

FOR IMMEDIATE RELEASE**Acetylon Pharmaceuticals' Selective HDAC Inhibitor Programs to be Featured at the 54th Annual Meeting of the American Society of Hematology**

Boston, Mass. – November 20, 2012 – Acetylon Pharmaceuticals Inc., a leader in targeted epigenetic drug discovery and development for enhanced therapeutic outcomes, today announced that its selective histone deacetylase (HDAC) inhibitors will be featured in five posters and one oral presentation at the 54th Annual Meeting of the American Society of Hematology (ASH), taking place December 8-11, 2012, in Atlanta, Georgia. These presentations will highlight three of Acetylon's development programs: ACY-1215, a selective HDAC6 inhibitor currently in two Phase 1b clinical trials for the treatment of relapsed or relapsed/refractory multiple myeloma, preclinical studies of ACY-1215 for the treatment of lymphoma, and a selective HDAC1/2 inhibitor in preclinical studies for sickle cell disease and beta-thalassemia.

"Acetylon is broadening its impact in epigenetic drug discovery and development with the expansion of ACY-1215 into lymphoma and the addition of an HDAC1/2 inhibitor in development for sickle cell disease and beta-thalassemia," said Walter C. Ogier, President and Chief Executive Officer and co-founder of Acetylon. "The lymphoma program is supported by three presentations at ASH from leading academic research groups, who demonstrate ACY-1215's synergistic effects with various other anti-cancer drugs in preclinical studies of lymphoma. The potential ability of ACY-1215 to enhance the activity of multiple anti-cancer drugs in multiple myeloma and lymphoma is highly encouraging for clinical development in these indications."

The details of the presentations are as follows:

Multiple Myeloma

Date: Monday, December 10, 2012

Time: 7:45 am ET (oral presentation)

Location: Thomas Murphy Ballroom 4

Session: 652. Myeloma – Pathophysiology and Pre-Clinical Studies, excluding Therapy: Modulating the Microenvironment in Multiple Myeloma

Abstract #: 328

Title: *Role of Selective HDAC6 Inhibition on Multiple Myeloma Bone Disease*

Authors Affiliated with the Following Institutions: Massachusetts General Hospital, Duke University, Harvard Medical School, Dana-Farber Cancer Institute, Beth Israel Deaconess Medical Center, Harvard School of Dental Medicine and Acetylon Pharmaceuticals Inc.

Description: *In vitro* data and the *in vivo* results from the xenograft models of human multiple myeloma in SCID mice, as well as data in HDAC6 genetic knock-out mice, indicate a potential beneficial role of HDAC6 inhibition on multiple myeloma-related bone disease.

Date: Monday, December 10, 2012

Time: 6:00-8:00 pm ET

Location: Hall B1-B2

Session: 653. Myeloma – Therapy, excluding Transplantation: Poster III

Abstract #: 4061

Title: *Rocilinostat (ACY-1215), a Selective HDAC6 Inhibitor, Alone and in Combination with Bortezomib in Multiple Myeloma: Preliminary Results from the First-in-Humans Phase I/II Study*

Authors Affiliated with the Following Institutions: Massachusetts General Hospital, Medical College of Wisconsin, University of Pennsylvania Perelman School of Medicine, Mount Sinai School of Medicine, University of Texas MD Anderson Cancer Center, Harvard Medical School, Rho Inc, Emory University and Acetylon Pharmaceuticals Inc.

Description: Preliminary results from this first clinical evaluation of ACY-1215 suggest that selective inhibition of HDAC6 with ACY-1215, alone or in combination with bortezomib and dexamethasone, may provide a well-tolerated treatment option for relapsed or relapsed/refractory multiple myeloma.

Lymphoma

Date: Saturday, December 8, 2012

Time: 5:30-7:30 pm ET

Location: Hall B1-B2

Session: 625. Lymphoma – Pre-Clinical – Chemotherapy and Biologic Agents: Poster I

Abstract #: 1660

Title: *Combination of ACY-1215, a Selective Histone Deacetylase 6 (HDAC6) Inhibitor with the Bruton Tyrosine Kinase (BTK) Inhibitor, Ibrutinib, Represents a Novel Therapeutic Strategy in Mantle Cell Lymphoma (MCL)*

Authors Affiliated with the Following Institutions: H. Lee Moffitt Cancer Center & Research Institute, Weill Cornell Medical College and Acetylon Pharmaceuticals Inc.

Description: The viability of mantle cell lymphoma (MCL) cells was decreased when they were treated *in vitro* with either PCI-32765 or ACY-1215. However, combination of these two agents resulted in a 3-fold increase in apoptosis induction, pointing to a synergistic effect of BTK and HDAC6 inhibition in MCL. The additional findings that this approach can increase the immunogenicity of MCL cells and anti-MCL immune responses has provided the proper framework for combining the selective HDAC6 inhibitor ACY-1215 with BTK inhibition as a novel therapeutic strategy in MCL.

