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Onconova Therapeutics Announces Presentation of Nonclinical Data on Lead Compound ON 01910.Na and New Jak 2/Bcr-abl Inhibitors at AACR

APRIL 21, 2009 – NEWTOWN, PA -- Onconova Therapeutics, Inc. today announced the presentation of four scientific studies relating to its lead anticancer drug ON 01910.Na and a new class of anticancer compounds at the Annual Meeting of the American Association for Cancer Research (AACR), being held April 18-22, 2009 in Denver.

The data will be presented in one oral presentation and three posters on Monday and Tuesday during the AACR meeting.

The oral presentation and two posters will focus on ON 01910.Na, a novel, targeted, small-molecule anti-cancer compound undergoing clinical trials at several major centers in the USA and abroad. In clinical studies ON 01910.Na has shown broad-spectrum anti-tumor activity against both solid tumors and hematological malignancies and in nonclinical studies has demonstrated remarkable synergistic activity when combined with several classes of conventional chemotherapeutic agents.

Extensive Phase I data from more than 125 patients with solid tumors recruited in eight different clinical protocols is now available and indicate a good tolerability profile. Onconova is conducting additional single agent trials and new combination therapy clinical trials of ON 01910.Na with leading investigators at major oncology clinical centers in the USA.

The third poster introduces a new Onconova drug candidate, ON 044580, which is a dual JAK2/BCR-ABL kinase inhibitor which has therapeutic potential to treat JAK2V617F positive myeloproliferative disorders (MPDs), other MPDs and imatinib-resistant chronic myeloid leukemia (CML) and may provide an option for patients who develop resistance to current therapies.

AACR Presentations Relating to Onconova Compounds:

Tuesday, April 21, 11:55 AM -12:10 PM

Room 505-507, Colorado Convention Center
Session Title: Mechanisms of Drugs Targeting Cell Cycle Controls

Abstract # 3866

“Modulation of Chk1, Chk2, p-Chk2 dimer and p-ATM in cancer cells treated with ON 01910.Na, a clinical stage mitotic inhibitor”

Irina Oussenko, James F. Holland, E. Premkumar Reddy, Takao Ohnuma. Mount Sinai School of Medicine, New York, NY, Fels Institute for Cancer Research and Molecular Biology, Temple University School of Medicine, Philadelphia, PA

Dr. Oussenko will report assessment of DNA damage checkpoints throughout the cell cycle and effects on signaling molecules upstream of Cdc25C following treatment with ON 01910.Na. These results suggest that effects on DNA damage checkpoint pathways may be a part of ON 01910.Na's multi-targeted mode of action.

Tuesday, April 21, 8:00 AM -12:00 PM

Hall B-F, Denver Convention Center
Session Title: Conjugates and Other Targeted Approaches
Poster Section: 33
Poster Board Number: 28

Abstract# 3654

“ON 01910.Na, a novel clinical grade PLK-1 inhibitor, selectively induces apoptosis in human B-cell chronic lymphocytic leukemia (B-CLL)”

Colby M. Chapman, Patricia Perez-Galan, Adrian Wiestner. NHLBI, NIH, Bethesda, MD

This study aimed to determine activity of ON 01910.Na in CLL and investigate the mechanism of action because ON 01910.Na is a multi-kinase inhibitor. ON 01910.Na has anti-tumor activity in solid tumor models and is currently undergoing clinical testing in other cancers. Results of this study show that over expression of the antiapoptotic protein Mcl-1 is a central mechanism of CLL cell survival, and ON 01910.Na decreased Mcl-1 protein expression in leukemic cells and this preceded the onset of apoptosis. This suggests that a reduction of Mcl-1 expression could play an important role in the antileukemic activity of ON 01910.Na. These results support the development of ON 01910.Na in CLL, and a clinical trial for this indication is being developed.

