Targeting the genetic and immunological drivers of cancer
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August 9, 2019
Mirati Therapeutics
Answering Cancer’s Challenges with a Targeted Approach

Who We Are:

- A Team of Knowledgeable and Experienced Oncology Drug Developers with a Track Record of Success in Rapidly Developing Multiple Therapies from Preclinical Development to the Market
- Applying Proven Approaches to the Development of Sitravatinib and MRTX849

Where We Are Focused:

- Addressing Areas of Unmet Need for Large Patient Populations
  - Treating cancer progression after checkpoint inhibitor therapy
  - Targeting KRAS mutations across multiple tumor types
- Utilizing our Knowledge to Expedite Development
  - From Identifying Lead to IND for MRTX849 in 9 months

Oncology Experience

- Ibrance (palbociclib)
- Xalkori (crizotinib)
- Sutent (sunitinib malate)
- Inlyta (axitinib)

3
## Mirati’s Development Pipeline Has Multiple Near-Term Milestones

<table>
<thead>
<tr>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase 1/1b</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Next Milestone</th>
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<tbody>
<tr>
<td><strong>Immuno-oncology</strong>&lt;br&gt;Sitravatinib + Nivolumab</td>
<td></td>
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<tr>
<td>NSCLC</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Interim Analysis Q4 2020 Final Analysis Q4 2021</td>
</tr>
<tr>
<td>Bladder</td>
<td></td>
<td></td>
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<td></td>
<td>Clinical Data Update Q4 2019</td>
</tr>
<tr>
<td>MOA Trials&lt;br&gt;RCC, Head &amp; Neck</td>
<td></td>
<td></td>
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<td>Mechanism of Action Clinical Data Q4 2019</td>
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<tr>
<td><strong>Immuno-oncology</strong>&lt;br&gt;Sitravatinib + Tislelizumab (anti-PD-1)</td>
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<tr>
<td>NSCLC, RCC, HCC, Gastric, Ovarian, other</td>
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<td></td>
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<td>Early Proof of Concept Clinical Data Q4 2019</td>
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<tr>
<td><strong>Targeted Single Agent</strong>&lt;br&gt;CBL mutations</td>
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<tr>
<td>NSCLC Melanoma</td>
<td></td>
<td></td>
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<td></td>
<td>Clinical Data Update Q4 2019</td>
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<tr>
<td><strong>KRAS</strong>&lt;br&gt;Oral KRAS Inhibitors</td>
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<tr>
<td>MRTX849&lt;br&gt;G12C Inhibitor</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Clinical Data Update Q4 2019</td>
</tr>
<tr>
<td>NSCLC CRC Pancreatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Clinical Candidate Selection 2019</td>
</tr>
<tr>
<td>G12D Inhibitor</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRC, NSCLC, Pancreatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**NSCLC** = non-small cell lung cancer; **CRC** = colorectal cancer; **RCC** = renal cell cancer; **HCC** = hepatocellular cancer; **IND** = Investigational New Drug application; **Tislelizumab** (BeiGene’s anti-PD-1) **MOA** = Mechanism of Action
Immuno-Oncology Combinations
Sitravatinib + Checkpoint Inhibitors
Sitravatinib in the Tumor Microenvironment Aims to Restore Immune Responses Through Inhibition of Immunosuppressive Signaling

**Tyro, Axl, Mer**
- Macrophages shift to Type 1 resulting in production of immune stimulating cytokines
- Enhances innate and adaptive immune response

**VEGFR2, KIT + Tyro, Axl, Mer**
Increase:
- Dendritic cell dependent antigen presentation
- NK cell response
- T cell trafficking

**VEGFR2 & KIT**
- Reduction in Tregs and MDSCs
- Enhance CD8+ T-cell response

**Pre-Treatment**

**Post-Treatment**

---

Garton et al., Anti-KIT Monoclonal Antibody Treatment Enhances the Antitumor Activity of Immune Checkpoint Inhibitors by Reversing Tumor-Induced Immunosuppression. Mol Cancer Ther, 2017. 16(4)
Currently Enrolling Phase 3 Program in 2nd Line NSCLC
Sitravatinib + Nivolumab Randomized Phase 3 Trial Design

