UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): January 9, 2024

REVOLUTION MEDICINES, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-39219**	**47-2029180**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification Number)**

700 Saginaw Drive
Redwood City, California 94063
(Address of principal executive offices, including Zip Code)

Registrant's telephone number, including area code: (650) 481-6801

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol	Name of each exchange on which registered
Common Stock, $0.0001 par value per share	RVMD	The Nasdaq Stock Market LLC (Nasdaq Global Select Market)

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 2.02 Results of Operations and Financial Condition.

On January 9, 2024, Revolution Medicines, Inc. (the "Company") posted a corporate presentation to the investor section of the Company's website at: ir.revmed.com/events-and-presentations. The Company's corporate presentation is attached hereto as Exhibit 99.1.

The information furnished under this Item 2.02 and in the presentation attached as Exhibit 99.1 to this Current Report on Form 8-K shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section or Sections 11 or 12(a)(2) of the Securities Act of 1933, as amended (the "Securities Act"). The information contained in this Item 2.02 and in the presentation attached as Exhibit 99.1 to this Current Report on Form 8-K shall not be incorporated by reference into any filing with the Securities and Exchange Commission (the "SEC") made by the Company, whether made before or after the date hereof, regardless of any general incorporation language in such filing.

Item 7.01 Regulation FD Disclosure.

On January 9, 2024, the Company posted a corporate presentation to the investor section of the Company's website at: ir.revmed.com/events-and-presentations. The Company's corporate presentation is attached hereto as Exhibit 99.1.

The furnishing of the attached presentation is not an admission as to the materiality of any information therein. The information contained in the slides is summary information that is intended to be considered in the context of more complete information included in the Company's filings with the SEC and other public announcements that the Company has made and may make from time to time by press release or otherwise. The Company undertakes no duty or obligation to update or revise the information contained in this Current Report on Form 8-K, although it may do so from time to time as its management believes is appropriate. Any such updating may be made through the filing of other reports or documents with the SEC, through press releases or through other public disclosures. For important information about forward looking statements, see the slide titled "Legal Disclaimer" in Exhibit 99.1 attached hereto.

The information furnished under this Item 7.01 and in the presentation attached as Exhibit 99.1 to this Current Report on Form 8-K shall not be deemed "filed" for purposes of Section 18 of the Exchange Act or otherwise subject to the liabilities of that section or Sections 11 or 12(a)(2) of the Securities Act. The information contained in this Item 7.01 and in the presentation attached as Exhibit 99.1 to this Current Report on Form 8-K shall not be incorporated by reference into any filing with the SEC made by the Company, whether made before or after the date hereof, regardless of any general incorporation language in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Company presentation dated January 9, 2024.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

REVOLUTION MEDICINES, INC.

Date: January 9, 2024

By: /s/ Jack Anders
Jack Anders
Chief Financial Officer

Exhibit 99.1

Revolution Medicines

On Target to Outsmart Cancer

January 9, 2024

Legal Disclaimer

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, prospective products, availability of funding, ability to manage existing collaborations and establish new strategic collaborations, licensing or other arrangements, the scope, progress, results and costs of developing our product candidates or any other future product candidates, conducting clinical trials, the potential market size and size of the potential patient populations for our product candidates, the timing and likelihood of success of obtaining product approvals, plans and objectives of management for future operations, the scope of protection we are able to establish and maintain for intellectual property rights covering our product candidates, future results of anticipated products the impact of global events and other macroeconomic conditions on our business, and the expected benefits of the transaction with EQRx, Inc. are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. The information included in these materials is provided as of January 9, 2024 unless specified elsewhere herein, and is qualified as such. Except as required by applicable law, we undertake no obligation to update any forward-looking statements or other information contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

