Filed by Revolution Medicines, Inc.

Pursuant to Rule 425 under the Securities Act of 1933
and deemed filed pursuant to 14a-12 under the Securities Exchange Act of 1934
Subject Company: EQRx, Inc.
Commission File No.: 001-40312
Date: August 1, 2023

This filing relates to the proposed transaction between Revolution Medicines, Inc. a Delaware corporation ("Revolution Medicines"), and EQRx, Inc., a Delaware corporation ("EQRx"), pursuant to the terms of that certain Agreement and Plan of Merger, dated as of July 31, 2023 (the "Merger Agreement"), by and among Revolution Medicines, EQRx, Equinox Merger Sub I, Inc., a Delaware corporation and a wholly owned subsidiary of Revolution Medicines ("Merger Sub I"), and Equinox Merger Sub II LLC, a Delaware limited liability company and a wholly owned subsidiary of Revolution Medicines ("Merger Sub II" and, together with Merger Sub I, the "Merger Subs" and each a "Merger Sub").

On August 1, 2023, Revolution Medicines made available the following presentation:

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 the expected timing of closing of the proposed transaction with EQRx, Inc. (EQRx), the expected benefits of the proposed transaction, 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, 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, and the impact of the COVID-19 pandemic on our business 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 May 8, 2023, 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 May 8, 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.

Summary of Transaction and Business Updates Announced August 1, 2023

- RVMD to acquire EQRx in an all-stock transaction to gain more than $1B in additional capital
- Strengthened balance sheet intended to support RVMD's parallel, late-stage development for RAS(ON) Inhibitor pipeline, which includes RMC-6236, RMC-6291and RMC-9805
  - RMC-6236 clinical activity data to be presented at ESMO 2023* on October 22
  - RMC-6236 supporting clinical data to be presented at 2023 Triple Meeting^ in October
  - RMC-6291 initial clinical findings to be presented at 2023 Triple Meeting in October
- Deal expected to close in November 2023, subject to satisfaction of customary closing conditions
- Stock exchange ratio to be determined using a blended average share price to account for developments in our business and potential movement in RVMD share price
  - ~20% based on a determined RVMD share price at signing
  - ~80% based on RVMD share price as determined in close proximity to shareholder vote (subject to 6% discount)
- RVMD to continue focus on mission to discover, develop and deliver pioneering RAS(ON) Inhibitor drugs on behalf of patients with RAS-addicted cancers

* European Society for Medical Oncology Congress (ESMO); Presentation No. 6520
^ AACR-NCI-EORTC International conference on Molecular Targets and Cancer Therapeutics ("Triple Meeting")

On Target to Outsmart Cancer

HIGH UNMET NEED IN RAS-ADDICTED CANCERS

30% of human cancers[1], largely unserved by targeted therapeutics

STRONG CLINICAL VALIDATION OF RAS^{MUTANT} AS CANCER DRIVER

Proof-of-principle from first-gen $KRAS^{G12C}$ inhibitors[2]

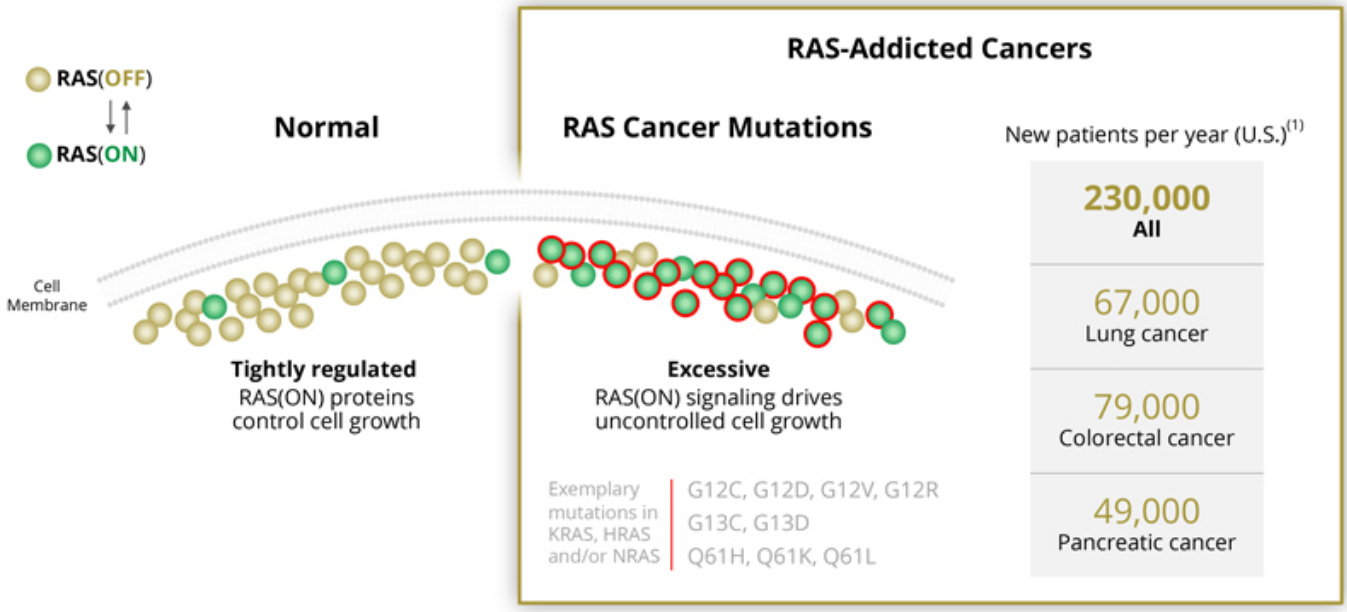
DEEP, SCIENCE-DRIVEN CLINICAL AND PRECLINICAL PIPELINE

RAS(ON) Inhibitors
Groundbreaking class of drug candidates designed for robust cancer suppressior

RAS Companion Inhibitors
Class-leading drug candidates designed to counter treatment resistance

(1) Prior et al., *Cancer Research* 2020
(2) Lumakras approved by the FDA in May 2021, Krazati approved by the FDA in December 2022

Excessive RAS(ON) Signaling Drives 30% of Human Cancers, Targeted by Our Pipeline Strategy

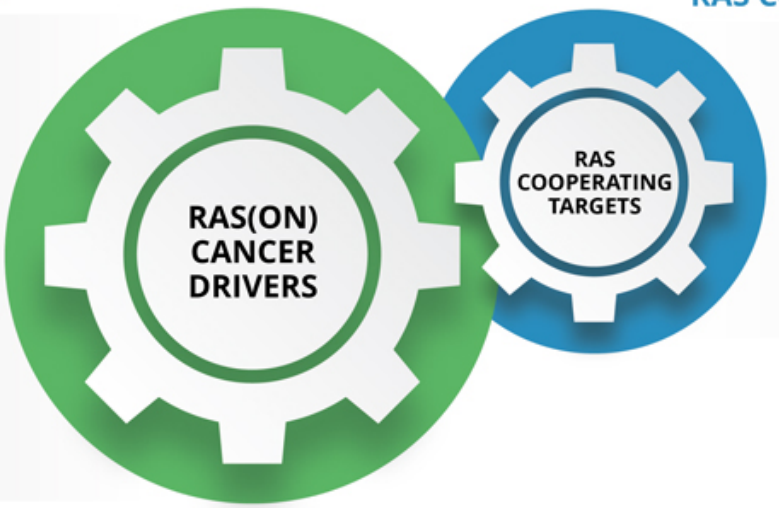


(1) Estimated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020 (see appendix for additional detail); lung cancer = non-small cell lung cancer

Deep, Science-Driven Clinical and Preclinical Pipeline of Targeted Therapies for RAS-Addicted Cancers

RAS(ON) Inhibitors

- 3 Clinical-stage Drug Candidates
- 2 Development-stage Drug Candidates
- \+ Multiple pipeline expansion programs

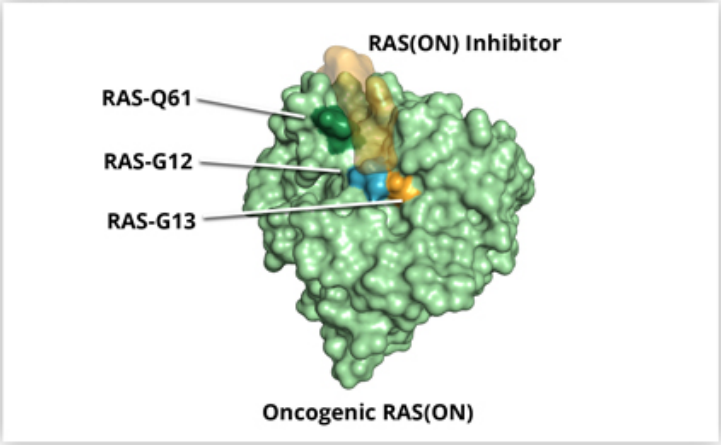


RAS Companion Inhibitors

- 2 Clinical-stage Drug Candidates
- 1 IND-ready Drug Candidate

Groundbreaking RAS(ON) Inhibitors Bind Near RAS Cancer Mutation Hotspots and Suppress Cancer Signaling

Bind



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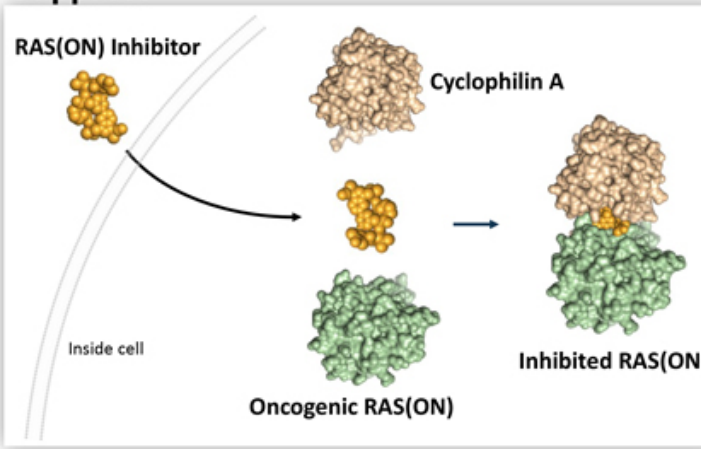


- **Potent, selective, oral and drug-like inhibitors**
- **Deep and sustained suppression of RAS(ON) cancer signaling**

Current Portfolio of RAS(ON) Inhibitors Targets Every RAS Cancer Mutation Hotspot[1]

Relative Frequency in Human RAS Cancers[2]

