Jefferies Global Healthcare Conference
June 2017

Chris LeMasters
Chief Business Officer
Safe Harbor Statement

Certain statements contained in this presentation, other than statements of fact that are independently verifiable at the date hereof, contain "forward-looking" statements, within the meaning of the Private Securities Litigation Reform Act of 1995, that involve significant risks and uncertainties. Forward looking statements can be identified by the use of forward looking words such as “believes,” “expects,” “hopes,” “may,” “will,” “plan,” “intends,” “estimates,” “could,” “should,” “would,” “continue,” “seeks,” “pro forma,” or “anticipates,” or other similar words (including their use in the negative), or by discussions of future matters such as the development of current or future product candidates, timing of potential development activities and milestones, business plans and strategies, possible changes in legislation and other statements that are not historical. These statements include but are not limited to statements under the captions “Business,” “Risk Factors” and “Management’s Discussion and Analysis of Financial Condition and Results of Operations” and in other sections incorporated by reference from our Annual Report on Form 10-K and Quarterly Reports on Form 10-Q, as applicable, as well as our other filings with the SEC. You should be aware that the occurrence of any of the events discussed under the heading “Risk Factors” in any applicable prospectus supplement and any documents incorporated by reference herein or therein could substantially harm our business, operating results and financial condition and that if any of these events occurs, it could adversely affect the value of an investment in our securities.
Mirati’s Leadership Team

Applying Proven Approaches to Oncology Development

Charles M. Baum, M.D., Ph.D.
CHIEF EXECUTIVE OFFICER
SVP Clinical Research, Pfizer
Leader of Key Oncology Programs: Ibrance, Xalkori, Sutent, Inlyta, Temodar

Isan Chen, M.D.
CHIEF MEDICAL OFFICER
CMO at Aragon Pharmaceuticals
Previously VP at Pfizer with Ibrance, Xalkori, Sutent, Inlyta

James Christensen, Ph.D.
CHIEF SCIENTIFIC OFFICER
Head of Precision Research, Oncology Research Unit, Pfizer
Deep experience in precision oncology with Ibrance, Xalkori, Sutent, Inlyta

Chris LeMasters
CHIEF BUSINESS OFFICER
CBO/VP at Tragara Pharmaceuticals, Cabrellis Pharmaceuticals, Conforma Therapeutics
Led successful sales of two clinical-stage oncology companies

Jamie Donadio
CHIEF FINANCIAL OFFICER
Corporate Finance lead at Amylin Pharmaceuticals
Significant capital markets transaction experience. Participated in sale to BMS for $7 Billion

Mirati’s leadership is supported by an operational team with deep oncology drug development expertise across all disciplines
Successful Oncology Drug Development is not Linear
Strong scientific rationale driven by continuous feedback to identify the best approach and support decision making

DEEP
scientific understanding and continual research to refine understanding of the disease and molecule

IDENTIFYING
oncogenic drivers or scientifically based combinations

RELENTLESS
focus on finding patients most likely to benefit from our medicines

DESIGN
trials that are adaptive and answer multiple questions in a single trial

EMBRACE
change and make real time adjustments to follow the data and the science as it emerges
### Mirati’s Clinical Programs

**Targeted Agents and Immuno-Oncology Combinations**

<table>
<thead>
<tr>
<th>Targeted Single Agents</th>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase 1/1b</th>
<th>Phase 2</th>
<th>Sponsor</th>
<th>2017 Milestones</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Glesatinib</strong>&lt;br&gt;MET and Axl</td>
<td>Targeted Agent&lt;br&gt;MET alterations</td>
<td>NSCLC</td>
<td></td>
<td></td>
<td></td>
<td><strong>Phase 2 data: 2H 2017</strong></td>
</tr>
<tr>
<td><strong>Sitravatinib</strong>&lt;br&gt;RET, CBL, Chr4q12</td>
<td>Targeted Agent&lt;br&gt;RET, CBL, Chr4q12 alterations</td>
<td>NSCLC</td>
<td></td>
<td></td>
<td></td>
<td><strong>Phase 1b data: 3Q 2017</strong></td>
</tr>
</tbody>
</table>

