 2022: Operating Expenses net of stock-based compensation***	$148.9M

* This amount is comprised of cash, cash equivalents and short-term investments

** Shares outstanding as of September 30, 2022, includes 57.6 million shares of common stock outstanding and pre-funded warrants to purchase a total of 7.6 million shares of common stock. The pre-funded warrants have a per share exercise price of $0.001.

*** Amount disclosed is calculated as Q3 2022 operating expenses ($191.9M) less Q3 2022 stock-based compensation expense ($43.0M).

NASDAQ: MRTX

MIRATI THERAPEUTICS

Targeting the genetic and immunological drivers of cancer

Corporate Overview Presentation

November 2022