RMC-6236 MULTI/G12X

RMC-6291 G12C

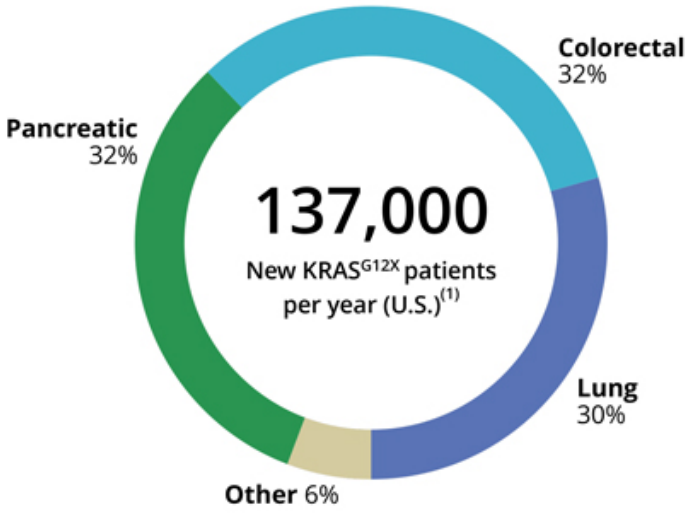
RMC-9805 G12D

RMC-0708 Q61H

RMC-8839 G13C

(1) RAS cancer mutation hotspots defined as G12, G13 and Q61
(2) Calculated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020 (see appendix for additional detail)

RMC-6236: First-in-Class RAS^{MULTI}(ON) Inhibitor with Broad Potential Against RAS-Addicted Cancers



KRAS^{G12X} includes KRAS^{G12D}, KRAS^{G12V}, KRAS^{G12R} and KRAS^{G12C}

Preclinical Profile

Highly Potent and Selective RAS(ON) Inhibitor

- Suppresses diverse mutant RAS cancer drivers and cooperating wild-type RAS proteins

Robust Anti-tumor Activity in Cancer Models

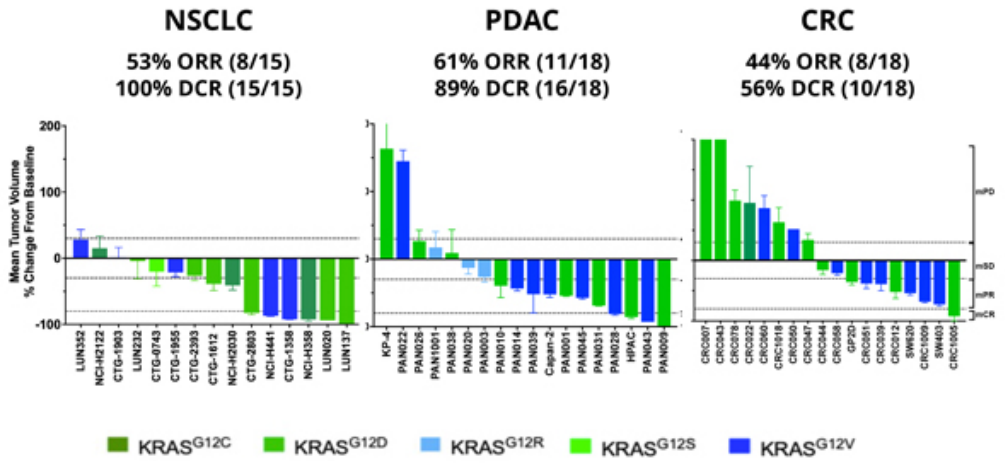
- Deep and sustained inhibition drives durable anti-tumor activity in tumors with common RAS variants

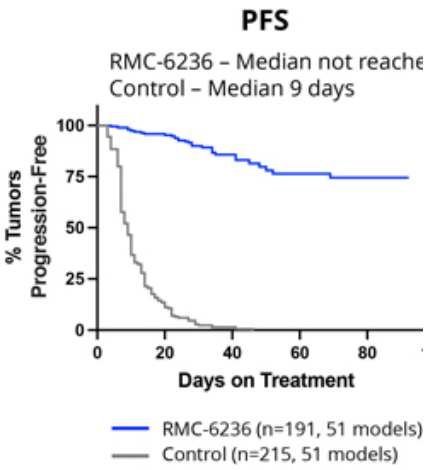
Attractive PK/ADME Profile

- Favorable *in vivo* oral bioavailability, clearance and concentration in tumors for effective target coverage in RAS-addicted cancer cells

(1) Calculated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020 (see appendix for additional detail); lung cancer = non-small cell lung cancer

RMC-6236: Highly Active with Durable Benefit Across *in Vivo* Models of Major Human Cancers with KRAS^{G12X} Drivers





RVMD preclinical research as of 06/01/22
RMC-6236 dosed at 25 mg/kg po qd; n=1-10/group
Progression defined as tumor doubling from baseline
NSCLC = non-small cell lung cancer; PDAC = pancreatic ductal adenocarcinoma; CRC = colorectal cancer
Responses assigned according to mRECIST (see appendix)
ORR = objective response rate; DCR = disease control rate; PFS = progression-free survival

RMC-6236 Phase 1/1b Trial: Clinical Translation of Preclinical Single Agent Profile and Initial Platform Validation

	Preclinical Profile	Aims of Phase 1/1b Clinical Trial[2]
Dosing and Safety	✓ **Oral dosing** (daily and intermittent): drug levels that drive sustained RAS pathway suppression ✓ **Safety**: well-tolerated in active range, dose-limiting toxicities "on target" and reversible ✓ **Long-term treatment**[1] at active doses	○ **Oral dosing**: complete dose optimization and determine a dose for initiation of pivotal trials ○ **Safety**: characterize the safety and tolerability profile of RMC-6236
Anti-Tumor Activity	✓ **Tumor selection**: active in diverse RAS^{MUTANT} NSCLC, pancreatic and CRC models; $KRAS^{G12X}$ most sensitive ✓ **Activity**: deep regressions across $KRAS^{G12X}$ tumors, especially NSCLC and pancreatic models	○ **Patient selection**: signal-seeking across diverse $KRAS^{G12X}$ tumors ○ **Efficacy**: characterize the ORR, and durability of response and SD, of RMC-6236 in patients with $KRAS^{G12X}$ tumors

(1) Long-term in mouse models defined as up to 90 days of treatment
(2) Ongoing study, RMC-6236-001 - ClinicalTrials.gov Identifier: NCT05379985 https://clinicaltrials.gov/ct2/show/NCT05379985?term=RMC-6236&draw=2&rank=1
$KRAS^{G12X}$ includes $KRAS^{G12A}$, $KRAS^{G12D}$, $KRAS^{G12R}$, $KRAS^{G12S}$, $KRAS^{G12V}$
ORR = overall response rate; SD = stable disease

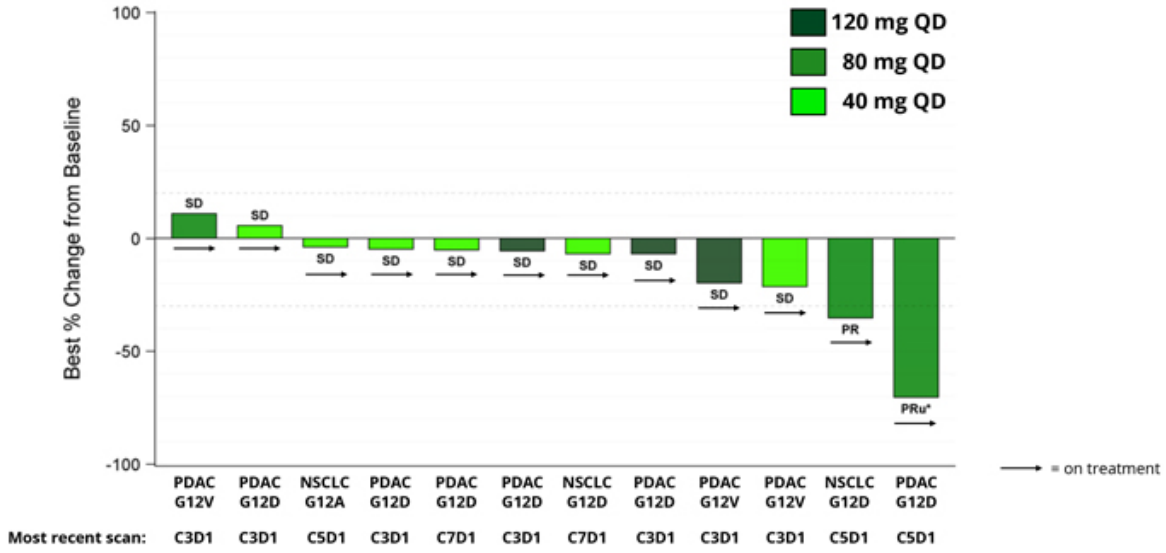
RMC-6236-001: Treatment-Related AEs Occurring in ≥ 10% of All Patients

	10 mg QD (N=3)		20 mg QD (N=13)		40 mg QD (N=9)		80 mg QD (N=7)		120 mg QD (N=4)		Overall (N=36)	
Preferred Term	**Any Grade**	**Grade ≥3**	**Any Grade**	**Grade ≥3**	**Any Grade**	**Grade ≥3**	**Any Grade**	**Grade ≥3**	**Any Grade**	**Grade ≥3**	**Any Grade**	**Grade ≥3**
Rash (CMQ)*	0	0	2 (15.4%)	0	4 (44.4%)	0	6 (85.7%)	0	4 (100%)	0	16 (44.4%)	0
Nausea	1 (33.3%)	0	2 (15.4%)	0	6 (66.7%)	0	2 (28.6%)	0	1 (25.0%)	0	12 (33.3%)	0
Diarrhoea	0	0	1 (7.7%)	0	2 (22.2%)	0	1 (14.3%)	0	2 (50.0%)	0	6 (16.7%)	0
Fatigue	0	0	0	0	2 (22.2%)	0	0	0	2 (50.0%)	0	4 (11.1%)	0
Vomiting	0	0	1 (7.7%)	0	2 (22.2%)	0	0	0	1 (25.0%)	0	4 (11.1%)	0

One related grade 4 adverse event of bowel perforation (also considered a serious adverse event) was reported in a patient receiving 80 mg daily.
The likely cause of the perforation was considered to be shrinkage of metastatic $KRAS^{G12V}$ pancreatic cancer at the site of full-thickness bowel infiltration.

