Revolution Medicines

On Target to Outsmart Cancer

February 26, 2024

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For a further description of the risks and uncertainties that could cause actual results to differ from those anticipated in these forward-looking statements, as well as risks relating to the business of Revolution Medicines in general, see Revolution Medicines' Annual Report on Form 10-K filed with the Securities and Exchange Commission on February 26, 2024, and its future periodic reports to be filed with the Securities and Exchange Commission.

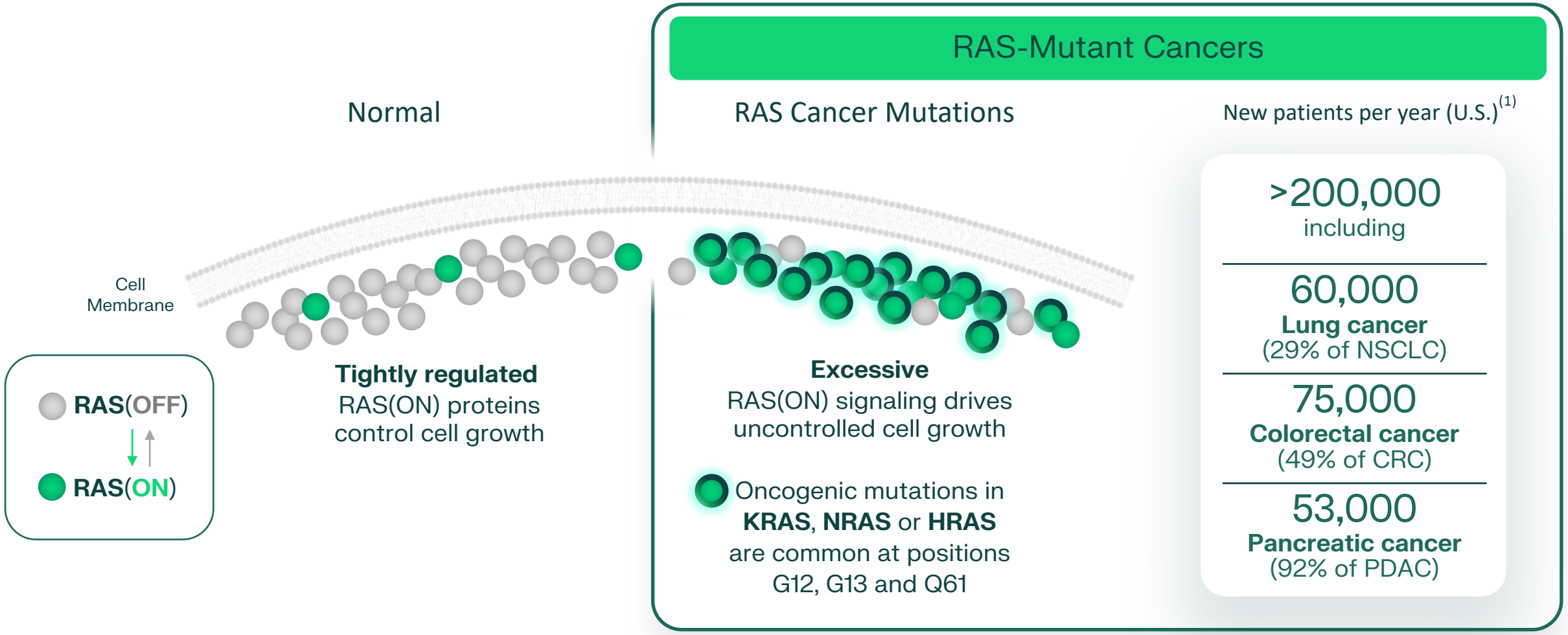
This presentation concerns product candidates that are under clinical investigation and which have not yet been approved for marketing by the U.S. Food and Drug Administration (FDA). These product candidates are currently limited by Federal law to investigational use, and no representation is made as to their safety or effectiveness for the purposes for which they are is being investigated.

Revolution Medicines

Mission: to revolutionize treatment for patients with RAS-addicted cancers through the discovery, development and delivery of innovative, targeted medicines.

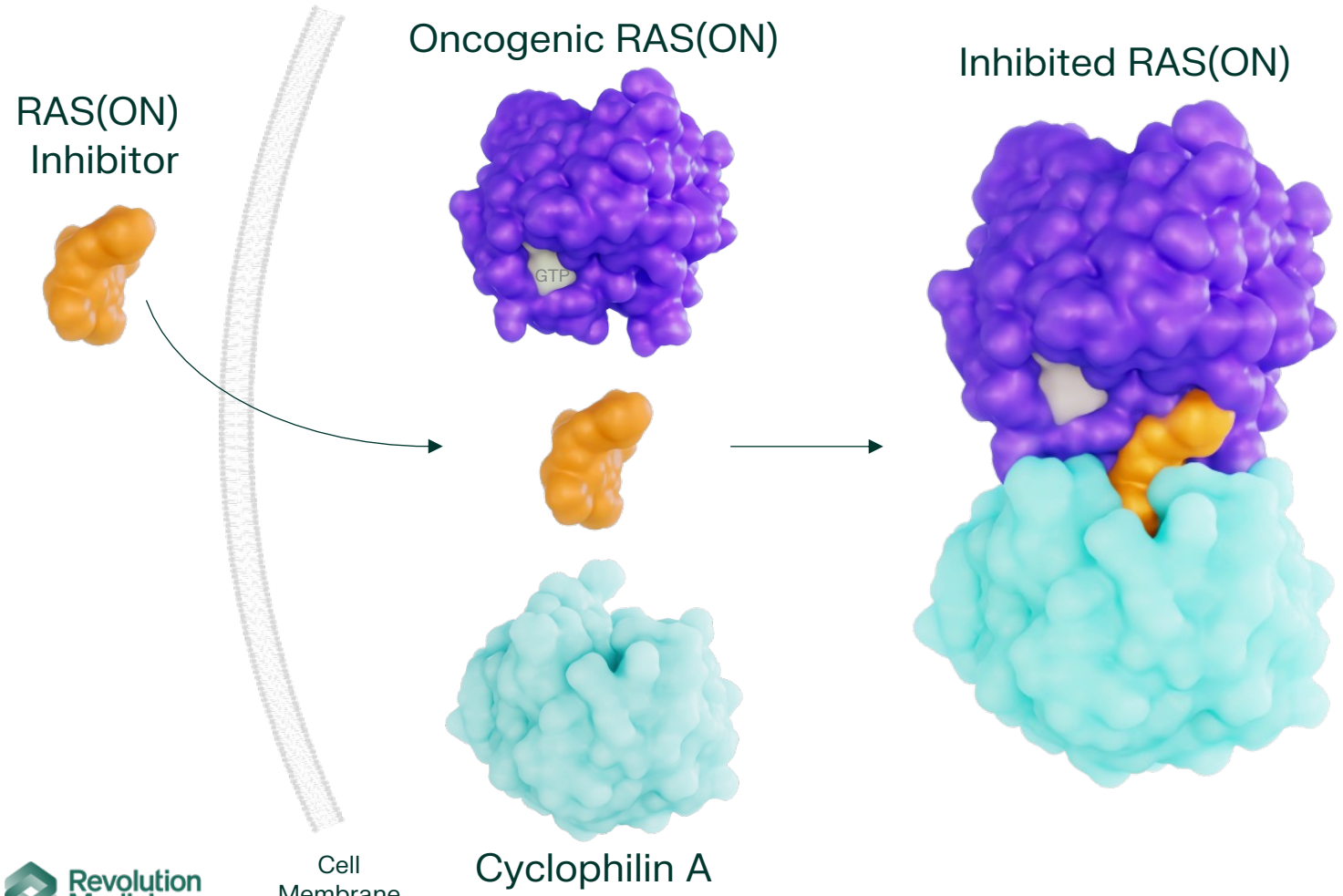
- **Pioneering class of RAS(ON) inhibitor drug candidates** targeting oncogenic drivers of common, life-threatening cancers
- **Unprecedented RAS(ON) multi-selective inhibitor (RMC-6236) and RAS(ON) G12C-selective inhibitor (RMC-6291)** show promising and highly differentiated initial clinical profiles
- **On track toward late-stage development of RMC-6236** and advancement of mutant-selective inhibitors led by RMC-6291 and RMC-9805

Portfolio of RAS(ON) Inhibitors Designed to Target 30% of Human Cancers



(1) Estimated using tumor mutation frequencies from Foundation Medicine Insights March 2022 and scaled to estimated patient numbers using cancer incidence from ACS Cancer Facts and Figures 2023 (see appendix for additional detail); NSCLC = non-small cell lung cancer; CRC = colorectal cancer; PDAC = pancreatic ductal adenocarcinoma

Pioneering Tri-complex RAS(ON) Inhibitors Designed to Deliver Robust and Durable Anti-tumor Activity



- **Direct inhibition of RAS(ON) cancer drivers**
- **Deep and durable suppression of RAS cancer signaling designed to defy common drug resistance mechanisms**
- **Clinical validation of first two RAS(ON) Inhibitors studied as single agents**

Initial Clinical Profiles of RAS(ON) Inhibitors Support Broad Set of Potential Opportunities to Treat RAS-Addicted Cancers

		Target Genotypes
Multi-Selective		
RMC-6236	Clinical validation in NSCLC and PDAC	**G12X and expansion**[1]
Mutant-Selective		
RMC-6291	Evidence of differentiated clinical activity in NSCLC and CRC	**G12C**
RMC-9805	Dose escalation ongoing	**G12D**

