Data Published in the Proceedings of the National Academy of Sciences highlight

The Potential of Cyclacel's targeted CDK inhibitors in breast cancer
-Targeting CDK2/cyclin E with CYC065 Overcomes Herceptin® Resistance in HER2 Positive Breast Cancer -

Berkeley Heights, NJ, February 17, 2011 – Cyclacel Pharmaceuticals, Inc. (NASDAQ: CYCC, NASDAQ: CYCCP; Cyclacel or the Company), today announced the publication of preclinical data in the Proceedings of the National Academy of Sciences (PNAS), demonstrating that cyclin E plays a major role in making Human Epidermal growth factor Receptor 2 positive (HER2+) breast cancer resistant to trastuzumab (Herceptin®), a widely used medicine for breast cancer patients who test positive for HER2. The publication provides a rationale for exploring Cyclacel's orally available CDK inhibitors in this patient population.

Elevated expression levels of cyclin E in HER2+ patients treated with trastuzumab resulted in a reduced rate of clinical benefit and lower survival compared with patients whose cancers did not overexpress cyclin E. Treatment of HER2+ breast cancer cells resistant to trastuzumab with CYC065, Cyclacel's cyclin-dependent kinase (CDK) inhibitor, blocked CDK2/cyclin E activity, dramatically slowed tumor growth and killed resistant breast cancer cells.

"For over a decade Cyclacel has emerged as a leader in the study of cell cycle biology and the identification of novel anticancer drugs that exploit mechanisms of cell cycle control. We are excited about CYC065's promising activity in breast cancer resistant to trastuzumab, one of the mainstay therapies for early-stage breast cancer," said Spiro Rombotis, Cyclacel's President & Chief Executive Officer. "The publication by a leading breast cancer group extends and supports previous reports showing that targeting CDK2/cyclin E with Cyclacel's novel CDK inhibitors, seliciclib and CYC065, can kill cancer cells, such as breast and lung cancer, that fail to respond to current standards of care."

The CDK2 enzyme and its partner protein, cyclin E, have been extensively investigated as therapeutic targets in light of their frequent deregulation in breast cancers with a poor prognosis. In the PNAS article titled "*Cyclin E amplification/overexpression is a mechanism of trastuzumab resistance in HER2 positive breast cancer patients*," investigators from Massachusetts General Hospital Cancer Center, (Boston, MA), Vall d'Hebron University Hospital (Barcelona, Spain), Memorial Sloan-Kettering Cancer Center (New York, NY) and Manitoba Institute of Cell Biology (Winnipeg, Canada) performed a genome-wide analysis to pinpoint causes of resistance in HER2+ breast cancer. Their analysis identified cyclin E as the common genetic signature of resistance to trastuzumab in HER2+ breast cancer cells. Cyclacel's CYC065, a second generation CDK inhibitor, was found to be effective in killing trastuzumab-resistant breast cancer cells in vitro and in vivo. The mechanism of action of CYC065 included inhibition of CDK2/cyclin E, cell cycle arrest and induction of cell death by apoptosis.

"We have determined that breast cancer cells resistant to therapeutic agents targeting HER2 are highly sensitive to CDK inhibition by CYC065," said Maurizio Scaltriti, Ph.D., research scientist from the Division of Hematology and Oncology of the Massachusetts General Hospital Cancer Center, and first author of the manuscript. "Amplification and overexpression of cyclin E is a mechanism by which breast cancer cells develop resistance to trastuzumab. Modulations of cyclin E levels by genetic means result in different sensitivity towards the anti-HER2 agent. Cyclin E overexpressing, trastuzumab-resistant cells have higher CDK2 activity and are more sensitive to pharmacological inhibition by inhibitors, such as seliciclib or its more potent derivative, CYC065. CYC065 has promising in vivo activity in xenograft models of resistant cells, and this activity appears to be enhanced by the addition of trastuzumab."

An estimated 15 to 20 percent of breast cancers have an amplification of the HER2/neu gene or overexpression of its protein product, which results in an aggressive tumor phenotype and reduced survival. HER2 targeted agents such as trastuzumab are highly effective in adjuvant and metastatic breast cancer. However clinical effectiveness is strongly diminished by primary or acquired resistance. Identification and targeting the causative mechanisms for such resistance, such as CDK2/cyclin E activation, may have a significant impact in improving therapeutic outcomes in HER2+ breast cancer patients. In a small retrospective study the authors of the PNAS article demonstrated that approximately 35% of HER2+ breast tumors had overexpressed/amplified cyclin E and this correlated with decreased sensitivity to trastuzumab. Frequently, cyclin E overexpression was not associated with other mechanisms of trastuzumab resistance underscoring the potential contribution that CDK inhibitors, such as CYC065 and seliciclib, may confer in this patient population.