EDC data as of 02/17/2023
CMQ = Customized MedDRA Query
*Consists of dermatitis acneiform, dermatitis psoriasiform, palmar-plantar erythrodysesthesia syndrome, rash maculo-papular, and rash pustular.

RMC-6236-001: Change in Tumor Burden from Patients with KRAS^{G12X} NSCLC or Pancreatic Cancer Treated at ≥ 40 mg Daily



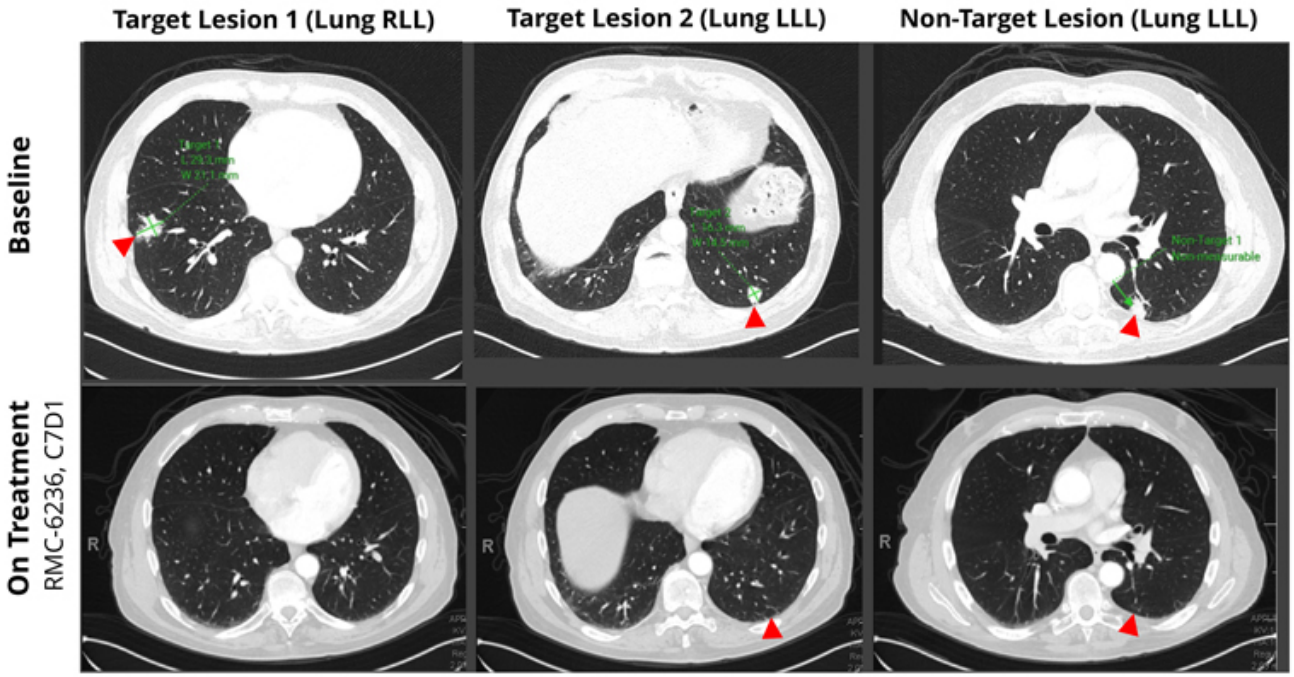
EDC data as of 02/17/2023; efficacy evaluable patients defined as those in this data set with at least one post baseline response assessment or who have died or have experienced clinical progression prior to the first post baseline scan (n=12). Cycle time is 21 days. SD = stable disease, PR = partial response. NSCLC = non-small cell lung cancer; PDAC = pancreatic ductal adenocarcinoma.
*PR subsequently confirmed as of 03/16/23.

RMC-6236-001 Case Report: $KRAS^{G12D}$ Pancreatic Cancer Patient

- 76 year-old male
- $KRAS^{G12D}$ pancreatic cancer diagnosed November 2017
- Treated with neoadjuvant chemotherapy, surgery, adjuvant chemotherapy 2018
- Progressed with metastatic disease January 2022; treated with gemcitabine + nab-paclitaxel + investigational therapy with SD as best response

- November 2022 lung metastases
- $KRAS^{G12D}$ with co-occurring loss of P53, CDKN2A, CDKN2B and MTAP
- Treated with RMC-6236 80 mg daily

Updated RMC-6236-001 Case Report: Confirmed PR for KRAS^{G12D} Pancreatic Cancer Patient



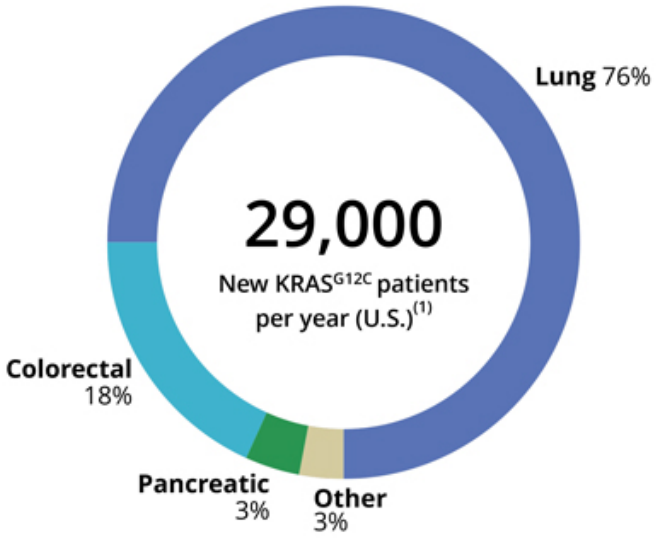
RLL=right lower lobe; LLL=left lower lobe
SLD = sum of longest diameters per RECIST 1.1

Images courtesy of RMC-6236-001 study site with additional annotation by RVMD
(SLD values and red arrows highlighting detectable lesions)

Planning is Underway for Next Steps with RMC-6236

	Clinical Development
Single Agent	○ **Current RMC-6236-001 trial:** dose optimization for pivotal trials is ongoing ○ **One or more pivotal trials:** late-stage evaluation of treatment with RMC-6236 in patients with $KRAS^{G12X}$–bearing tumors potentially to begin in 2024
Combination	○ **Phase 1/2 clinical trial:** evaluation of combination treatment with RMC-6291 and RMC-6236 in patients with $KRAS^{G12C}$–bearing tumors potentially to begin in early 2024

RMC-6291: Mutant-Selective RAS(ON) Inhibitor with Best-in-Class Potential for KRAS^{G12C} Cancers



Preclinical Profile

Highly Potent and Selective RAS(ON) Inhibitor

- Highly active against KRAS^{G12C}
- Covalent for irreversible inhibition
- Low off-target risk and acceptable safety profile

Robust Anti-tumor Activity in Cancer Models

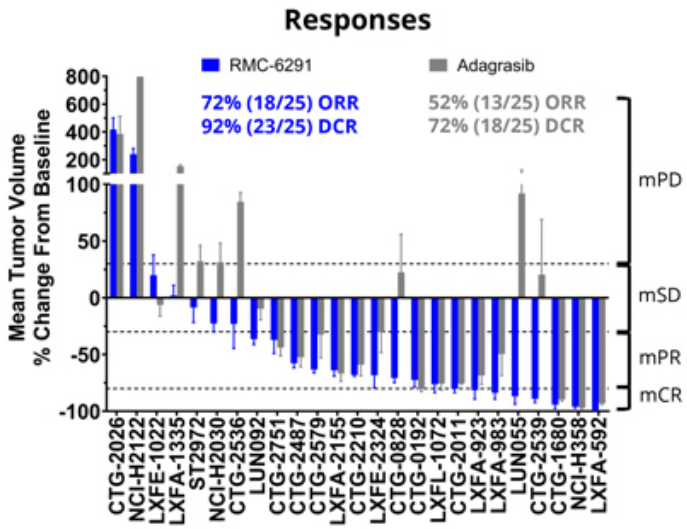
- Rapid, deep and sustained inhibition drives durable anti-tumor effects across multiple KRAS^{G12C} tumor types, with complete responses in some models

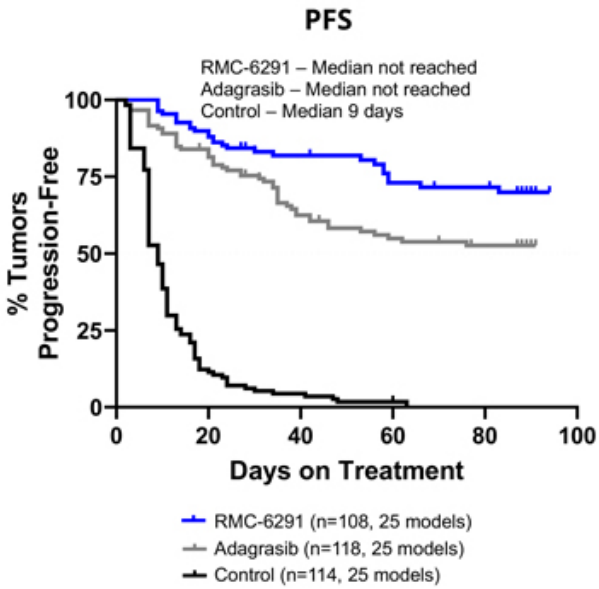
Attractive PK/ADME Profile

- Favorable *in vivo* oral bioavailability and clearance for effective target coverage in KRAS^{G12C}-addicted cancer cells

(1) Calculated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020 (see appendix for additional detail); lung cancer = non-small cell lung cancer

RMC-6291: Superior Response Rates and Durability in Mouse Clinical Trial with 25 KRAS^{G12C} NSCLC Models