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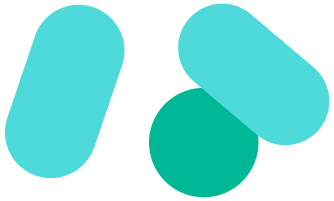
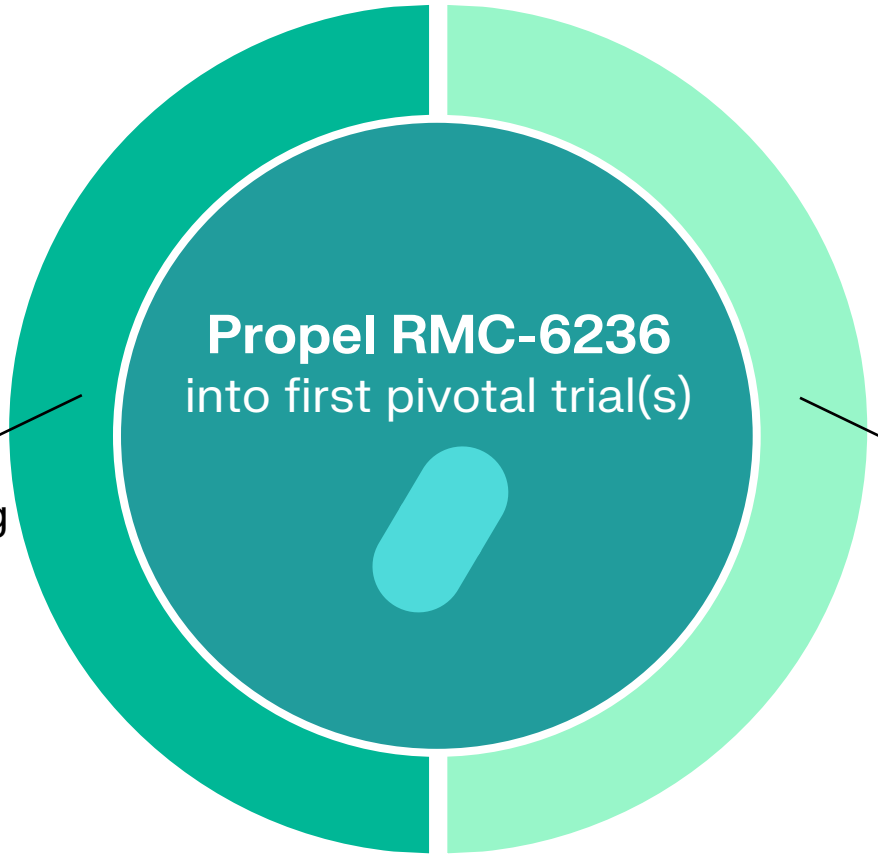
(1) KRAS G12X initially defined as mutation at codon 12 which encodes glycine (G) to X where X = A, D, R, S, or V. RMC-6236-001 protocol amended in August 2023 to broaden enrollment, now allowing patients with tumors bearing mutations in any of the three hotspots (G12X/G13X/Q61X) in any of the three major RAS isoforms (KRAS/NRAS/HRAS); G12X broadened to include G12C.

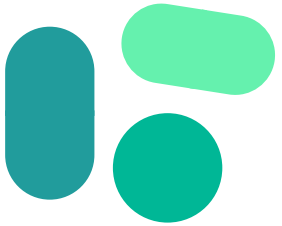
2024 Capital Allocation Priorities to Advance Pioneering RAS(ON) Inhibitor Pipeline …

Expand reach of RMC-6236
by clinically assessing opportunities (1L, types, mutations)





Qualify mutant-selective inhibitors
led by RMC-6291 and RMC-9805 for late-stage development



… driving to

Industry-Leading Targeted Medicines Franchise for RAS-Addicted Cancers

RAS(ON) Multi-Selective Inhibitor RMC-6236

RMC-6236-001 Phase 1 Study Design

Key Eligibility Criteria

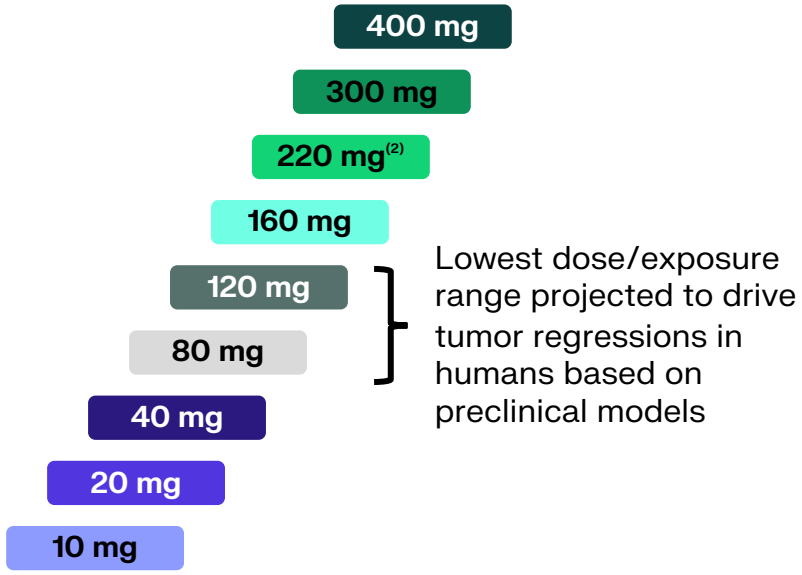
- Advanced solid tumors with KRAS G12X mutations[1] (initially excluding KRAS G12C)
- Received prior standard therapy appropriate for tumor type and stage
- ECOG PS 0–1
- No active brain metastases

Key Endpoints

- Safety and tolerability
- Pharmacokinetics
- Anti-tumor activity

Dose Escalation

RMC-6236 administered orally QD



Additional patients with PDAC or NSCLC were enrolled at dose levels that cleared DLT evaluation

Dose Optimization + RAS Genotype and Tumor Type Expansion

RMC-6236-001 Clinical Trial: https://clinicaltrials.gov/study/NCT05379985

(1) KRAS G12X initially defined as mutation at codon 12 which encodes glycine (G) to X where X = A, D, R, S, or V.

(2) 220 mg cleared DLT evaluation and a dose of 200 mg was selected for further expansion/optimization.

DLT, dose-limiting toxicity; ECOG PS, Eastern Cooperative Oncology Group Performance Status; QD, once daily.

RMC-6236-001: Summary of Treatment-Related Adverse Events

	Total (n=131)				
Maximum severity of TRAEs	**Grade 1**	**Grade 2**	**Grade 3**	**Grade 4**	**Any Grade**
TRAEs occurring in ≥10% of patients, n (%)					
Rash*	57 (44)	29 (22)	6 (5)	0	92 (70)
Nausea	41 (31)	14 (11)	0	0	55 (42)
Diarrhea	32 (24)	9 (7)	1 (1)	0	42 (32)
Vomiting	27 (21)	9 (7)	0	0	36 (28)
Stomatitis	10 (8)	9 (7)	2 (2)	0	21 (16)
Fatigue	12 (9)	4 (3)	0	0	16 (12)
Other select TRAEs, n (%)					
ALT elevation	6 (5)	1 (1)	1 (1)‡	0	8 (6)
AST elevation	6 (5)	0	1 (1)‡	0	7 (5)
Electrocardiogram QT prolonged	1 (1)	0	0	0	1 (1)
TRAEs leading to dose reduction†, n (%)	0	9 (7)	2 (2)	0	11 (8)
TRAEs leading to treatment discontinuation, n (%)	0	0	0	1 (1)	1 (1)

- Median duration of treatment at the time of data extraction was 2.27 months (range: 0.2–14)
- One Grade 4 TRAE occurred in a patient with PDAC treated at 80 mg who had a large intestine perforation at the site of an invasive tumor that reduced in size while on treatment (TRAE leading to treatment discontinuation)
- No fatal TRAEs were observed. Two patients discontinued study treatment due to death: one patient with PDAC (120 mg) died due to PD; one patient with NSCLC (200 mg) died due to unknown cause reported as unrelated to RMC-6236

‡ Post-data extraction, the Grade 3 ALT and AST elevations were associated with biliary obstruction and reported as unrelated to RMC-6236