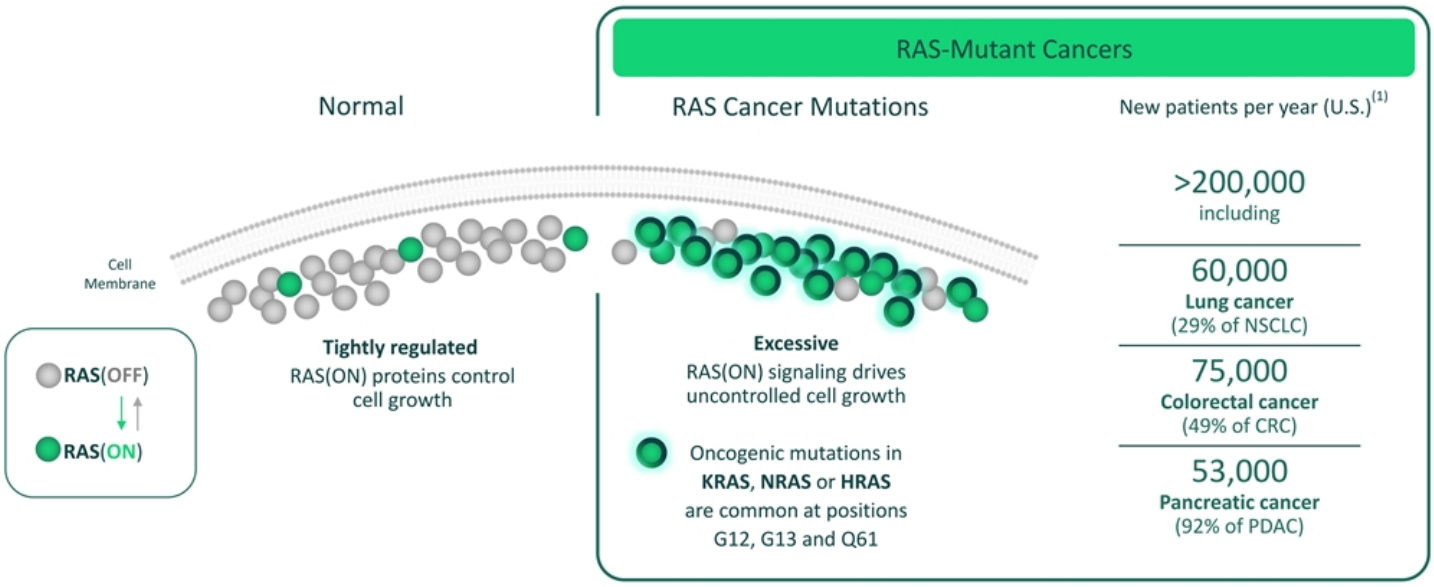
For a further description of the risks and uncertainties that could cause actual results to differ from those anticipated in these forward-looking statements, as well as risks relating to the business of Revolution Medicines in general, see Revolution Medicines' Quarterly Report on Form 10-Q filed with the Securities and Exchange Commission on November 6, 2023, 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.

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



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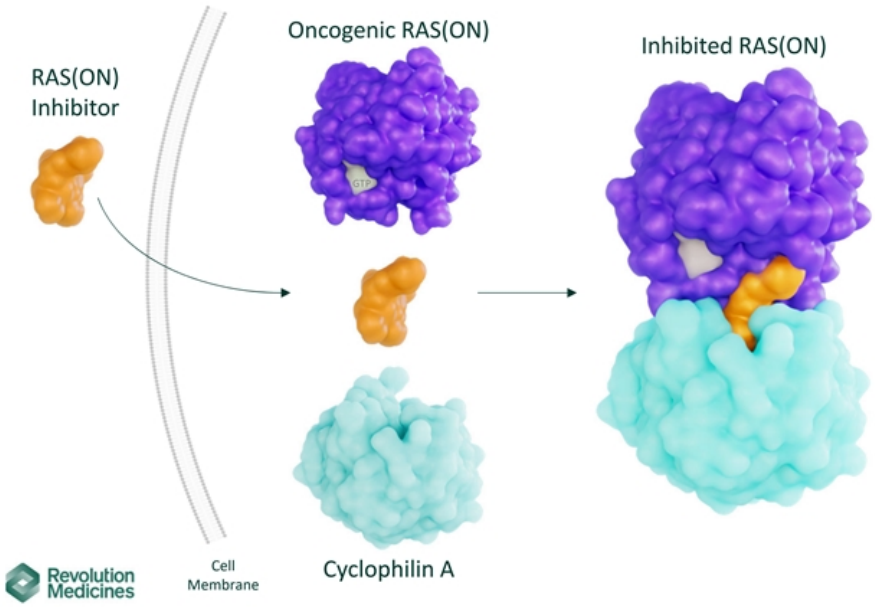
(1) Estimated using tumor mutation frequencies from Foundation Medicine Insights March 2022 and scaled to estimated patient numbers using cancer incidence from ACS Cancer Facts and Figures 2023 (see appendix for additional detail); NSCLC = non-small cell lung cancer; CRC = colorectal cancer; PDAC = pancreatic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