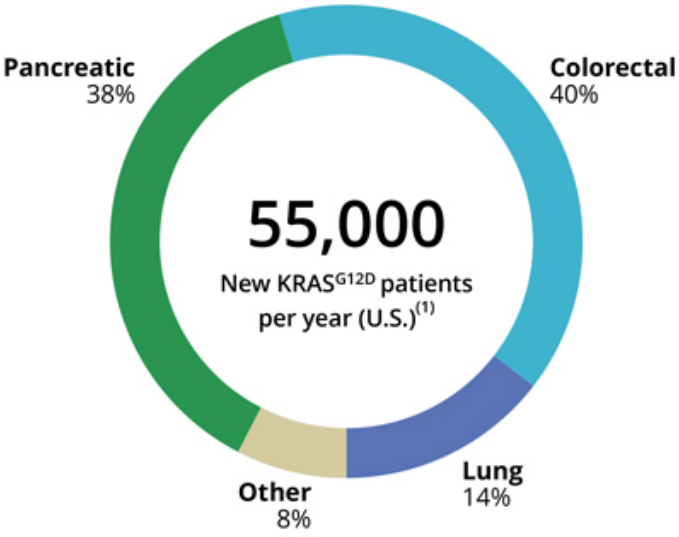
RVMD preclinical research as of 11/20/22
Adagrasib dosed at 100 mg/kg po qd; RMC-6291 dosed at 200 mg/kg po qd; n=3 to 10/group
Progression defined as tumor doubling from baseline
p<0.001 by Log-rank test (RMC-6291 vs adagrasib treatment in the KM analysis)
NSCLC = Non-small cell lung cancer
Responses assigned according to mRECIST (see appendix)

RMC-6291 Phase 1/1b Trial: Clinical Translation of Preclinical Single Agent Profile and Initial Platform Validation

	Preclinical Profile	Aims of Phase 1/1b Clinical Trial[2]
Dosing and Safety	✓ **Oral dosing** (daily): drug levels that drive maximal target crosslinking and sustained RAS pathway suppression ✓ **Safety**: well-tolerated in active range, highly selective for KRAS^{G12C} ✓ **Long-term treatment**[1] at active doses	○ **Oral dosing**: once daily or BID to reach active exposures; surrogate markers of activity (ctDNA ○ **Safety**: short- and long-term safety and tolerability at active exposures ○ **Dose**: complete dose optimization and determine a dose for initiation of pivotal studies
Anti-Tumor Activity	✓ **Tumor selection**: active in KRAS^{G12C} NSCLC and CRC tumor models, including some resistant to KRAS^{G12C}(OFF) inhibitors ✓ **Activity**: deep and durable regressions across KRAS^{G12C} tumors, especially NSCLC	○ **Patient selection**: KRAS^{G12C} solid tumors; KRAS^{G12C}(OFF) inhibitor-treated patients included in dose escalation ○ **Efficacy**: initial clinical responses by RECIST; formal proof-of-concept via expansion cohorts focused on NSCLC patients and potentially other tumor types, including CRC

(1) Long-term in mouse models defined as up to 90 days of treatment
(2) Ongoing study, RMC-6291-001 - ClinicalTrials.gov Identifier: NCT05462717 https://www.clinicaltrials.gov/ct2/show/NCT05462717?term=RMC-6291&draw=2&rank=1
ctDNA = circulating tumor DNA

RMC-9805: First-in-Class Mutant-Selective RAS(ON) Inhibitor for KRAS^{G12D} Cancers



Preclinical Profile

Highly Potent and Selective RAS(ON) Inhibitor

- Highly active against KRAS^{G12D}
- Covalent for irreversible inhibition
- Low off-target risk and acceptable safety profile

Robust Anti-tumor Activity in Cancer Models

- Rapid, deep and sustained inhibition drives durable regressions in KRAS^{G12D} lung, pancreatic and colorectal cancers

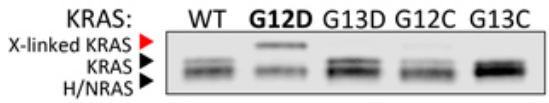
Attractive PK/ADME Profile

- Favorable *in vivo* oral bioavailability and clearance for effective target coverage in KRAS^{G12D}-addicted cancer cells

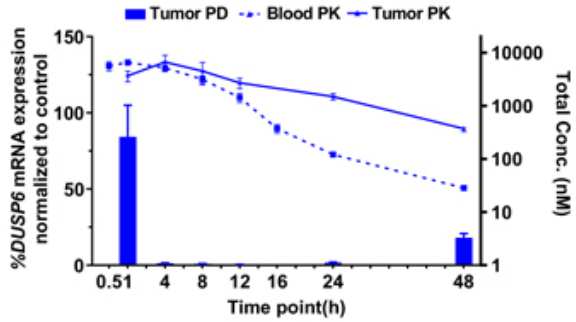
(1) Calculated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020 (see appendix for additional detail); lung cancer = non-small cell lung cancer

RMC-9805: Selective, Covalent Binding and Inhibition of KRAS^{G12D}(ON) with Apoptosis Induction *in Vivo*

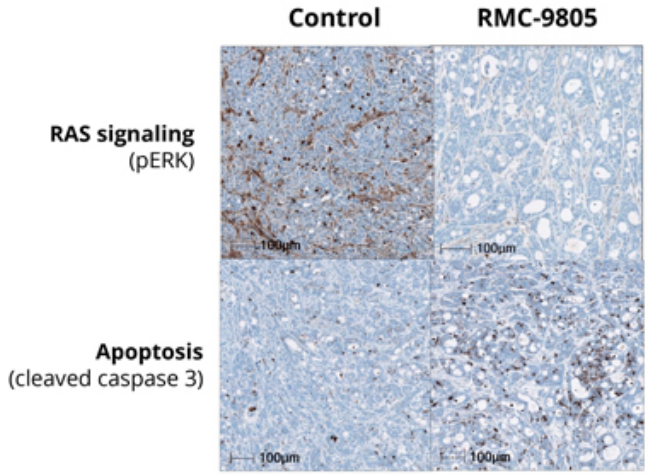
Selective Covalent Binding to KRAS^{G12D}(ON)



Drug Exposure and Pathway Suppression after Oral Dosing[1]

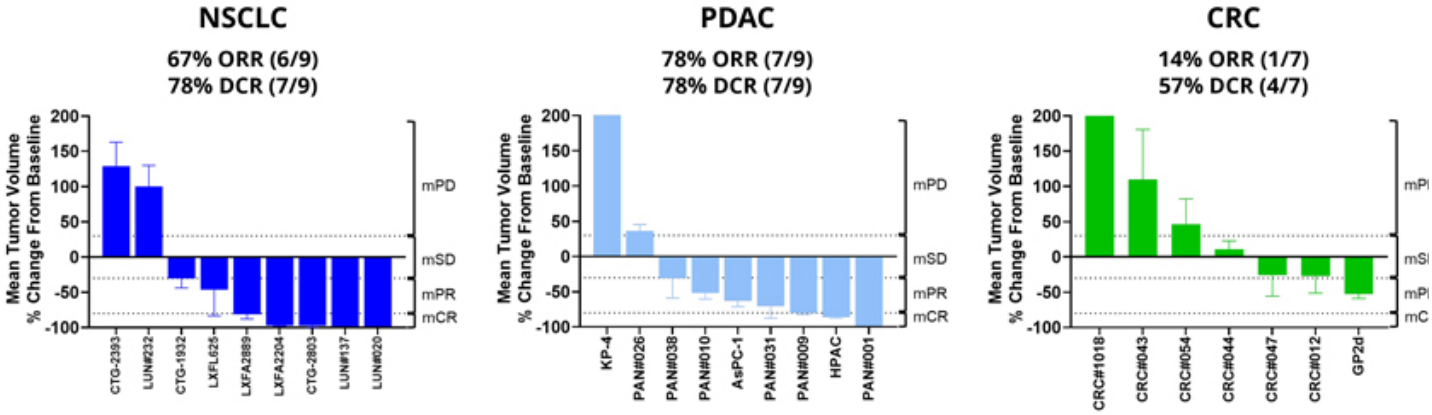


RAS Signaling Inhibition and Apoptosis Induction[2]



RVMD preclinical research
RMC-9805 dosed at 100 mg/kg po in HPAC subcutaneous xenograft model (PDAC, KRAS$^{G12D/WT}$)
(1) PK/PD data collected at indicated timepoints after a single dose
(2) Histopathology data collected 24h after a single dose

RMC-9805: Highly Active *in Vivo* Across Diverse KRAS^{G12D} Cancer Models



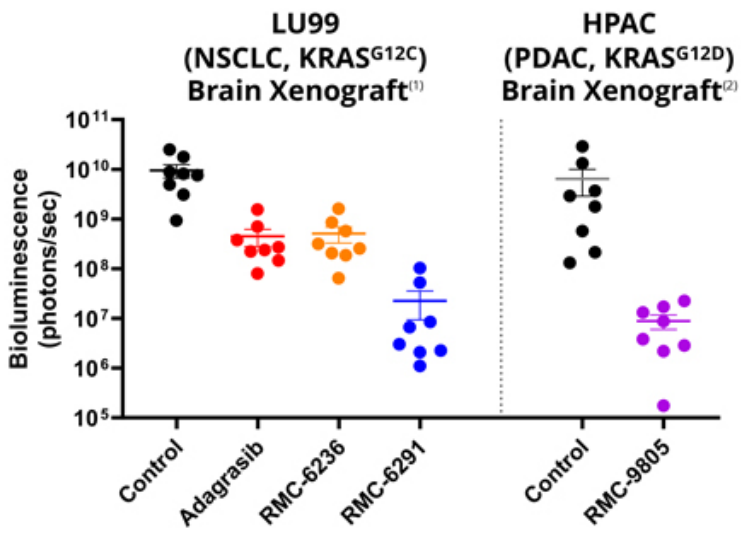
RVMD preclinical research as of 11/02/22
RMC-9805 dosed at 100 mg/kg po qd; n=2-8/group
NSCLC = non-small cell lung cancer; PDAC = pancreatic ductal adenocarcinoma; CRC = colorectal cancer
Responses assigned according to mRECIST (see appendix)
ORR = objective response rate; DCR = disease control rate

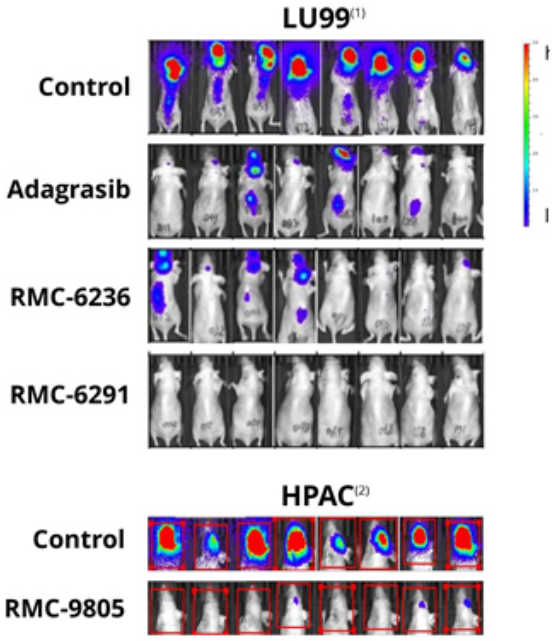
RMC-9805 Phase 1/1b Trial: Clinical Translation of Preclinical Single Agent Profile

