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Personalized Medicine Potential of Cyclacel's Innovative and Diverse Oncology Pipeline Highlighted at NCRI Cancer Conference

Translational Findings Highlight Combination Potential of Sapacitabine in BRCA-Defective Cancers; Clinical Trials Ongoing

BERKELEY HEIGHTS, N.J., Nov. 6, 2012 (GLOBE NEWSWIRE) -- Cyclacel Pharmaceuticals, Inc. (Nasdaq:CYCC) (Nasdaq:CYCCP) (Cyclacel or the Company), announced today multiple posters presented on the Company's sapacitabine and on its Polo-Like Kinase 1 (Plk1) inhibitors during the 8th National Cancer Research Institute (NCRI) Cancer Conference being held November 4-7, 2012, Liverpool, United Kingdom.

Sapacitabine:

"Sapacitabine efficacy is enhanced in homologous recombination defective tumours"
Date/Time: Tuesday November 6, 2012, 8:30 — 17:30 Greenwich Mean Time
Poster Number: B21

Cyclacel researchers reported that in vitro studies of sapacitabine's active metabolite CNDAC showed that sapacitabine may be effective in patients with mutations of the breast cancer susceptibility proteins BRCA1/2 or homologous recombination repair (HRR)-deficient tumors, such as subsets of triple negative breast, ovarian, colon and non-small cell lung cancer (NSCLC). In addition, CNDAC synergized with either PARP inhibitors or cisplatin in both NSCLC and ovarian cancer cell lines. Cyclacel's cyclin-dependent kinase (CDK) inhibitor seliciclib reduces expression of BRCA1 and BRCA2 and can potentiate sapacitabine/CNDAC activity, as well as other agents enhanced in double strand break (DSB) repair-defective backgrounds. Sapacitabine treatment in combination with seliciclib is currently under investigation in a Phase 1 trial in patients with solid tumors at the Dana Farber Cancer Institute (Boston, MA).

"Therapeutic potential of sapacitabine in cancers defective in homologous recombination"
Date/Time: Tuesday November 6, 2012, 8:30 — 17:30 Greenwich Mean Time
Poster Number: B207

Cyclacel collaborators from the Northern Institute for Cancer Research, Newcastle University, UK reported confirmation that ovarian and breast cancer cell lines defective in HRR are highly sensitive to CNDAC, supporting a possible role for sapacitabine/CNDAC in HRR-defective diseases. Primary ovarian cancer samples were also highly sensitive to CNDAC, and correlation of HRR status with CNDAC sensitivity is being assessed.

The studies further support the potential for sapacitabine to be used alone or in combinations to treat HRR- defective tumors, such as ATM- or BRCA-defective tumors. An investigator-sponsored Phase 2 study of sapacitabine in chronic lymphocytic leukemia (CLL) and small lymphocytic lymphoma (SLL) with deletion 11q22-23 is ongoing at The University of Texas MD Anderson Cancer Center (Houston, TX). A Phase 1, study of sapacitabine in combination with seliciclib in patients with advanced solid tumors, is ongoing at the Dana Farber Cancer Institute (Boston, MA). As reported at the American Society of Clinical Oncology 2012 annual meeting among 19 patients in this study treated at the recommended Phase 2 doses, 3 with advanced breast, ovarian and pancreatic cancer achieved partial responses (PRs) and 1 with ovarian cancer showed stable disease. All 4 responding patients were reported by the investigator to be BRCA defective.

Polo-Like Kinase 1 (Plk1) inhibitors:

"Parameters improving the therapeutic window of Compound 4, a potent and selective Polo-like kinase 1 inhibitor: in vitro studies"
Date/Time: Tuesday November 6, 2012, 8:30 — 17:30 Greenwich Mean Time
Poster Number: B15

Cyclacel scientists and academic collaborators reported the biological characterization of Compound 4, a potent and selective, preclinical-stage, Plk1 inhibitor, selected for further development from Cyclacel's novel Plk1 inhibitor series. In a panel of esophageal cancer cell lines, sensitivity to Compound 4 correlated with p53 status. Esophageal cell lines lacking functional p53 showed the greatest sensitivity to Compound 4. Short drug exposure times demonstrated differential sensitivity between

cancerous esophageal cells versus control, outlining the potential broad therapeutic index for Compound 4 in treating esophageal cancers, and in particular those with non-functional p53. Status of p53 could be used as a predictive biomarker in clinical trials to identify responders.