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



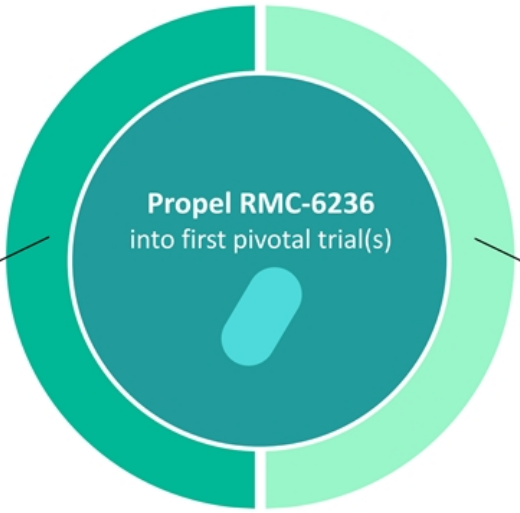
- **Direct inhibition of RAS(ON) cancer drivers**
- **Deep and durable suppression of RAS cancer signaling designed to defy common drug resistance mechanisms**
- **Clinical validation of first two RAS(ON) Inhibitors studied as single agents**

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 begun 3Q23	**G12D**

(1) 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

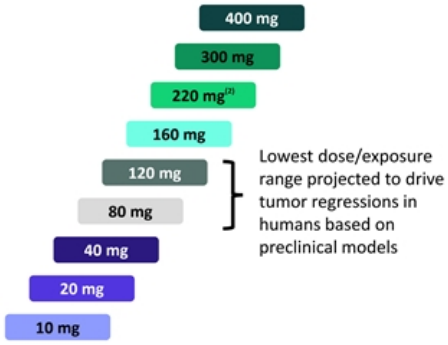
- Advanced solid tumors with KRAS G12X mutations[1] (initially excluding KRAS G12C)
- Received prior standard therapy appropriate for tumor type and stage
- ECOG PS 0–1
- No active brain metastases

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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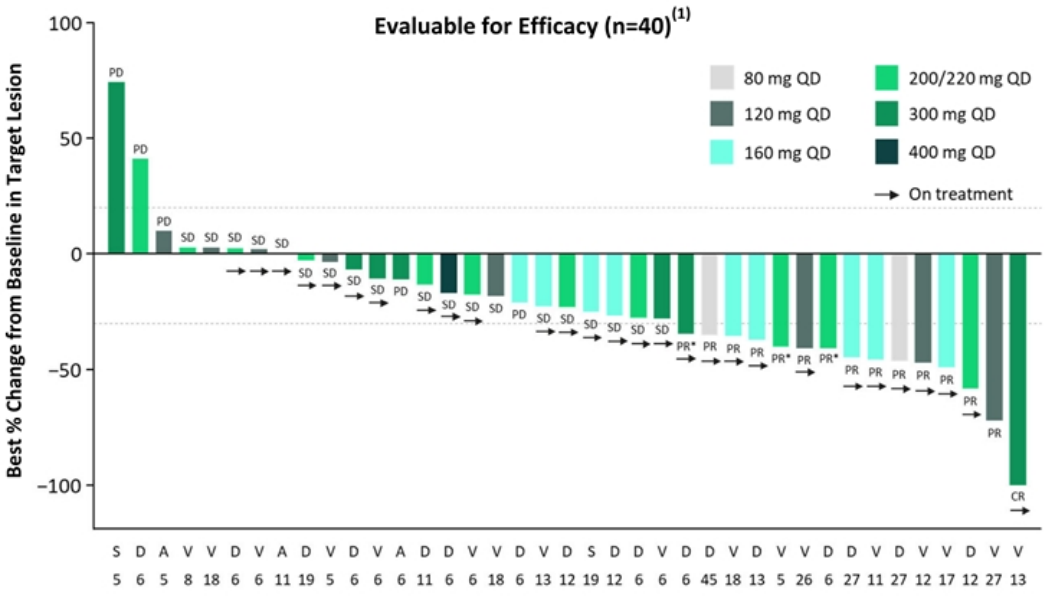
*Includes preferred terms of dermatitis acneiform, rash maculopapular, rash, rash pustular, dermatitis psoriasiform, erythema, rash erythematous; multiple types of rash may have occurred in the same patient; †The most common TRAE leading to dose reduction was rash (acneiform or maculopapular); there 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