	Preclinical Profile	Aims of Phase 1/1b Clinical Trial[2]
Dosing and Safety	✓ **Oral dosing** (daily): drug levels that drive maximal target crosslinking and sustained RAS pathway suppression ✓ **Safety**: well-tolerated in active range, highly selective for $KRAS^{G12D}$ ✓ **Long-term treatment**[1] at active doses	○ **Oral dosing**: once daily or BID to reach active exposures; surrogate markers of activity (ctDNA ○ **Safety**: short- and long-term safety and tolerability at active exposures ○ **RP2DS**
Anti-Tumor Activity	✓ **Tumor selection**: active in $KRAS^{G12D}$ tumor models across histotypes, including NSCLC, PDAC and CRC ✓ **Activity**: deep and durable regressions across $KRAS^{G12D}$ tumors, especially NSCLC and PDAC	○ **Patient selection**: $KRAS^{G12D}$ solid tumors ○ **Efficacy**: initial clinical responses by RECIST; formal proof-of-concept via expansion cohorts focused on distinct histotypes

(1) Long-term in mouse models defined as up to 90 days of treatment
(2) Site activation ongoing under an investigational new drug application
RP2DS = recommended Phase 2 dose and schedule; ctDNA = circulating tumor DNA

RAS(ON) Inhibitors Drive Tumor Regressions in Models of Brain Metastasis





RVMD preclinical research
Adagrasib dosed at 100 mg/kg po bid, RMC-6236 dosed at 25 mg/kg po qd, RMC-6291 dosed at 100 mg/kg po bid, RMC-9805 dosed at 100 mg/kg po qd
(1) LU99 intracranial xenograft model (NSCLC, KRAS$^{G12C/WT}$). Bioluminescence measured at end of study.
(2) HPAC intracranial xenograft model (PDAC, KRAS$^{G12D/WT}$). Bioluminescence measured at end of study.

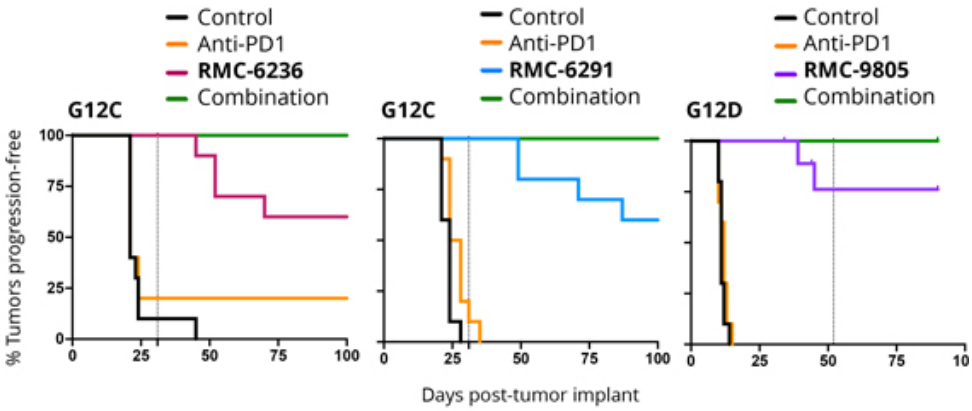
RAS(ON) Inhibitors Induce Anti-Tumor Immunity via Multiple Mechanisms in Immunocompetent Models

Mechanisms of Action

↑ Antigen presentation

↓ Myeloid suppressive cells

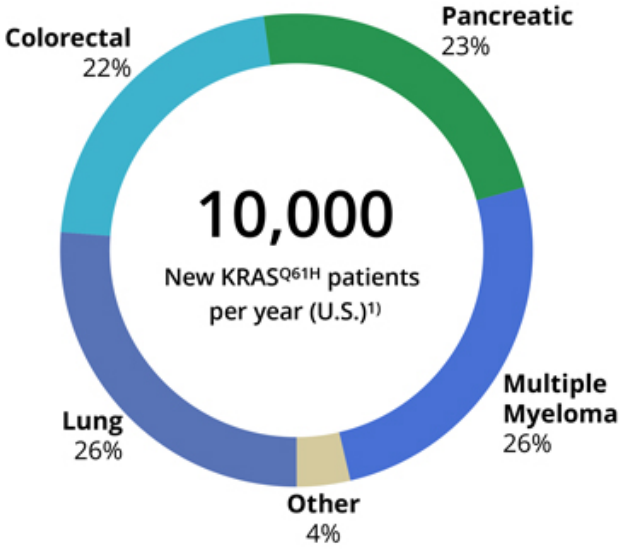
↑ T-cell infiltration



Additive Activity Supports Clinical Combination Strategies with Immune Therapies

RVMD preclinical research
RMC-9805 experiment conducted in CT26 syngeneic tumor model ($KRAS^{G12D}$); 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; RMC-9805 (100 mg/kg po qd) dosed for 42 days; anti-PD-1 (10 mg/kg ip biw, for 21 days)
Vertical dashed lines represent treatment stop; Kaplan-Meier progression defined as tumor doubling from baseline

RMC-0708: First-in-Class Mutant-Selective RAS(ON) Inhibitor for KRAS^{Q61H} Cancers



Preclinical Profile

Highly Potent and Selective RAS(ON) Inhibitor

- Highly active against KRAS^{Q61H}
- Non-covalent, highly selective over wild-type RAS
- Low off-target risk and acceptable safety profile

Robust Anti-tumor Activity in Cancer Models

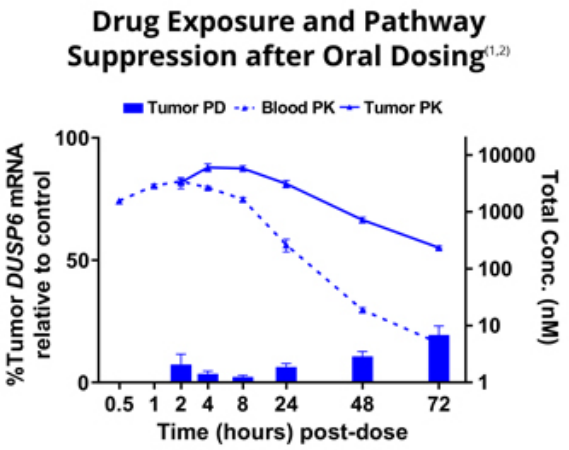
- Rapid, deep and sustained inhibition drives durable regressions in KRAS^{Q61H} lung, pancreatic and colorectal cancers

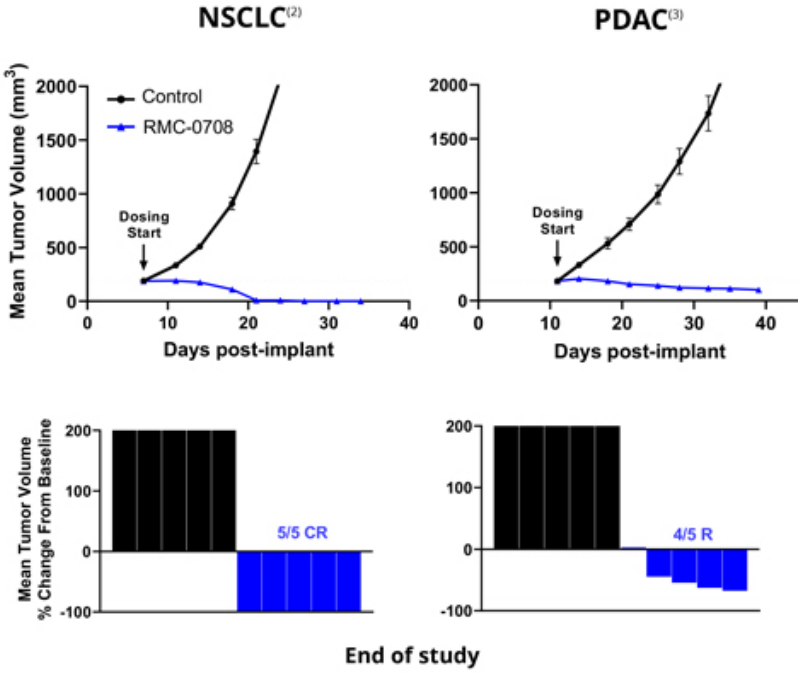
Attractive PK/ADME Profile

- Favorable *in vivo* oral bioavailability and clearance for effective target coverage in KRAS^{Q61H}-addicted cancer cells

(1) Calculated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020 (see appendix for additional detail); lung cancer = non-small cell lung cancer

RMC-0708: Sustained Pathway Inhibition *in Vivo* and Tumor Regressions in KRAS^{Q61H} Cancer Models





RVMD preclinical research
RMC-0708 dosed at 30 mg/kg po qd
(1) PK/PD data collected at indicated timepoints after a single dose
(2) HCC2108 subcutaneous xenograft model (NSCLC, KRAS$^{Q61H/Q61H}$)
(3) T3M-4 subcutaneous xenograft model (PDAC, KRAS$^{Q61H/WT}$)
R = number of regressions >10% from initial; CR = number of regressions ≥80% from initial
Each animal represented as a separate bar in waterfall plots

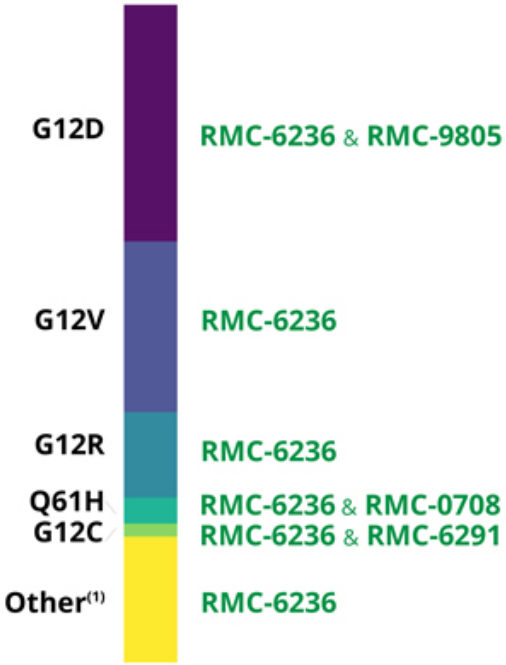
On Target to Outsmart **Pancreatic Cancer**: RAS(ON) Inhibitors Designed to Cover All KRAS^{MUTANT} Drivers[1]

