REVOLUTION MEDICINES

On Target to Outsmart Cancer™

JANUARY 11, 2022

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 maintain existing collaborations, including with Sanofi, 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 part of an oral presentation on January 11, 2022 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 10, 2021, 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.

On Target to Outsmart Cancer

HIGH UNMET NEED IN RAS-ADDICTED CANCERS

RAS proteins drive 30% of human cancers[(1)], and are largely unserved by targeted therapeutics

STRONG CLINICAL VALIDATION OF RAS AS CANCER DRIVER

Proof-of-principle from first-gen $KRAS^{G12C}$ inhibitors[(2)] predicts favorable impact of targeted inhibitors across numerous RAS cancer drivers

DEEP SCIENCE-DRIVEN PIPELINE

Comprehensive collection of groundbreaking *RAS(ON) Inhibitors* with best-in-class preclinical profiles and/or first-in-class potential covering RAS space broadly; first candidates planned to enter clinic in 2022

Leading *RAS Companion Inhibitors* in clinic designed for combination treatment strategies to counter resistance to RAS targeted therapies

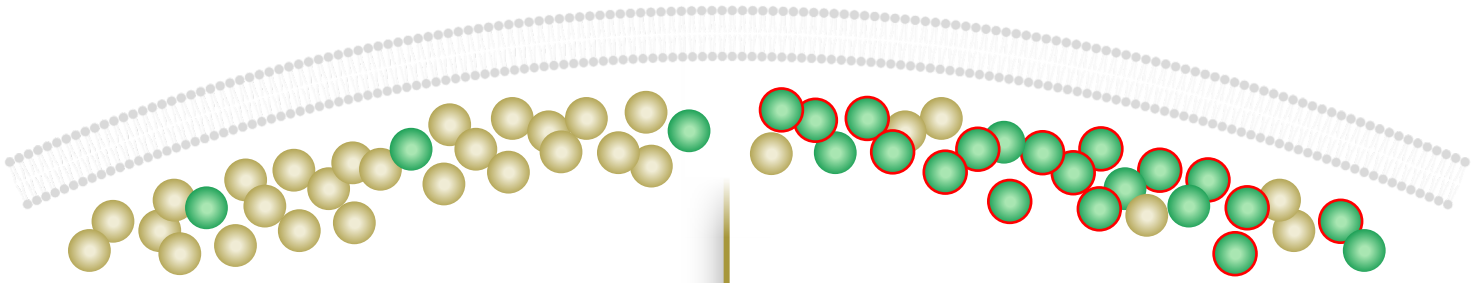
(1) Prior et al., *Cancer Research* 2020
(2) Lumakras approved by the FDA in May 2021

Excessive RAS(ON) Signaling Drives 30% of Human Cancers

RAS(OFF)

RAS(ON)

Normal



Tightly regulated
RAS(ON) proteins control cell growth

RAS-Addicted Cancers

RAS Cancer Mutations

Excessive
RAS(ON) signaling drives uncontrolled cell growth

Exemplary mutations in KRAS, HRAS and/or NRAS	G12C, G12D, G12V, G12R G13C, G13D Q61H, Q61K, Q61L

New patients per year (U.S.)[1]

230,000 **All**	
67,000	Lung cancer
79,000	Colorectal cancer
49,000	Pancreatic cancer

(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

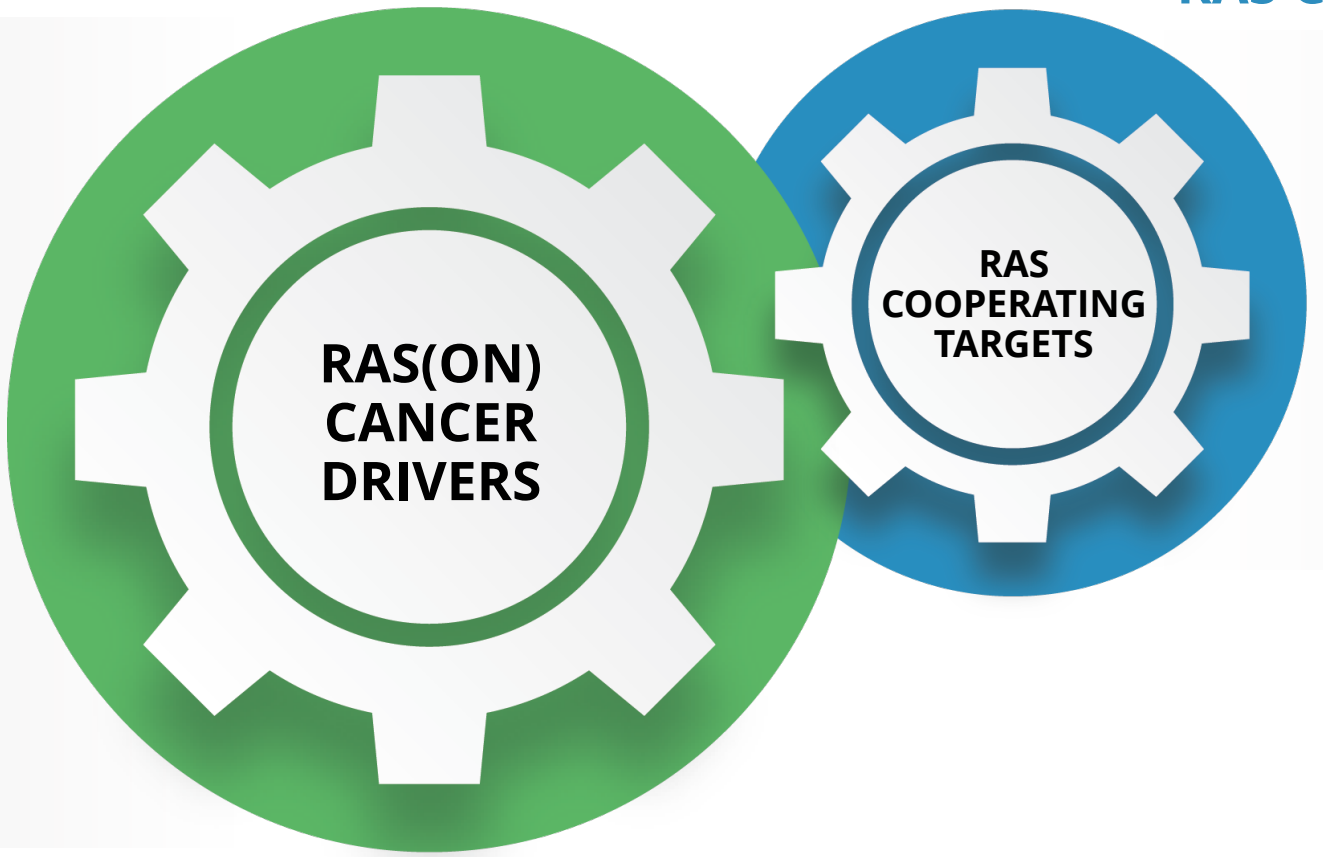
Deep Science-Driven Pipeline of Targeted Therapies for RAS-Addicted Cancers