### Immuno-Oncology Combinations

<table>
<thead>
<tr>
<th>Immuno-Oncology Combinations</th>
<th>Indication</th>
<th>Preclinical</th>
<th>Phase 1/1b</th>
<th>Phase 2</th>
<th>Sponsor</th>
<th>2017 Milestones</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sitravatinib</strong>&lt;br&gt;TAM Family Split Family</td>
<td>Immuno-oncology&lt;br&gt;Combination with nivolumab</td>
<td>NSCLC</td>
<td></td>
<td></td>
<td></td>
<td><strong>Phase 2 data: 2H 2017</strong>&lt;br&gt;<strong>Investigator Sponsored Trial</strong></td>
</tr>
<tr>
<td><strong>Mocetinostat</strong>&lt;br&gt;HDAC</td>
<td>Immuno-oncology&lt;br&gt;Combination with durvalumab</td>
<td>NSCLC</td>
<td></td>
<td></td>
<td></td>
<td><strong>Phase 2 data: mid-2017</strong></td>
</tr>
</tbody>
</table>

### Preclinical Programs

<table>
<thead>
<tr>
<th>Preclinical Programs</th>
<th>Indication</th>
<th>2017 Milestones</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LSD1 inhibitor</strong></td>
<td>SCLC, AML</td>
<td><strong>File IND: 4Q</strong></td>
</tr>
<tr>
<td><strong>KRAS inhibitor</strong></td>
<td>Solid Tumors</td>
<td><strong>IND Candidate Selection: 2H 2017</strong></td>
</tr>
</tbody>
</table>

---

* nivolumab owned by BMS and durvalumab owned by AstraZeneca
** NSCLC = non-small cell lung cancer; RCC = Renal Cell Carcinoma, SCLC = Small Cell Lung Cancer, AML = Acute Myeloid Leukemia
Precision Medicine
Single Agent Programs

Glesatinib (MGCD265)
Sitravatinib (MGCD516)
Mirati’s Precision Medicine Oncology Programs Supported by Novel Diagnostic Collaborations

- Development of comprehensive companion diagnostics -
- Proactive physician outreach for patients with targeted driver mutations -
- Matching patients to targeted therapies and clinical trials -
- Patient finding efforts underway and highly productive -

- Leader in tissue-based next-gen sequencing (NGS) using a comprehensive genomic profiling assay for solid tumors
- Tissue-based genomic profiling assay can detect relevant mutations from one biopsy

- Utilizing NGS in circulating tumor DNA (ctDNA) to identify multiple tumor mutations with a single blood sample
- Enables additional NSCLC patients who have insufficient tumor tissue for biopsy to be screened
Glesatinib (MGCD265)
Multi-Targeted Kinase Inhibitor for NSCLC
MET is a Driver of Tumor Growth in NSCLC

*MET is abnormally activated by mutations or gene amplification*

*MET mutations define a unique NSCLC segment and do not overlap with other drivers (e.g., ALK, ROS, EGFR)*
Glesatinib: A MET Inhibitor With Differentiated Binding
Type II binding mode confers advantage against secondary MET mutations

Glesatinib binds independently of the activation loop, where resistance mutations typically occur

100- to 1,000-fold greater activity than other MET inhibitors against some MET mutations

<table>
<thead>
<tr>
<th>Emergent Mutations</th>
<th>Wild Type</th>
<th>D1228H</th>
<th>D1228N</th>
<th>Y1230A</th>
<th>Y1230C</th>
<th>Y1230D</th>
<th>Y1230H</th>
<th>Y1235D</th>
</tr>
</thead>
<tbody>
<tr>
<td>GLESATINIB</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AMG-208</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>crizotinib</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>INC280</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Biochemical IC50s:
- < 100nM
- 100 – 500nM
- > 1000nM

* Data on file, AACR 2016 Abstract

- Selective pressure produces these resistance mutations *in vitro* and *in vivo*
- Resistance mutations also observed clinically
- Glesatinib is active against these mutations
AMETHYST Phase 2 Trial (Glesatinib in NSCLC)