Two Opportunities for Approval in 2nd Line NSCLC:
- Interim Analysis: ORR, for Accelerated Approval (Q4’20)
- Primary Analysis: OS, for Full Approval (Q4’21)

Key Inclusion/Exclusion Criteria:
- Advanced, Non-Squamous NSCLC
- Progression on or following first line combination of PD-(L)1 inhibitor plus chemotherapy
- Excludes patients with known driver mutations

Endpoints:
- Interim: ORR (for potential Subpart H accelerated approval)
- Primary: OS (for potential full approval)
- Secondary: PFS, duration of response, safety, tolerability

Projected Phase 3 Timing:
- Study Initiation: Currently Enrolling
- Interim ORR Analysis: Q4 2020
- Primary OS Analysis: Q4 2021

ORR = Overall Response Rate; DOR = Duration of Response; OS = Overall Survival; PFS = Progression-Free Survival; NSCLC = non-small cell lung cancer
2nd Line NSCLC Represents a Significant Market Opportunity for Sitravatinib in Checkpoint Refractory Patients

Keytruda + Chemo 1st Line
*Has Created an Opportunity in 2nd Line*

<table>
<thead>
<tr>
<th>No/Low PD-L1</th>
<th>High PD-L1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1L Keytruda + Chemo</td>
<td>Keytruda</td>
</tr>
<tr>
<td>2L Checkpoint Refractory (Docetaxel Standard of Care)</td>
<td>Platinum Doublet</td>
</tr>
<tr>
<td>3L</td>
<td>Docetaxel</td>
</tr>
</tbody>
</table>

2nd Line NSCLC is a Significant Market Opportunity

2017 NSCLC Market - US Sales

- 1st Line = $4.4B
- 2nd Line = $2.8B

1st Line Treated Patients (US): 101,000
2nd Line Treated Patients (US): 61,000

Sources: EvaluatePharma, CancerMPact Patient Metrics and Kantar Health analysis
# Sitravatinib + Checkpoint Inhibitors

## Docetaxel: An Efficacy Comparator Used in Prior 2nd Line NSCLC Trials

### Advanced NSCLC

#### 2nd Line or Subsequent Therapy

<table>
<thead>
<tr>
<th>Trial</th>
<th>Compound</th>
<th>Pts</th>
<th>ORR, %</th>
<th>DOR, Mo.</th>
<th>PFS, Mo. (Range)</th>
<th>SD, %</th>
<th>CBR, % (CR+PR+SD)</th>
<th>OS, Mo. (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Checkpoint Naïve Patients</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CheckMate 057[1]</td>
<td>nivolumab</td>
<td>n=292</td>
<td>19%</td>
<td>17.2</td>
<td>2.3 (2.2-3.3)</td>
<td>25%</td>
<td>44%</td>
<td>12.2 (9.7-15.0)</td>
</tr>
<tr>
<td></td>
<td>docetaxel</td>
<td>n=290</td>
<td>12%</td>
<td>5.6</td>
<td>4.2 (3.5-4.9)</td>
<td>42%</td>
<td>54%</td>
<td>9.4 (8.1-10.7)</td>
</tr>
<tr>
<td>OAK[2]</td>
<td>atezolizumab</td>
<td>n=425</td>
<td>14%</td>
<td>16.3</td>
<td>2.8 (2.6-3.0)</td>
<td>35%</td>
<td>49%</td>
<td>13.8 (11.8-15.7)</td>
</tr>
<tr>
<td></td>
<td>docetaxel</td>
<td>n=425</td>
<td>13%</td>
<td>6.2</td>
<td>4.0 (3.3-4.2)</td>
<td>42%</td>
<td>55%</td>
<td>9.6 (8.6-11.2)</td>
</tr>
<tr>
<td><strong>Checkpoint Refractory Patients</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>KEYNOTE 010[3]</td>
<td>pembrolizumab</td>
<td>n=345</td>
<td>18%</td>
<td>Not yet reported</td>
<td>3.9 (3.1-4.1)</td>
<td>10.4 (9.4-11.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>docetaxel</td>
<td>n=343</td>
<td>9%</td>
<td>6.1</td>
<td>4.0 (3.1-4.2)</td>
<td></td>
<td></td>
<td>8.5 (7.5-9.8)</td>
</tr>
<tr>
<td><strong>MRTX-500[4]</strong></td>
<td>sitravatinib + nivolumab</td>
<td>n=56</td>
<td>20%</td>
<td>9.2*</td>
<td>6.8*</td>
<td>55%</td>
<td>75%</td>
<td>15.1*</td>
</tr>
</tbody>
</table>