Tuesday, April 21, 1:00 PM - 5:00 PM

Hall B-F, Denver Convention Center

Session Title: Novel Agents 3

Poster Section: 37

Poster Board Number: 27

Abstract #4702

“Pathway-based comparison approach for the identification of responders to the mitotic modulator ON 01910.Na in head and neck cancer (HNC)”

Aik Choon Tan, Ryan Anderson, Barbara Frederick, David Raben, Antonio Jimeno.

University of Colorado Denver, Aurora, CO

This study was designed to determine the efficacy of the novel clinical stage mitotic inhibitor ON 01910.Na in head and neck cancer (HNC), and to elucidate gene expression-based predictors of efficacy, in comparison with approved therapeutic agents. These results show that ON 01910.Na is a highly effective mitotic inhibitor active in a broad panel of HNC cell lines, with potency equivalent or superior to approved agents. Expression analysis revealed that sensitive strains had over-expression of the cell cycle pathway (providing further evidence of mechanisms relevant in the action of ON 01910.Na in HNC).

Tuesday, April 21, 8:00 AM -12:00 PM

Hall B-F, Denver Convention Center

Session Title: Kinase Inhibitors 3

Poster Section: 36

Poster Board Number: 1

Abstract # 3717

“Identification of a dual JAK-2/BCR-ABL kinase inhibitor for treatment of myeloproliferative disorders”

Shashidhar S. Jatiani, Stephen C. Cosenza, Venkat R. Pallela, Ji Hee Ha, Stacey J. Baker, M V Ramana Reddy, E. Premkumar Reddy. Fels Institute for Cancer Research, Temple University School of Medicine, Philadelphia, PA

A specific JAK2 mutation has been observed in about 95% of patients with the myeloproliferative disorder polycythemia vera (PV), and 50% of those with both essential thrombocythemia (ET) and primary myelofibrosis (PMF). This mutation has also been found in patients with non-classic MPDs such as refractory anemia with ringed sideroblasts associated with thrombocytosis (RARS-T), chronic neutrophilic leukemia (CNL), atypical chronic myeloid leukemia (CML) and chronic myelomonocytic leukemia (CMML) at incidences of 50%, 20%, 20% and 3%, respectively. JAK2V617F has been found to confer erythropoietin-independent growth of the mutant cells in vitro due to deregulation of signaling pathways downstream of JAK2. These findings have opened new avenues for the diagnosis and classification of patients with these disorders, and identify a new molecular target for drug discovery. These study results identify ON 044580 as a first in class dual Jak2/Bcr-abl inhibitor. The results suggest that ON

044580 may have therapeutic potential to treat JAK2V617F positive MPDs and CML and provide an option for patients who develop resistance to current therapies such as imatinib and dasatinib.

About Onconova's Product-Pipeline

Onconova is developing therapeutic candidates directed at critical targets involved in signal transduction, cell-cycle and DNA repair. These candidates are derived from the Company's proprietary library of new chemical entities and non-ATP competitive chemotypes. In addition to ON 01910.Na, Onconova is also developing Ex-RAD™, an injectable and oral radioprotectant, and inhibitors of Cyclin D, JAK and Bcr-abl pathways. These compounds were invented by Dr. E. Premkumar Reddy and colleagues at the Fels Institute and are exclusively licensed to Onconova Therapeutics, Inc.

About Onconova Therapeutics, Inc.

Onconova, based in Newtown, PA, and Lawrenceville, NJ, discovers and develops novel small molecule therapeutic agents for cancer, radiation protection and hematological disorders. Currently, the Company is conducting clinical trials at major centers in the USA and abroad. The novel chemical library platform is permitting identification of non-ATP competitive kinase inhibitors directed at validated and novel targets, and a new immunoconjugate technology (comprising potent active compounds and proprietary linkers) for arming monoclonal antibodies for cancer therapy. All of the Company's products and technologies are being developed internally.

For more information on Onconova Therapeutics, Inc., please visit www.onconova.com.

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