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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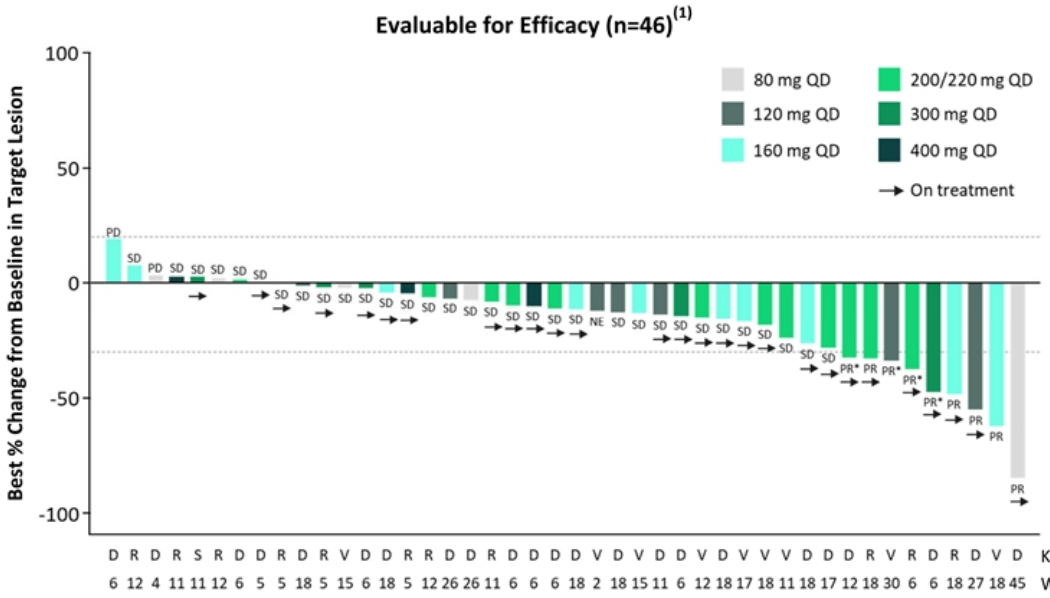
Data Extracted 12 Oct 2023.

KRAS G12X PDAC: Best Overall Response to RMC-6236



KRAS G12 Mutation

Week of Most Recent Scan

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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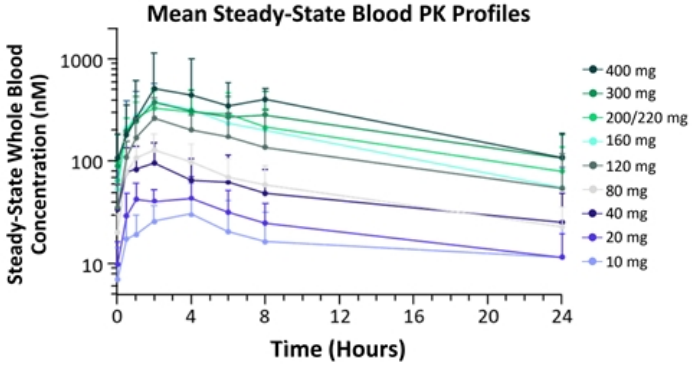
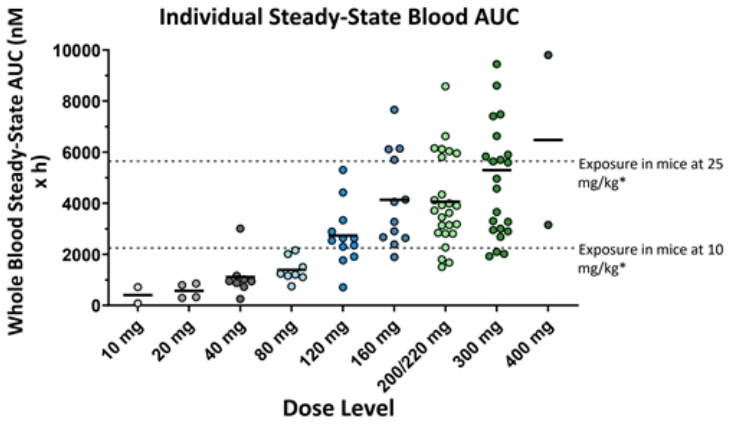
Data Extracted 12 Oct 2023.