Registration-enabling open-label Phase 2 study

Phase 2 open-label, parallel arm study in recurrent or metastatic NSCLC with selected activating genetic alterations in MET

Second Line: Patients must have received at least one prior treatment with platinum-based combination therapy or checkpoint inhibitor therapy

Genetic Patient Selection: via tissue or blood Next-Generation Sequencing (NGS)

Patient Cohorts:

- **MET Exon 14 Deletion**
- **MET Gene Amplification**

Endpoints:

- Primary: Objective Response Rate (ORR)
- Secondary: Progression Free Survival (PFS)
Glesatinib Combined Phase 2 and Phase 1b Activity

**MET Ex14 deletion – New Formulation patients (n=13 evaluable)**

**Best Response**

- **Confirmed PR**: 11%
- **Unconfirmed PR**: 0%
- **SD**: 17%
- **PD**: 18%
- **Nonmeasurable Disease**: 20%
- **PD in non-target lesion on 1st scan**: 24%
- **PD**: 31%
- **PD**: 40%
- **PD**: 47%
- **PD**: 50%
- **PD**: 50%
- **PD**: 66%

**Total enrolled patients**: 16

**Patients with no scan data to date**: 3

**Data cutoff**: 2-Dec-2016

**Initial Indication of Response (n=13)**

- **ORR (confirmed & unconfirmed)**: 46% (6/13)
- **Tumor Regression**: 85% (11/13)

**Duration**

- **Confirmed PR**
- **Unconfirmed PR**
- **SD**
- **PD**
- **Response First Achieved on Study**
- **Withdrawal of consent (AE)**
- **Off study**
- **Nonmeasurable Disease**

**Weeks**: 3 6 9 12 15 18 21 24 27 30 33 36 39 42 45 48 51 54 57

Investigator-reported activity data
Pfizer: Crizotinib Ph1b NSCLC Advanced \textit{MET} Exon 14-altered

Data Update: IASLC World Lung Cancer Dec 2016*

---

**Crizotinib Phase 1b PROFILE 1001 Study**  
\textit{(MET Exon 14-altered)}

\textbf{n}=30 (9/30 1\textsuperscript{st} line patients)  
Local molecular profiling, central confirmation not required

\textbf{Objective Response Rate:} 11/28 (39\%, CI: 22,59)

\textbf{Best Response} (\textbf{n}=28)  
\begin{tabular}{ll}
CR & 2 (7\%) \\
PR & 9 (32\%) \\
SD & 10 (36\%) \\
PD & 2 (7\%) \\
Unkn & 5 (18\%) \\
\end{tabular}

\textbf{Durability} (\textbf{n}=30)  
\begin{tabular}{ll}
Median Duration & 5.4 mo (range: 0.4-18.2) \\
Median PFS & 8.0 mo (CI: 6.9, 10.8) \\
\end{tabular}

---

Efficacy data for first line patients treated (\textbf{n}=9/30) was not reported separately

\* Data reported by Pfizer. Mirati has not conducted a head to head study of glesatinib and crizotinib.
Glesatinib: Phase 2 Activity

MET Gene Amp – New Formulation patients (n=8 evaluable)

**Best Response**

- **Confirmed PR**: 25%
- **Unconfirmed PR**: 15%
- **SD**: -6%, -8%, -14%, -18%
- **PD**: -30%, -39%

- **Total enrolled selected patients**: 8
- **Patients with no scan data to date**: 0

- **3 patients on study**

**Initial Indication of Response (n=8)**

- **ORR (unconfirmed)**: 25% (2/8)
- **Tumor Regression**: 75% (6/8)

*Data cutoff: 2-Dec-2016*

**Investigator-reported activity data**
**Pfizer: Crizotinib Phase 2 NSCLC MET Alterations**

*Data Update: IASLC World Lung Cancer Dec 2016*

---

### Crizotinib Phase 2 METROS Trial (Italy)

n=16 (2nd line)  
Dose: 250mg BID

**MET AMP (n=12, 10 evaluable)**

<table>
<thead>
<tr>
<th>Best Response</th>
<th>1/10</th>
<th>5/10</th>
<th>4/10</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**MET Exon Deletion (n=4, 3 evaluable)**

