



Syndax Pharmaceuticals' Entinostat in Combination Shows Activity in Breast Cancer

--Animal data being presented at the San Antonio Breast Cancer Symposium--

Waltham, Mass. – December 10, 2010 – Syndax Pharmaceuticals, Inc., a clinical-stage epigenetics oncology company, announces results from two preclinical studies on entinostat, an orally bioavailable, highly selective, class I histone deacetylase (HDAC) inhibitor, in animal models of breast cancer. The data are being presented as oral poster discussions at the San Antonio Breast Cancer Symposium December 8 through December 12 in San Antonio Texas.

The following poster discussions are being presented:

- “A Combination of HDAC Inhibitor Entinostat (MS-275), All Trans Retinoic Acid (ATRA) and Chemotherapy Drug(s) Causes Regression of Established Xenografts of Triple Negative Breast Cancer” December 9, 2010 from 5:30 to 7:30 PM CT by Nguyen K. Nguyen from Johns Hopkins University School of Medicine
- “HDAC Inhibitor Entinostat Restores Responsiveness of Letrozole Resistant MCF-7Ca Xenografts to Als through Modulation of Her-2” December 10, 2010 from 5:30 to 7:30 PM CT by Gauri J. Sabnis, Ph.D. from the University of Maryland School of Medicine

“These promising results in predictive animal models provide important insight into the molecular mechanisms by which entinostat can target resistance pathways in breast cancer,” said Joanna Horobin, M.D., president and chief executive officer of Syndax. “In the phase 2 ENCORE 303 study presented at ASCO, we showed that the addition of entinostat could halt disease progression emerging on treatment with all of the commercially available aromatase inhibitors. Dr. Sabnis’ work provides an elegant hypothesis supporting our clinical findings. We are optimistic that our ongoing ENCORE 301 study, a double-blind, randomized, placebo-controlled phase 2 study of entinostat in combination with the aromatase inhibitor exemestane, will provide further evidence supporting the clinical benefit and tolerability of entinostat in postmenopausal women with progressing metastatic breast cancer. We expect to see results in the first half of next year.”

The University of Maryland study looked at the ability of entinostat to restore responsiveness of letrozole resistant MCF-7Ca xenografts to aromatase inhibitors. Entinostat was able to increase estrogen receptor expression and aromatase activity in the letrozole resistant tumors. The results suggest that entinostat may modulate Her-2 signaling and reverse the acquired resistance to letrozole caused by up-regulation of estrogen independent growth factor signaling pathways.

Triple negative breast cancer (TNBC) is a sub-group of breast cancer that is normally unresponsive to hormone therapy as well as many forms of chemotherapy. Researchers from Johns Hopkins University School of Medicine reasoned that combining epigenetic therapy, entinostat, with differentiation therapy, retinoic acid receptor beta 2 agonist (ATRA) will provide an effective combination of drugs against TNBC. Low-doses of chemotherapy also were added to the combination. The results showed that entinostat plus ATRA and doxorubicin has the potential to be an effective treatment against TNBC.

For more information regarding the SABCS presentations please visit the SABCS website at <http://www.sabcs.org/ProgramSchedule/ScheduleGlance.asp>.

About Entinostat

Entinostat is an orally bioavailable, highly selective, class I histone deacetylase (HDAC) inhibitor with a long half-life that allows for weekly or every-other-week dosing. Entinostat is currently being investigated in multiple phase 2 clinical studies: in advanced breast cancer in combination with aromatase inhibitors; in combination with erlotinib in metastatic lung cancer and as a single agent in Hodgkin's lymphoma. Entinostat also is being studied in advanced non-small-cell lung cancer and in advanced colorectal cancer in combination with azacitidine under a Cooperative Research and Development Agreement (CRADA) with the NCI.

Research has shown that HDACs are involved in the expression of various genes, such as the estrogen receptor, that regulate cell growth, differentiation and apoptosis. Such genes are frequently silenced in cancer cells through the over-expression of enzymes including HDACs. HDACs are therefore recognized as promising targets for cancer treatment. Further, studies have demonstrated that HDAC inhibition can significantly enhance anti-cancer activity when used in combination with a broad range of anti-cancer agents. The potential therefore exists to overcome tumor resistance to targeted agents.

About Syndax

Syndax Pharmaceuticals, Inc. is a Waltham, MA-based, oncology-focused pharmaceutical company. Syndax is building a portfolio of new oncology products to extend and improve the lives of patients by developing and commercializing novel cancer therapies in optimized, mechanistically driven combination regimens. Formed in 2005, the company's intellectual property is based on work from scientific founder Ronald Evans, Ph.D., recipient of the 2004 Albert Lasker Prize for Basic Medical Research, a Member of the National Academy of Sciences, a professor at the Salk Institute for Biological Studies and a Howard Hughes Medical Institute Investigator. Syndax has worldwide rights to develop and commercialize entinostat and is backed by top-tier Venture Capital firms: Domain Associates, MPM Capital, Avalon, Pappas and Forward Ventures. For more information please visit www.syndax.com.

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