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Peregrine's Vascular Targeting Agent Inhibits Growth of Breast Cancer Tumor Metastases by 50 Percent in Pre-Clinical Studies

Pre-Clinical Data Was Presented at the American Association of Cancer Research Annual Meeting

TUSTIN, Calif., Jul 14, 2003 /PRNewswire-FirstCall via COMTEX/ -- Peregrine Pharmaceuticals, Inc. (Nasdaq: PPHM) today announced that researchers at The University of Texas Southwestern Medical Center at Dallas and the University of Texas MD Anderson Cancer Center have demonstrated in animal models that a fusion protein comprised of vascular endothelial growth factor (VEGF) with a toxin (gelonin) inhibited the growth of breast cancer metastatic tumors by 50 percent. The VEGF construct is part of a Vascular Targeting Agent (VTA) compound family that Peregrine licensed to SuperGen, Inc. (Nasdaq: SUPG) in February 2001.

The study, which was presented at the American Association of Cancer Research's Annual Meeting, involved administering VEGF121/rGelonin (VEGF/rGel) in mice injected with human breast cancer cells. The study showed that in the VEGF121/rGel-treated group the number lung metastases was reduced by 50 percent over control and the mean area of the lung metastases was reduced by 58 percent over control. Approximately 62 percent of metastatic colonies from the VEGF121/rGel-treated group had fewer than 10 vessels per colony as compared to 23 percent in the control group. The study also showed the treatment was well tolerated. No significant morphological changes were visible in either VEGF121/rGel-treated or gelonin control mice. These data strongly suggest that anti-tumor vascular effect of VEGF121/rGel could be utilized not only for treating primary tumors but also for inhibiting metastatic spread.

Similar data from these studies was recently published in the Proceedings of the National Academy of Sciences, which involved administering VEGF121/rGelonin (VEGF/rGel) in mice injected with human melanoma and human prostate cancer cells. Researchers found that tumors in mice treated with VEGF/rGel had been reduced by up to 84 percent compared to tumors in untreated mice. Furthermore, VEGF/rGel selectively destroyed blood vessels supplying human solid tumors without harming the vasculature of normal tissue.

VEGF is one of the predominant factors responsible for angiogenesis -- the ability of a tumor to create new blood vessels to maintain growth and metastasize. The receptors for VEGF are over-expressed on the endothelium of tumor vasculature (tumor blood vessels) but are almost undetectable in the adjacent normal tissue, so they make excellent targets for the development of therapeutic agents that inhibit tumor growth and metastatic spread by inhibiting the new blood vessel formation.

"The anti-tumor effects of the VEGF/rGel fusion construct against breast cancer metastases in animal models were impressive," said Dr. Philip Thorpe, professor of pharmacology at UT Southwestern, and the inventor of the VTA technology. "These studies suggest that VEGF/rGel has significant potential as an anti-tumor agent for treating cancer patients."

About Vascular Target Agents -- The Next Generation of Cancer Therapy

Virtually all detectable tumors rely on a vascular network to obtain oxygen and nutrients. Disruption of this network can have a devastating effect on a tumor. In pre-clinical animal studies, VTAs have shown to be potent anti-cancer agents that act by cutting off the supply of oxygen and nutrients to tumor cells by causing blood clots to form within the tumor's blood supply network. VTAs localize within the tumor vasculature by selectively binding to the flat endothelial cells that line tumor blood vessels. Once the VTA binds to its target, it initiates thrombosis (blood clotting) through a coagulation cascade, which leads to complete clotting of the tumor blood vessels within a matter of minutes. Because blockage of a single capillary results in the destruction of thousands of tumor cells, only a small quantity of VTAs localized in the tumor's vascular system may cause an avalanche of tumor cell death.

Vascular targeting agents offer several advantages as potentially powerful anti-cancer treatments. By targeting receptors unique to tumor cell vasculature, VTAs can kill tumors by cutting off oxygen and nutrients without causing damage to surrounding healthy tissue. Additionally, VTAs reduce the risk of potential side effects by operating at lower dosages than traditional cancer therapies because they do not need to penetrate the innermost layer of a tumor to take effect. Lastly, while drug resistance caused by the instability and mutability of cancer cells is a significant problem with conventional therapies that target tumor cells, cells targeted by VTAs do not mutate to become drug resistant.

About Peregrine Pharmaceuticals

Peregrine Pharmaceuticals is a biopharmaceutical company focused on the development, commercialization and licensing of unique technologies for the treatment of cancer, primarily based on three collateral targeting technologies. Peregrine's Tumor Necrosis Therapy (TNT), Vasopermeation Enhancement Agents (VEA), and Vascular Targeting Agents (VTA) technologies target cell structures and cell types that are common among solid tumor cancers, giving them broad applicability across various tumor types. The company has received approval from the FDA to start a Cotara™ Phase III clinical trial for brain cancer. Cotara is also being studied in a Phase I trial for colorectal, pancreas, soft tissue sarcoma and biliary cancers at Stanford University. The company is focused on licensing collaborations for all of its technologies under development. The company's Oncolym® technology to treat non-Hodgkin's B-cell lymphoma in Phase I/II of development is available for licensing. The company operates a cGMP contract manufacturing facility for monoclonal antibodies and recombinant proteins through its wholly owned subsidiary Avid Bioservices, Inc. (www.avidbio.com). Copies of Peregrine press releases, SEC filings, current price quotes and other valuable information for investors may be found on the website www.peregrineinc.com .

Safe Harbor Statement: This release may contain certain forward-looking statements that are made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Actual events or results may differ from the company's expectations as a result of risk factors discussed in Peregrine's reports on file with the U.S. Securities and Exchange Commission, including, but not limited to, the company's report on Form 10-K for the year ended April 30, 2002 and on Form 10-Q for the quarter ended January 31, 2003. Investor Relations


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