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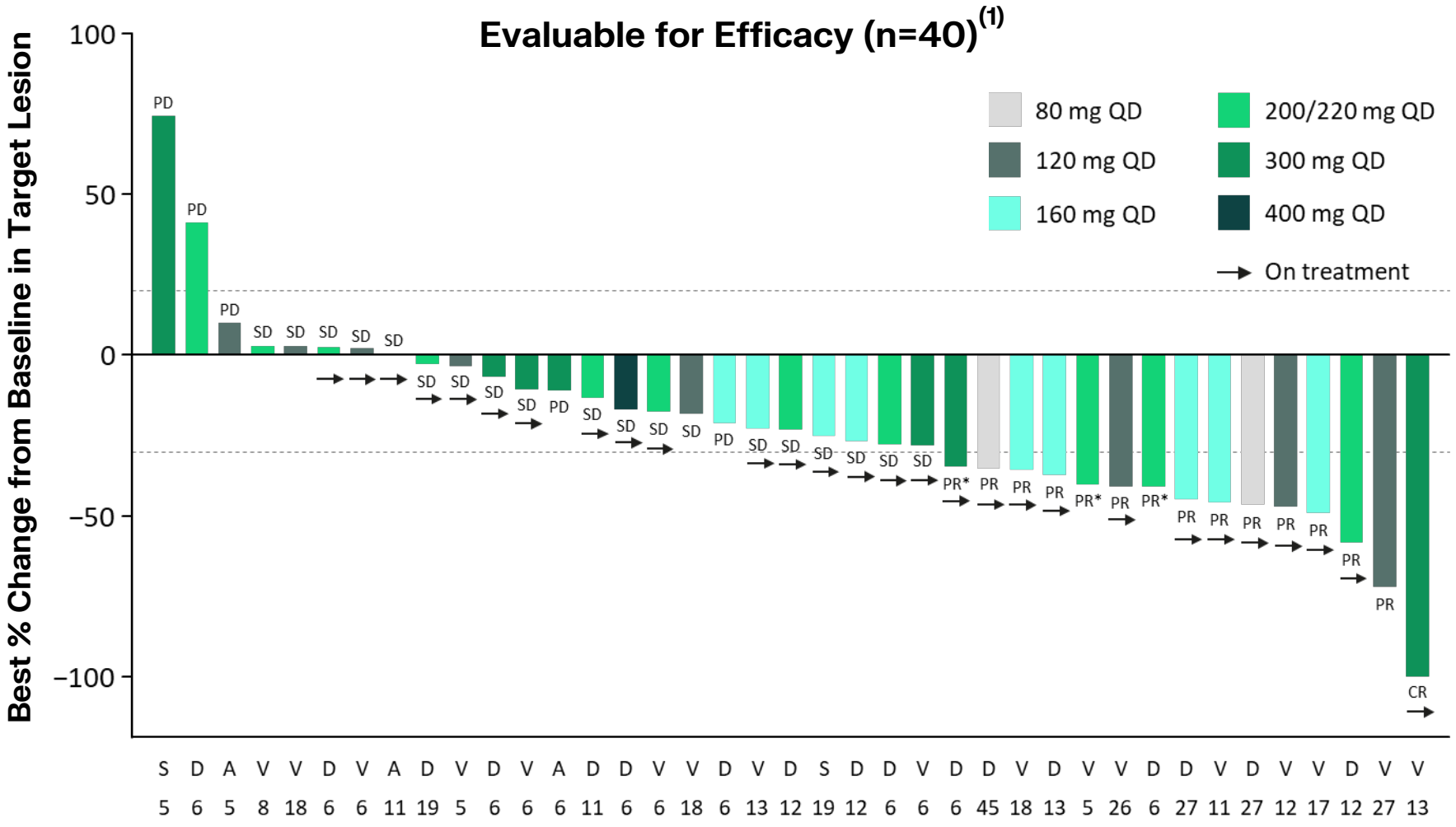
*Includes preferred terms of dermatitis acneiform, rash maculopapular, rash, rash pustular, dermatitis psoriasiform, erythema, rash erythematous; multiple types of rash may have occurred in the same patient; †The most common TRAE leading to dose reduction was rash (acneiform or maculopapular); there were no reductions at doses ≤80 mg. AE, adverse event; ALT, alanine transaminase; AST, aspartate transferase; PD, progressive disease; TRAEs, treatment-related adverse events.

Data Extracted 11 Sep 2023.

KRAS G12X NSCLC: Best Overall Response to RMC-6236



KRAS G12 Mutation

Week of Most Recent scan

RMC-6236-001: Clinical Activity in KRAS G12X NSCLC(2)	
Best overall response, n (%)	
Complete response	1 (3)
Partial response	14 (35)
Stable disease	19 (48)
Progressive disease	5 (13)
Not evaluable(3)	1 (3)
ORR, n (%)	15 (38)
Confirmed, n	12
DCR (CR+PR+SD), n (%)	34 (85)
SOC Benchmark(4)	
Docetaxel, ORR (%)	(13)
DCR (%)	(60)

(1) Patients who received first dose of RMC-6236 at least 8 weeks prior to data extract date.
(2) Tumor response per RECIST 1.1.
(3) One subject withdrew from study without post-baseline scans.
(4) SOC=standard of care; efficacy benchmark for docetaxel taken from CodeBreaK 200, Lancet (2023) 401: 733-746.
*Unconfirmed PR per RECIST 1.1.

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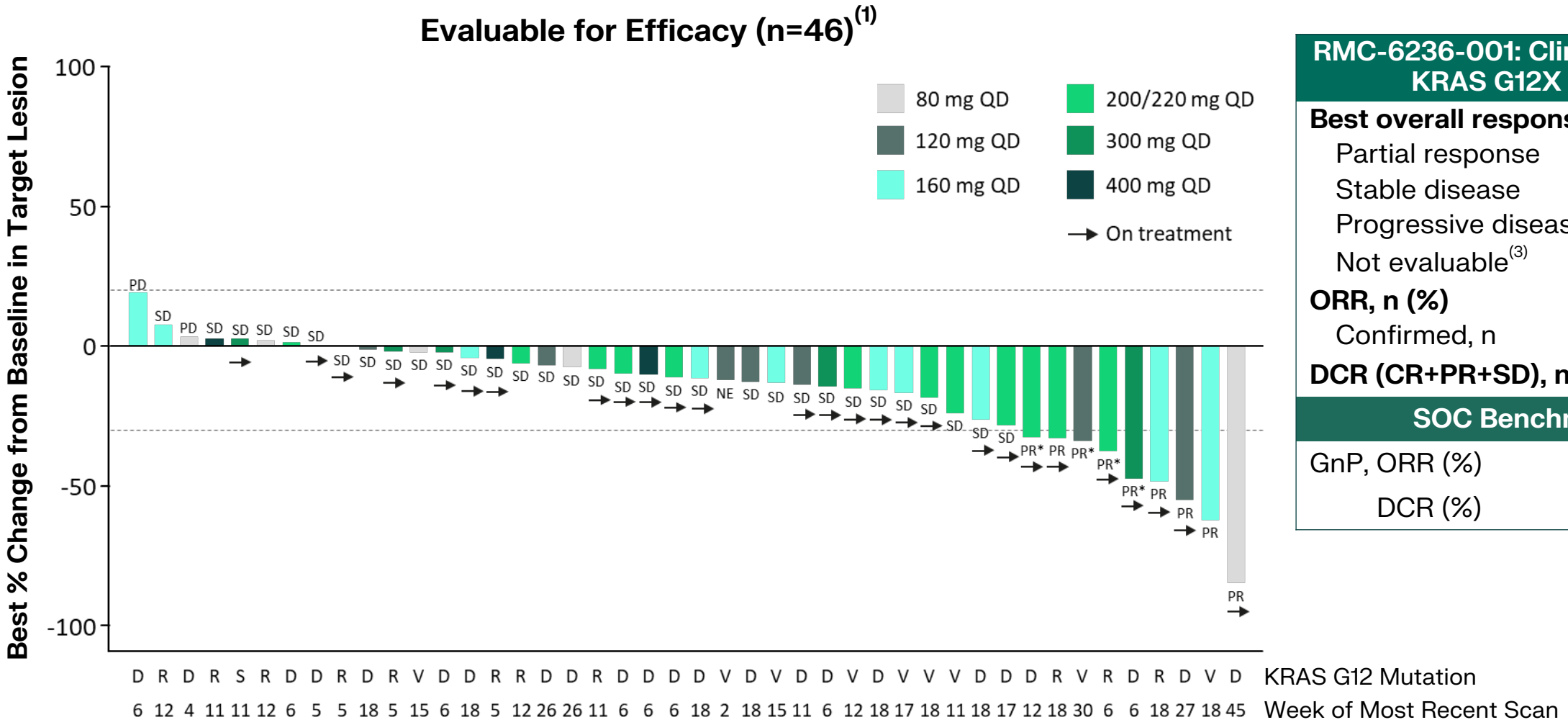
Data Extracted 12 Oct 2023.

KRAS G12X PDAC: Best Overall Response to RMC-6236



RMC-6236-001: Clinical Activity in KRAS G12X PDAC(2)	
Best overall response, n (%)	
Partial response	9 (20)
Stable disease	31 (67)
Progressive disease	3 (7)
Not evaluable(3)	3 (7)
ORR, n (%)	9 (20)
Confirmed, n	5
DCR (CR+PR+SD), n (%)	40 (87)
SOC Benchmarks(4)	
GnP, ORR (%)	(11)
DCR (%)	(56)

(1) Patients who received first dose of RMC-6236 at least 8 weeks prior to data extract date.
(2) Tumor response per RECIST 1.1.
(3) Two patients died prior to first post-baseline scan; 1 patient had scan after 11 days of treatment and subsequently died due to PD.
(4) SOC=standard of care; no clearly established standard of care in 2L PDAC; GnP=Gemcitabine plus nab-paclitaxel; efficacy benchmarks for GnP taken from Br J Cancer (2022) 126:1394-1400.
*Unconfirmed PR per RECIST 1.1.