RAS(ON) Inhibitors

2 Drug Candidates expected to enter clinic in 2022

2 Drug Candidates expected to file INDs in 2023

4+ Pipeline expansion programs



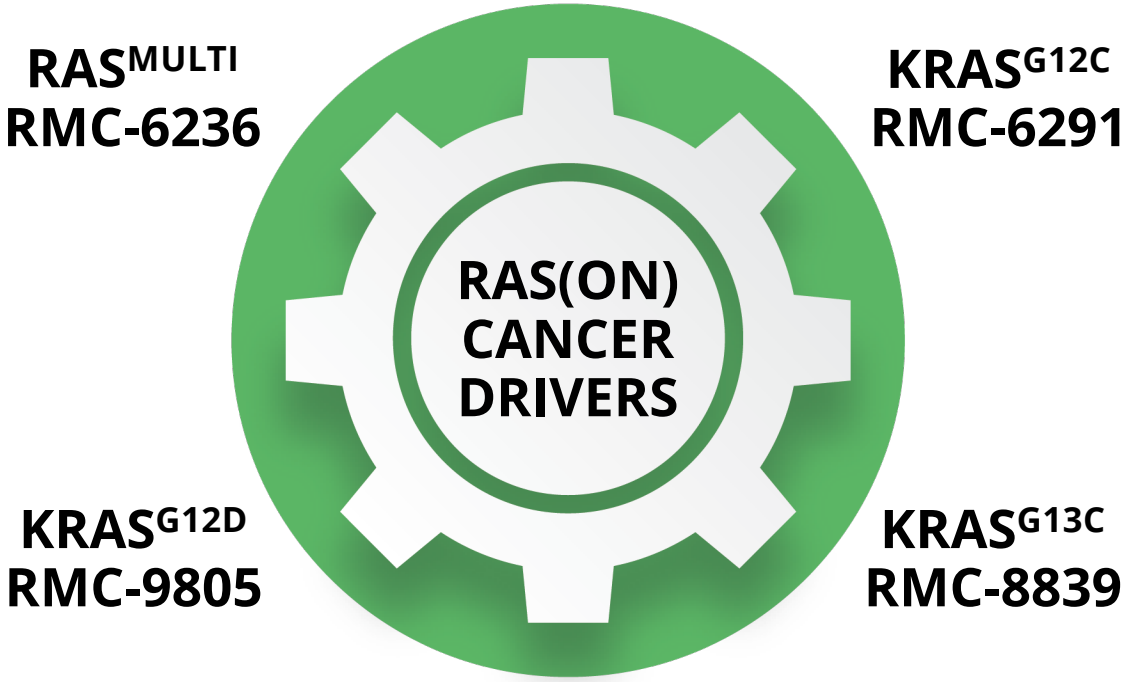
RAS Companion Inhibitors

2 Clinical-stage Drug Candidates

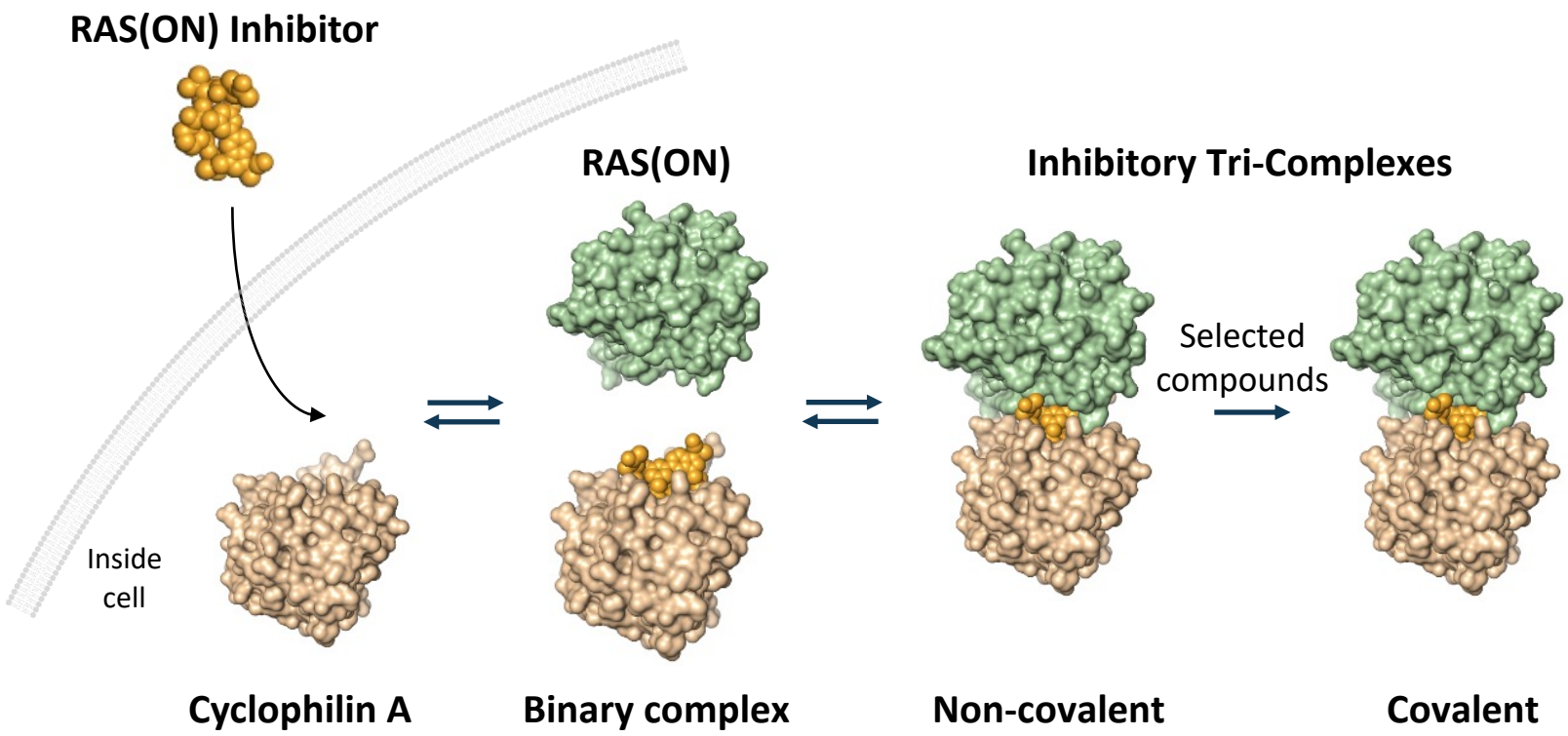
1 IND-ready Drug Candidate

RAS(ON) Inhibitors

Induce Rapid, Deep and Sustained Suppression of RAS(ON) Cancer Drivers



Distinctive RAS Drug Discovery: Innovation Engine Targets Oncogenic RAS(ON) Proteins

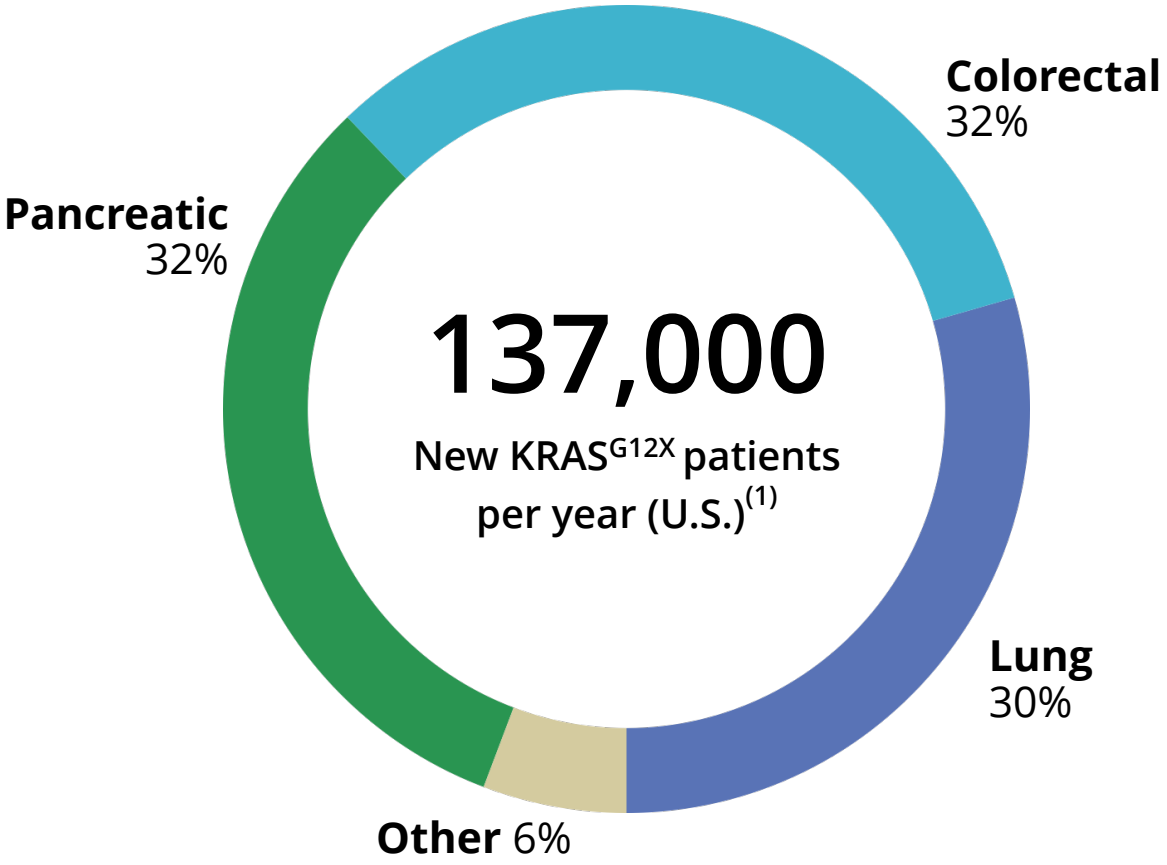


RAS(ON) Inhibitors

Deep and Diverse Collection

- Highly potent and selective
- Oral and drug-like
- Rapid, deep and sustained suppression of RAS(ON) signaling

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}

Highly Potent and Selective RAS(ON) Inhibitor

- Inhibits canonical RAS family members, suppressing the mutant cancer driver 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 including KRAS^{G12D}, KRAS^{G12V}, KRAS^{G12R} and KRAS^{G12C}

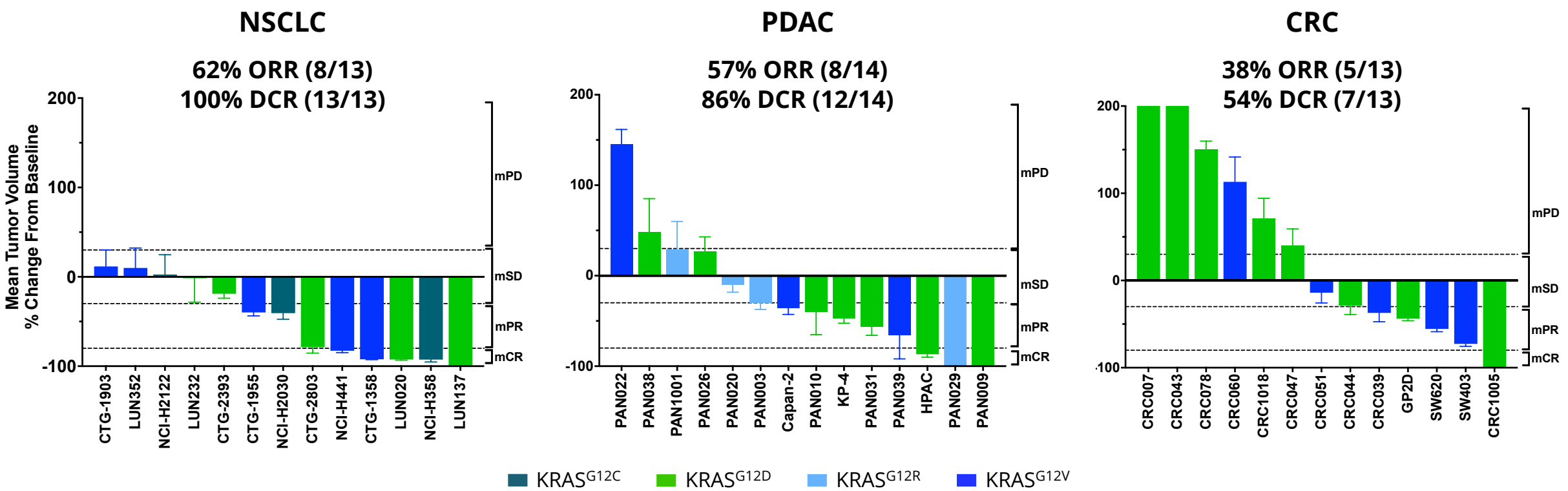
Attractive PK/ADME Profile

- 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

Characterization above is based on RVMD preclinical research

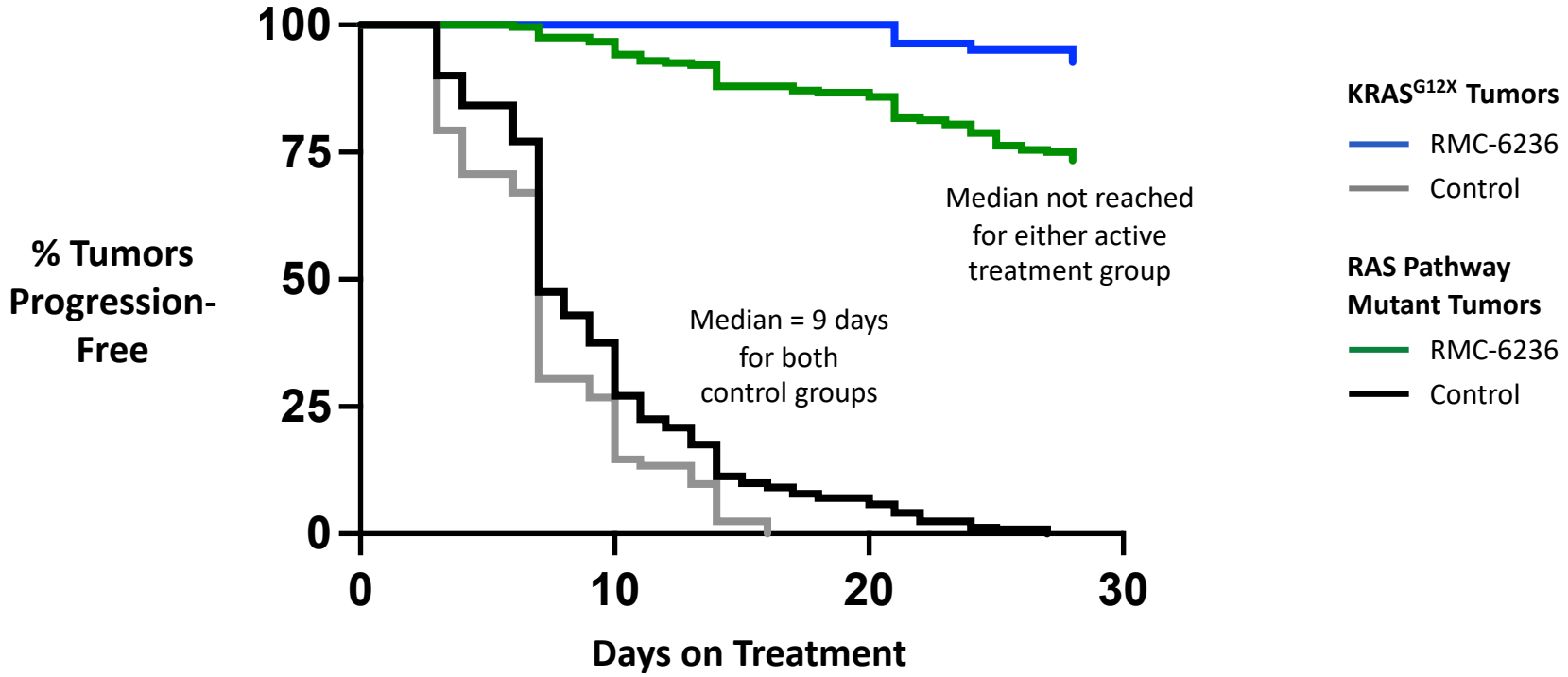
RMC-6236: Highly Active *in Vivo* Across Cancer Models with KRAS^{G12X} Drivers