Data presented is from separate studies and does not reflect results that might have been obtained from head-to-head studies. Results from Mirati’s planned Phase 3 trial comparing sitravatinib + nivolumab to docetaxel may differ materially from prior studies presented.

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4. Mirati ESMO 2018 Congress

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**ORR** = Overall Response Rate  
**DOR** = Duration of Response  
**CBR** = Clinical Benefit Rate (CR + PR + SD>14 weeks)  
**SoC** = Standard of Care  
**CPI** = Checkpoint Inhibitor Therapy  
* Preliminary Kaplan-Meier Estimate
Neo-Adjuvant Studies in RCC and HNSCC Present Near-Term Indication Expansion Opportunities for Sitravatinib

Presurgical Settings with Accessible Tumors

- Tissue biopsies following treatment with Sitravatinib alone and in combination
- Pharmacodynamic data from Tumor Micro Environment
- Evaluate clinical activity in neo-adjuvant setting

Timing

- Studies initiated in H2 2018
- Initial data planned for Q4 2019

Study 1: Renal Cell Carcinoma
MD Anderson Cancer Center

Study 2: Head and Neck Cancer
Princess Margaret – Toronto

RCC = renal cell cancer; HNSCC = head and neck squamous cell carcinoma
# Sitravatinib Has Opportunity for Broad Indication Expansion Across Multiple Tumor Types

<table>
<thead>
<tr>
<th>Sponsor</th>
<th>Phase 1/1b</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Milestones</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NSCLC</strong></td>
<td>2nd line NSCLC checkpoint refractory sitravatinib + nivolumab vs. docetaxel</td>
<td>2nd/3rd line NSCLC checkpoint refractory sitravatinib + tislelizumab</td>
<td>POC Data H2 2019</td>
<td>Currently Enrolling</td>
</tr>
<tr>
<td>BeiGene</td>
<td>1st line NSCLC PD-L1 High sitravatinib + tislelizumab</td>
<td></td>
<td>POC Data H1 2020</td>
<td></td>
</tr>
</tbody>
</table>

**HCC and RCC**
- Pursue indications with strong clinical rationale for sitravatinib

<table>
<thead>
<tr>
<th>Sponsor</th>
<th>Phase 1/1b</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Milestones</th>
</tr>
</thead>
<tbody>
<tr>
<td>BeiGene</td>
<td>1st line HCC checkpoint naïve sitravatinib + tislelizumab; Sitravatinib alone</td>
<td></td>
<td>POC Data H1 2020</td>
<td></td>
</tr>
<tr>
<td>BeiGene</td>
<td>1st/2nd line RCC checkpoint naïve &amp; refractory sitravatinib + tislelizumab</td>
<td></td>
<td>POC Data H2 2019</td>
<td></td>
</tr>
</tbody>
</table>

