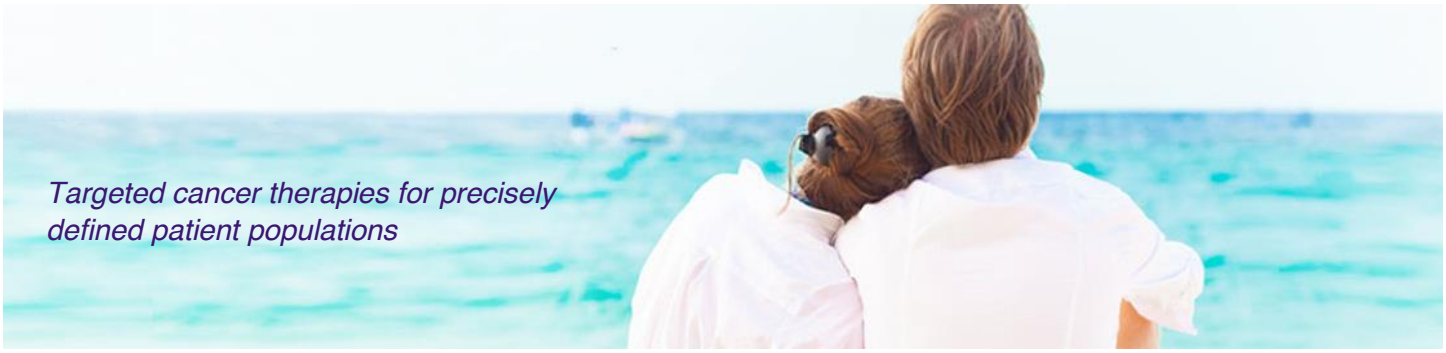
MIRATI THERAPEUTICS

NASDAQ: MRTX
www.mirati.com



An Experienced Targeted Oncology Team

Charles M. Baum, M.D., Ph.D.
Chief Executive Officer

Isan Chen, M.D.
Chief Medical Officer

James Christensen, Ph.D.
Chief Scientific Officer

Mark J. Gergen
Chief Operating Officer

Jamie Donadio
Vice President Finance

Perry C. Johnston, J.D.
Vice President Legal

2014 Catalysts

MGCD265

Initial proof of concept data in NSCLC and Head and Neck Cancer in Q4 2014

MGCD516

Start Phase 1 mid-year 2014

Identify Phase 2 dose YE 2014

Mocetinostat

Start Phase 2 trials in DLBCL and bladder cancer mid-year 2014

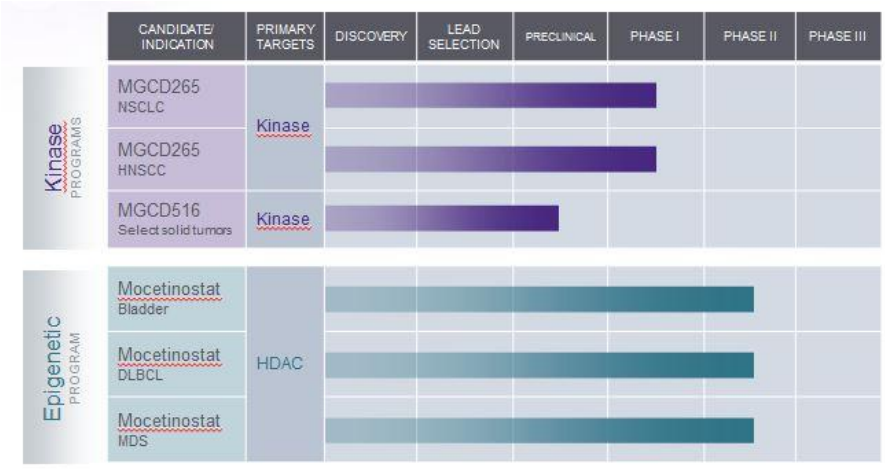
Initial Phase 2 data in MDS, DLBCL and bladder cancer by

Mirati Therapeutics is a targeted oncology company developing an advanced pipeline of breakthrough medicines for precisely defined patient populations. Mirati's approach combines the three most important factors in oncology drug development:

- Targeting genetic and epigenetic drivers of cancer as well as pathways of treatment resistance
- Creative and agile clinical development focused on selecting patient subpopulations most likely to respond to treatment, and
- A highly accomplished precision medicine team.