Deep Tumor Regressions and Complete Responses Observed Across Cancer Models

RVMD preclinical research, as of 10/12/21
RMC-6236 dosed at 25 mg/kg po qd; n = 3-10/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-6236: Highly Active *in Vivo* Across Cancer Models with Diverse RAS Drivers

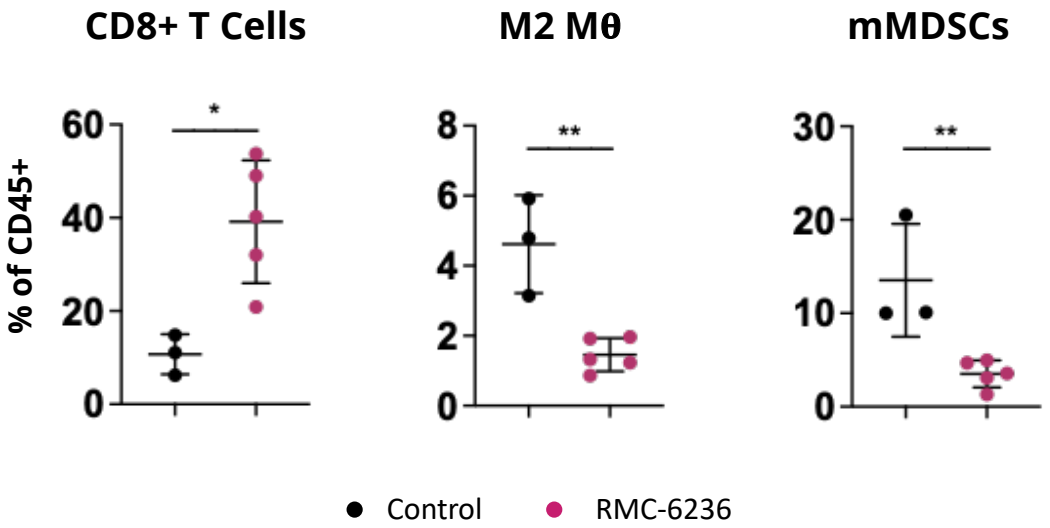


Durable Anti-Tumor Benefit Observed in KRAS^{G12X} Cancer Models and Beyond

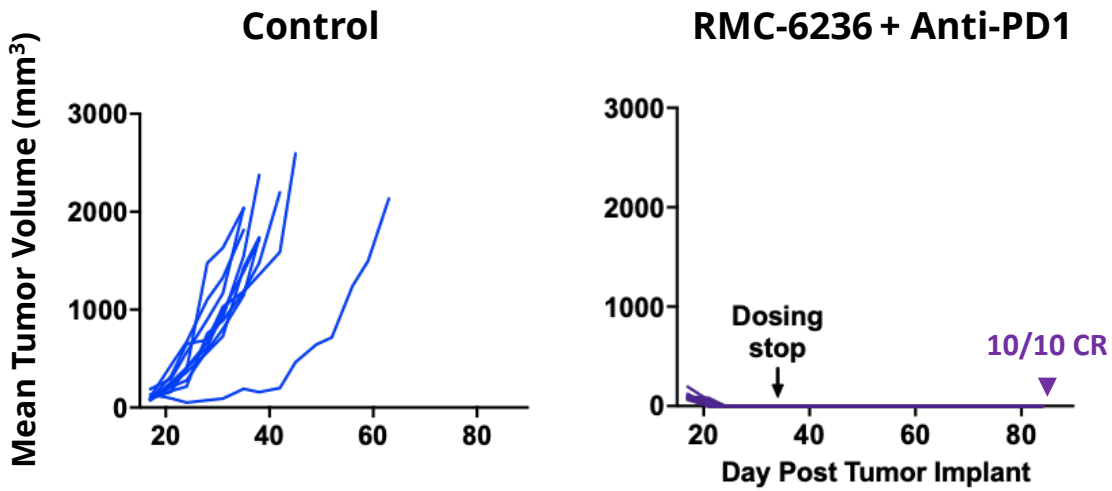
RVMD preclinical research, as of 10/12/21
RMC-6236 dosed at 25 mg/kg po qd
Progression defined as tumor doubling from baseline over 28 days
p<0.0001 by Log-rank test (control vs RMC-6236 treatment)
See appendix for composition of KRAS^{G12X} Tumors and RAS Pathway Mutant Tumors

RMC-6236: Anti-Tumor Immunity *in Vivo* and Strong Additivity with Checkpoint Inhibitor

Favorable Transformation of Tumor Immune Microenvironment



Durable Complete Responses with Checkpoint Inhibitor Combination



Modulation of the Tumor Microenvironment Primes for Anti-Tumor Immunity in Cancer Models

RVMD preclinical research, as of 09/10/2021
Syngeneic tumor model with CT26 cell line engineered to express $KRAS^{G12C}$
RMC-6236 dosed at 25 mg/kg po qd; Anti-PD1 dosed at 10 mg/kg ip biw; n = 10/group
M2 Mθ = M2 macrophages; mMDSCs = Monocytic myeloid derived suppressor cells

RMC-6236: Clinical Priorities to Pursue First-in-Class Activity Against KRAS^{G12X} Tumors

Activities

2022

- Submit IND^
- Initiate single agent dose escalation in patients with cancers with KRAS^{G12X} mutations (focused on NSCLC, pancreatic cancer and CRC)
- Include 'below MTD' expansion cohorts in select populations during dose escalation

Further development

- Define RP2DS
- Single agent expansion cohorts in KRAS^{G12X} tumors (NSCLC, pancreatic cancer and CRC)
- Combinations in KRAS^{G12X} tumors (NSCLC, pancreatic cancer and CRC)

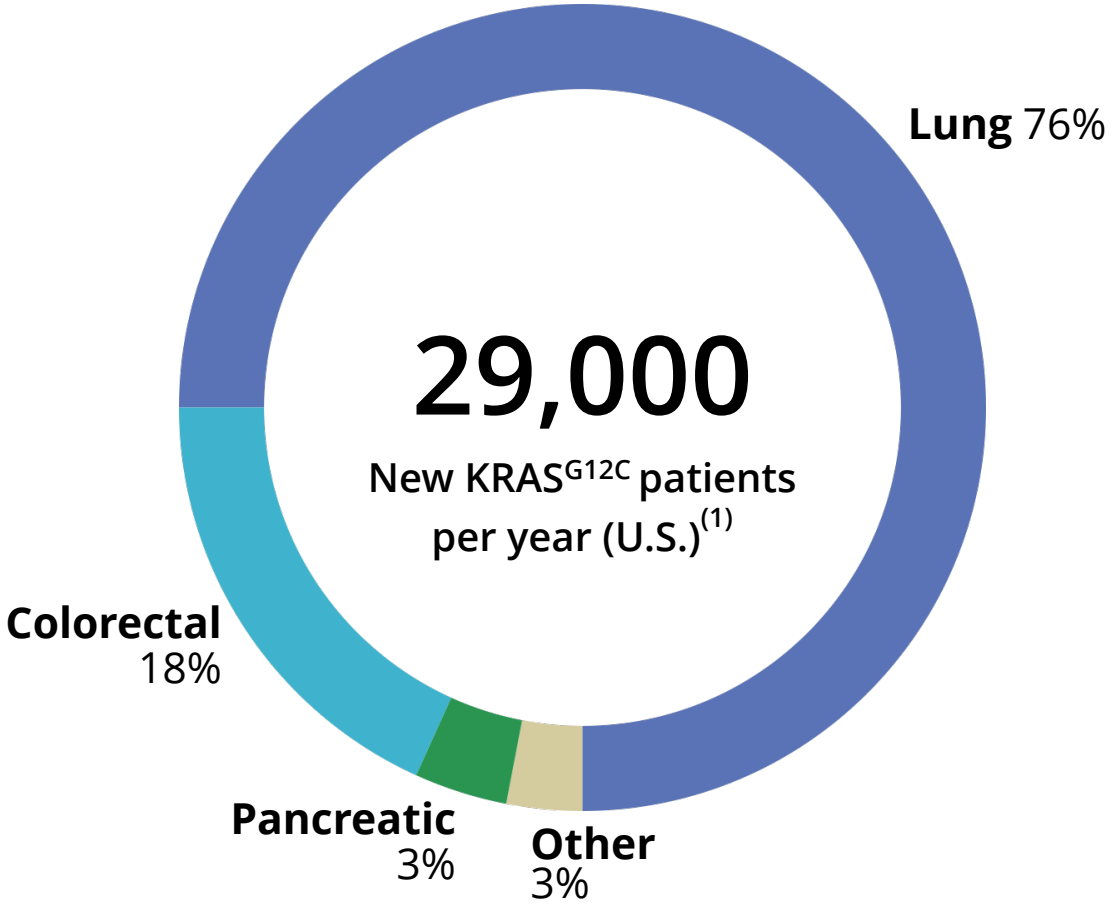
Aims

Evidence of first-in-class single agent activity against KRAS^{G12X} tumors^

^See Milestones table

KRAS^{G12X} may include KRAS^{G12D}, KRAS^{G12V}, KRAS^{G12R} and/or KRAS^{G12C}
RP2DS = Recommended Phase 2 dose and schedule
MTD = maximum tolerated dose
NSCLC = non-small cell lung cancer; CRC = colorectal cancer