**Additional Indications**
- Expand to additional indications with opportunity to extend treatment options

<table>
<thead>
<tr>
<th>Sponsor</th>
<th>Phase 1/1b</th>
<th>Phase 2</th>
<th>Phase 3</th>
<th>Milestones</th>
</tr>
</thead>
<tbody>
<tr>
<td>BeiGene</td>
<td>2nd/3rd line Bladder checkpoint refractory sitravatinib + nivolumab</td>
<td></td>
<td>POC Data Q4 2019</td>
<td></td>
</tr>
<tr>
<td>BeiGene</td>
<td>RCC and HNSCC (pre-surgical) sitravatinib + nivolumab</td>
<td></td>
<td>POC Data Q4 2019</td>
<td></td>
</tr>
<tr>
<td>BeiGene</td>
<td>Ovarian cancer following platinum sitravatinib + tislelizumab</td>
<td></td>
<td>POC Data H2 2019</td>
<td></td>
</tr>
<tr>
<td>BeiGene</td>
<td>2nd/3rd line Gastric following chemotherapy sitravatinib + tislelizumab</td>
<td></td>
<td>POC Data H1 2020</td>
<td></td>
</tr>
</tbody>
</table>

POC = proof of concept; NSCLC = non-small cell lung cancer; RCC = renal cell cancer; HCC = hepatocellular carcinoma; HNSCC = head and neck squamous cell carcinoma
Sitravatinib Summary: Multiple Opportunities for I/O Combinations Across A Broad Range of Tumors

Positive Interim Phase 2 Efficacy Data in Checkpoint Refractory NSCLC\(^1\) (n=56)

75% 42/56 demonstrated Clinical Benefit\(^{(2)}\)

20% 11/56 achieved a Confirmed Partial or Complete Response

Preliminary Kaplan-Meier estimates\(^{(3)}\)

<table>
<thead>
<tr>
<th>Median DoR:</th>
<th>Median PFS:</th>
<th>Median OS:</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.2 months</td>
<td>6.8 months</td>
<td>15.1 months</td>
</tr>
</tbody>
</table>

Registration Path for 2\(^{nd}\) Line NSCLC: Phase 3 Randomized Trial Initiated in Q2 2019

- Sitravatinib + nivolumab vs. docetaxel in 2\(^{nd}\) line checkpoint refractory NSCLC
- Interim Analysis (ORR) for accelerated approval expected by year end 2020
- Primary Analysis (OS) for full approval by year end 2021

Phase 2 Clinical Trials in Other Indications

- **Checkpoint Refractory Bladder Cancer**: Initial Phase 2 data expected in Q4 2019
- **Neo-adjuvant/Mechanism of Action Studies in RCC and HNSCC**: Initial data expected in Q4 2019
- **BeiGene Phase 2 Trials**: Initial Phase 2 proof of concept data in NSCLC, HCC, RCC, Ovarian and Gastric expected in H2 2019 / H1 2020

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1. Data presented at ESMO 2018 Congress; Data cut-off: 27-Aug-2018; Response data per investigator assessment, response confirmations updated after data cut-off.
2. Clinical Benefit defined as Stable Disease (SD), Partial Response (PR) or Complete Response (CR)
3. DoR = Duration of Response; PFS = Progression Free Survival; OS = Overall Survival;
Targeted Oncology

Sitravatinib Single Agent: *CBL Loss of Function Mutations*
Sitravatinib Has a Unique Opportunity to Address CBL Loss-of-Function Mutations in Cancer

Loss-of-Function Mutations in CBL Result in Increased Target RTK Activation in Tumor Cells

Single Agent Sitravatinib Program Focused on CBL in NSCLC and Melanoma

- 1.5% of NSCLC and 3.5% of Melanoma
- Circulating tumor DNA assay (liquid biopsy) is now available to identify CBL loss of function mutations

Initial Clinical Activity\(^1\) in CBL Mutations is Encouraging

- 1/2 confirmed Partial Response in NSCLC
- 1/2 confirmed Partial Response in Melanoma

Single Agent Sitravatinib Has Been Well Tolerated with Manageable Side Effects

Clinical update expected in H2 2019 to define single agent registration pathway

1. Presented at ESMO 2018 Congress; data cut-off 4-Sep-2018
Targeted Oncology

MRTX849: KRAS G12C Inhibitor
KRAS is the Most Frequently Mutated Gene in Human Cancer