Cyclacel continues to collaborate with the scientific team led by José Baselga, M.D., Ph.D., Chief of the Division of Hematology/Oncology and Associate Director of the Massachusetts General Hospital Cancer Center in order to further validate the therapeutic potential of CDK inhibitors in a cyclin E-mediated, trastuzumab-resistant patient population.

A copy of the publication is accessible from the PNAS website: www.pnas.org.

About Cyclacel's CDK Inhibitors

Seliciclib is an orally-available molecule that selectively targets multiple CDKs, in particular CDK2, CDK7 and CDK9, that are central to the process of cell division and cell cycle control. Seliciclib has been evaluated to date in approximately 380 patients and is currently being evaluated in randomized Phase 2 trials in patients with previously treated lung cancer and nasopharyngeal cancer.

CYC065 is a novel, orally available, cell cycle kinase inhibitor currently in investigational new drug (IND)-enabling studies. CYC065 and other compounds in a related series target the same key CDK/cyclin complexes, which are targeted by seliciclib. CYC065 retains the specificity and mechanism of action of seliciclib, but has increased anti-proliferative potency and improved pharmaceutical properties. Cyclacel has developed CYC065 and other novel derivatives of seliciclib in collaboration with the Cancer Research UK Centre for Cancer Therapeutics at The Institute of Cancer Research (ICR), London, UK.

About CDKs and cyclins

Cyclin-dependent kinases (CDKs) are a group of signalling molecules that play a direct role in the regulation and progression of the cell cycle. CDK activity is dependent on the availability of their regulatory subunits called cyclins. Production and destruction of cyclins are tightly regulated in coordination with cell cycle progression. Targeting CDK/cyclin macromolecular complexes is an attractive strategy for the design of novel anticancer drugs.

In 2001, the Nobel Prize in Physiology and Medicine was awarded for the discovery of cyclins and CDKs, key regulators of the cell cycle. By selectively modulating cell cycle regulation in cancer cells, inhibition of CDK/cyclin complexes represents a promising strategy for cancer therapy. For example Cyclacel's seliciclib (CYC202, R-roscovitine), a novel, first-in-class, orally available CDK inhibitor, currently in Phase 2 clinical trials, selectively targets multiple CDKs, and in particular CDK2, CDK7 and CDK9. Seliciclib also induces apoptosis in neutrophil granulocytes that mediate inflammation, indicating that CDK inhibitors may also hold promise in applications outside oncology, such as the treatment of chronic autoimmune and inflammatory diseases, such as arthritis or asthma.

About Cyclacel Pharmaceuticals, Inc.

Cyclacel is a biopharmaceutical company developing oral therapies that target the various phases of cell cycle control for the treatment of cancer and other serious diseases. Sapacitabine (CYC682), a cell cycle modulating nucleoside analog, is in Phase 3 development for the treatment of acute myeloid leukemia in the elderly under a Special Protocol Assessment agreement with the U.S. Food and Drug Administration, and is in Phase 2 studies for myelodysplastic syndromes and lung cancer. Seliciclib (CYC202 or R-roscovitine), a CDK (cyclin dependent kinase) inhibitor, is in Phase 2 studies for the treatment of lung cancer and nasopharyngeal cancer and in a Phase 1 trial in combination with sapacitabine. Cyclacel's ALIGN Pharmaceuticals subsidiary markets directly in the U.S. Xclair® Cream for radiation dermatitis, Numoisyn® Liquid and Numoisyn® Lozenges for xerostomia. Cyclacel's strategy is to build a diversified biopharmaceutical business focused in hematology and oncology based on a portfolio of commercial products and a development pipeline of novel drug candidates. Please visit www.cyclacel.com for additional information.

Forward-looking Statements

This news release contains certain forward-looking statements that involve risks and uncertainties that could cause actual results to be materially different from historical results or from any future results expressed or implied by such forward-looking statements. Such forward-looking statements include statements regarding, among other things, the efficacy, safety, and intended utilization of Cyclacel's product candidates, the conduct and results of future clinical trials, plans regarding regulatory filings, future research and clinical trials and plans regarding partnering activities. Factors that may cause actual results to differ materially include the risk that product candidates that appeared promising in early research and clinical trials do not demonstrate safety and/or efficacy in larger-scale or later clinical trials, the risk that Cyclacel will not obtain approval to market its products, the risks associated with reliance on outside financing to meet capital requirements, and the risks associated with reliance on collaborative partners for further clinical trials, development and commercialization of product candidates. You are urged to consider statements that include the words "may," "will," "would," "could," "should," "believes," "estimates," "projects," "potential," "expects," "plans," "anticipates," "intends," "continues," "forecast," "designed," "goal," or the negative of those words or other comparable words to be uncertain and forward-looking. For a further list and description of the risks and uncertainties the Company faces, please refer to our most recent Annual Report on Form 10-K and other periodic and current filings that have been filed with the Securities and Exchange Commission and are available at www.sec.gov. Such forward-looking statements are current only as of the date they are made, and we assume no obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise.

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