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



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

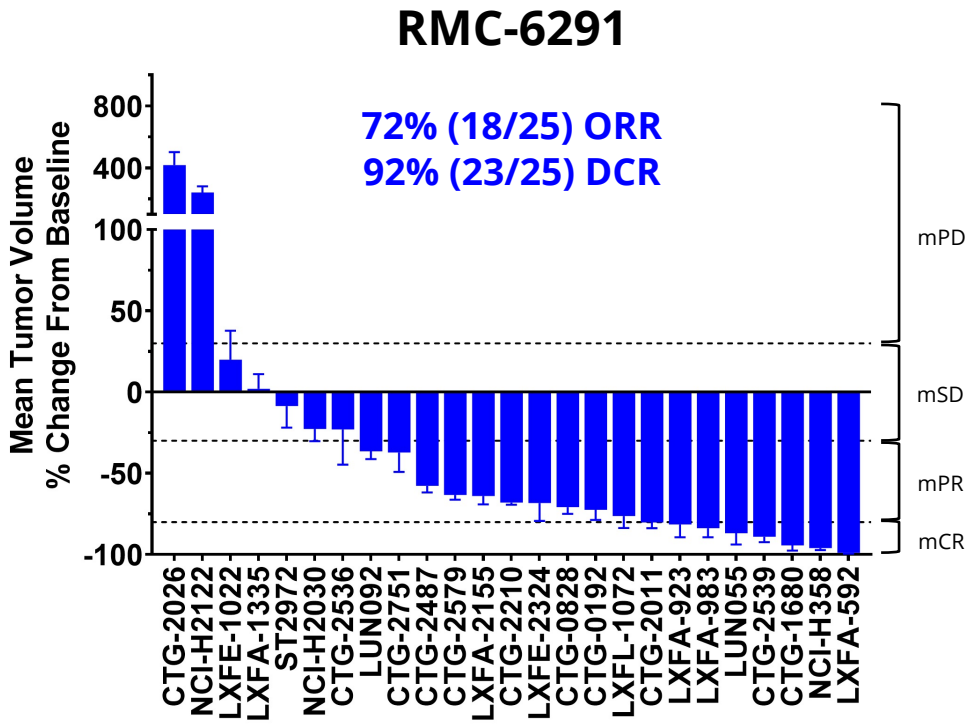
Attractive PK/ADME Profile

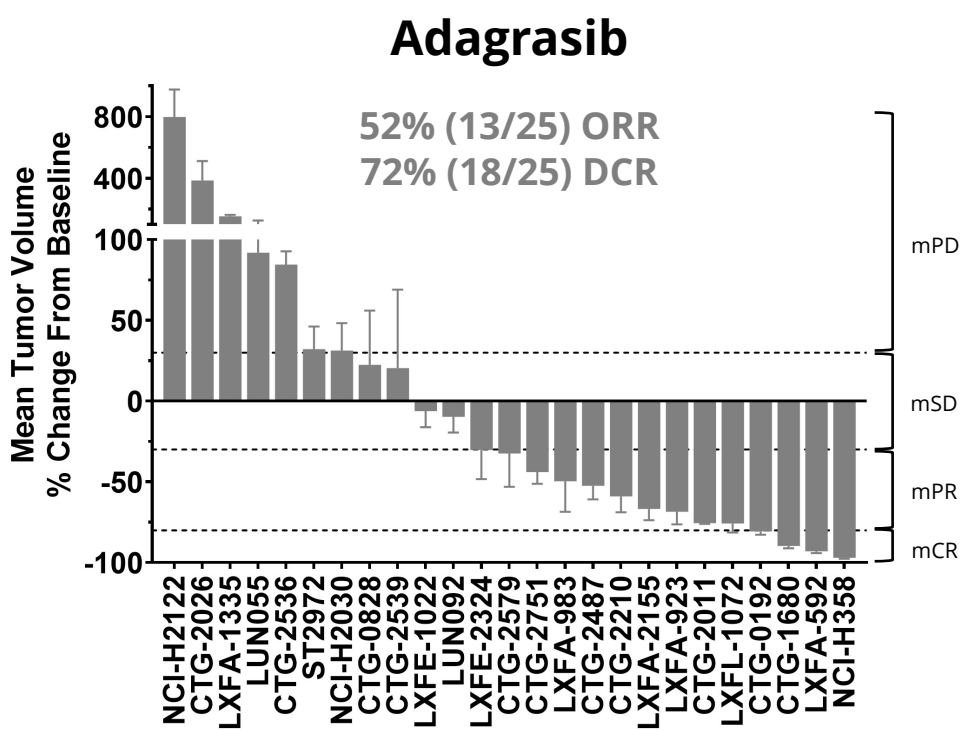
- Favorable *in vivo* oral bioavailability and clearance for effective target coverage in KRAS^{G12C}-addicted cancer cells

(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

Characterization above is based on RVMD preclinical research

RMC-6291: Superior Outcomes in Mouse Clinical Trial with KRAS^{G12C} NSCLC Models



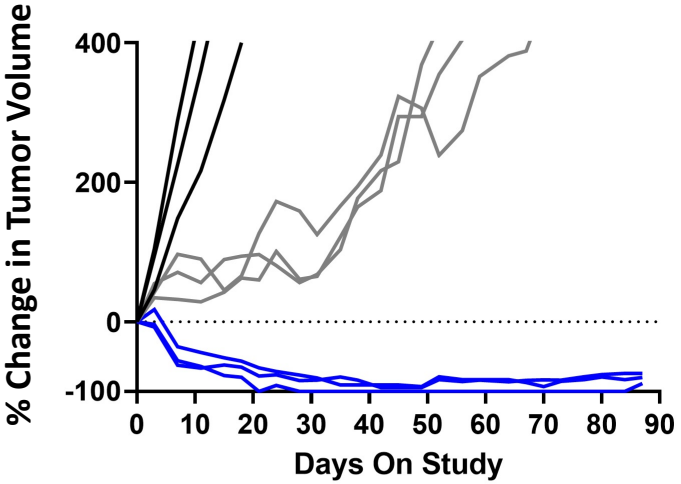


Best-in-Class Potential in KRAS^{G12C} NSCLC

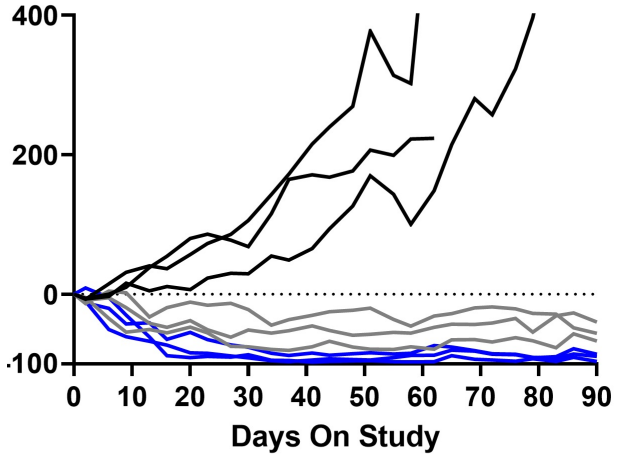
RVMD preclinical research as of 10/21/21
Adagrasib dosed at 100 mg/kg po qd; RMC-6291 dosed at 200 mg/kg po qd; n = 3 to 10/group
NSCLC = Non-small cell lung cancer
Responses assigned according to mRECIST (see appendix)

RMC-6291 May Improve on KRAS^{G12C} (OFF) Inhibitor Class Across Three Outcome Measures in NSCLC

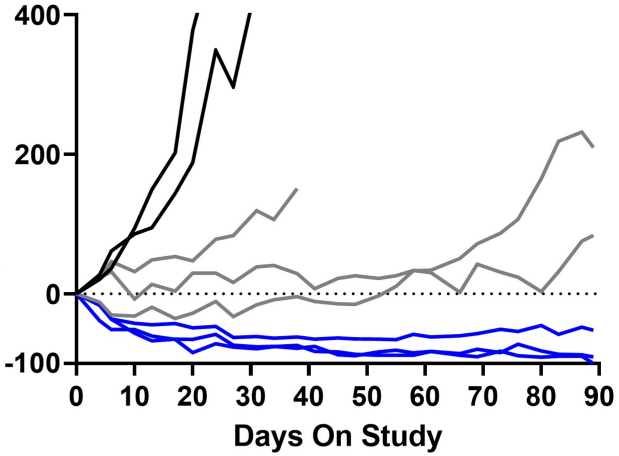
Increased Rate Of Response[(a)]



Increased Depth Of Response[(b)]



Increased Duration Of Response[(c)]



Control RMC-6291 Adagrasib

Best-in-Class Potential in KRAS^{G12C} NSCLC

RVMD preclinical research as of 07/28/21
RMC-6291 dosed at 200 mg/kg po qd; Adagrasib dosed at 100 mg/kg po qd
NSCLC = Non-small cell lung cancer
PDX Models: (a) LUN055; (b) LXFA-983; (c) CTG-0828
Nichols. Targeting KRAS^{G12C}(ON) and Potential Application to Overcoming Drug Resistance in RAS-Addicted Tumors. RAS-Targeted Drug Development Summit. Sept. 22, 2021.

RMC-6291: Clinical Priorities to Pursue Best-in-Class Activity Against KRAS^{G12C} Tumors

Activities

2022

- Submit IND^
- Initiate single agent dose escalation in KRAS^{G12C} tumors
- Include 'below MTD' expansion cohorts in select populations (e.g., NSCLC) during dose escalation

Further development

- Define RP2DS
- Single agent expansion cohorts in KRAS^{G12C} NSCLC and pancreatic cancer (RAS inhibitor naïve +/- failure)
- Combinations in KRAS^{G12C} NSCLC & CRC