**Controlled KRAS Signaling**
- A key regulator of cell growth and survival

**Abnormal KRAS Signaling**
- Mutated KRAS activity is uncontrolled

**KRAS Signaling**
- KRAS is tightly controlled by growth factor receptor signaling

**Abnormal KRAS Signaling**
- KRAS activated independently of growth factor receptor signaling

Uncontrolled cell proliferation, survival, and growth
Mutant KRAS is an Oncogenic Tumor Driver That is Exclusive from Other Common Driver Mutations

Mutational Prevalence and Overlap in 1,357 Lung Adenocarcinomas¹

KRAS mutations occur early and do not overlap with other driver mutations

KRAS G12 (346)
KRAS G13 (37)
RET Fusion (20)
HER2 (44)
BRAF (65)
MET (47)
EGFR Exon 19 Deletion (102)
KRAS Q61 (18)
ROS1 (29)
ALK Fusion (42)
EGFR L858R (109)
HER2 (44)
RET Fusion (20)
KRAS G13 (37)
MET (47)
BRAF (65)
KRAS G12 (346)
KRAS Q61 (18)
ROS1 (29)
ALK Fusion (42)
EGFR L858R (109)

Mutant KRAS signaling is required for tumor cell survival

Sensitivity of Cancer Cell Lines to shRNA Knockdown of KRAS²

Sensitivity Score

Cancer Cell Lines

Wild Type KRAS
Mutant KRAS

2. McDonald et al, Project DRIVE: A Compendium of Cancer Dependencies and Synthetic Lethal Relationships Uncovered by Large-Scale, Deep RNAi Screening, Cell 2017
KRAS G12C Mutations Occur Frequently in Multiple Tumor Types

**KRAS G12C Mutation Frequency**

- **NSCLC adenocarcinoma**: 14%
- **Colorectal**: 4%
- **Pancreatic**: 2%

**Approx. US & EU Patients (2)**

<table>
<thead>
<tr>
<th>Tumor Type</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSCLC</td>
<td>44,400</td>
</tr>
<tr>
<td>Colorectal</td>
<td>19,600</td>
</tr>
<tr>
<td>Pancreatic</td>
<td>4,000</td>
</tr>
</tbody>
</table>

Total Addressable Pts: ~68,000


2. Mirati estimates based on epidemiology data reported in Globocan 2022 (accessed January 2019) and frequencies by mutation. Rounded to the nearest 100.
Addressing KRAS G12C+ Tumors is a Potential ~$7B Market Opportunity

Large Patient Population
US and EU Patients

<table>
<thead>
<tr>
<th>Condition</th>
<th>US and EU Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>KRAS G12C</td>
<td>68,000</td>
</tr>
<tr>
<td>ALK</td>
<td>10,000</td>
</tr>
<tr>
<td>RET</td>
<td>20,000</td>
</tr>
<tr>
<td>TRK</td>
<td>30,000</td>
</tr>
</tbody>
</table>

Significant Commercial Potential
Projected US and EU Markets for KRAS G12C

- **NSCLC** $4.9B
- **Colorectal** $2.2B
- **Pancreatic** $400M

~$7+ Billion Potential Market

1. Mirati estimates based on epidemiology data reported in Globocan 2022 (accessed January 2019) and frequencies by mutation; RET estimate does not include thyroid cancer
2. Mirati estimate, assuming revenue of $150,469 per US patient (net price of $16,719/mon and median duration of treatment of 9 months) and $90,281 per EU patient (40% discount to US)

Duration of treatment modeled from clinical observations with MEK/RAF combination therapy in BRAF-mutated NSCLC
Covalent KRAS G12C Inhibition is a Major Breakthrough in a Previously “Undruggable” Target

KRAS G12C Can Be Inhibited Covalently

1. KRAS G12C has a cysteine present in its inactive form

2. Binding to the cysteine opens an adjacent Switch II pocket

3. Inhibitor covalently binds to the cysteine and the induced Switch II pocket

4. KRAS G12C is irreversibly locked in the inactive state

KRAS tumor cell death
MRTX849 is a Potent and Selective KRAS G12C Inhibitor

**Potent**

Antiproliferation IC50s
MRTX849, 3D format

**Selective**

Cysteine Selectivity in H358 Cells
MRTX849 (10 µM)