Mirati is developing MGCD265 and MGCD516, kinase inhibitors that target compelling receptor kinases that are drivers of tumor cell growth and cancer progression. Mirati is also conducting proof-of-concept trials for mocetinostat, a spectrum-selective histone deacetylase (HDAC) inhibitor, for the treatment of patients with diffuse large cell B-cell lymphoma (DLBCL) or bladder cancer who have defects in histone acetylation due to mutations in CREBBP and/or EP300. Mocetinostat is also in a Phase 2 dose confirmation study as a potential first line treatment for patients with myelodysplastic syndromes (MDS) in combination with standard of care Vidaza, a hypomethylating agent.

Pipeline Driving Towards Multiple Inflection Points



Kinase Inhibitor Programs

Receptor tyrosine kinases (RTKs) are a family of kinases involved in the transmission of signals that regulate the expression of many genes, including those that control cell growth and cell division. Aberrant kinase function, caused by genetic alterations or over-expression, underlies many cancer cell processes. Mirati is developing two RTK inhibitors: MGCD265 and MGCD516.

MGCD265

MGCD265 is a potent small molecule multi-targeted kinase inhibitor of Met, Axl and is in Phase 1 development initially as a treatment for non-small cell lung cancer (NSCLC) and head and neck cancer (HNSCC). Certain genetic alterations of Met and Axl (~8%) appear to be oncogenic drivers, so targeting patients with specific mutations and gene amplification is the most promising opportunity for patient selection. In addition to single agent opportunities, Met and Axl are implicated as drivers of tumor progression in patients whose tumors become resistant to EGFR inhibitors providing a potential opportunity for combination therapy.

MGCD516

MGCD516 is a potent inhibitor of multiple closely related RTKs including the Trk, RET and DDR families, which are reported oncogenic drivers in NSCLC and other cancer types. In preclinical studies, MGCD516 has shown potent inhibition in vitro of cell proliferation, cell motility and angiogenesis. It has also demonstrated anti-tumor activity including tumor regression in multiple human xenograft tumor models in mice. The IND has been filed and Mirati expects to initiate Phase 1 in mid 2014.

Epigenetics Program

Epigenetic mechanisms affect the regulation of gene expression independent of the primary DNA sequence. Epigenetic pathways are often dysregulated during cancer progression, resulting in the silencing of tumor suppressor genes, activation of oncogenes, and uncontrolled tumor growth and metastatic progression of certain malignancies including MDS, lymphomas and selected solid tumors such as bladder cancer.

Mocetinostat in DLBCL and solid tumors

Mocetinostat is a spectrum-selective HDAC inhibitor being developed for the treatment of diffuse large b-cell lymphoma (DLBCL) and certain solid tumors such as bladder cancer in patients with defects in histone acetylation resulting from mutations in CREBBP and/or EP300. In an analysis of 30 evaluable patients with DLBCL, treatment with mocetinotstat as a single agent resulted in reduction in tumor size in 77% of patients including 1CR and 6 PR's. Mirati has identified certain loss of function mutations (CREBBP and EP300) that appear in up to 30% of DLBCL and certain solid tumors such as bladder cancer, representing a promising patient enrichment strategy for clinical development.

Mocetinostat in MDS

The epigenetic mechanism of mocetinostat has been shown to have synergistic antitumor activity when combined with hypomethylating (HMAs) agents such as Vidaza (azacitidine) and may be complementary in the treatment of MDS. First-line treatments for intermediate- to high-risk patients are limited to hypomethylating agents (HMAs) such as azacitidine, but the majority of patients fail to respond to these drugs. Mocetinostat showed a 93 percent disease control rate in 28 patients with MDS.

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Mirati Therapeutics, Inc.
9363 Towne Centre Drive, Suite 200
San Diego, CA 92121
(858) 332-3410
www.mirati.com