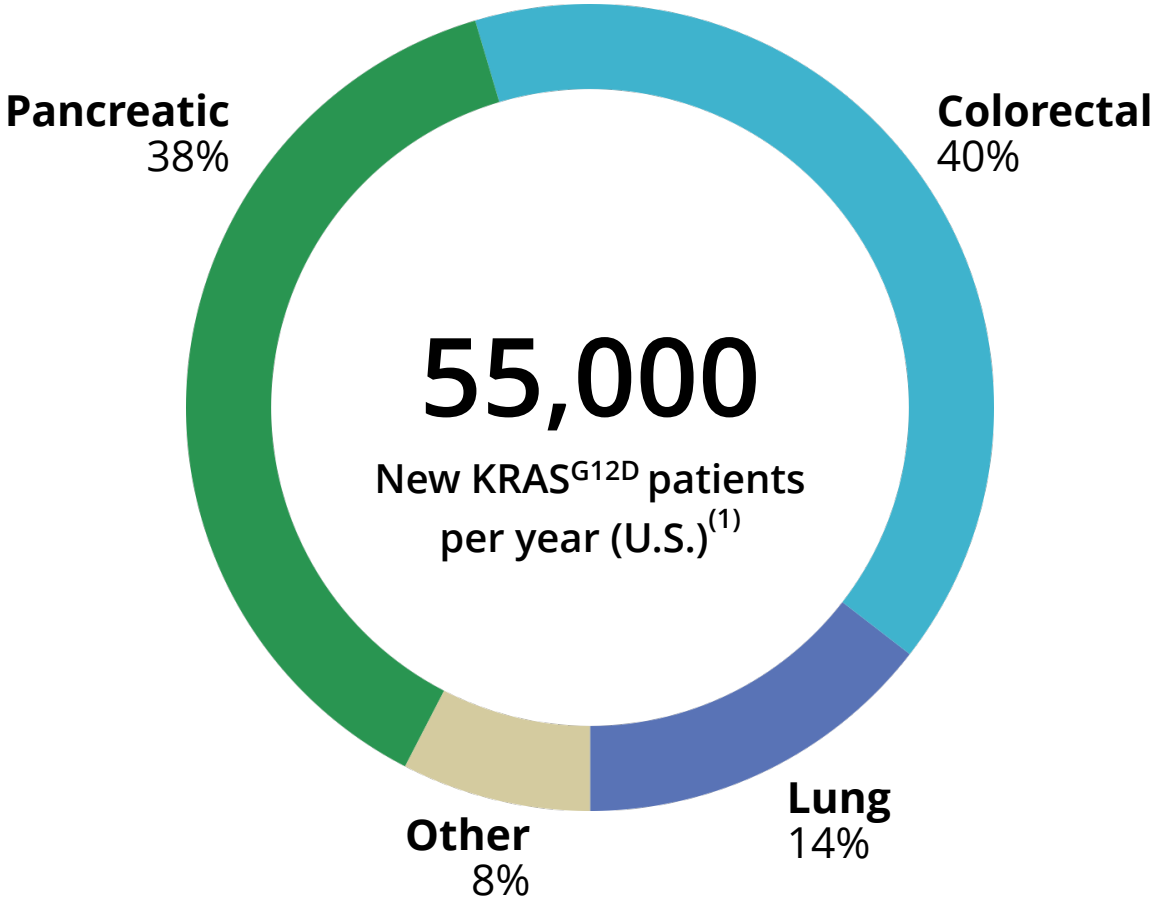
Aims

Preliminary evidence of superior activity against KRAS^{G12C} tumors^

^See Milestones table

RP2DS = Recommended Phase 2 dose and schedule
MTD = maximum tolerated dose
NSCLC = non-small cell lung cancer; CRC = colorectal cancer

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



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

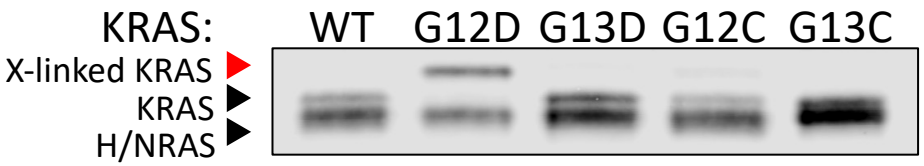
- Favorable *in vivo* oral bioavailability and clearance for effective target coverage in KRAS^{G12D}-addicted cancer cells

(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

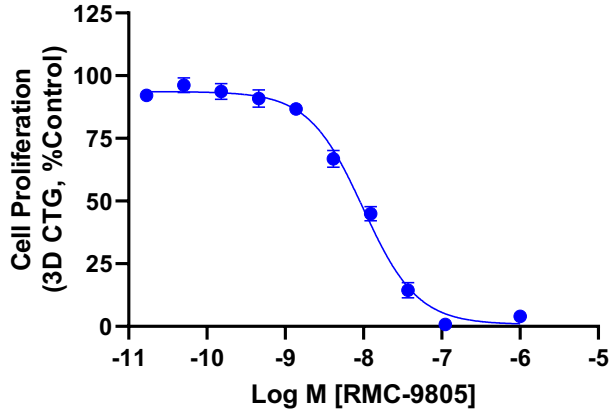
Characterization above is based on RVMD preclinical research

RMC-9805: Selective, Covalent and Orally Active with Sustained Inhibition of KRAS^{G12D} *in Vivo*

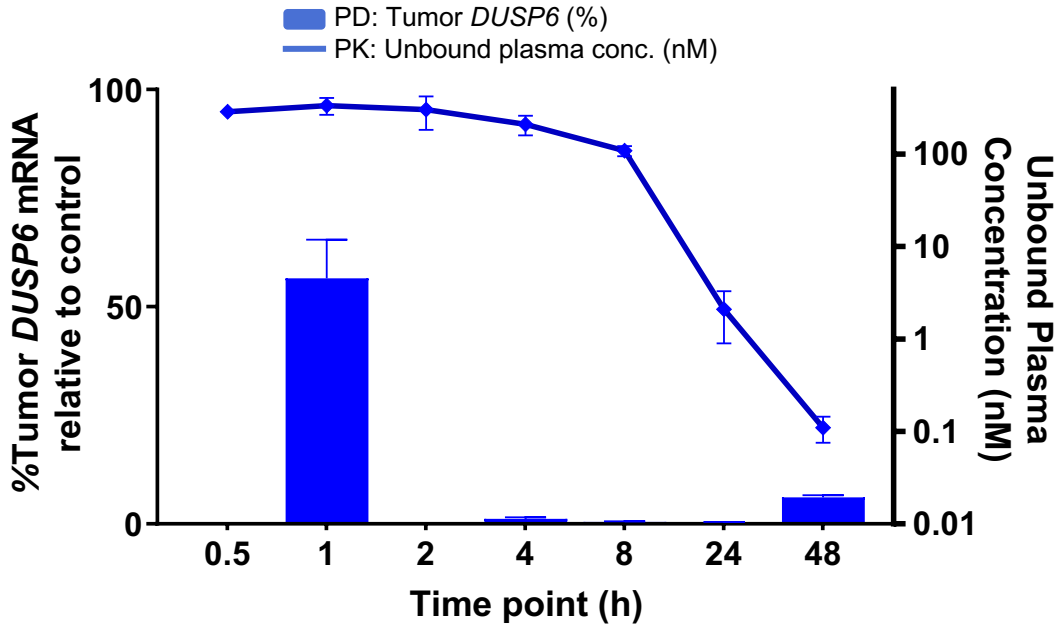
Selective Covalent Modification of KRAS^{G12D}



Potent Inhibition of KRAS^{G12D} Cancer Cell Growth



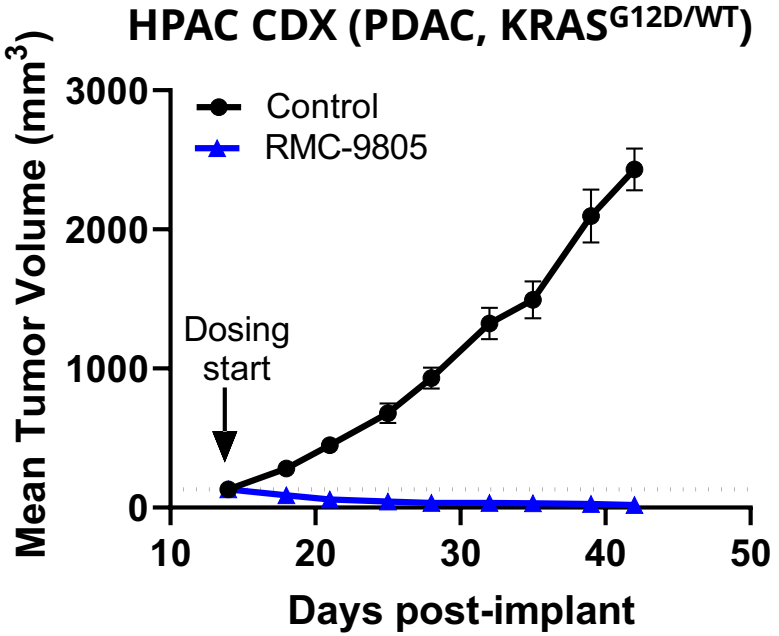
Single Dose PK/PD HPAC CDX (PDAC, KRAS$^{G12D/WT}$)

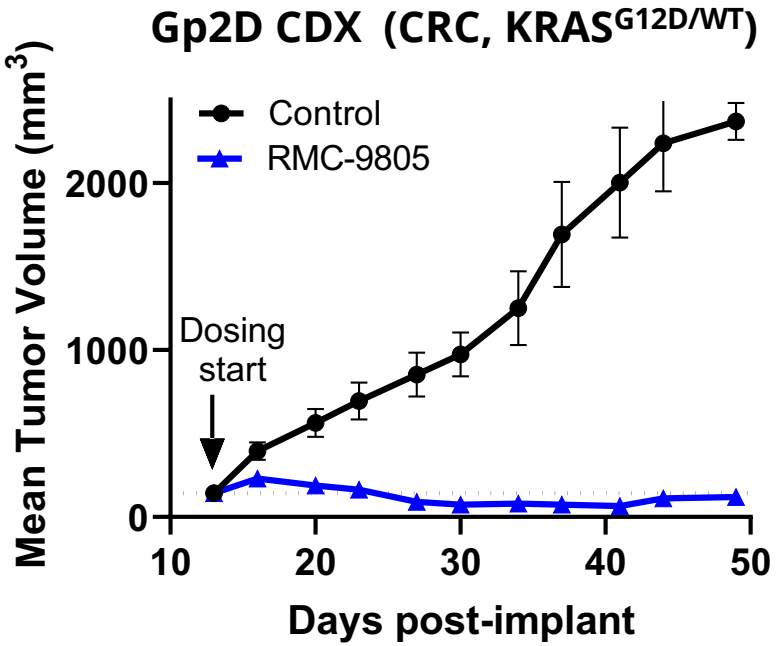


RVMD preclinical research, as of 10/13/2021
RMC-9805 dosed at 100 mg/kg po
PDAC = pancreatic ductal adenocarcinoma