# MRTX849: KRAS G12C Inhibitor
MRTX849: Preclinical Attributes Have Potential to be Best-in-Class

<table>
<thead>
<tr>
<th>Attribute</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potency</td>
<td>Low nM potency across multiple cellular models of KRAS G12C</td>
</tr>
<tr>
<td>Selectivity</td>
<td>1,000+ fold selective for KRAS G12C vs. KRASWT and other protein cysteines</td>
</tr>
<tr>
<td>Oral Bioavailability</td>
<td>Orally Bioavailable</td>
</tr>
<tr>
<td>Half Life</td>
<td>Long Half Life</td>
</tr>
<tr>
<td>Therapeutic Index</td>
<td>Wide Therapeutic Index</td>
</tr>
<tr>
<td>Volume of Distribution</td>
<td>Extensive Tissue Distribution</td>
</tr>
</tbody>
</table>

Preclinical projection of:
- >50% human bioavailability
- >20 hour human half life
- >10 fold safety margin
- Projected human volume of distribution exceeds 10 L/Kg
MRTX849 Has Shown Promising *In Vivo* Antitumor Activity in CDX and PDX Models as a Single Agent

*MRTX849: KRAS G12C Inhibitor – Single Agent*

Tumors did not recur at 100mg/kg despite cessation of treatment, indicating durable and complete responses.

### MIA PaCa-2 (CDX pancreatic)

- **MRTX849 oral administration**

<table>
<thead>
<tr>
<th>Study Day</th>
<th>Tumor volume (mm³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start Dosing</td>
<td>1000</td>
</tr>
<tr>
<td>10</td>
<td>Vehicle</td>
</tr>
<tr>
<td>20</td>
<td>3mg/kg</td>
</tr>
<tr>
<td>30</td>
<td>10mg/kg</td>
</tr>
<tr>
<td>40</td>
<td>30 &amp; 100mg/kg*</td>
</tr>
<tr>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

### LU2529 (PDX Lung)

- **MRTX849 oral administration**

<table>
<thead>
<tr>
<th>Study Day</th>
<th>Tumor volume (mm³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start Dosing</td>
<td>1000</td>
</tr>
<tr>
<td>10</td>
<td>Vehicle</td>
</tr>
<tr>
<td>20</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td></td>
</tr>
<tr>
<td>40</td>
<td></td>
</tr>
<tr>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

* Tumors did not recur at 100mg/kg despite cessation of treatment, indicating durable and complete responses
Currently Enrolling Phase 1/2 Clinical Trial of MRTX849
Phase 1/2 Trial Design: Single Agent

Phase 1: ACCELERATED DOSE ESCALATION
- 150mg QD
- Dose Level 2
- Dose Level 3
- Dose Level 4
- Intra-Patient Dose Escalation

Phase 2: SINGLE AGENT EXPANSION COHORTS
- NSCLC Cohort
- CRC Cohort
- “Basket” Cohort
- Other Tumors

Potential Registration Path:
- Single Arm Accelerated Approval
- Objective Response Rate

Phase 1 Initiated in January 2019
- Only enrolling patients with G12C+ tumors
- Single patient cohorts with intra-patient dose escalation; may expand at any dose level
- 100% dose escalations, continuing up to the MTD
- Objective: Identify Recommended Phase 2 Dose (RP2D)

Phase 2 Expansions Expected in H2 2019
- RP2D cohorts will be expanded, by tumor type
- Potential for single arm accelerated approval based upon ORR
- “Basket” cohort may provide support for tumor agnostic approval
In-Vivo Activity Suggests a Synergistic Effect of Combining MRTX849 with a PD-1 Inhibitor

KRAS G12C Inhibition Combined with Checkpoint Inhibitor Therapy Demonstrates Durable Complete Regressions In-Vivo

- KRAS G12C NSCLC tumors are primed for checkpoint inhibitor therapy with high levels of TMB and PD-L1 expression
- MRTX849 treatment stimulates anti-tumor immunity and recruits CD4+ & CD8+ T cells in KRAS G12C-mutant tumors.
- MRTX849 combined with a PD-1 inhibitor may provide another path to first line treatment.