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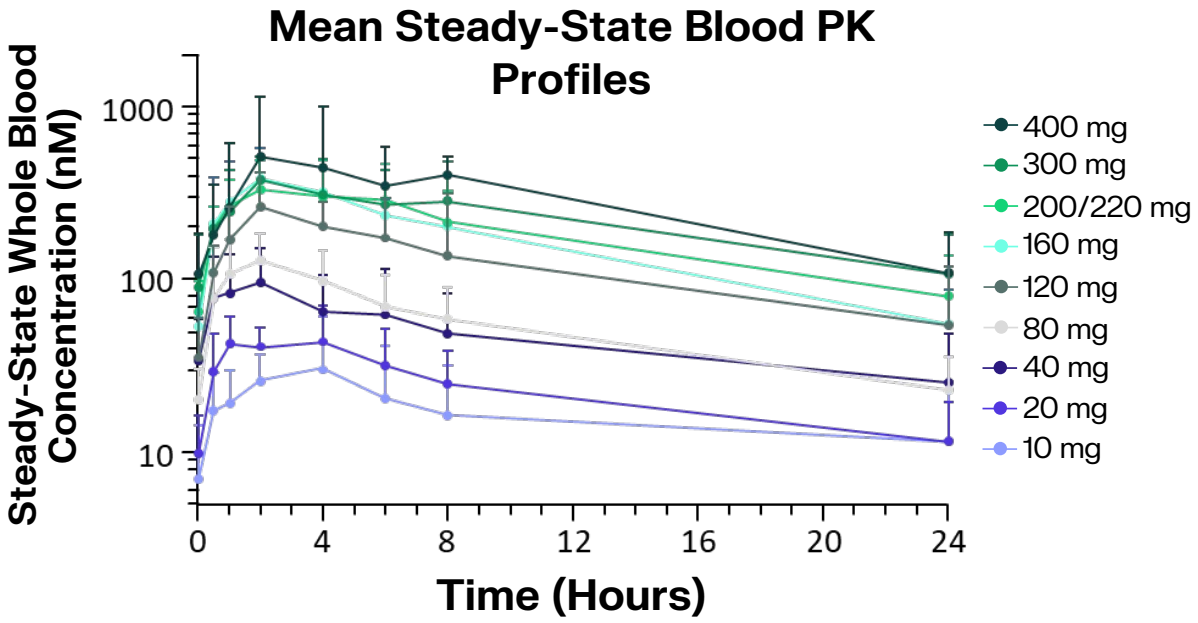
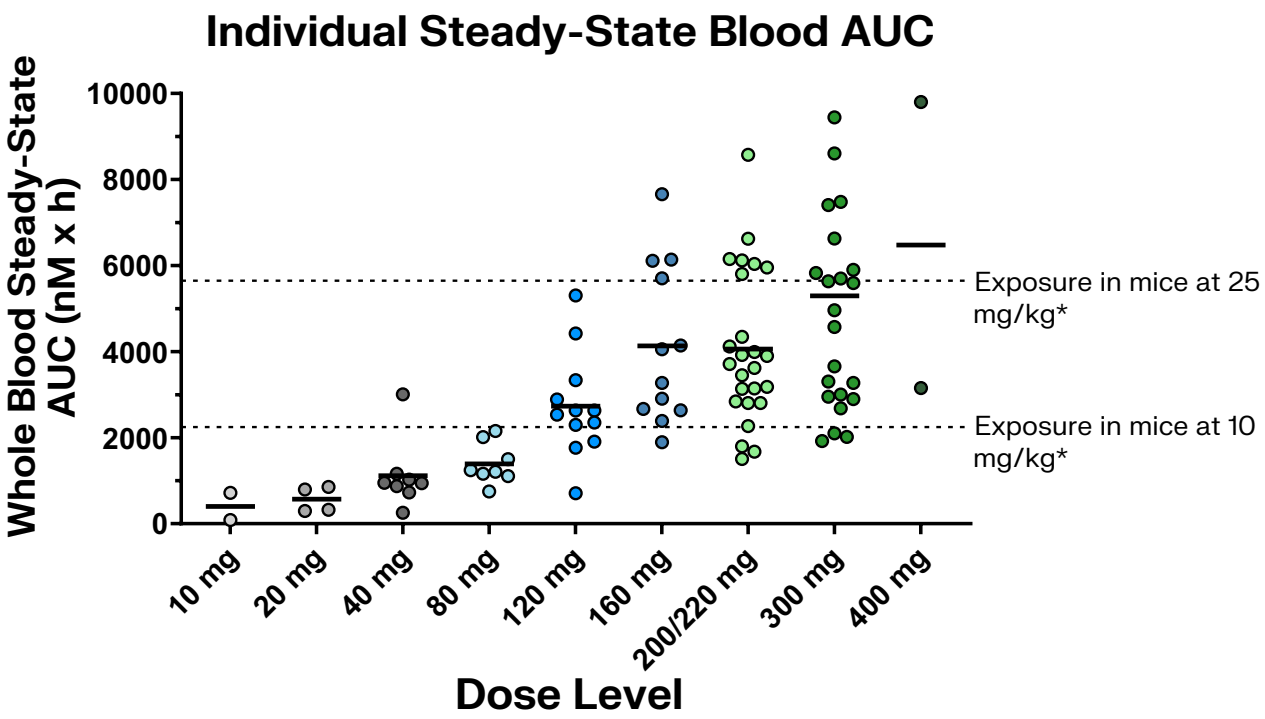
Data Extracted 12 Oct 2023.

Zeroing In on RMC-6236 Monotherapy Dose Selection





- Dose-dependent increases in exposure with minimal accumulation were observed after repeat daily dosing
- Dose levels ≥80 mg achieved exposures that induced tumor regressions in human xenograft models with $KRAS^{G12X}$ mutations in mice[(1)]
  - 10 mg/kg QD induces tumor regressions in sensitive models
  - 25 mg/kg QD induces tumor regressions in the majority of models

*Exposure corrected with cross-species protein binding and blood/plasma partitioning. Left: steady-state concentrations from Cycle 1 Day 15. Error bars represent standard deviation; right: steady-state AUC is Cycle 1 Day 15 AUC_{last}. Each circle represents an individual patient AUC. Horizontal bars represent mean AUC for each dose level (10 mg: n=2; 20 mg: n=4; 40 mg: n=7; 80 mg: n=8; 120 mg: n=12; 160 mg: n=12, 200 mg: n=13; 220 mg: n=4; 300 mg: n=9; 400 mg: n=2); AUC, area under the curve; PK, pharmacokinetics.

(1) Singh M, et al. Presentation at American Association for Cancer Research Annual Meeting, 8–13 April 2022, New Orleans, USA; abstract #3597.

Data Extracted 22 Sep 2023.

Key RMC-6236-001 Monotherapy Expansion Cohorts Underway

Corporate Priority	Cohort	Dosing	Purpose
Expand reach of RMC-6236	**NSCLC**		
	G12X dose optimization (300 mg and below)	✓	Dose selection for pivotal trial
	RAS G13X and Q61X expansion (300 mg)	✓	Pivotal trial design
	PDAC		
	G12X dose optimization (300 mg and below)	✓	Dose selection for pivotal trial
	RAS G13X and Q61X expansion (300 mg)	✓	Pivotal trial design
	CRC		
	G12X expansion (300 mg)	✓	Signal seeking
	RAS G13X and Q61X expansion (300 mg)	✓	Signal seeking

- G12C included in G12X across all tumor types and cohorts

RMC-6236-001 protocol amended in August 2023 to broaden enrollment, now allowing patients with tumors bearing mutations in any of the three hotspots (G12X/G13X/Q61X) in any of the three major RAS isoforms (KRAS/NRAS/HRAS); G12X broadened to include G12C

Proposed Global Randomized Phase 3 Trial in Patients with Previously-Treated RAS Mutant NSCLC

Trial Design[1]

R

RMC-6236

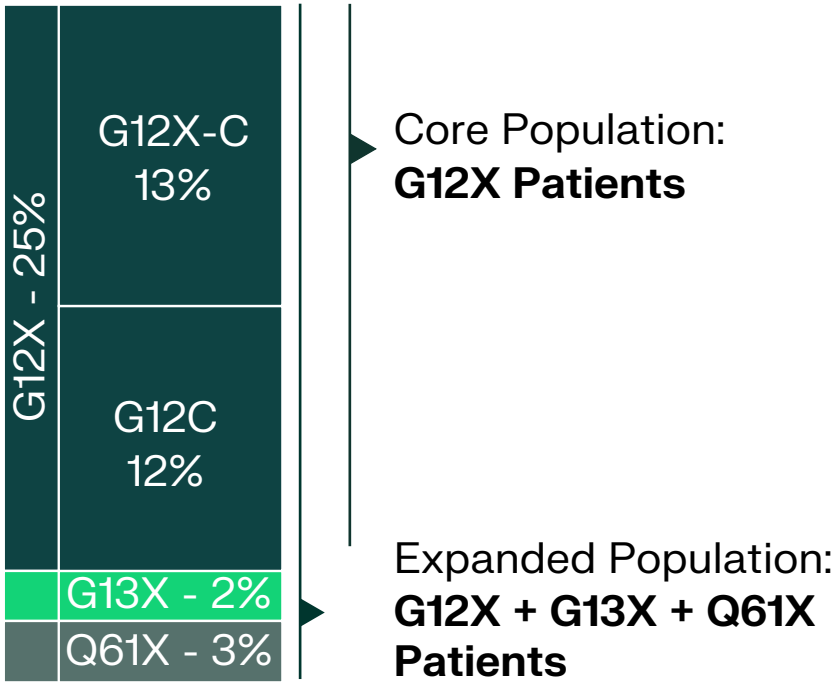
Docetaxel

Endpoints

PFS
OS
Patient Reported Outcomes

- **N** > 400 patients
- **Prior therapies**: Anti-PD-(L)1 and platinum-containing regimen in metastatic setting; RAS inhibitor naïve (including G12C inhibitor)
- **Biomarker**: RAS G12X, G13X, or Q61X mutation
- **Study Initiation**: Aiming for 2024

Potential Patient Populations[1,2]



- Potential for nested trial design to enable evaluation of core and expanded patient populations[1]