RMC-9805: Tumor Regressions in Models of KRAS^{G12D} Cancers

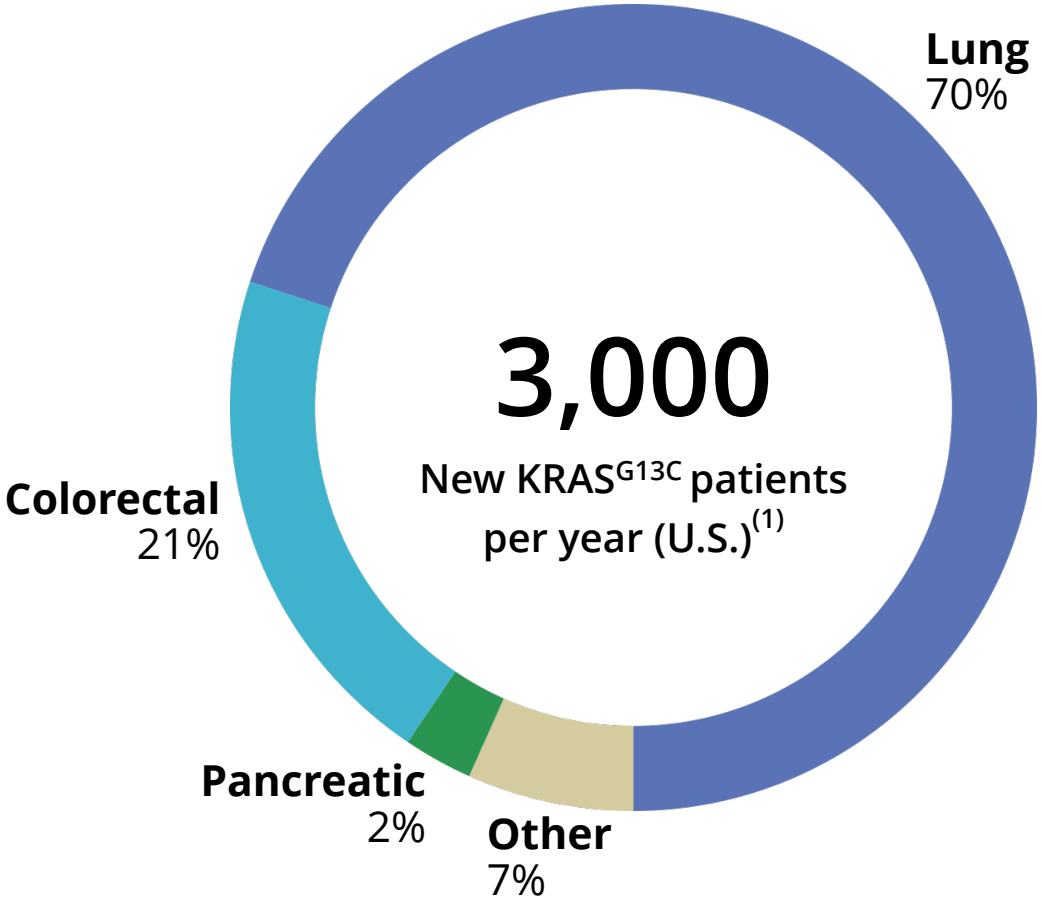
- First-in-class mutant-selective covalent inhibitor of KRAS^{G12D}
- Deep and durable anti-tumor responses *in vivo* in pancreatic and colorectal cancer models
- Oral dosing, well tolerated





RVMD preclinical research, as of 11/05/2021
RMC-9805 dosed at 100 mg/kg pd qd; n = 10/group (top), 6/group (bottom); PDAC = pancreatic ductal adenocarcinoma; CRC = colorectal cancer

RMC-8839: First-in-Class Mutant-Selective RAS(ON) Inhibitor for KRAS^{G13C} Cancers



Highly Potent and Selective RAS(ON) Inhibitor

- Highly active against KRAS^{G13C}
- 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^{G13C} lung cancers

Attractive PK/ADME Profile

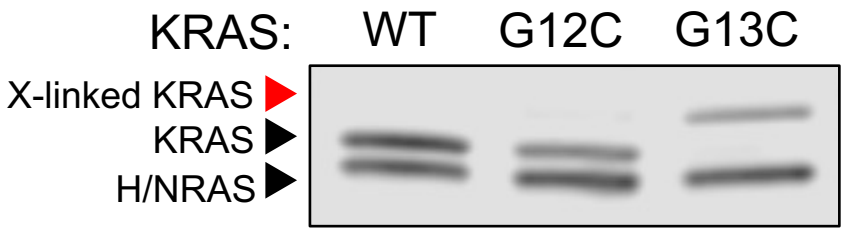
- Favorable *in vivo* oral bioavailability and clearance for effective target coverage in KRAS^{G13C}-addicted cancer cells

(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

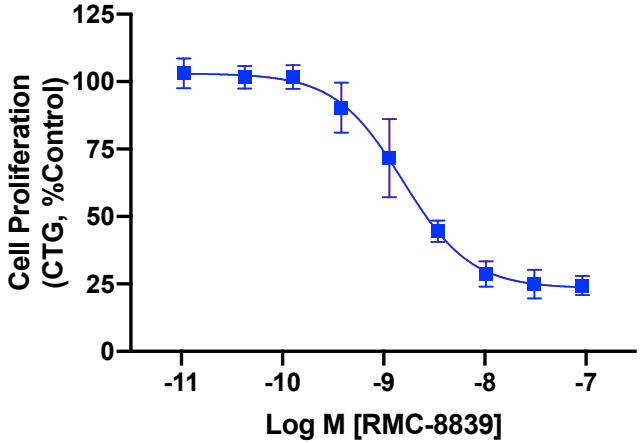
Characterization above is based on RVMD preclinical research

RMC-8839: Selective, Covalent and Orally Active with Sustained Inhibition of KRAS^{G13C} *in Vivo*

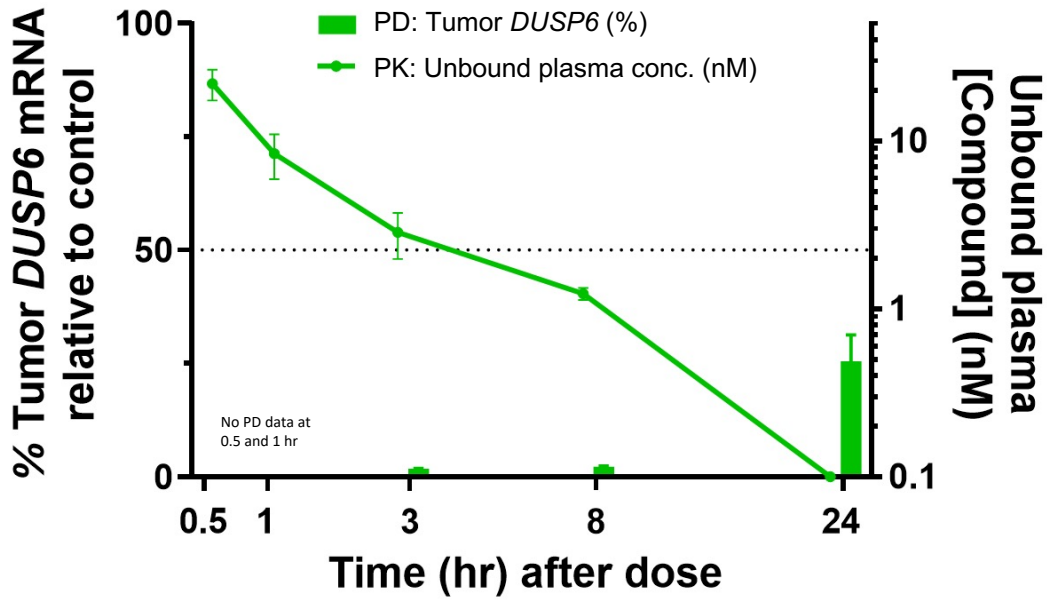
Selective Covalent Modification of KRAS^{G13C}



Potent Inhibition of KRAS^{G13C} Cancer Cell Growth



Single Dose PK/PD NCI-H1734 (NSCLC CDX, KRAS^{G13C})

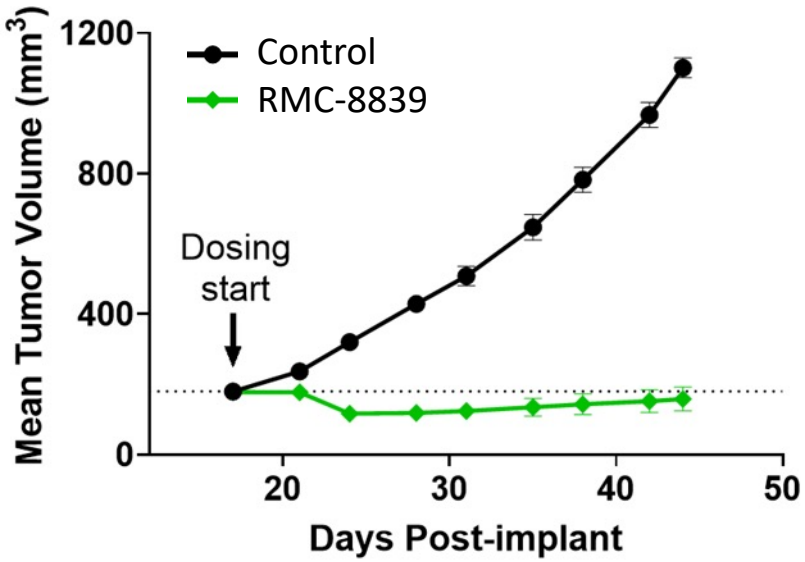


RVMD preclinical research, as of 12/22/2021
RMC-8839 dosed at 100 mg/kg po
NSCLC = non-small cell lung cancer

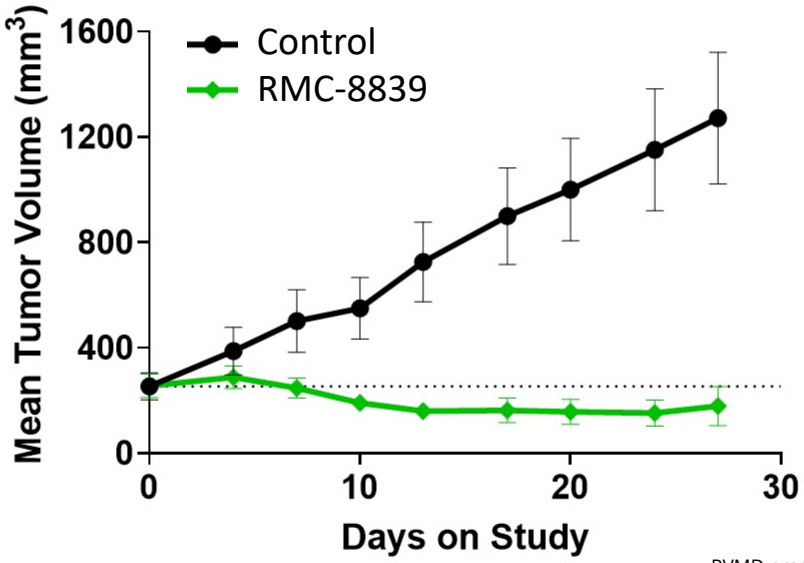
RMC-8839: Tumor Regressions in Models of KRAS^{G13C} Cancers

- First-in-class mutant-selective covalent inhibitor of KRAS^{G13C}
- Deep anti-tumor responses *in vivo* in non-small cell lung cancer models
- Oral dosing, well tolerated

NCI-H1734 CDX (NSCLC, KRAS$^{G13C/WT}$)



ST2822B PDX (NSCLC, KRAS$^{G13C/WT}$)



RVMD preclinical research, as of 11/17/2021
RMC-8839 dosed at 100 mg/kg pd qd; n = 5/group; NSCLC = Non-small cell lung cancer