1. Preliminary results from MRTX849 testing in CT26 Clone E3 G12C syngeneic mouse model
**In-Vivo Activity Also Suggests a Synergistic Effect of Combining MRTX849 with a SHP2 Inhibitor**

1. KYSE-410 xenograft model

**MRTX849 and SHP2 Inhibitor**

<table>
<thead>
<tr>
<th>Dosing</th>
<th>Tumor Volume (mm³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vehicle</td>
<td></td>
</tr>
<tr>
<td>MRTX849 100mg/kg</td>
<td></td>
</tr>
<tr>
<td>RMC-4550 30mg/kg</td>
<td></td>
</tr>
<tr>
<td>MRTX849 + RMC-4550</td>
<td></td>
</tr>
</tbody>
</table>

- **Dosing Stopped**

**MRTX849: KRAS G12C Inhibitor – Combinations**

- **July 2019:** Mirati announced a clinical collaboration with Novartis’ TNO155 (SHP-2 inhibitor)

**MRTX849 inhibits KRAS G12C which suppresses MAPK/ERK signaling and tumor growth**

**ERK inhibition hyperactivates EGFR and SHP2 signaling as an adaptive response to KRAS inhibition**

**Combinations of MRTX849 with EGFR or SHP2 inhibitors combat upstream reactivation of the RAS pathway**
Planned Initiation of Combination Clinical Trials in H2 2019

- Combination trials will enroll in parallel with single agent trials
- Combinations will be evaluated in patients with KRAS G12C+ tumors
- Opportunities for further patient selection will be evaluated

Signal Finding Phase 1/2 Trials
Active Combinations will be Advanced to Pivotal Trials
MRTX849 Summary: A Potential Best-in-Class KRAS G12C Inhibitor

Preclinical Attributes of MRTX849

- **Potent**: 1-20 nM potency in cellular assays
- **Selective**: 1,000 fold selective for KRAS G12C mutations over KRAS\(^{\text{wild type}}\)
- **Highly active in vivo**: complete durable regressions in KRAS G12C CDX and PDX models

- **Orally bioavailable**: preclinical projection of >50% human bioavailability
- **Long half life**: preclinical projection of >20 hour human half life maintains full inhibition of KRAS G12C

Significant Commercial Potential

**Large Patient Population**: KRAS G12C incidence is ~24,500 in the U.S. alone \(^{(1)}\)

**Large Commercial Opportunity**: Patient population is 3X larger than the $1.8B ALK market \(^{(2)}\)

Near-Term Milestones

- **Single-Agent**: Current Phase 1/2 Clinical Trial ongoing, with possibility of expansion into registrational
  - *Phase 1/2 Data Update Planned in Q4 2019*
- **Combination**: Could increase response rate and durability in resistant patient populations. Planned initiation of combination trials expected in H2 2019

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1. Mirati estimates based on epidemiology data reported in Globocan 2022 (accessed January 2019) and frequencies by mutation
2. ALK market: 2018 worldwide sales
Targeted Oncology

KRAS G12D Program
Mirati’s Next KRAS Program: A Direct Inhibitor of KRAS G12D

Prevalent in Tumors with High Unmet Need
KRAS G12D Mutational Frequencies

<table>
<thead>
<tr>
<th>Tumor Type</th>
<th>KRAS G12D Mutational Frequency</th>
<th>Total US/EU Pts (2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatic</td>
<td>35%</td>
<td>68,500</td>
</tr>
<tr>
<td>Colorectal</td>
<td>12%</td>
<td>58,900</td>
</tr>
<tr>
<td>Endometrial</td>
<td>5%</td>
<td>45,000</td>
</tr>
<tr>
<td>NSCLC adenocarcinoma</td>
<td>5%</td>
<td>9,800</td>
</tr>
</tbody>
</table>