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

Cohort	Patients Enrolled	Purpose
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

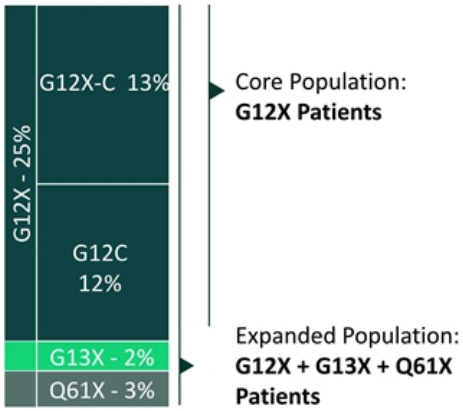
- RMC-6236
- Docetaxel

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]

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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);

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

Trial Design[1]

R

RMC-6236

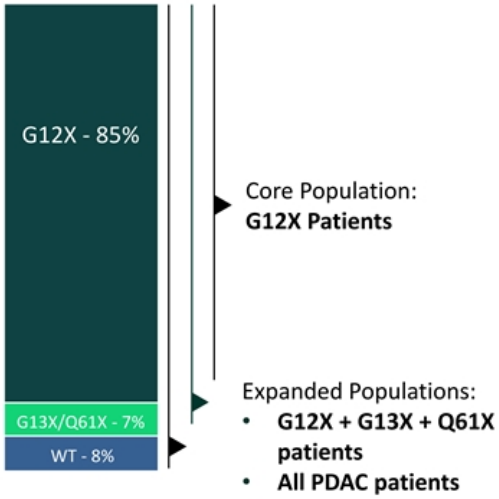
Physician's choice:
e.g., GnP or mFOLFIRINOX

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]



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

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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

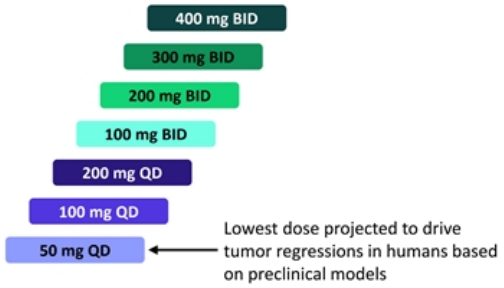
- Advanced solid tumors with $KRAS^{G12C}$ mutations
- Received prior standard therapy including treatment with $KRAS^{G12C}$(OFF) inhibitors
- ECOG PS 0–1
- No active brain metastases

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

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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