Devastating disease
>90% driven by KRAS mutations

49,000

New KRAS^{MUTANT} pancreatic cancer patients per year (U.S.)[1].
No approved targeted therapies

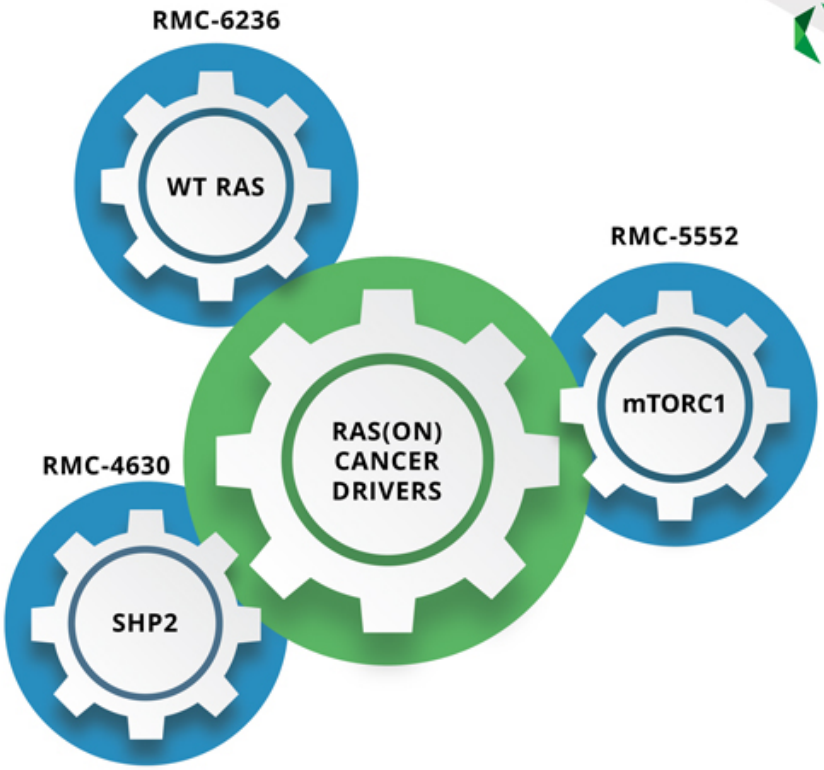
Dismal survival rates



(1) Calculated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020 (see appendix for additional detail); RMC-6236 tested against all mutations occurring at >2% frequency in pancreatic cancer

RAS Companion Inhibitors

Designed to Suppress Cooperating Targets and Pathways that Sustain RAS-Addicted Cancers



Evaluation of RMC-4630 in Combination with Sotorasib in KRAS^{G12C} Cancer Patients

Phase 1/1b Clinical Trial[1]

- ✓ Selected single agent RP2DS: Oral dosing of 200 mg D1D2 weekly: well-tolerated, safety profile consistent with on-pathway inhibition
- ✓ Anti-tumor activity in certain KRAS^{MUTANT} and NF1^{LOF} cancers evidenced by SD, PR and/or CR

Amgen's CodeBreaK 101c Clinical Trial[2]

- ✓ In KRAS^{G12C} patients, "the combination of sotorasib with RMC-4630 was safe and tolerable"[3] with sotorasib at 960 mg po qd and RMC-4630 at 140-200 mg po D1D2 weekly
- ✓ 75% ORR/100% DCR among KRAS^{G12C} inhibitor-naïve NSCLC patients treated at top two doses of RMC-4630 (n=4)

Aims of RMC-4630-03 Phase 2 Trial[4]

Dosing and Safety

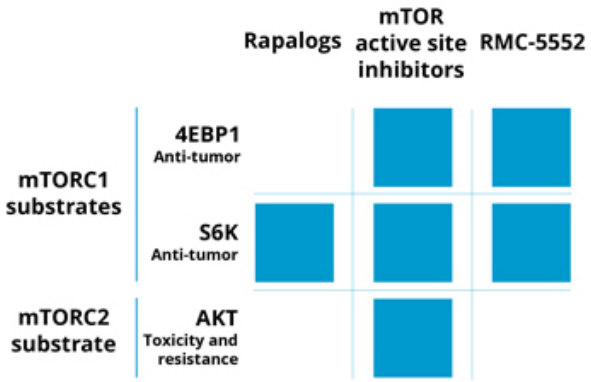
- **Dosing**: Focused primarily on 200 mg D1D2 weekly combined with sotorasib at 960 mg daily
- **Safety**: short- and long-term safety and tolerability

Anti-Tumor Activity

- **Patient Selection**: NSCLC patients without prior KRAS^{G12C} inhibitor treatment stratified into two cohorts: KRAS^{G12C} with or without co-mutations such as KEAP1 or STK11
- **Efficacy**: demonstrate clinical benefit additive to sotorasib

(1) Ongoing study, RMC-4630-01 - ClinicalTrials.gov Identifier: NCT03634982 https://clinicaltrials.gov/ct2/show/NCT03634982?term=RMC-4630&draw=2&rank=1
(2) Ongoing study, CodeBreaK 101c - ClinicalTrials.gov Identifier: NCT04185883 https://clinicaltrials.gov/ct2/show/NCT04185883?term=codebreak+101&draw=2&rank=1
(3) Falchook et. al. Sotorasib in Combination with RMC-4630, a SHP2 Inhibitor, in *KRAS* p.G12C-Mutated NSCLC and Other Solid Tumors. 2022 World Conference on Lung Cancer. August 6-9, 2022. Vienna, Austria. Abstract #OA03.03.
(4) Ongoing study, RMC-4630-03 - ClinicalTrials.gov Identifier: NCT05054725 https://clinicaltrials.gov/ct2/show/NCT05054725?term=RMC-4630&draw=2&rank=2

RMC-5552: First-in-Class Bi-steric mTORC1-Selective Inhibitor for Cancers with Hyperactive mTOR Signaling



Highly Potent and Selective mTORC1 Inhibitor

- Bi-steric mechanism enables selectivity for mTORC1
- Capable of reactivating the tumor suppressor 4EBP1

Robust Anti-tumor Activity in Cancer Models

- Selective inhibition of mTORC1 drives durable regressions in mTOR pathway-mutant models

Attractive PK/ADME Profile

- Weekly dosing provides favorable PK exposure and prolonged target modulation *in vivo*

Characterization above is based on RVMD preclinical research

RMC-5552: Compelling Profile as a Potential RAS Companion Inhibitor

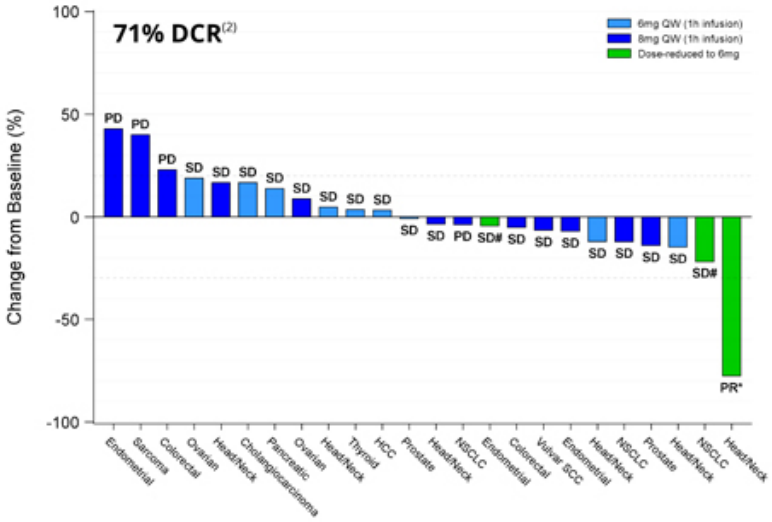
Preliminary radiologic and molecular evidence of activity at tolerated doses:

- Disease control across diverse tumors, including durable stable disease
- Objective response and regressions
- Favorable changes in surrogate markers
  - 3 of 6 patients with stable disease and oncogenic mTOR pathway variants had molecular responses(1)

(1) n = 6 molecular response-evaluable patients with oncogenic mTOR pathway variants detected by ctDNA treated at 6 mg or higher majority dose. "Oncogenic" defined as pathogenic or likely pathogenic by blinded adjudication process using publicly available variant data. Molecular response defined by 50% decrease or greater in mean VAF at C3D1 by Guardant360® Molecular Response algorithm. VAF = variant allele fraction

Phase 1/1b Single Agent Study

Best Tumor Change in Efficacy Evaluable Patients Treated at 6 mg or 8 mg IV Weekly



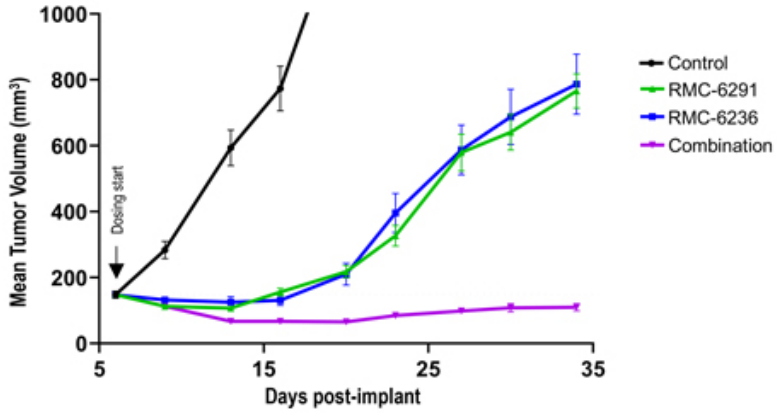
(2) n = 28 efficacy evaluable subjects. DCR = disease control rate.
*Patient received one dose of 12 mg, followed by weekly doses of 6 mg, had complete loss of oncogenic PTEN variant b ctDNA, and has been on RMC-5552 for >12 months.
#Patient received one dose of 10 mg, followed by weekly doses of 6 mg. Both patients were on RMC-5552 for >24 week Data as of 12/19/2022. PD = progressive disease, SD = stable disease, PR = partial response, mo = months

RMC-5552 Phase 1/1b Trial: Clinical Optimization of Single Agent Profile for Combination with RAS(ON) Inhibitor Portfolio