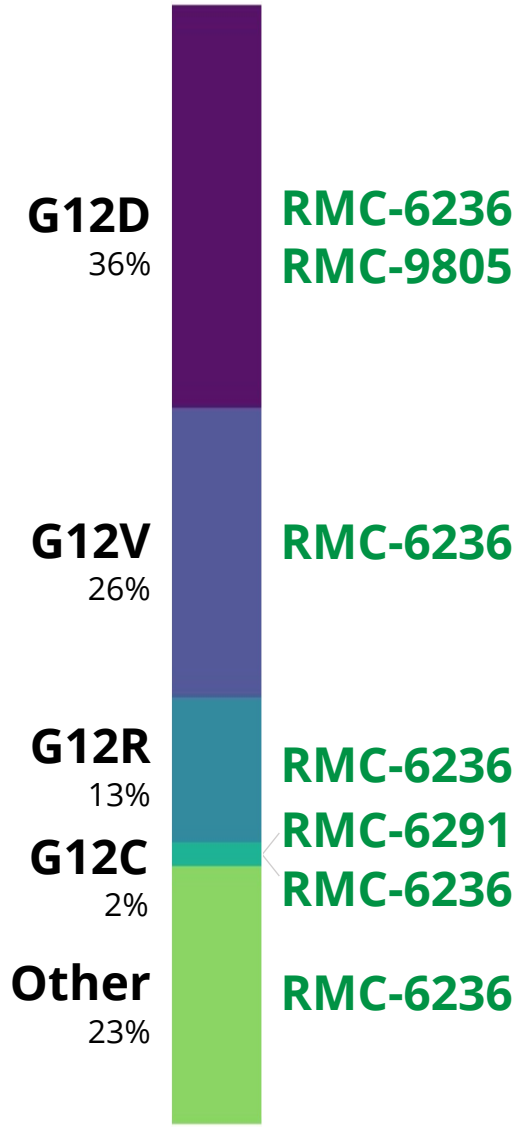
On Target to Outsmart
Pancreatic Cancer

Devastating disease
>90% driven by KRAS mutations

49,000

New KRAS^{MUTANT} pancreatic cancer patients per year (US)[1]

Dismal survival rates
No approved targeted therapies



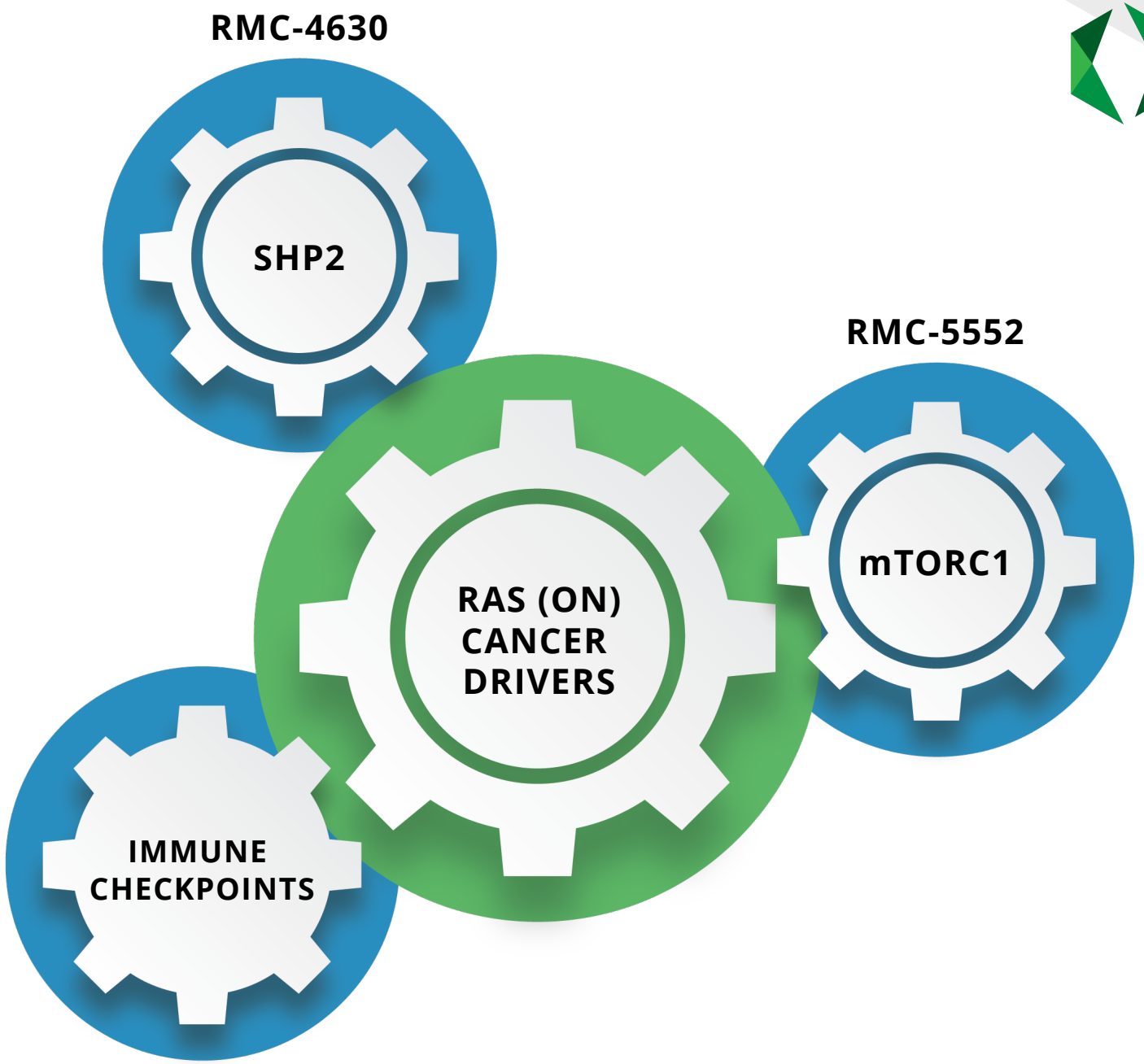
Our development-stage RAS(ON) Inhibitors

- Inhibit >90% of pancreatic cancer drivers in cancer models[1]
- Exhibit strong anti-tumor activity in preclinical models of pancreatic cancer

(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)

RAS Companion Inhibitors

Suppress Cooperating Targets and Pathways that Sustain RAS-Addicted Cancers



RMC-4630: Ongoing and Planned Clinical Combination Studies

STUDY	SPONSOR	COMBINED WITH	INDICATION(S)	STATUS
CodeBreaK 101c (U.S.)	Amgen	sotorasib	2L+ $KRAS^{G12C}$ solid tumors	Ongoing (Phase 1b/2)
RMC-4630-03 (Global)	RevMed	sotorasib	2L+ $KRAS^{G12C}$ NSCLC	Ongoing (Phase 2)
TCD16210 (Global)	Sanofi	adagrasib	2L+ $KRAS^{G12C}$ NSCLC	In preparation (Phase 1/2)
TBD	RevMed	RMC-6291	$KRAS^{G12C}$ TBD	Planning
TCD16210 (Global)	Sanofi	pembrolizumab	1L $PDL1^{+}$ NSCLC	Ongoing (Phase 1/2)

RMC-4630/SAR442720 under 2018 partnership

NSCLC = non-small cell lung cancer

RMC-4630: Clinical Priorities to Pursue Best-in-Class Combination Activity in KRAS^{G12C} Tumors

Activities

2022

- Complete enrollment in RMC-4630-03 and preliminary evaluation^

Further development

- Registration study in combination with KRAS^{G12C}(OFF) inhibitor in KRAS^{G12C} NSCLC
- Combination study(ies) with KRAS^{G12C}(OFF) inhibitor in KRAS^{G12C} CRC and/or pancreatic cancer
- Combination study(ies) with RMC-6291

Aims

Evidence of clinical benefit as RAS Companion Inhibitor against KRAS^{G12C} NSCLC^
Evidence of clinical benefit as a RAS Companion Inhibitor against additional KRAS^{G12C} tumors

^See Milestones table

RMC-4630/SAR442720
under 2018 partnership

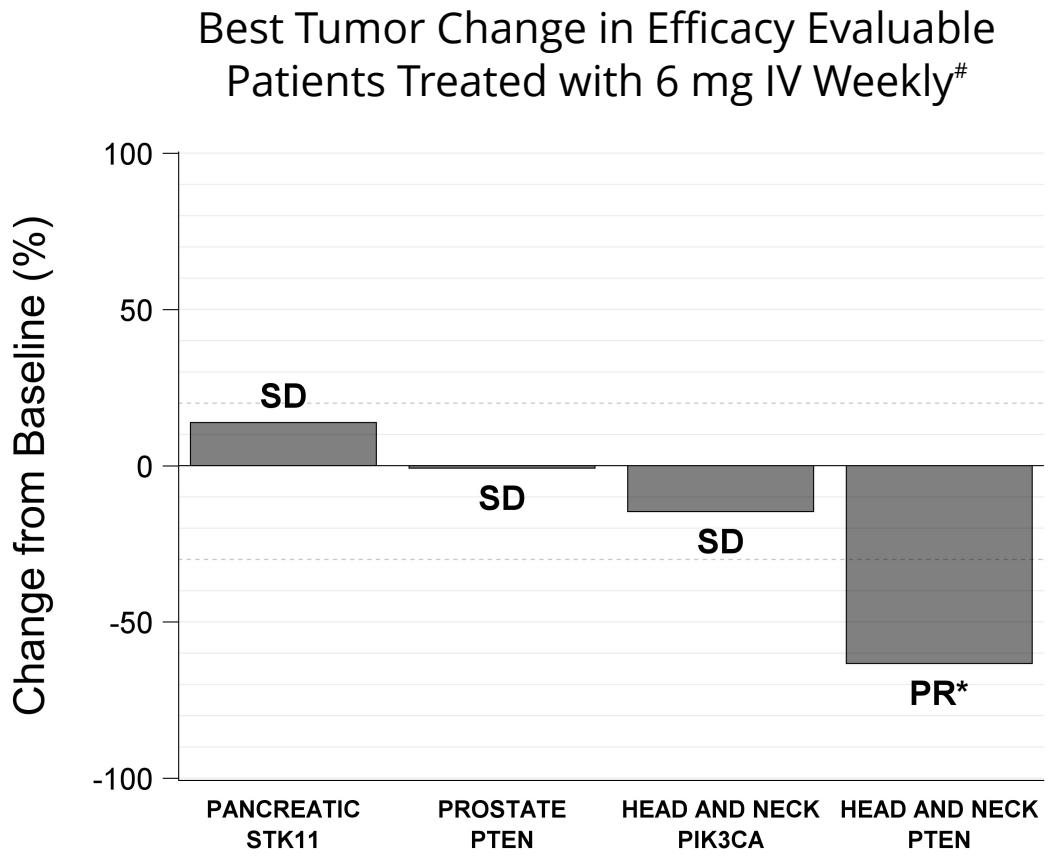
NSCLC = non-small cell lung cancer
CRC = colorectal cancer

RMC-5552 Clinical Opportunity

- Potent, selective inhibitor of hyperactivated mTORC1 to reactivate the tumor suppressor 4EBP1
- Designed for combination with RAS(ON) inhibitors in patients with cancers harboring RAS/mTOR pathway co-mutations(1)
  - >30,000 new patients per year across lung, colorectal and pancreatic cancers (U.S.)(2)
- Single agent Phase 1b dose escalation underway, focused on tumor genotypes linked to hyperactivated mTORC1 signaling

(1) mTOR pathway co-mutations include genetic changes with likely oncogenic activity in one or more of PIK3CA, PTEN, TSC1, TSC2, STK11, and/or mTOR
(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

Preliminary Evidence of Clinical Activity



Preliminary assessments suggest mucositis as the major dose-limiting toxicity. 6 mg weekly was well tolerated. Further enrollment at doses above 6 mg is ongoing to define the RP2DS; *Patient received one dose of 12 mg, followed by weekly doses of 6 mg. Data as of 01/07/2022.