<table>
<thead>
<tr>
<th>Best Response</th>
<th>1/3</th>
<th>2/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Principal Investigator: Federico Cappuzzo, MD

---

* Data reported by primary investigator. Mirati has not conducted a head to head study of glesatinib and crizotinib.
Glesatinib Summary

- Trial enrollment is progressing well as planned
  - 88 sites open in 9 countries
- In January, we reported an Phase 2 update:
  - In the MET Exon 14 deletion cohort, 4/13 confirmed and 2/13 unconfirmed PRs with tumor regression in 11/13
  - In the MET Gene amplification cohort, 2/8 unconfirmed PRs with tumor regression in 4/8
- Glesatinib has been Well-Tolerated
- MET Exon 14 deletion (~2% NSCLC) and MET Gene Amp (~2% NSCLC) are commercially attractive
- Phase 2 data update is expected in 2H 2017
Sitravatinib (MGCD516)
Multi-Targeted Kinase Inhibitor
Sitravatinib: A Novel Kinase Inhibitor

Sitravatinib targets genetic alterations in up to 5.5% of NSCLC patients

- **RET fusions**: 2%
- **CHR4q12 amplicons**: 2%
- **CBL mutations**: 1.5%
- **Other and Unknown**: 42%
- **KRAS**: 15%
- **EGFR**: 15%
- **MET**: 4%
- **ALK**: <1%
- **TrkA/C fusions**: <1%
- **TrkB/C mutations**: <1%
- **DDR2 mutations**: <1%
- **MET Exon 14del**: 2%
- **MET Gene amp**: 2%

Sitravatinib: Rationale For Targeting CBL Mutations

- **CBL: A Receptor Brake**
  - CBL normally acts as a brake for activated receptors
  - Turns off activated receptors via receptor recycling

- **CBL is inactivated by mutations**
  - Results in hyperactivated signaling of receptors normally modulated by CBL

- **Inactivated CBL is an oncogenic driver**
  - CBL mutations present in NSCLC (1.5%), melanoma (3.5%) and cancers of unknown origin (2%)

- **Sitravatinib potently inhibits tumor cells expressing CBL mutations**

**Key CBL Targets and Unique Binding Motifs**

- AXL / MER
- PDGFR / KIT
- MET / RON
- LINEpYSSDPT
- ESIFDNLPYTTLSD
- NESVDPYRATFP
Sitravatinib: Rationale For Targeting CHR4q12 Amplification

• CHR4q12 amplification increases expression of oncogenic drivers
  – PDGFR, KIT and VEGFR

• Sitravatinib is a potent inhibitor of oncogenic kinases driven by CHR4q12 amplification
  – IC50 values < 10 nM in cellular PDGFRA, KIT & KDR activity assays

• Sitravatinib is active in tumors with CHR4q12 amplification
  – Tumor regression in CHR4q12 amplified NSCLC models at 20 mg/kg
  – Mouse dose matches clinical exposure

• CHR4q12 is amplified in ~2% of NSCLC
Sitravatinib Phase 1b Expansion Trial

**Primary Focus**
NGS Screen for Patients with:

- **RET fusions**
- **CBL mutations**
- **CHR4q12 amplicons**

**Cohort 1**
2nd-line or later NSCLC

**Cohort 2**
2nd-line or later “Basket” (solid tumors)

**Cohort 3**
Advanced solid tumors

Solid tumors likely to respond to sitravatinib profile, including mRCC and CRPC

mRCC = metastatic renal cell carcinoma; CRPC = castration-resistant prostate cancer
Sitravatinib: NSCLC Phase 1b Activity Update

NSCLC patients with RET Fusion (n=4 evaluable)

**Initial Indication of Response** (n=4)
- ORR (confirmed & unconfirmed) 50% (2/4)
- Tumor Regression 100% (4/4)

*Data cutoff: 9-Dec-2016

**Investigator-reported activity data

---

**Best Response**

- Confirmed PR
- Unconfirmed PR
- SD
- PD
- RECIST Cutoff for PR, 30%

- Maximum % Change from Baseline
- 0% - 10%
- 10% - 20%
- 20% - 30%
- 30% - 40%
- 40% - 50%
- 50% - 60%
- 60% - 70%
- 70%