About sapacitabine

Sapacitabine (CYC682), an orally-available nucleoside analogue, is in the SEAMLESS, registration-directed, Phase 3 trial in elderly patients with newly diagnosed acute myeloid leukemia (AML) who are unfit or have refused intensive chemotherapy. In addition, sapacitabine is in Phase 2 trials in patients with hematological malignancies, including AML, myelodysplastic syndromes (MDS), cutaneous T-cell lymphoma (CTCL), chronic lymphocytic leukemia (CLL) and small lymphocytic lymphoma (SLL), and non-small cell lung cancer (NSCLC), and a Phase 1 trial in combination with seliciclib in patients with advanced solid tumors. Sapacitabine acts through a novel DNA single-strand breaking mechanism, leading to production of DNA double strand breaks (DSBs) and/or checkpoint activation. Unrepaired DSBs cause cell death. Repair of sapacitabine-induced DSBs is dependent on the homologous recombination DNA repair (HRR) pathway. Both sapacitabine and CNDAC, its major metabolite, have demonstrated potent anti-tumor activity in preclinical studies.

Over 500 patients have received sapacitabine in Phase 2 studies in AML, MDS, CTCL and NSCLC and Phase 1 studies in both hematological malignancies and solid tumors. At the 2009 Annual Meeting of the American Society of Hematology (ASH), Cyclacel reported data from a randomized Phase 2, single-agent study of sapacitabine including promising 1-year survival in elderly patients with AML aged 70 years or older. At the 2011 Annual Meeting of the American Society of Clinical Oncology (ASCO), Cyclacel reported data from a pilot Phase 1/2 study including promising response rate, low 4-week and 8-week mortality in elderly patients with AML aged 70 years or older receiving sapacitabine alternating with decitabine. The FDA and the European Medicines Agency have designated sapacitabine as an orphan drug for the treatment of both AML and MDS. Sapacitabine is part of Cyclacel's pipeline of small molecule drugs designed to target and stop uncontrolled cell division.

About Plk1 (Polo-Like Kinase 1)

Polo-like kinases are enzymes that were first discovered in the fruit fly model of human cancer by Prof. David Glover, Cyclacel's Chief Scientist. Professor Glover is a world authority on Aurora kinases, Polo kinases and related mechanisms controlling cell division. Activity of the mitotic kinase Plk1 is strongly associated with cancer progression. Several studies have shown correlations between elevated Plk1 expression, histological grade and poor prognosis in several types of cancer.[1] Plk1 may have a role in oncogenesis through its regulation of tumor suppressors such as p53 and BRCA2. The inhibition of Plk1 by small molecules or siRNA has been shown to interfere with several stages of mitosis. Therefore Cyclacel's Plk1 inhibitors may represent an opportunity to treat cancer with a targeted anti-mitotic approach that will inhibit several important regulatory events in tumor cells.

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. The Company's most advanced oral product candidate, sapacitabine, is the subject of SEAMLESS, a Phase 3 trial being conducted under an SPA with the FDA as front-line treatment of acute myeloid leukemia (AML) in the elderly and Phase 2 studies for AML, myelodysplastic syndromes (MDS), chronic lymphocytic leukemia (CLL) and solid tumors including breast, lung, ovarian and pancreatic cancer. Cyclacel's pipeline includes seliciclib, a CDK inhibitor, in Phase 2 for lung and nasopharyngeal cancer and in Phase 1 in combination with sapacitabine; and CYC065, a second generation CDK inhibitor, in IND-directed development. Cyclacel's strategy is to build a diversified biopharmaceutical business focused in hematology and oncology based on 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, trials may have difficulty enrolling, Cyclacel may not obtain approval to market its product candidates, 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 other filings we file 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.

[1]Kanaji et al, Oncology 2006:70:126. Weichert et al, Cancer Sci. 2006:97:271. Yamada et al, Oncogene 2004:23: 5901.

CONTACT: Investors/Media: Corey Sohmer

(908) 517-7330

csohmer@cyclacel.com