R = Randomized
(1) Study design subject to change based on regulatory authority feedback
(2) Percentages of all NSCLC patients with tumors bearing RAS G12X, G13X, or Q61X genotypes; estimated using tumor mutation frequencies from Foundation Medicine Insights March 2022 and scaled to estimated patient numbers using cancer incidence from ACS Cancer Facts and Figures 2023 (see appendix for additional detail);

Proposed Global Randomized Phase 3 Trial of RMC-6236 in Patients with Previously-Treated PDAC

Trial Design[1]

R

RMC-6236

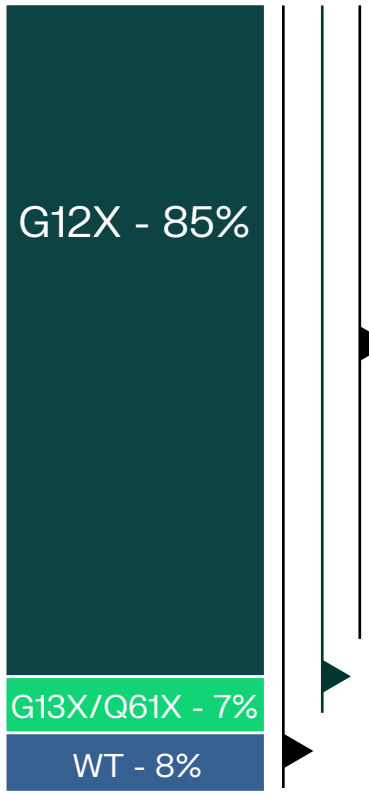
Physician's choice: e.g., GnP or mFOLFIRINOX

Endpoints

PFS
OS
Patient Reported Outcomes

- **N** > 500 patients
- **Prior therapies**: Fluoropyrimidine or gemcitabine-based regimen; RAS inhibitor naïve (including G12C inhibitor)
- **Biomarker**: All comers, RAS mutation testing (G12X, G13X, or Q61X) to allow stratification
- **Study Initiation**: Potentially in 2024

Potential Patient Populations[1,2]



Core Population:
G12X Patients

Expanded Populations:
- **G12X + G13X + Q61X patients**
- **All PDAC patients**

- Potential for nested trial design to enable evaluation of core and expanded patient populations[1]

R = Randomized; WT=wild-type
(1) Study design subject to change based on regulatory authority feedback
(2) Percentages of all PDAC patients with tumors bearing RAS G12X, G13X, Q61X or WT genotypes; estimated using tumor mutation frequencies from Foundation Medicine Insights March 2022 and scaled to estimated patient numbers using cancer incidence from ACS Cancer Facts and Figures 2023 (see appendix for additional detail);

Complementary RAS(ON) Inhibitors Designed for Monotherapy and Combination Strategies Against RAS-Addicted Cancers

RAS Multi-Selective

- Monotherapy with broad potential for RAS-addicted cancers
- Backbone of RAS(ON) inhibitor doublets with mutant-selective RAS(ON) inhibitors
- Targeted agent for SOC combinations, including immunotherapies

RAS Mutant-Selective

- Alternative monotherapy approaches
- Complementary to RAS multi-selective inhibitor in RAS(ON) inhibitor doublets
- Differentiated targeted agent profiles for SOC combinations, including immunotherapies

RAS(ON) G12C-Selective Inhibitor RMC-6291

RMC-6291-001 Phase 1 Study Design

Key Eligibility Criteria

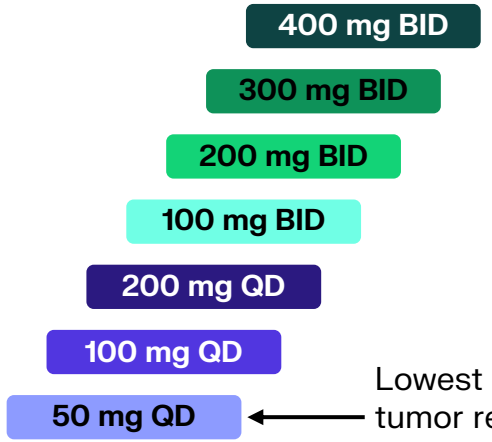
- Advanced solid tumors with $KRAS^{G12C}$ mutations
- Received prior standard therapy including treatment with $KRAS^{G12C}$(OFF) inhibitors
- ECOG PS 0–1
- No active brain metastases

Key Endpoints

- Safety and tolerability
- Pharmacokinetics
- Anti-tumor activity

Dose Escalation

RMC-6291 administered orally QD or BID



Additional patients with NSCLC or CRC were enrolled at dose levels that cleared DLT evaluation (backfill enrollment and dose optimization)

Dose Optimization

RMC-6291-001 Clinical Trial: https://clinicaltrials.gov/study/NCT05462717
DLT=dose-limiting toxicity; ECOG PS=Eastern Cooperative Oncology Group Performance Status; QD=once daily; BID=twice daily

RMC-6291-001: Summary of Treatment-Related Adverse Events

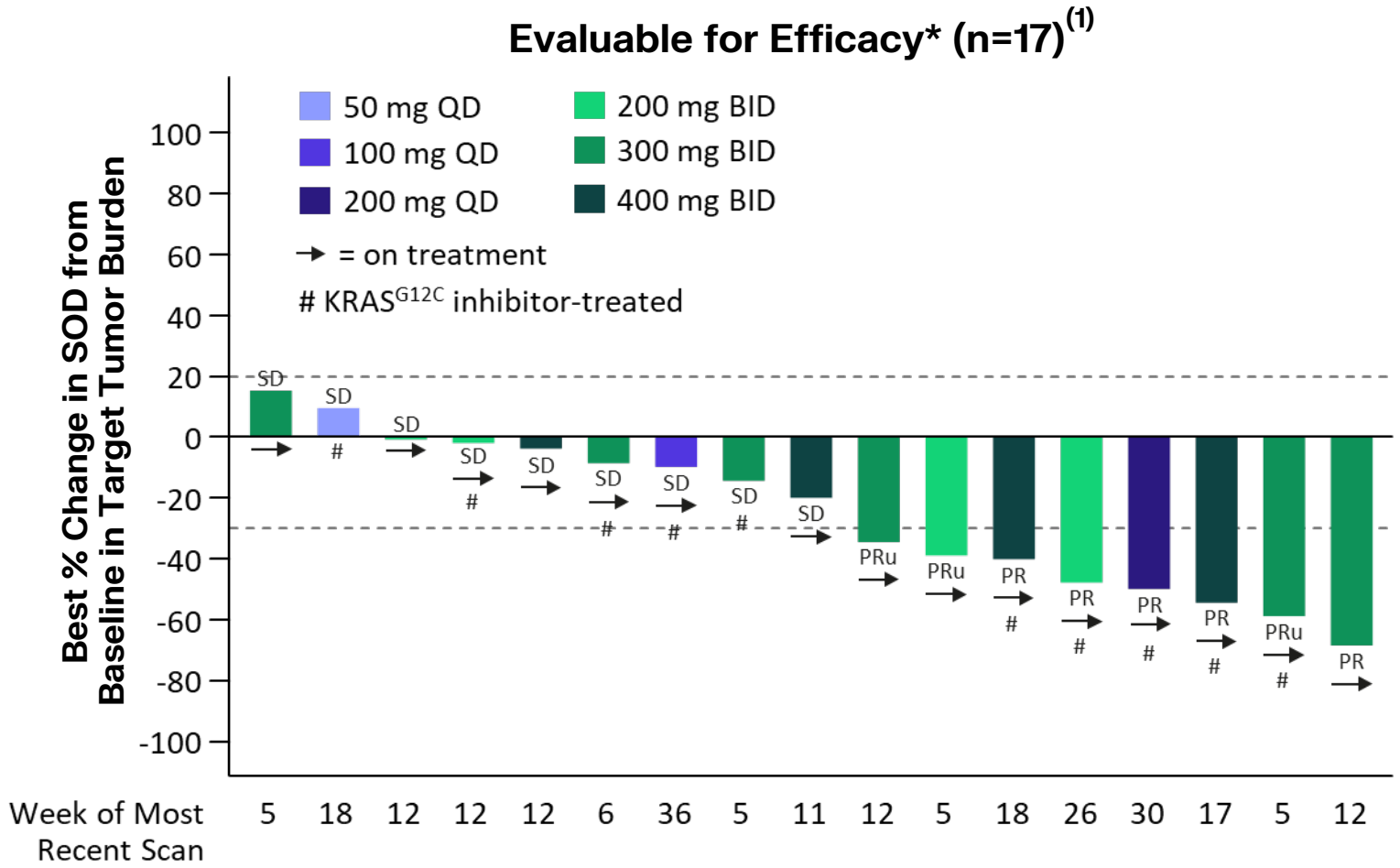
Total (n=63)				
Maximum Severity of TRAEs	**Grade 1**	**Grade 2**	**Grade 3**	**Any Grade**
TRAEs occurring in ≥10% of patients, n (%)				
Diarrhea	10 (16)	7 (11)	1 (2)	18 (29)
Nausea	14 (22)	3 (5)	0	17 (27)
ECG QT prolonged	8 (13)	1 (2)	7 (11)	16 (25)
QTcF* ≥501 ms	–	–	1 (2)	–
Fatigue	4 (6)	4 (6)	0	8 (13)
Vomiting	6 (10)	2 (3)	0	8 (13)
AST increased	7 (11)	0	0	7 (11)
TRAEs leading to dose reduction, n (%)	0	1 (2)	8 (13)	9 (14)
TRAEs leading to treatment discontinuation, n (%)	0	0	1 (2)	1 (2)

- No treatment-related Grade 4 or 5 AEs or SAEs have been reported
- No patients had cardiac sequelae (e.g., torsade de pointes) associated with an ECG QT prolonged event

*QTcF refers to QT interval corrected for heart rate by Fridericia's formula.
AE, adverse event; AST, aspartate transferase; ECG, electrocardiogram; SAE, serious adverse event, TRAE, treatment-related adverse event.