Large Patient Population
US and EU Patients

2. Mirati estimates based on epidemiology data reported in Globocan 2022 (accessed January 2019) and frequencies by mutation; RET estimate does not include thyroid cancer. Round to the nearest 100.
Mirati Therapeutics Has Multiple Potential Value-Driving Catalysts in 2H 2019

<table>
<thead>
<tr>
<th>Sitravatinib</th>
<th>Initiating Phase 3 Registrational Trial in NSCLC</th>
<th>Sitravatinib + Nivolumab in Checkpoint Refractory NSCLC Phase 3 Trial Initiation</th>
<th>Currently Enrolling</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Extending Proof of Concept into Additional Indications</td>
<td>Sitravatinib + Nivolumab in Checkpoint Refractory Bladder Phase 2 Initial Proof of Concept Data</td>
<td>Q4 2019</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sitravatinib + Tislelizumab in NSCLC, RCC, Ovarian Phase 2 Initial Proof of Concept Data (BeiGene)</td>
<td>H2 2019</td>
</tr>
<tr>
<td></td>
<td>Neo-Adjuvant Studies</td>
<td>Sitravatinib + Nivolumab in Pre-Surgical RCC, HNSCC Mechanism of Action and Initial Clinical Neo-Adjuvant Data</td>
<td>Q4 2019</td>
</tr>
</tbody>
</table>

| MRTX849 | Potential Best-in-Class KRAS G12C Inhibitor | MRTX849 in KRAS G12C Tumors Phase 1/2 Early Proof of Concept Data | Q4 2019 |

| KRAS G12D Program | Expanding KRAS Franchise | KRAS G12D Program Lead Candidate Identification | Q4 2019 |

NSCLC = non-small cell lung cancer; CRC = colorectal cancer; RCC = renal cell cancer; HCC = hepatocellular cancer; HNSCC = head and neck squamous cell carcinoma; Tislelizumab (BeiGene’s anti PD-1)
Select Company Financials

<table>
<thead>
<tr>
<th>NASDAQ</th>
<th>MRTX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cash (Q2 19)*</td>
<td>$485.5M</td>
</tr>
<tr>
<td>Shares Outstanding**</td>
<td>49.0M</td>
</tr>
</tbody>
</table>

* Reported cash and investments as of March 31, 2019.

** As of June 30, 2019, includes 38.6 million shares of common stock and pre-funded warrants to purchase a total of 10.4 million shares of common stock. The pre-funded warrants have a per share exercise price of $0.001.
Targeting the genetic and immunological drivers of cancer

Corporate Presentation
August 2019
Back-up Slides
Phase 2 Trial of Sitravatinib + Nivolumab in Checkpoint Refractory NSCLC

MRTX-500 Trial - Design

**Key Inclusion Criteria:**
- Advanced/metastatic NSCLC
- Documented radiographic progression on prior checkpoint inhibitor therapy

**Key Exclusion Criteria:**
- No intervening therapy following progression on checkpoint inhibitor therapy
- No significant immune-related AEs with prior checkpoint therapy
- Exclude patients with known driver mutations

**Endpoints:**
- Primary: ORR
- Secondary: Safety and tolerability, duration of response, PFS, OS

**Sitravatinib**:
- Dose 120mg daily

**Nivolumab**:
- Dose 3mg/kg every 2 weeks (full labeled dose)

Study cycles of 28 days, with disease assessment scans every 2 cycles
Phase 2 Trial of Sitravatinib + Nivolumab in Checkpoint Refractory NSCLC

MRTX-500 Trial – Initial Responses

Study cycles of 28 days, with disease assessment scans every 2 cycles


BEST RESPONSE
(Evaluable Patients, N=56)

42/56 (75%) Clinical Benefit (SD+PR+CR)
18/56 (32%) Tumor Regression >30%
11/56 (20%) Confirmed CR/PR

DURATION OF TREATMENT
(Evaluable Patients, N=56)

Preliminary Kaplan-Meier Estimate of Median Duration of Response: 9.2 months