	Preclinical Profile	Aims of Phase I/Ib Clinical Trial[1]
Dosing and Safety	✓ **Dosing**: Once weekly dosing achieves levels that drive sustained inhibition of mTORC1 signaling and activation of 4EBP1 ✓ **Safety**: Well-tolerated, highly mTORC1 selective	○ **Dosing**: Establish optimal IV regimen based on safety, anti-tumor activity and surrogate marker of activity (ctDNA) ○ **Safety**: Demonstrate short- and long-term safety and tolerability at active exposures
Anti-Tumor Activity	✓ **Single Agent**: Strong activity in tumor models with hyperactivated mTORC1 ✓ **RAS Companion**: Combinatorial activity with RAS(ON) inhibitors	○ **Single Agent**: Evidence of activity at tolerated doses in tumors with hyperactive mTORC1 signaling ○ **RAS Companion**: Identify appropriate dose and schedule for initiation of combinations with RAS(ON) inhibitors

(1) Ongoing study, RMC-5552-001 - ClinicalTrials.gov Identifier: NCT04774952 https://clinicaltrials.gov/ct2/show/NCT04774952?term=rmc-5552&draw=2&rank=1
ctDNA = circulating tumor DNA

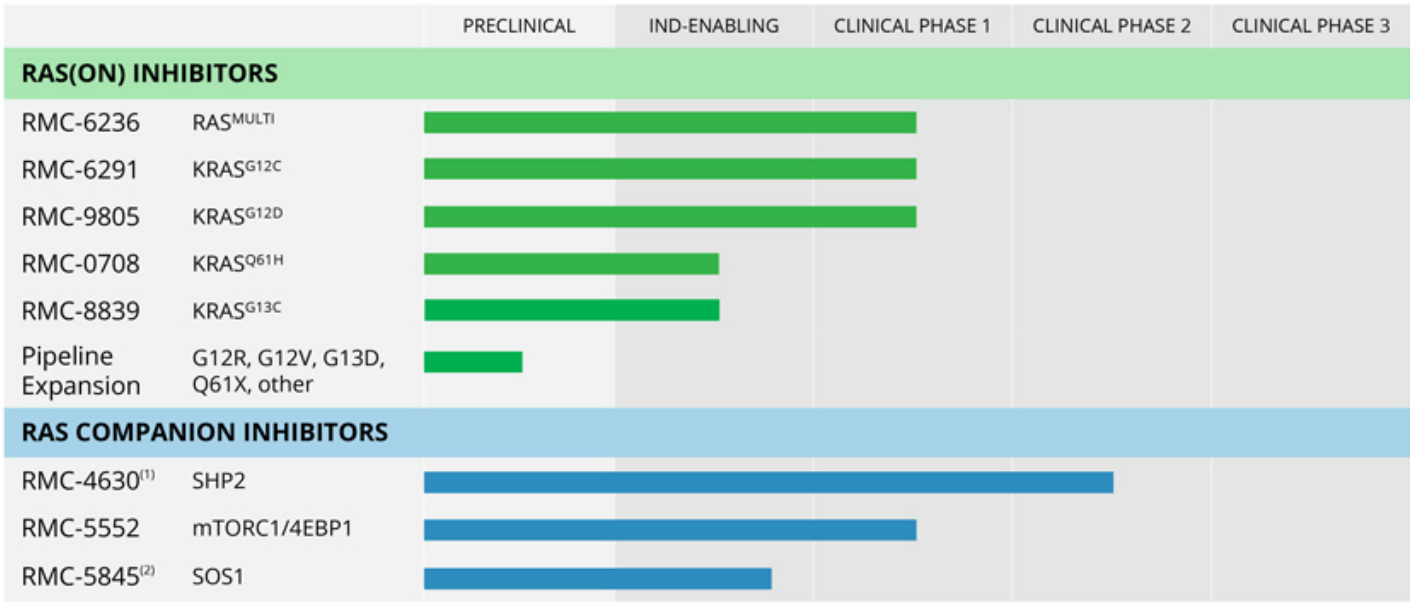
Overcoming Resistance: RMC-6291 + RMC-6236 Combination Induces Regressions in KRAS^{G12C} NSCLC Model



RAS^{MULTI}(ON) Inhibitor Deployed as a RAS Companion Inhibitor

RVMD preclinical research
NCI-H2122 subcutaneous xenograft model (NSCLC, KRAS$^{G12C/G12C}$)
RMC-6291 dosed at 100 mg/kg po qd; RMC-6236 dosed at 10 mg/kg po qd
RMC-6291, RMC-6236 and Combination - n = 15/group, Control – n=8
NSCLC = non-small cell lung cancer

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



(1) Sanofi collaboration on RMC-4630/SAR442720 terminated effective June 2023
(2) IND-ready, active development deferred

Anticipated Milestones

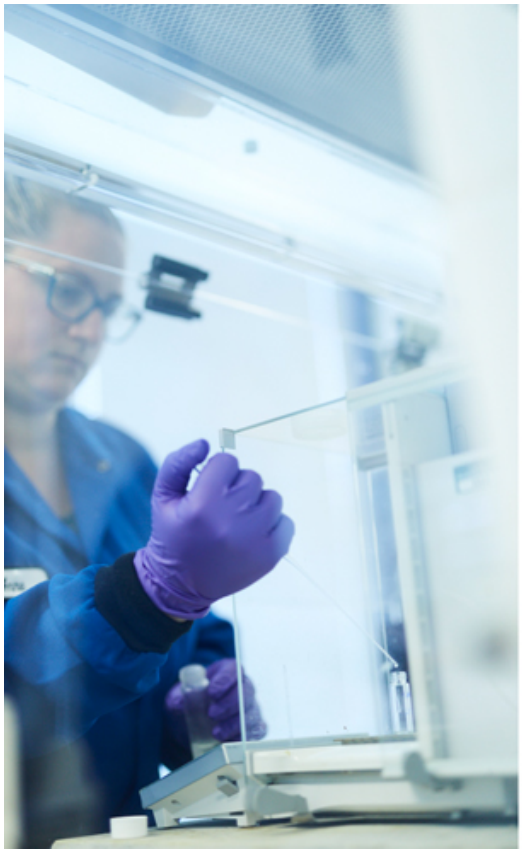
	MILESTONE (EXPECTED TIMING)
RAS(ON) INHIBITORS	
RMC-6236 (RAS^{MULTI})	Report further evidence of first-in-class single agent activity (Oct 2023)
RMC-6291 ($KRAS^{G12C}$)	Report preliminary evidence of differentiation from $KRAS^{G12C}$(OFF) inhibitors (Oct 2023)
RMC-9805 ($KRAS^{G12D}$)	Announce dosing of first patient (2H2023)
RAS COMPANION INHIBITORS	
RMC-4630 (SHP2)	Provide topline data from RMC-4630-03 (2H2023)
RMC-5552 (mTORC1/4EBP1)	Report additional evidence of single agent activity (4Q2023)

Financial Information

Financial Position	
Cash, cash equivalents and marketable securities as of March 31, 2023	$909.8 million[1]

2023 Financial Guidance
2023 GAAP net loss of $360 million to $400 million[2]

(1) With current cash, cash equivalents and marketable securities, the company projects it can fund planned operations into 2025
(2) Includes non-cash stock-based compensation expense of approximately $40 million to $50 million
Financial guidance does not include impact of proposed EQRx acquisition



REVOLUTION MEDICINES

On Target to Outsmart Cancer™

Focused on serving high unmet needs across numerous cancers driven by diverse RAS mutations

First two targeted ***RAS(ON) Inhibitors*** have shown initial clinical activity and evaluation continues

Targeted ***RAS Companion Inhibitors*** designed to counter drug resistance have shown initial clinical activity and evaluation continues

Development-stage portfolio covers RAS drivers of all major RAS-addicted cancers

Appendix

- RAS cancer epidemiology statistics are estimated using tumor mutation frequencies from Foundation Medicine Insights August 2020 and scaled to estimated patient numbers using cancer incidence from ACS *Cancer Facts and Figures* 2020:
  - RAS mutations include: KRAS G12(A,C,D,R,S,V), KRAS G13(C,D), KRAS Q61(H, K, L), KRAS A146T, KRAS wild-type amplification, NRAS G12C, NRAS Q61(H,K,L,R,P), HRAS mutations of known/likely function (including HRAS Q61(H,L)), BRAF class 3 mutations, NF1 loss of function mutations, PTPN11 mutations of known/likely function. NF1 LOF mutations = 50% of all NF1 mutations of known/likely function. BRAF class 3 mutations = D287H, D594(A,E,G,H,N,V,Y), F595L, G466(A,E,R,V,E,D,R), N581(I,S), S467L,T599I, V459L.
  - Includes 12 major types: non-small cell lung cancer, colorectal, pancreatic adenocarcinoma, renal, gastroesophageal, head and neck squamous cell, ovarian and biliary cancers, acute myeloid leukemia, and advanced melanoma, bladder and uterine/endometrial cancers causing mortality.
    - $KRAS^{Q61H}$ epidemiology statistics include multiple myeloma in addition to 12 major types named above
  - Est. worldwide annual incidence of RAS-mutated cancers is 3.4 million per Prior et al., *Cancer Research* 2020
- RAS mutations drive 30% of human cancers per Prior et al., *Cancer Research* 2020
- Mouse tumor responses on slides 9, 12 and 17 assigned according to mRECIST (modified from Gao et al. Nat Med. 2015):
  - mPD = progressive disease; mSD = stable disease; mPR = partial response; mCR = complete response
- PK = pharmacokinetic; ADME = absorption, distribution, metabolism, and excretion