Data Extracted 05 October 2023.

KRAS^{G12C} NSCLC Previously Treated with or Naïve to a KRAS^{G12C}(OFF) Inhibitor: Best Overall Response to RMC-6291



RMC-6291-001: Clinical Activity in KRAS G12C NSCLC(2)

Best overall response, n (%)	Prior G12Ci (n=10)	Naïve to G12Ci (n=7)
Partial response(3)	5 (50)	3 (43)
Stable disease	5 (50)	4 (57)
Progressive disease	0	0
ORR, n (%)	5 (50)	3 (43)
DCR (CR+PR+SD), n (%)	10 (100)	7 (100)

(1) All treated patients who received a first dose of RMC-6291 at least 8 weeks prior to data extract date.
(2) Tumor response per RECIST 1.1.
(3) PR includes 5 confirmed and 3 unconfirmed.
Pru=Unconfirmed PR per RECIST 1.1; G12Ci=G12C inhibitor.

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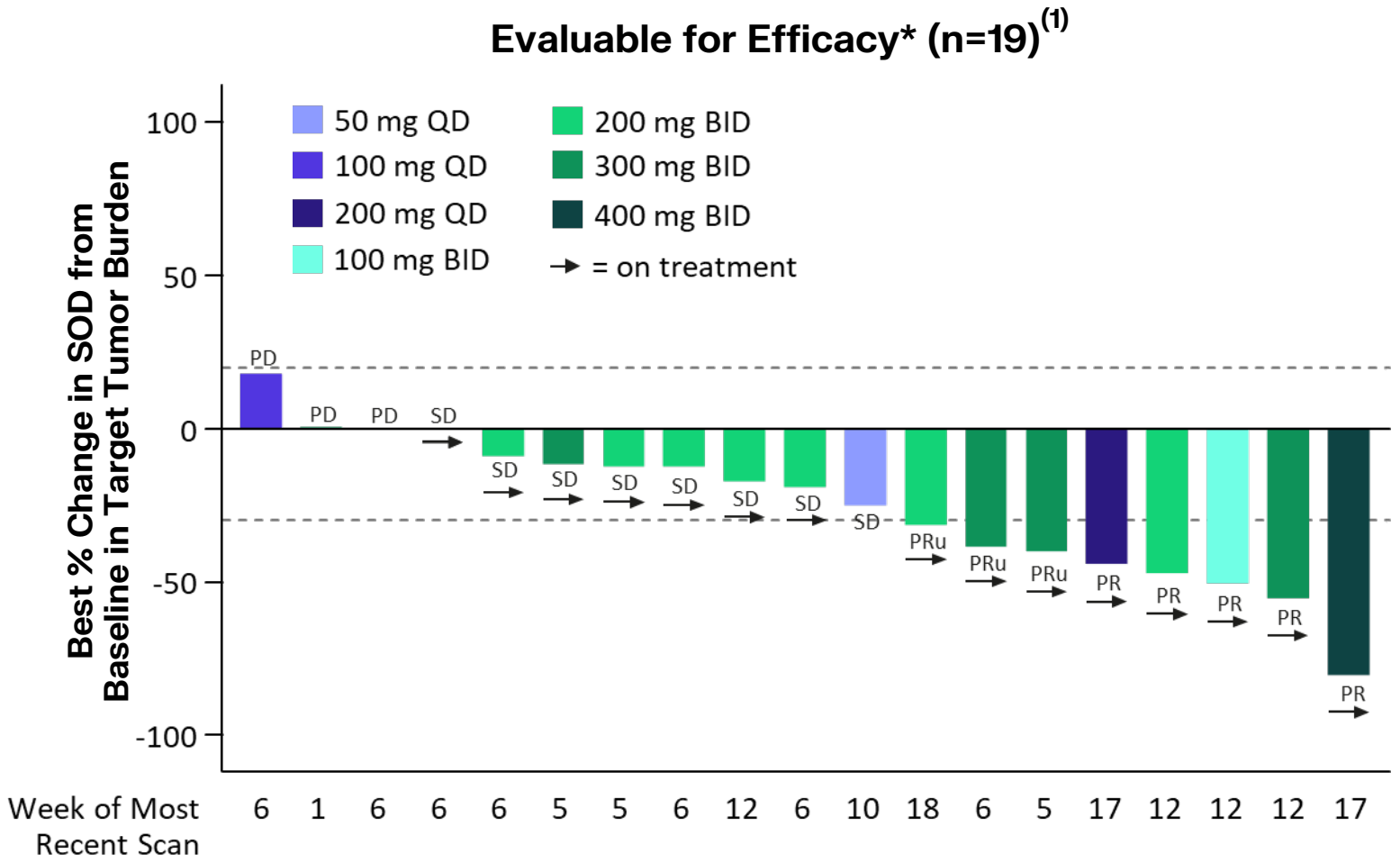
Data Extracted 05 October 2023.

KRAS^{G12C} CRC Naïve to KRAS^{G12C}(OFF) Inhibitor: Best Overall Response to RMC-6291



RMC-6291-001: Clinical Activity in KRAS G12C CRC(2)	
Best overall response, n (%)	**n=20†**
Partial response(3)	8 (40)
Stable disease	8 (40)
Progressive disease(4)	4 (20)
ORR, n (%)	8 (40)
DCR (CR+PR+SD), n (%)	16 (80)

(1) All treated patients who received first dose of RMC-6291 at least 8 weeks prior to data extract date.
(2) Tumor response per RECIST 1.1.
(3) PR includes 5 confirmed and 3 unconfirmed.
(4) One patient had PD due to a new lesion and target lesion measurements were not available.
Pru=Unconfirmed PR per RECIST 1.1.

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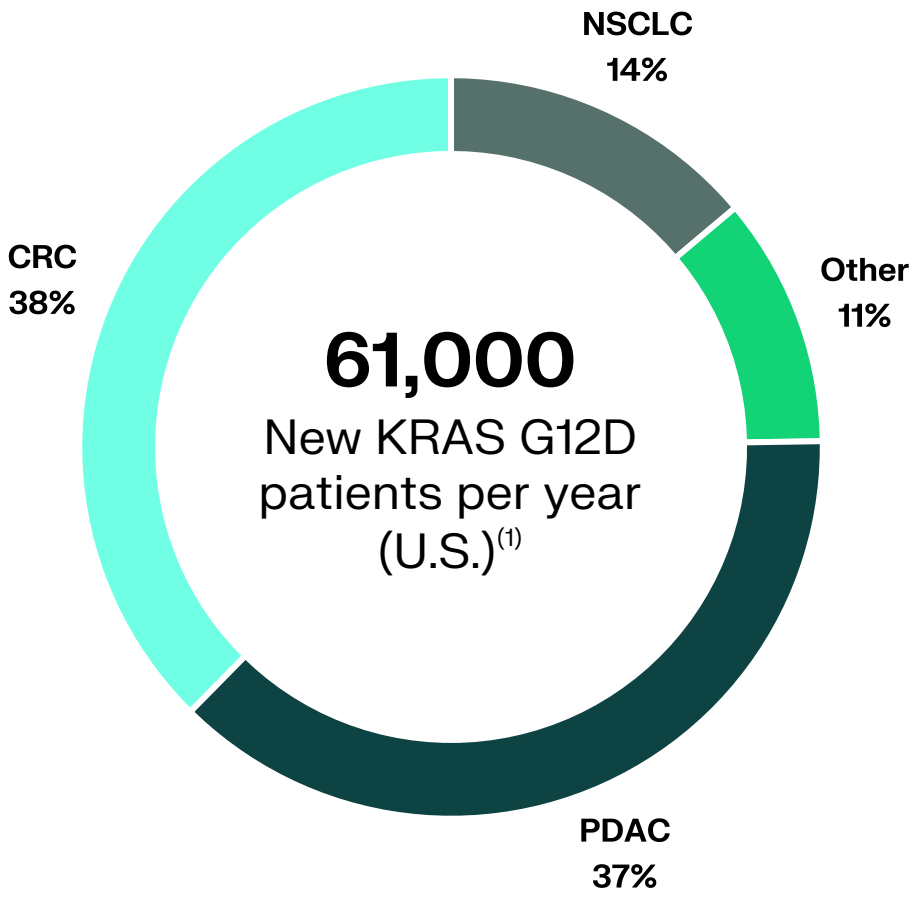
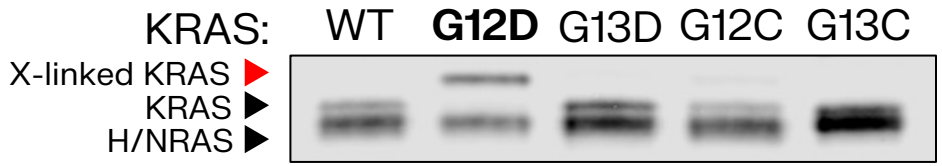
Data Extracted 05 October 2023.