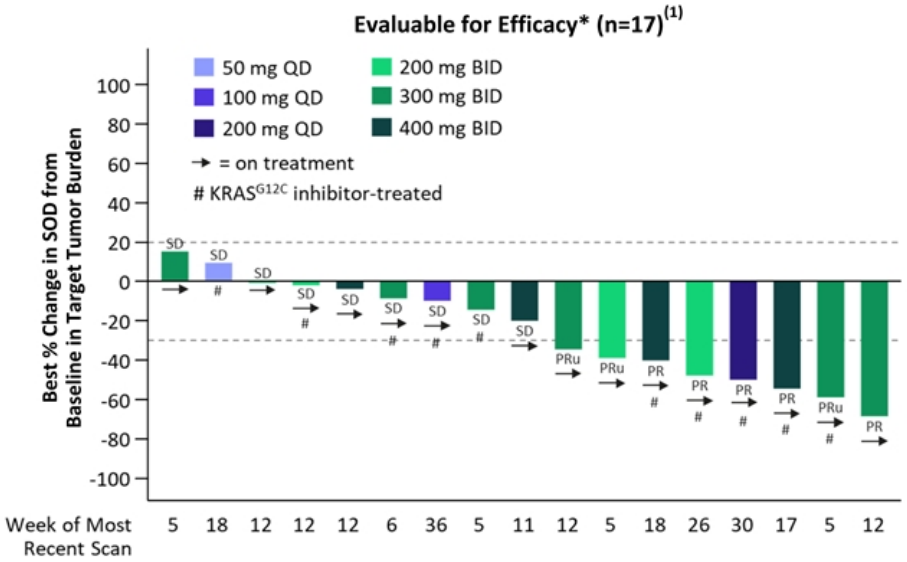
	Total (n=63)			
Maximum Severity of TRAEs	**Grade 1**	**Grade 2**	**Grade 3**	**Any Grade**
TRAEs occurring in ≥10% of patients, n (%)				
Diarrhea	10 (16)	7 (11)	1 (2)	18 (29)
Nausea	14 (22)	3 (5)	0	17 (27)
ECG QT prolonged	8 (13)	1 (2)	7 (11)	16 (25)
QTcF* ≥501 ms	–	–	1 (2)	–
Fatigue	4 (6)	4 (6)	0	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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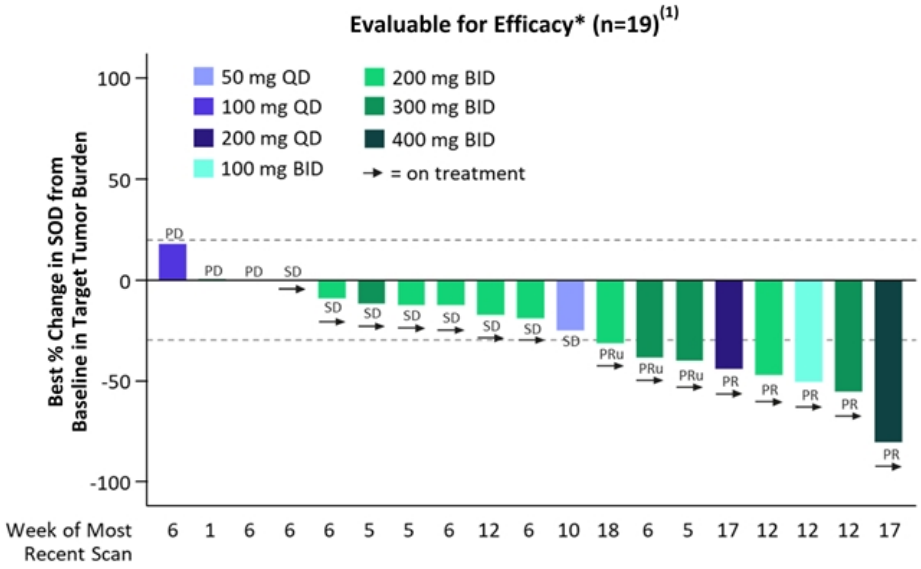
Data Extracted 05 October 2023.

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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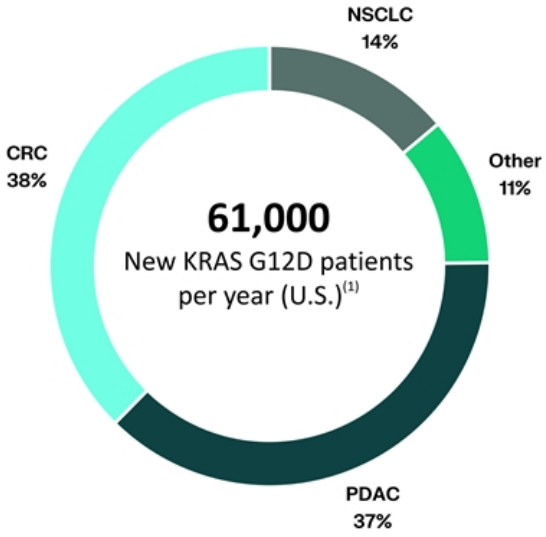
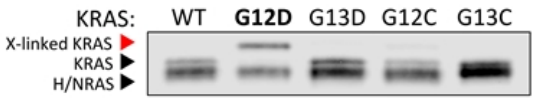
Data Extracted 05 October 2023.