Forward-Looking Statements

This presentation contains forward-looking statements within the meaning of federal securities laws, including the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995. Such statements are based upon current plans, estimates and expectations of management of Revolution Medicines and EQRx in light of historical results and trends, current conditions and potential future developments, and are subject to various risks and uncertainties that could cause actual results to differ materially from such statements. The inclusion of forward-looking statements should not be regarded as a representation that such plans, estimates and expectations will be achieved. Words such as "anticipate," "expect," "project," "intend," "believe," "may," "will," "should," "plan," "could," "continue," "target," "contemplate," "estimate," "forecast," "guidance," "predict," "possible," "potential," "pursue," "likely," and words and terms of similar substance used in connection with any discussion of future plans, actions or events identify forward-looking statements. All statements, other than historical facts, including express or implied statements regarding the proposed transaction; the conversion of equity interests contemplated by the Merger Agreement; the issuance of common stock of Revolution Medicines contemplated by the Merger Agreement; the expected filing by Revolution Medicines of a registration statement and Joint Proxy Statement/Prospectus to be included therein; the expected timing of the closing of the proposed transaction; the ability of the parties to complete the proposed transaction considering the various closing conditions; the expected benefits of the proposed transaction; the competitive ability and position of the combined company; and any assumptions underlying any of the foregoing, are forward-looking statements. Important factors that could cause actual results to differ materially from Revolution Medicines' and EQRx's plans, estimates or expectations described in such forward-looking statements could include, but are not limited to: (i) the risk that the proposed transaction may not be completed in a timely manner or at all, which may adversely affect Revolution Medicines' and EQRx's businesses and the price of their respective securities; (ii) uncertainties as to the timing of the consummation of the proposed transaction; (iii) the potential failure to receive, on a timely basis or otherwise, the required approvals of the proposed transaction, including stockholder approvals by both Revolution Medicines' stockholders and EQRx's stockholders, and the potential failure to satisfy the other conditions to the consummation of the transaction; (iv) that the proposed transaction may involve unexpected costs, liabilities or delays; (v) the effect of the announcement, pendency or completion of the proposed transaction on each of Revolution Medicines' or EQRx's ability to attract, motivate, retain and hire key personnel and maintain relationships with customers, distributors, suppliers and others with whom Revolution Medicines or EQRx does business, or on Revolution Medicines' or EQRx's operating results and business generally; (vi) that the proposed transaction may divert management's attention from each of Revolution Medicines' and EQRx's ongoing business operations; (vii) the risk of any legal proceedings related to the proposed transaction or otherwise, or the impact of the proposed transaction thereupon, including resulting expense or delay; (viii) that Revolution Medicines or EQRx may be adversely affected by other economic, business and/or competitive factors; (ix) the occurrence of any event, change or other

circumstance that could give rise to the termination of the Merger Agreement relating to the proposed transaction, including in circumstances which would require Revolution Medicines or EQRx to pay a termination fee; (x) the risk that restrictions during the pendency of the proposed transaction may impact Revolution Medicines' or EQRx's ability to pursue certain business opportunities or strategic transactions; (xi) the risk that Revolution Medicines or EQRx may be unable to obtain governmental and regulatory approvals required for the proposed transaction, or that required governmental and regulatory approvals may delay the consummation of the proposed transaction or result in the imposition of conditions that could reduce the anticipated benefits from the proposed transaction or cause the parties to abandon the proposed transaction; (xii) the risk that the anticipated benefits of the proposed transaction may otherwise not be fully realized or may take longer to realize than expected; (xiii) the impact of legislative, regulatory, economic, competitive and technological changes; (xiv) risks relating to the value of Revolution Medicines securities to be issued in the proposed transaction; (xv) the risk that integration of the proposed transaction post-closing may not occur as anticipated or the combined company may not be able to achieve the growth prospects expected from the transaction; (xvi) the effect of the announcement, pendency or completion of the proposed transaction on the market price of the common stock of each of Revolution Medicines and the common stock and publicly traded warrants of EQRx; (xvii) the implementation of each of Revolution Medicines' and EQRx's business model and strategic plans for product candidates and pipeline, and challenges inherent in developing, commercializing, manufacturing, launching, marketing and selling potential existing and new products; (xviii) the scope, progress, results and costs of developing Revolution Medicines' and EQRx's product candidates and any future product candidates, including conducting preclinical studies and clinical trials, and otherwise related to the research and development of Revolution Medicines' and EQRx's pipeline; (xix) the timing and costs involved in obtaining and maintaining regulatory approval for Revolution Medicines' and EQRx's current or future product candidates, and any related restrictions, limitations and/or warnings in the label of an approved product; (xx) the market for, adoption (including rate and degree of market acceptance) and pricing and reimbursement of Revolution Medicines' and EQRx's product candidates and their respective abilities to compete with therapies and procedures that are rapidly growing and evolving; (xxi) uncertainties in contractual relationships, including collaborations, partnerships, licensing or other arrangements and the performance of third-party suppliers and manufacturers; (xxii) the ability of each of Revolution Medicines and EQRx to establish and maintain intellectual property protection for products or avoid or defend claims of infringement; (xxiii) exposure to inflation, currency rate and interest rate fluctuations and risks associated with doing business locally and internationally, as well as fluctuations in the market price of each of Revolution Medicines' and EQRx's traded securities; (xxiv) risks relating to competition within the industry in which each of Revolution Medicines and EQRx operate; (xxv) the unpredictability and severity of catastrophic events, including, but not limited to, acts of terrorism or outbreak of war or hostilities; (xxvi) whether the termination of EQRx's license agreements and/or discovery collaboration agreements may impact its or Revolution Medicines' ability to license in additional programs in the future and the risk of delays or unforeseen costs in terminating such arrangements; (xxvii) risks that restructuring costs and charges may be greater than anticipated or incurred in different periods than anticipated; (xxviii) the risk that EQRx's restructuring efforts may adversely affect its programs and its ability to recruit and retain skilled and motivated personnel, and may be distracting to employees and management; and (xxix) the risk that EQRx's restructuring or wind-down efforts may negatively impact its business operations and reputation with or ability to serve counterparties or may take longer to realize than expected, as well as each of Revolution Medicines' and EQRx's response to any of the aforementioned factors. Additional factors that may affect the future results of Revolution Medicines and EQRx are set forth in their respective filings with the U.S. Securities and Exchange Commission (the "SEC"), including each of Revolution Medicines' and EQRx's most recently filed Annual Reports on Form 10-K, subsequent Quarterly Reports on Form 10-Q, Current Reports on Form 8-K and other filings with the SEC, which are available on the SEC's website at www.sec.gov. See in particular Item 1A of Revolution Medicines' Quarterly Report on Form 10-Q for the quarterly period ended March 31, 2023 under the heading "Risk Factors," and Item 1A of each of EQRx's Annual Report on Form 10-K for the fiscal year ended December 31, 2022 and Quarterly Report on Form 10-Q for the quarterly period ended March 31, 2023 under the headings "Risk Factors." The risks and uncertainties described above and in the SEC filings cited above are not exclusive and further information concerning Revolution Medicines and EQRx and their respective businesses, including factors that potentially could materially affect their respective businesses, financial conditions or operating results, may emerge from time to time. Readers are urged to consider these factors carefully in evaluating these forward-looking statements, and not to place undue reliance on any forward-looking statements, which speak only as of the date hereof. Readers should also carefully review the risk factors described in other documents that Revolution Medicines and EQRx file from time to time with the SEC. Except as required by law, each of Revolution Medicines and EQRx assume no obligation to update or revise these forward-looking statements for any reason, even if new information becomes available in the future.

Additional Information and Where to Find It

In connection with the proposed transaction, Revolution Medicines and EQRx plan to file with the SEC and mail or otherwise provide to their respective security holders a joint proxy statement/prospectus regarding the proposed transaction (as amended or supplemented from time to time, the "Joint Proxy Statement/Prospectus"). INVESTORS AND REVOLUTION MEDICINES' AND EQRX'S RESPECTIVE SECURITY HOLDERS ARE URGED TO CAREFULLY READ THE JOINT PROXY STATEMENT/PROSPECTUS IN ITS ENTIRETY WHEN IT BECOMES AVAILABLE AND ANY OTHER DOCUMENTS FILED BY EACH OF REVOLUTION MEDICINES AND EQRX WITH THE SEC IN CONNECTION WITH THE PROPOSED TRANSACTION OR INCORPORATED BY REFERENCE THEREIN BECAUSE THEY WILL CONTAIN IMPORTANT INFORMATION ABOUT THE PROPOSED TRANSACTION AND THE PARTIES TO THE PROPOSED TRANSACTION.

Revolution Medicines' investors and security holders may obtain a free copy of the Joint Proxy Statement/Prospectus and other documents that Revolution Medicines files with the SEC (when available) from the SEC's website at www.sec.gov and Revolution Medicines' website at ir.revmed.com. In addition, the Joint Proxy Statement/Prospectus and other documents filed by Revolution Medicines with the SEC (when available) may be obtained from Revolution Medicines free of charge by directing a request to Eric Bonach, H/Advisors Abernathy at eric.bonach@h-advisors.global.

EQRx's investors and security holders may obtain a free copy of the Joint Proxy Statement/Prospectus and other documents that EQRx files with the SEC (when available) from the SEC's website at www.sec.gov and EQRx's website at investors.eqrx.com. In addition, the Joint Proxy Statement/Prospectus and other documents filed by EQRx with the SEC (when available) may be obtained from EQRx free of charge by directing a request to EQRx's Investor Relations at investors@eqrx.com.

No Offer or Solicitation

This communication is not intended to and shall not constitute an offer to buy or sell or the solicitation of an offer to buy or sell any securities, nor shall there be any offer, solicitation or sale of securities in any jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such jurisdiction. No offering of securities shall be made except by means of a prospectus meeting the requirements of Section 10 of the Securities Act of 1933, as amended.

Participants in the Solicitation

Revolution Medicines, EQRx and their respective directors, executive officers, other members of management, certain employees and other persons may be deemed to be participants in the solicitation of proxies from the security holders of Revolution Medicines and EQRx in connection with the proposed transaction. Security holders may obtain information regarding the names, affiliations and interests of Revolution Medicines' directors and executive officers in Revolution Medicines' Annual Report on Form 10-K for the fiscal year ended December 31, 2022, which was filed with the SEC on February 27, 2023, and Revolution Medicines' definitive proxy statement on Schedule 14A for its 2023 annual meeting of stockholders, which was filed with the SEC on April 26, 2023. To the extent holdings of Revolution Medicines' securities by Revolution Medicines' directors and executive officers have changed since the amounts set forth in such proxy statement, such changes have been or will be reflected on subsequent Statements of Changes in Beneficial Ownership on Form 4 filed with the SEC. Security holders may obtain information regarding the names, affiliations and interests of EQRx's directors and executive officers in EQRx's Annual Report on Form 10-K for the fiscal year ended December 31, 2022, which was filed with the SEC on February 23, 2023, and in certain of EQRx's Current Reports on Form 8-K. To the extent holdings of EQRx's securities by EQRx's directors and executive officers have changed since the amounts set forth in such Annual Report on Form 10-K, such changes have been or will be reflected on subsequent Statements of Changes in Beneficial Ownership on Form 4 filed with the SEC. Additional information regarding the interests of such individuals in the proposed transaction will be included in the Joint Proxy Statement/Prospectus relating to the proposed transaction when it is filed with the SEC. These documents (when available) may be obtained free of charge from the SEC's website at www.sec.gov, Revolution Medicines' website at www.revmed.com and EQRx's website at www.eqrx.com.