RMC-5552: Clinical Priorities to Pursue Best-in-Class Combination Activity in RAS^{MUTANT}/mTORC1-Activated Tumors

Activities

2022

- Complete single agent dose-escalation
- Initiate single agent expansion cohorts in select tumors with mTOR pathway mutations

Further development

- Define single agent RP2DS
- Complete single agent expansion cohorts
- Combinations with RAS(ON) inhibitors from our portfolio in RAS^{MUTANT} tumors with mTOR pathway co-mutations

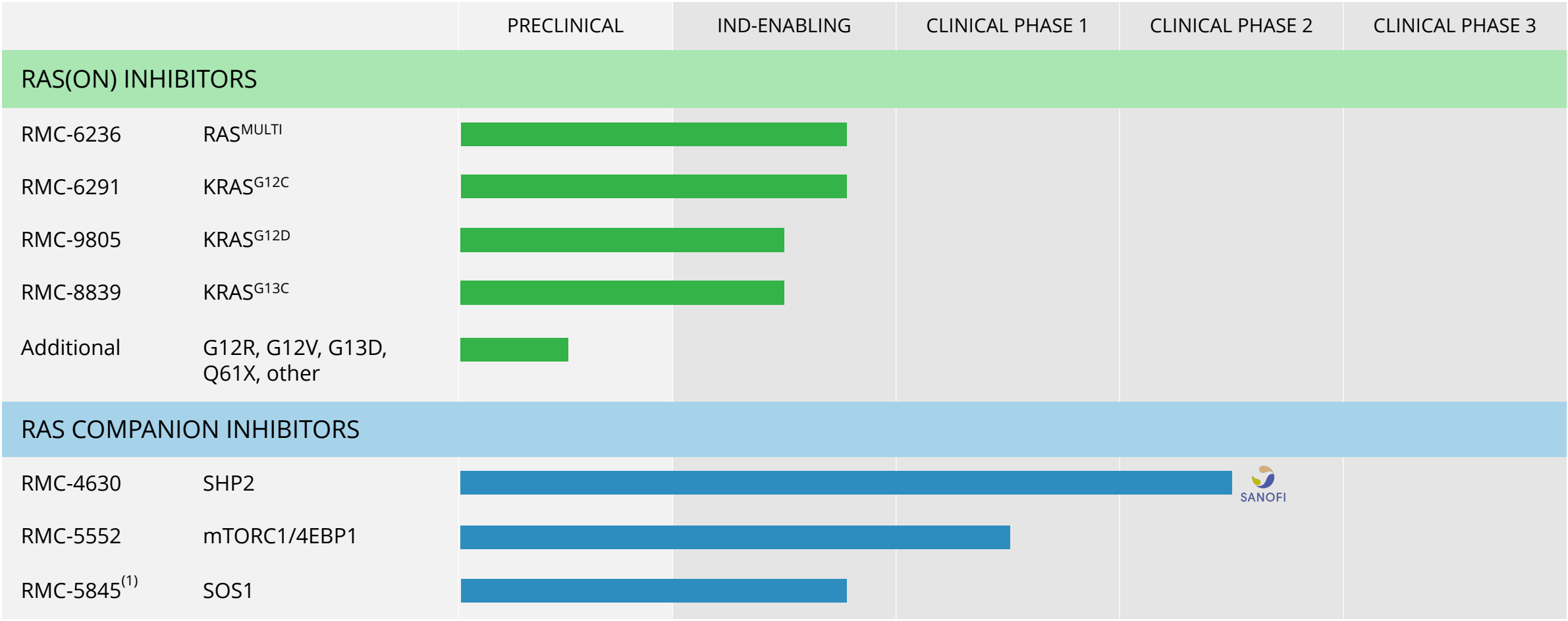
Aims

Additional evidence of single agent activity against tumors with mTOR pathway mutations^

^See Milestones table

RP2DS = Recommended Phase 2 dose and schedule

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



(1) IND-ready

Anticipated Milestones

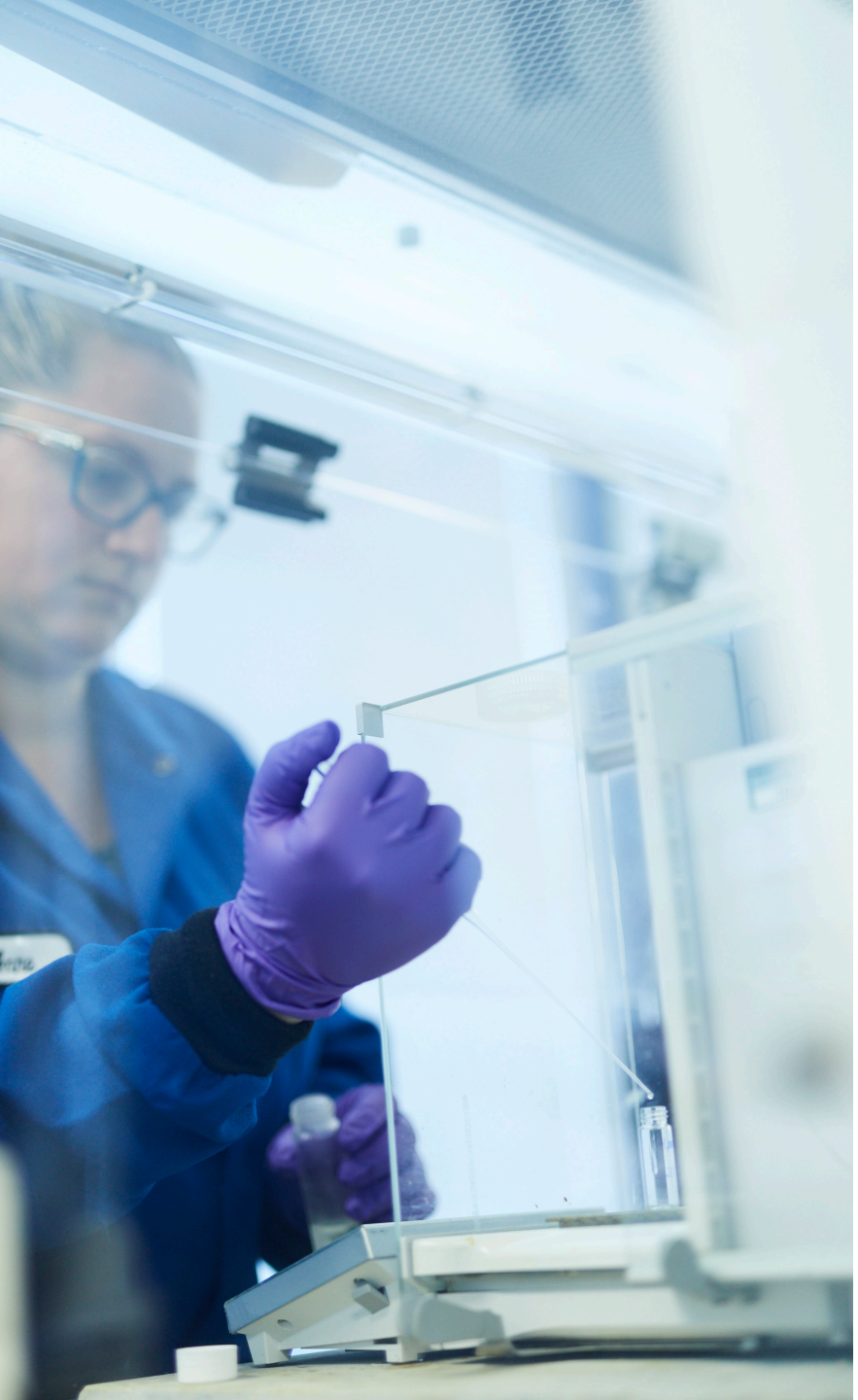
PROGRAM	MILESTONE (EXPECTED TIMING)
RAS(ON) INHIBITORS	
RMC-6236 (RAS^{MULTI})	Submit IND (1H22); Provide evidence of first-in-class single agent activity (2023)
RMC-6291 ($KRAS^{G12C}$)	Submit IND (1H22); Provide preliminary evidence of superior activity (2023)
RMC-9805 ($KRAS^{G12D}$)	Submit IND (1H23)
RMC-8839 ($KRAS^{G13C}$)	Submit IND (2H23)
Additional RAS^{MUTANT}-Selective Inhibitor	Nominate development candidate (2H22)
RAS COMPANION INHIBITORS	
RMC-4630 (SHP2)	Complete enrollment in RMC-4630-03 (2H22); Provide preliminary (2H22) and additional (2023) evidence of clinical benefit as a RAS Companion Inhibitor from RMC-4630-03
RMC-5552 (mTORC1/4EBP1)	Provide additional evidence of single agent activity (2023)

Financial Information

Financial Position	
Cash, cash equivalents and marketable securities @ 9/30/2021	$608.7 million[(1)]

2021 Financial Guidance
2021 GAAP net loss of $170 million to $190 million[(2)]

(1) Expected to fund planned operations to end of 2023
(2) Includes non-cash stock-based compensation expense of approximately $20 million



REVOLUTION MEDICINES

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

Targeted *RAS(ON) Inhibitors* with compelling preclinical profiles expected to begin entering clinic in 2022

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(K,L,R,P), HRAS mutations of known/likely function, 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.
  - 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
- $KRAS^{G12X}$ includes $KRAS^{G12D}$, $KRAS^{G12V}$, $KRAS^{G12R}$ and $KRAS^{G12C}$
- Mouse tumor responses on slides 9 and 14 assigned according to mRECIST (modified from Gao et al. Nat Med. 2015):
  - mPD = progressive disease; mSD = stable disease; mPR = partial response; mCR = complete response
- Kaplan-Meier progression on slide 10 defined as tumor doubling from baseline over 28 days:
  - $KRAS^{G12X}$ Tumors, where X = D,V,C, A or R: n = 207
  - RAS Pathway Mutant Tumors includes $KRAS^{G12X}$ and other RAS and RAS pathway mutant tumors: $KRAS^{G13C}$, $KRAS^{G13D}$, $KRAS^{K117N}$, $KRAS^{Q61H}$, $NF1^{LOF}$, $PTPN11^{E76K\ or\ G503V}$, $BRAF^{Class\ 3\text{-}mutant}$, and $KRAS^{WT\text{-}Amp}$: n = 332
- PDX = patient-derived xenograft; CDX = cell line-derived xenograft