RAS(ON) G12D-Selective Inhibitor
RMC-9805

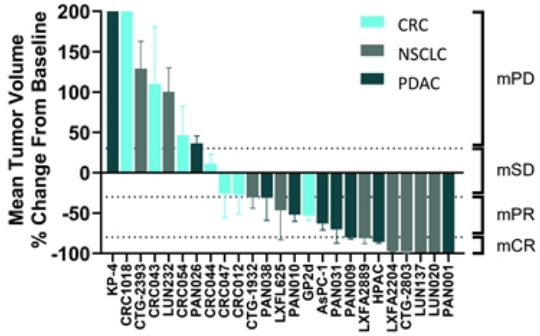
RMC-9805: Clinical Stage, RAS(ON) Mutant-Selective, Covalent Inhibitor for RAS G12D Cancers



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

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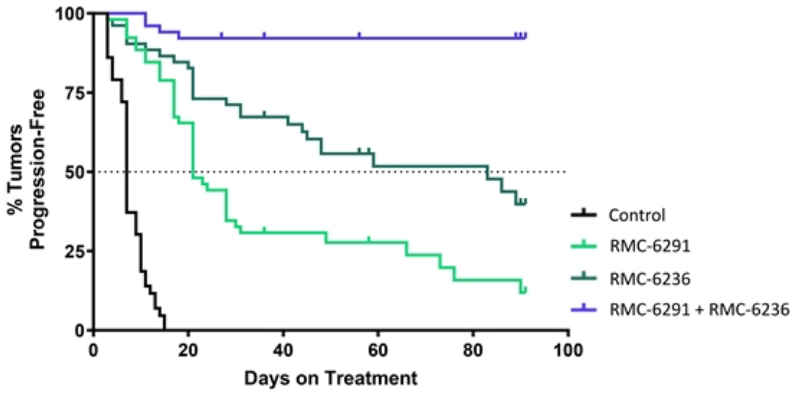
Phase 1b Combo: RMC-6236 + RMC-6291 Doublet Designed to Overcome Resistance and Prolong Durability in KRAS G12C NSCLC

Preclinical Validation[1]



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

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

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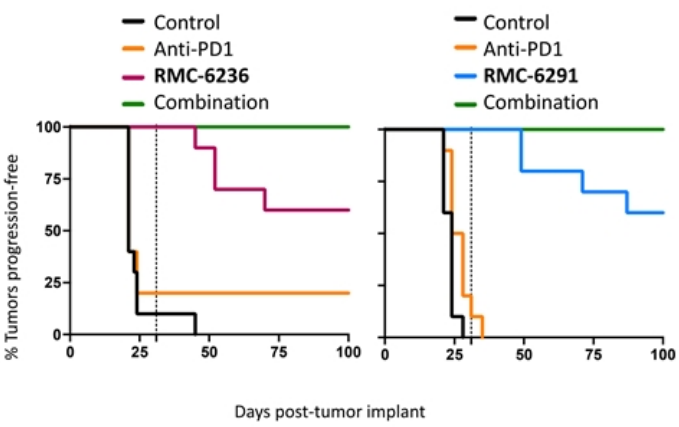
(1) RVMD preclinical research; NSCLC = non-small cell lung cancer; RMC-6236 dosed at 25 mg/kg po qd (n=52); RMC-6291 dosed at 100 or 200 mg/kg po qd (n=52); Combination (n=51). For each group, n = total number of animals from the seven models that comprise the dataset. Progression defined as tumor doubling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

Cohort	Status	Purpose
NSCLC[1]		
RMC-6236 + pembrolizumab +/- chemotherapy	recruiting	qualification for potential 1L
RMC-6291 + pembrolizumab +/- chemotherapy	recruiting	qualification for potential 1L
Solid tumors[2]		
RMC-6236 + RMC-6291	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

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(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**
  - Dose optimization underway at 200-300 mg daily
- **RMC-9805 exhibiting good oral bioavailability, including dose-dependent increases in exposure, consistent with preclinical projections**