Date: Saturday, December 8, 2012

Time: 5:30-7:30 pm ET

Location: Hall B1-B2

Session: 625. Lymphoma – Pre-Clinical – Chemotherapy and Biologic Agents: Poster I

Abstract #: 1650

Title: *Dual Targeting of Protein Degradation Pathways with the Selective HDAC6 Inhibitor Rocilinostat (ACY-1215) and Bortezomib, Demonstrates Synergistic Antitumor Activity in Preclinical Models of Lymphoma*

Authors Affiliated with the Following Institutions: Columbia University Medical Center and Acetylon Pharmaceuticals Inc.

Description: These are the first results to indicate that a selective HDAC inhibitor can have marked activity across a panel of lymphoma cell lines. These findings raise the prospect that dual targeting of the ubiquitin-proteasome and aggresomal protein degradation pathways can be synergistically effective. They provide excellent pre-clinical rationale for expanding the use of ACY-1215 in combination with bortezomib for patients with relapsed or refractory lymphoma.

Date: Sunday, December 9, 2012

Time: 6:00-8:00 pm ET

Location: Hall B1-B2

Session: 625. Lymphoma – Pre-Clinical – Chemotherapy and Biologic Agents: Poster II

Abstract #: 2765

Title: *The Irreversible Proteasome Inhibitor Carfilzomib Interacts Synergistically with the Selective HDAC6 Inhibitor ACY-1215 in ABC- and GC-DLBCL and Mantle Cell Lymphoma Sensitive or Resistant to Bortezomib*

Authors Affiliated with the Following Institutions: Virginia Commonwealth University and University of Rochester

Description: These findings indicate that combining the selective HDAC6 inhibitor ACY-1215 with carfilzomib (CFZ) synergistically in preclinical models induces apoptosis in low- and high-risk diffuse large B-cell lymphoma (DLBCL) and in mantle cell lymphoma (MCL) cells through a JNK-dependent process in association with G₂M arrest, down-regulation of HR23B, and induction of DNA damage. They also suggest that this strategy, which is active against sensitive as well as bortezomib-resistant DLBCL and MCL cells, warrants further exploration in NHL.

Sickle Cell Disease/Beta-Thalassemia

Date: Monday, December 10, 2012

Time: 6:00-8:00 pm ET

Location: Hall B1-B2

Session: 112. Thalassemia and Globin Gene Regulation: Poster III

Abstract #: 3259

Title: *Induction of Human Fetal Hemoglobin Expression by Selective Inhibitors of Histone Deacetylase 1 and 2 (HDAC1/2)*

Authors Affiliated with the Following Institutions: Dana-Farber Cancer Institute, Massachusetts General Hospital and Acetylon Pharmaceuticals Inc.

Description: These results suggest that inhibition of HDAC1 and 2 is sufficient to induce fetal globin expression. Our selective HDAC1/2 inhibitors have highly favorable oral pharmacokinetic profiles suitable for further development towards the treatment of SCD and beta-thalassemia.

About ACY-1215

Blood cancers such as multiple myeloma and lymphoma are characterized by successive genetic mutations resulting in uncontrolled cell proliferation and dysfunctional production of intracellular proteins. ACY-1215 selectively inhibits the intracellular enzyme HDAC6, which leads to an accumulation of excess protein and in addition may disrupt critical proliferative signals in malignant cells. Disruption of these molecular processes in cancer cells triggers programmed cell death, called "apoptosis," with little or no effect on normal cells. Currently available HDAC drugs affect the expression of numerous genes in normal cells as well as cancer cells, which can result in side effects such as gastrointestinal dysfunction, lowered blood platelet levels and risk of hemorrhage and profound fatigue as well as potential for significant cardiac complications. Selective inhibition of HDAC6 is expected to reduce or eliminate these often-severe side effects associated with non-selective HDAC inhibition and may enable the development of optimized treatment regimens, including maximally effective combination drug therapies.

About HDAC1/2 Inhibition

The induction of fetal hemoglobin (HbF) is an established therapeutic strategy for sickle cell disease and could potentially also be effective for beta-thalassemia. HDAC inhibition has been shown to induce HbF, however, clinical development of non-selective HDAC inhibitors has been limited due to the number of off-target side effects. Selective HDAC1/2 inhibition represents a novel treatment approach that could represent a safer and more effective treatment option for patients with sickle cell disease and beta-thalassemia.

About Acetylon

Acetylon Pharmaceuticals Inc. is the leader in the development of novel small molecule drugs targeting epigenetic mechanisms for the enhanced therapeutic outcome of cancer and other critical unmet medical needs. The Company's epigenetic drug discovery platform has initially yielded a proprietary library of optimized, orally-administered Class II histone deacetylase (HDAC)-selective compounds. Restoration of proper HDAC regulation through highly-selective HDAC inhibition is thought to be applicable to a broad range of diseases, including cancer, sickle cell disease, beta-thalassemia and autoimmune diseases. Acetylon's lead drug candidate, ACY-1215, is a selective HDAC6 inhibitor in clinical development for the treatment of multiple myeloma.

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