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Data Presented at AACR Meeting Shows Peregrine's Selective Anti-VEGF Antibodies are as Effective as Avastin(R) in Preclinical Cancer Models

- Peregrine's Selective Anti-VEGF Antibodies Inhibited Tumor Growth By 90% In a Preclinical Cancer Model
- Peregrine's Antibodies that Selectively Inhibit VEGF Receptor 2 Could Potentially Have Advantages over Non-Selective Approaches
- These Results Along with Data Presented on a Fully Human Antibody Support Advancing Peregrine's Anti-VEGF Program towards Clinical Trials

LOS ANGELES and TUSTIN, Calif., April 16 /PRNewswire-FirstCall/ -- Peregrine Pharmaceuticals, Inc. (Nasdaq: PPHM) a clinical stage biopharmaceutical company developing targeted monoclonal antibodies for the treatment of cancer and hepatitis C virus (HCV) infection, today reported that preclinical data presented at the Centennial Annual Meeting of the American Association for Cancer Research (AACR) showed that its monoclonal antibody 2C3, which selectively blocks vascular endothelial growth factor (VEGF) from binding to the second of the two VEGF receptors, demonstrates potent anti-cancer efficacy in a preclinical orthotopic model of pancreatic cancer. In this model, treatment with 2C3 reduced the growth of pancreatic tumors by 90%. The researchers also assessed the anti-tumor activity of 2C3 and the marketed anti-cancer agent Avastin® in a number of preclinical cancer models. The anti-tumor activity of 2C3, which only blocks VEGF receptor 2 (VEGFR2) compared favorably to that of Avastin, which blocks VEGF binding to both VEGF receptor 1 (VEGFR1) and VEGFR2.

VEGF is a primary stimulant of the development and maintenance of the blood vessels needed by tumors to survive and grow. It is thought to work by binding to and activating two receptors that are expressed on endothelial and other types of cells. The series of studies reported today tested the hypothesis that selectively inhibiting VEGFR2 is an equally effective anti-tumor strategy to blocking both receptors. Most of the anti-angiogenic agents currently being developed or marketed for cancer, such as Avastin, block the activity of both VEGF receptors.

"The success of Avastin underscores the potential of blocking VEGF as a major strategy for fighting solid cancers," said Steven W. King, president and CEO of Peregrine. "The finding that our selective anti-VEGF antibody demonstrates potent anti-tumor effects in preclinical models of pancreatic cancer is important both for our understanding of the underlying physiology of cancer and because selective VEGF inhibitors may ultimately prove to have advantages compared to non-selective approaches. Now that we have also demonstrated that a fully human anti-VEGF antibody (R3) is comparable to our earlier murine versions, we intend to accelerate our anti-VEGF program, with the goal of initiating clinical trials as soon as next year."

In support of the observed anti-tumor effects of 2C3, the researchers reported that tumor-associated macrophages, immune system cells found in the tumors and peritoneal cavities of these animals, preferentially express VEGFR2, which appears to dominate the VEGF signaling that promotes tumor progression and metastasis in this model of pancreatic cancer.

In this pancreatic cancer model, 2C3 significantly reduced the metastatic spread of tumor cells as monotherapy and also augmented the anti-tumor activity of chemotherapy when administered in a combination regimen. In addition, when given for a prolonged period, 2C3 produced decreased microvessel density, decreased expression of the VEGFR2 receptor and decreased tumor-associated macrophage invasion, which was consistent with the finding that the VEGFR2 receptor dominates VEGF-induced macrophage recruitment into tumors.

The researchers also tested an equivalent fully human monoclonal antibody, R3, in similar pancreatic cancer models, where it significantly reduced the growth, vascularization and macrophage infiltration of pancreatic tumors in mice. These positive results using a fully human antibody support Peregrine's intent to advance its anti-VEGF program towards clinical trials.

This research, which was conducted under the direction of Dr. Rolf Brekken, assistant professor of surgery at UT Southwestern Medical Center in Dallas, was supported in part by a sponsored research agreement with Peregrine Pharmaceuticals. The fully human antibody R3 used in these studies was developed in collaboration with Affitech AS of Norway. These studies were also supported in part by a postdoctoral fellowship from the Susan G. Komen for the Cure, the Effie Marie Cain Scholarship in Angiogenesis Research and the Department of Surgery at UT Southwestern.

Number 2128: Blockade of tumor-derived VEGF activation of VEGF receptor 2 reduces macrophage infiltration into tumors and decreases metastasis in a pre-clinical orthotopic model of pancreatic cancer, Juliet G. Carbon, Shane E. Holloway, Andrew F.

Miller, Anita Kavlie, Kyle Schlunegger, Jason B. Fleming, Rolf A. Brekken. UT Southwestern Medical Ctr., Dallas, TX, Affitech AS, Oslo, Norway, Peregrine Pharmaceuticals Inc., Tustin, CA, UT-MD Anderson, Houston, TX, Apr 16, 2007, 8:00 AM -12:00 PM PDT

About Peregrine Pharmaceuticals

Peregrine Pharmaceuticals, Inc. is a biopharmaceutical company with a portfolio of innovative product candidates in clinical trials for the treatment of cancer and hepatitis C virus (HCV) infection. The company is pursuing three separate clinical programs for HCV infection and a range of solid cancers in the U.S. and India with its lead product candidates bavituximab and Cotara®. Peregrine also has in-house manufacturing capabilities through its wholly owned subsidiary Avid Bioservices, Inc. (www.avidbio.com), which provides development and bio-manufacturing services for both Peregrine and outside customers. Additional information about Peregrine can be found at www.peregrineinc.com.

Safe Harbor Statement: Statements in this press release which are not purely historical, including statements regarding Peregrine Pharmaceuticals' intentions, hopes, beliefs, expectations, representations, projections, plans or predictions of the future are forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. The forward-looking statements involve risks and uncertainties including, but not limited to, the risk that pre-clinical mouse model results for pancreatic cancer will not correlate to efficacy studies in human clinical trials for pancreatic or other solid tumor cancers, the risk that selective VEGF inhibitors will not prove to have advantages compared to non-selective approaches, and the risk that the Company may experience delays in initiating clinical trials using its 2C3 antibody. It is important to note that the company's actual results could differ materially from those in any such forward-looking statements. Factors that could cause actual results to differ materially include, but are not limited to, uncertainties associated with completing preclinical and clinical trials for our technologies; the early stage of product development; the significant costs to develop our products as all of our products are currently in development, preclinical studies or clinical trials; obtaining additional financing to support our operations and the development of our products; obtaining regulatory approval for our technologies; anticipated timing of regulatory filings and the potential success in gaining regulatory approval and complying with governmental regulations applicable to our business. Our business could be affected by a number of other factors, including the risk factors listed from time to time in the Company's SEC reports including, but not limited to, the annual report on Form 10-K for the year ended April 30, 2006 and the quarterly report on Form 10-Q for the quarter ended January 31, 2007. The Company cautions investors not to place undue reliance on the forward-looking statements contained in this press release. Peregrine Pharmaceuticals, Inc. disclaims any obligation, and does not undertake to update or revise any forward-looking statements in this press release

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