RAS(ON) G12D-Selective Inhibitor RMC-9805

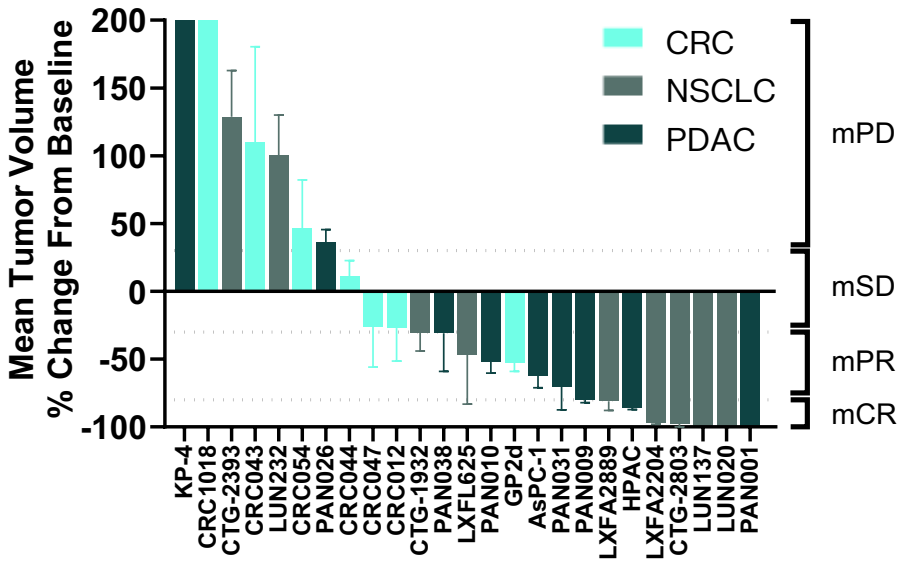
RMC-9805: Clinical Stage, RAS(ON) Mutant-Selective, Covalent Inhibitor for RAS G12D Cancers



Selective Covalent Binding to KRAS G12D



In Vivo Anti-Tumor Activity across KRAS G12D Cancer Models



(1) Estimated using tumor mutation frequencies from Foundation Medicine Insights March 2022 and scaled to estimated patient numbers using cancer incidence from ACS Cancer Facts and Figures 2023 (see appendix for additional detail)

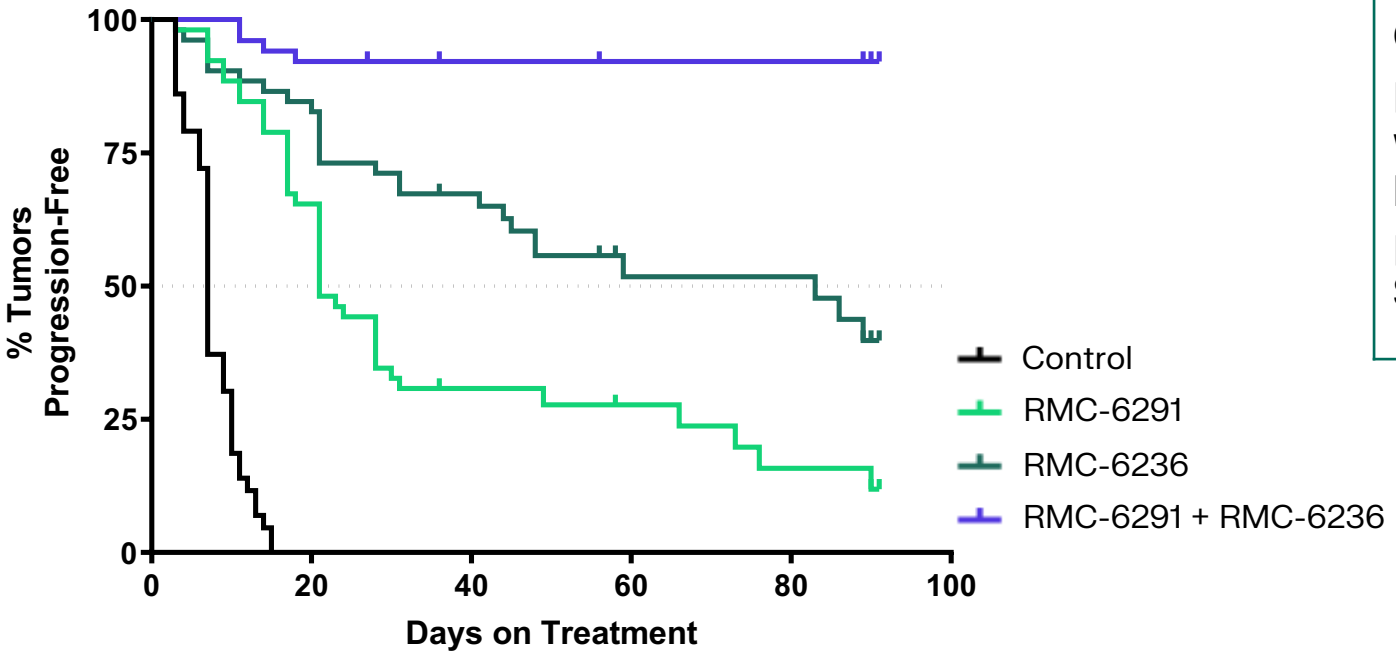
RVMD preclinical research as of 11/02/22; NSCLC = non-small cell lung cancer; PDAC = pancreatic ductal adenocarcinoma; CRC = colorectal cancer

RMC-9805 dosed at 100 mg/kg po qd; n=3-8/group; Responses assigned according to mRECIST: mPD = progressive disease; mSD = stable disease; mPR = partial response; mCR = complete response

RMC-9805-001 Clinical Trial: https://clinicaltrials.gov/study/NCT06040541

Phase 1b Combo: RMC-6236 + RMC-6291 Doublet Designed to Overcome Resistance and Prolong Durability in KRAS G12C NSCLC

Preclinical Validation[1]



RMC-6291-101 Clinical Trial[2]

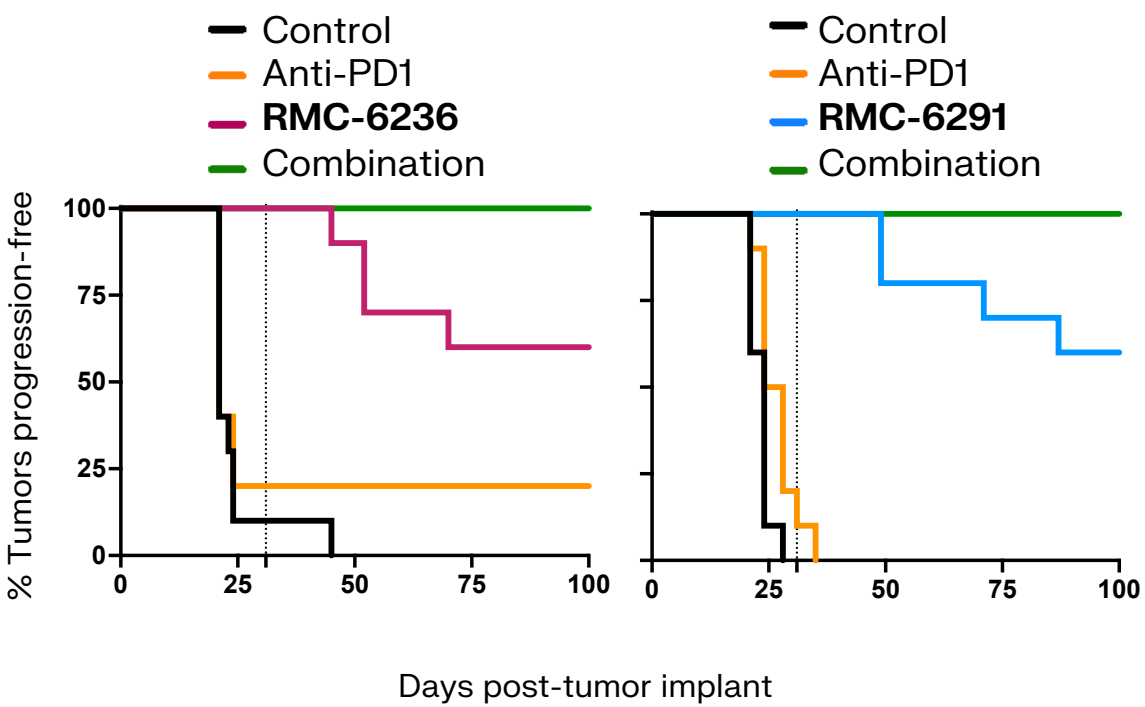
Objectives: evaluate safety, tolerability and preliminary activity of RMC-6236 combined with RMC-6291
Patient Population: KRAS G12C solid tumors, primarily NSCLC and CRC
Study Status: First patient dosed 4Q23

- RAS(ON) inhibitor doublet evaluated across seven models, including five identified as resistant to RMC-6291 monotherapy

(1) RVMD preclinical research; NSCLC = non-small cell lung cancer; RMC-6236 dosed at 25 mg/kg po qd (n=52); RMC-6291 dosed at 100 or 200 mg/kg po qd (n=52); Combination (n=51). For each group, n = total number of animals from the seven models that comprise the dataset. Progression defined as tumor doubling from baseline.
(2) RMC-6291-101 Clinical Trial: https://www.clinicaltrials.gov/study/NCT06128551

Phase 1b Combos: RAS(ON) Inhibitor Combinations with Pembrolizumab to Inform Potential Evaluation in 1L NSCLC

Preclinical Validation[1]



RMC-LUNG-101 Clinical Trial: Pembrolizumab[2]

Objectives: evaluate safety, tolerability and preliminary activity of RMC-6236 and RMC-6291 each combined with pembrolizumab
Patient Population: RMC-6236 in KRAS-mutant NSCLC, RMC-6291 in KRAS G12C NSCLC
Study Status: Recruiting