- 4 patients on study
- 1 patient withdrew before 1st scan
- 1 patient with 0 scans to date

**Duration**

- Confirmed PR
- SD
- PD
- Off study
- Response First Achieved on Study

- Days:
  - 3
  - 6
  - 9
  - 12
  - 15
  - 18
  - 21
  - 24
  - 27
  - 30
  - 33
  - 36
  - 39
  - 42
  - 45
  - 48

- Withdrew due to AE
Sitravatinib Single Agent Summary

• Early Phase 1b Data in RET Fusion NSCLC is Encouraging
  – Well-tolerated with manageable side effects
  – NSCLC RET Fusion is commercially attractive (~2% NSCLC)

• CBL mutations may act as oncogenic drivers in NSCLC (1.5%), melanoma (3.5%) and cancers of unknown origin (2%)
  – Sitravatinib demonstrated tumor regression in CHR4q12 amplified NSCLC models in early preclinical models

• CHR4q12 amplified NSCLC comprises approximately 2% of NSCLC
  – Early preclinical research shows that sitravatinib potently inhibits tumor cells expressing CBL inactivating mutation

• Phase 1b Safety: Sitravatinib has been Generally Well-Tolerated
• Phase 1b Single Agent Update Expected in Q3 2017
Immuno-Oncology Combination Programs

Sitravatinib + Checkpoint Inhibitors

Mocetinostat + Checkpoint Inhibitors
Sitravatinib: Potent Inhibitor of TAM and Split RTKs

Inhibition of TAM and Split may enhance responses to checkpoint inhibitors

**Split (VEGFR2 and KIT)**

- Targeting VEGFR2 reduces Tregs and MDSCs
- Targeting KIT depletes MDSCs
- Releases brakes for expansion of CD8+ T cells via PD-1 inhibition

**TAMs (MERTK and Axl)**

- Targeting MERTK & Axl shifts tumor associated macrophage (TAM) type to M1
- M1 macrophages secrete cytokines that enhance immune response (IL-12, TNF)

Both TAM & Split RTKs cooperate:

- To increase dendritic cell maturity and antigen presentation capacity
- To increase NK cell response
- To increase T cell trafficking into tumors

Sitravatinib is a first-in-class potent TAM and Split inhibitor
Sitravatinib + PD-1 Exhibits Marked Anti-tumor Activity

CT26 model (colon)

- Significant tumor growth inhibition and durable response for PD-1 + sitravatinib combination arm
- Expansion of systemic CD4 & CD8 T effector cells observed by Day 6
- Depletion of systemic myeloid-derived suppressor cells (MDSCs) by Day 6
Early clinical data supports utility of VEGF + Checkpoint

*VEGF inhibition is only half the Immuno-Oncology story for Sitravatinib*

### VEGF Inhibitors + Checkpoint Inhibitors*

**Reported Clinical Activity**

<table>
<thead>
<tr>
<th>Tumor</th>
<th>VEGF Inhibitor</th>
<th>Combination Partner</th>
<th>ORR</th>
<th>VEGF alone**</th>
<th>Checkpoint alone**</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCC (naïve)</td>
<td>Axinitib</td>
<td>Pembrolizumab</td>
<td>67% (35/52)</td>
<td>47%</td>
<td>-</td>
</tr>
<tr>
<td>RCC (2nd line)</td>
<td>Sunitinib</td>
<td>Nivolumab</td>
<td>52% (17/33)</td>
<td>34%</td>
<td>19%</td>
</tr>
<tr>
<td>RCC (2nd line)</td>
<td>Pazopanib</td>
<td>Nivolumab</td>
<td>45% (9/20)</td>
<td>34%</td>
<td>19%</td>
</tr>
<tr>
<td>NSCLC (2nd line)</td>
<td>Ramucirumab</td>
<td>Pembrolizumab</td>
<td>38% (10/26)</td>
<td>&lt;10%</td>
<td>20%</td>
</tr>
</tbody>
</table>