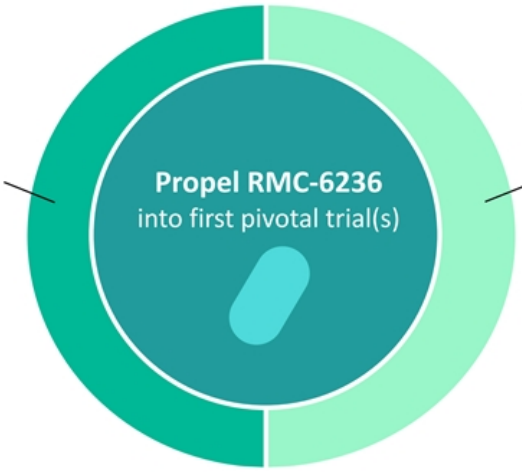
Financial

- **EQRx transaction completed, 2023 EOY estimated "flash" cash and marketable securities balance $1.85 billion (unaudited)**

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

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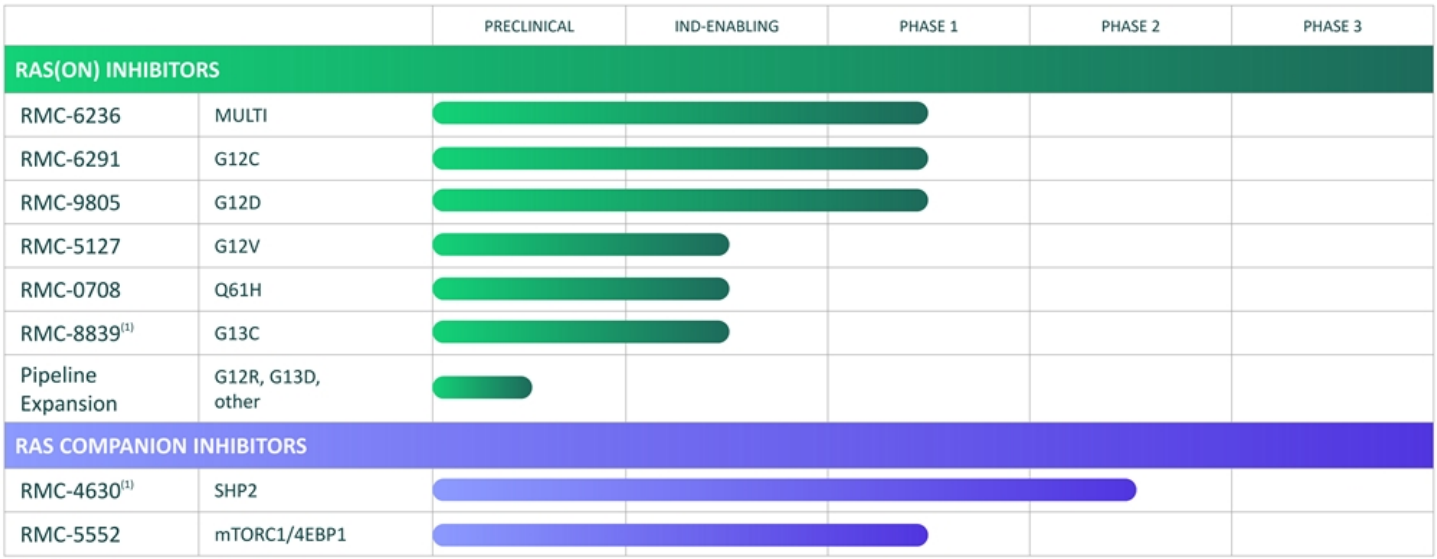
Deep Pipeline of Targeted Therapies for Majority of RAS-Addicted Cancers



(1) Development activities paused.

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Financial Information

Financial Position	
Preliminary estimate of cash, cash equivalents and marketable securities as of December 31, 2023 (unaudited)	$1.85 billion[1,2]

2023 Financial Guidance
2023 net loss of $385 million to $415 million[3]

(1) Includes cash, cash equivalents and marketable securities acquired in the EQRx transaction. We have not yet completed our year-end financial close and review processes, and the results of our ongoing review of our financial statements could result in changes to this amount.
(2) With current cash, cash equivalents and marketable securities, the company projects it can fund planned operations into 2027.
(3) Includes non-cash stock-based compensation expense of approximately $45 million to $50 million. 2023 net loss guidance does not include impact of the acquisition of EQRx. The company has not provided net loss guidance that includes the impact of the acquisition of EQRx because the company is not able to quantify the impact of this acquisition on the company's net loss without unreasonable effort.

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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