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(1) RVMD preclinical research; RMC-6236 and RMC-6291 experiments conducted in CT26 model engineered to express $KRAS^{G12C}$; RMC-6236 (25 mg/kg po qd) or RMC-6291 (200 mg/kg po qd) dosed for 14 days; Vertical dashed lines represent treatment stop; Kaplan-Meier progression defined as tumor doubling from baseline
(2) RMC-LUNG-101 Clinical Trial: https://www.clinicaltrials.gov/study/NCT06162221

Key RAS(ON) Inhibitor Combination Cohorts

Corporate Priority	Cohort	Status	Purpose
Expand reach of RMC-6236	**NSCLC**[1]		
	RMC-6236 + pembrolizumab +/- chemotherapy	dosing	qualification for potential 1L
	PDAC		
	RMC-6236 + chemotherapy	pending	qualification for potential 1L
	CRC		
	RMC-6236 + anti-EGFR	pending	signal seeking
	RMC-6236 + chemotherapy	pending	signal seeking
Qualify mutant-selective inhibitors for late-stage development	**NSCLC**[1]		
	RMC-6291 + pembrolizumab +/- chemotherapy	dosing	qualification for potential 1L
	Solid tumors[2]		
	RMC-6291 + RMC-6236	dosing	qualification for potential 1L

Revolution Medicines

(1) RMC-LUNG-101 Clinical Trial: https://www.clinicaltrials.gov/study/NCT06162221
(2) RMC-6291-101 Clinical Trial: https://www.clinicaltrials.gov/study/NCT06128551

Highlights of 2H-2023

RAS(ON) Multi-Selective Inhibitor

- **Encouraging initial RMC-6236 monotherapy safety, tolerability and antitumor activity profiles in both NSCLC and PDAC reported in October**
  - Favorable safety and response trends continue to build, including in 300 mg daily cohort
  - Favorable dose intensity observed across doses, including 300 mg daily
- **Clinical profiles from dose escalation, including exposures, support 300 mg daily and below for ongoing dose optimization in both NSCLC and PDAC to inform dose selection for pivotal trials**

RAS(ON) Mutant-Selective Inhibitors

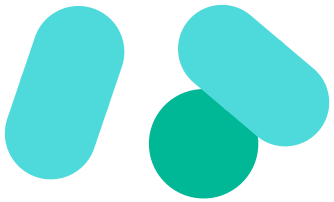
- **Encouraging initial RMC-6291 adverse event and monotherapy antitumor activity profiles in NSCLC and CRC reported in October**
  - Dosing ongoing at 200 mg BID
- **RMC-9805 exhibiting good oral bioavailability, including dose-dependent increases in exposure, consistent with preclinical projections**

Financial

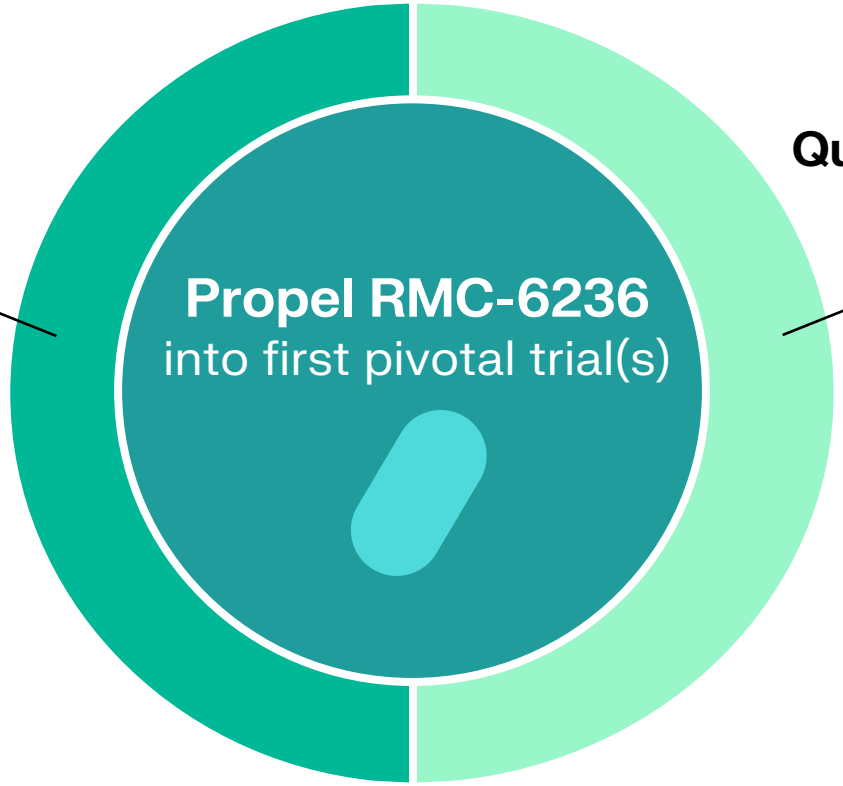
- **EQRx transaction completed, $1.85 billion cash and investment balance as of December 31, 2023**

Broad Clinical Validation Across RAS Genotypes and Tumor Types *in 2023* Driving Late-Stage Development *in 2024*

Expand reach of RMC-6236
by clinically assessing opportunities
(1L, types, mutations)

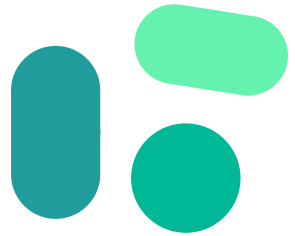


- Mono cohorts
- Combination cohorts



- Dose selection
- Durability of response
- Trial designs

Qualify mutant-selective inhibitors
led by RMC-6291 and RMC-9805 for late-stage development



- Mono profiles and dose selection
- Combination cohorts
- Late-stage plans

Corporate Priorities & Anticipated Milestones

Corporate Priorities	Milestone (Expected Timing)
Begin first RMC-6236 monotherapy pivotal trials	• Disclose updated clinical safety, tolerability and activity data from ongoing Phase 1 study in patients with NSCLC (2H 2024) • Initiate Phase 3 2L NSCLC study (2H 2024) • Disclose updated clinical safety, tolerability and activity data from ongoing Phase 1 study in patients with PDAC (2H 2024) • Initiate Phase 3 2L PDAC study (2H 2024)
Expand reach of RMC-6236	• Disclose initial combination RMC-6236 + pembrolizumab clinical PK, safety, tolerability and activity data (2H 2024) • Disclose initial data from Phase 1 expansion monotherapy cohort for additional tumor types and genotypes (Q2-Q3 2024)
Qualify mutant-selective inhibitors for late-stage development	<u>RMC-6291 | G12C-selective inhibitor</u> • Disclose initial combination RMC-6291 + pembrolizumab clinical PK, safety, tolerability and activity data (2H 2024) • Disclose initial combination RMC-6291 + RMC-6236 clinical PK, safety, tolerability and activity data (2H 2024) <u>RMC-9805 | G12D-selective inhibitor</u> • Disclose initial monotherapy clinical PK, safety, tolerability and activity data from ongoing Phase 1 study (2H 2024)

Deep Pipeline of Targeted Therapies for Majority of RAS-Addicted Cancers

		PRECLINICAL	IND-ENABLING	PHASE 1	PHASE 2	PHASE 3
RAS(ON) INHIBITORS						
RMC-6236	MULTI					
RMC-6291	G12C					
RMC-9805	G12D					
RMC-5127	G12V					
RMC-0708	Q61H					
RMC-8839[1]	G13C					
Pipeline Expansion	G12R, other					
RAS COMPANION INHIBITORS						
RMC-4630[1]	SHP2					
RMC-5552	mTORC1/4EBP1					

(1) Development activities paused.

Financial Information

Financial Position	
Cash, cash equivalents and marketable securities as of December 31, 2023	$1.85 billion[(1)]

2024 Financial Guidance
2024 GAAP Net Loss of $480 million to $520 million[(2)]

(1) With current cash, cash equivalents and marketable securities, the company projects it can fund planned operations into 2027, based on its current operating plan.
(2) Includes non-cash stock-based compensation expense of approximately $70 million to $80 million.

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Appendix

- All RAS cancer epidemiology statistics are estimated using tumor mutation frequencies from Foundation Medicine Insights March 2022 and scaled to estimated patient numbers using cancer incidence from ACS Cancer Facts and Figures 2023:
  - RAS mutations include: KRAS G12(A,C,D,F,L,R,S,V), KRAS G13(C,D,R,V), KRAS Q61(E,H,K,L,P,R) NRAS G12(A,C,D,R,S,V), NRAS G13(C,D,R,V), NRAS Q61(H,K,L,R), HRASG12(C,D,S,V), HRASG13(C,D,N,R,S,V), HRASQ61(K,L,R).
  - Includes 13 major solid cancer types: non-small cell lung cancer, colorectal, pancreatic ductal adenocarcinoma, renal, esophageal, head and neck squamous cell, ovarian, stomach, biliary, and carcinomas of unknown primary (CUP), and advanced melanoma, bladder and endometrial cancers causing mortality.
    - KRASQ61H epidemiology statistics include multiple myeloma in addition to 13 major solid cancer types named above

- RAS mutations drive 30% of human cancers per Prior et al., *Cancer Research* 2020

- Mouse tumor responses assigned according to mRECIST (modified from Gao et al. Nat Med. 2015):
  - mPD = progressive disease; mSD = stable disease; mPR = partial response; mCR = complete response