- Early clinical data suggests that VEGF inhibition expands the response to checkpoint inhibitors
- **SITRAVATINIB**, which potently inhibits TAM family kinases as well as VEGF and KIT, may further improve upon the effectiveness of checkpoint inhibitors

* Data as reported by Pfizer, GSK and Eli Lilly

** Comparative data on VEGF alone and Checkpoint alone represent amalgamations of published single agent trial data
Sitravatinib Immuno-Oncology Combination: NSCLC
Sitravatinib May Enhance Activity of Checkpoint Inhibitors Through TAM, VEGF and KIT Inhibition

**SITRAVATINIB**
~1nm inhibitor of TAM kinases
Potent inhibitor of KIT and VEGF

- **Inhibiting TAM kinases:**
  Stimulates anti-tumor immunity by changing tumor microenvironment from a tolerogenic to an immunogenic state

- **Inhibiting KIT and VEGF:**
  Depletes immunosuppressive cell subsets including Tregs and MDSCs

**Sitravatinib + nivolumab Phase 2 Study**
Non-squamous Second Line NSCLC
*Nivolumab dose 3mg/kg every 2 weeks (full labeled dose)*

**Trial Update:**
- Well tolerated at the 120mg daily dose
- Enrollment is on-going in the Phase 2 trial
- Initial Phase 2 data is expected in 2H 2017
**Sitravatinib Immuno-Oncology Combination: RCC**

*Investigator sponsored trial at MD Anderson Cancer Center*

---

**Sitravatinib**

- **~1nm inhibitor of TAM kinases**
- **Potent inhibitor of KIT and VEGF**

---

**Sitravatinib + nivolumab Phase 1/2 Study**

**Advanced Clear Cell Renal Cancer following progression on VEGF Therapy, Checkpoint Naive**

*Nivolumab dose 240mg every 2 weeks (full labeled dose)*

---

- **Phase I Dose Escalation**
- **Phase II Expansion**

---

**Trial Update:**

- Patient enrollment is on-going
- The first dose escalation cohort has enrolled

---

**Nizar Tannir, MD**

Primary Investigator
Mocetinostat
Immuno-Oncology
Class I & IV HDAC Inhibitor
Mocetinostat: Class I and IV HDAC Inhibitor

Shifting the tumor microenvironment (TME)

Immunosuppressive TME  

Mocetinostat  

Immune-stimulated TME

**Immune Cell Effects**

- **Tregs** and **MDSCs** decreased by mocetinostat
- **CD8** increased by mocetinostat

**Tumor Cell Effects**

PD-L1 and antigen presentation machinery upregulated by mocetinostat in tumor cells
Mocetinostat + PD-1 Exhibits Marked Anti-tumor Activity

- Mocetinostat significantly improved the ratio of CD8+ T cells to Treg cells
- Mocetinostat also significantly increases T-cell clonality and T-cell fraction
- The combination of mocetinostat and anti-PD-L1 antibody produces greater antitumor activity than either agent alone
Mocetinostat Immuno-Oncology Combination
Mocetinostat May Enhance Activity of Checkpoint Inhibitors Through HDAC Inhibition

MOCETINOSTAT
Class I & IV HDAC Inhibitor

Potential to enhance anti-tumor efficacy when combined with immunotherapy by
• Increasing HLA expression and tumor immunogenicity
• Depleting regulatory T-cells and myeloid-derived suppressor cells
• Increasing tumor PD-L1 expression (tumors more likely to respond to checkpoint inhibitors)

Does not inhibit Class II HDACs which may be immunosuppressive

Mocetinostat + durvalumab Phase 2 Study
Non-squamous Second Line NSCLC

Durvalumab dose 1500mg every 4 weeks (full labeled dose)

Trial Update:
• The 70mg, three times a week dose is well tolerated in combination with full dose durvalumab
• Enrollment is on-going for the Phase 2 trial
• Initial Phase 2 data is expected in mid 2017
Immuno-oncology Program Summary

Enhancing immunotherapy response

Sitravatinib + Nivolumab:

- Blocks immune suppressive factors while priming immune stimulatory pathways
- Emerging clinical data for VEGF in checkpoint combinations is supportive and the addition of TAM family inhibition may position sitravatinib as first and best-in-class
  - *Phase 2 in NSCLC is enrolling – initial data in 2H 2017*
  - *Phase 2 IST in RCC at MDACC is enrolling patients*

Mocetinostat + durvalumab:

- Preclinical data supports that mocetinostat increases CD8+ T cells while depleting immunosuppressive cells
  - *Phase 2 in NSCLC is enrolling – initial data in mid-2017*

*MDACC = MD Anderson Cancer Center*
Preclinical Programs

LSD1
KRAS
# Research and Preclinical Programs

**Genomically-Informed Precision Medicine**

<table>
<thead>
<tr>
<th>Initial Indications</th>
<th>Research</th>
<th>Candidate Selection</th>
<th>Preclinical</th>
<th>Phase I</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mirati Kinase Discovery Platform</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>LSD1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lysine-Specific Histone Demethylase 1</td>
<td>Small Cell Lung Cancer</td>
<td>2017</td>
<td>2018</td>
<td>2017</td>
</tr>
<tr>
<td>Critical regulator of H3K4 methylation</td>
<td>Acute Myeloid Leukemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>KRAS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>V-Ki-ras2 Kirsten rat sarcoma</td>
<td>Non-Small Cell Lung Cancer</td>
<td>2017</td>
<td>2018</td>
<td>2017</td>
</tr>
<tr>
<td>A Top 5 most commonly mutated Cancer gene</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**LSD1 Program:** Achieved Candidate Selection in December 2016
- Observed potency 10-100 fold better than competitors with high bioavailability and exposures
- **IND submission expected Q4 2017**

**KRAS Program:** Advancing towards Candidate
- Potent and selective compounds demonstrating tumor regression in **KRAS** mutant models
- **Candidate Selection anticipated in 2H 2017**
KRAS Inhibitor Program

Potent and selective compounds targeting KRAS G12C mutations

The KRAS Opportunity

KRAS is a Meaningful Target
Most frequently mutated oncogene in human cancer

KRAS: Difficult Protein to Drug
Lack of well-defined pocket at the effector site

KRAS G12C Mutant
KRAS G12C mutation offers a reactive handle for a specific irreversible inhibitor
KRAS G12C mutation occurs in 10-15% of all NSCLC

Mirati’s KRAS Inhibitor Program

• Partnership with Array Biopharma
  – Access to Covalent Chemistry expertise
  – Access to first-rate Crystallography

• Potent and Selective Compounds Identified
  – Highly potent in cellular assays
  – Orally bioavailable drug properties
  – Target inhibition and repeat-dose efficacy demonstrated in pre-clinical cancer models

• Provisional Patents Filed

KRAS Candidate Selection Expected in 2H 2017
## Mirati Therapeutics 2017 Milestones

*Meaningful data points across all programs in 2017*

<table>
<thead>
<tr>
<th>Program</th>
<th>2017 Milestone</th>
<th>Expected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glesatinib (265)</td>
<td>• Phase 2 update (<em>MET</em> Exon 14del and <em>MET</em> amp)</td>
<td>2H 2017</td>
</tr>
</tbody>
</table>
| Sitravatinib (516) | • Phase 1b update in NSCLC *RET* fusion patients  
|                  | • Initial Phase 2 data from dose escalation NSCLC patients in combination with anti-PD-1 | Q3 2017, 2H 2017 |
| Mocetinostat (103) | • Initial Phase 2 data in combination with anti-PD-L1                          | Mid-2017   |
| Pre-clinical    | • IND for *LSD1* inhibitor  
|                  | • IND candidate for *KRAS* inhibitor                                          | Q4 2017, 2H 2017 |
Company Financials

NASDAQ: MRTX

Cash (Q1 17)*
$105.4M

Jan Offering (net proceeds)
$66.8M

Shares Outstanding**
24.9M

* Estimated cash as of March 31, 2017. Current cash is expected to last into late 2018.

** Shares outstanding as of March 31, 2017. Does not include pre-funded warrants to purchase a total of 7,258,263 which have a per share exercise price of $0.001.
Thank you