November 10, 2015

DelMar Pharmaceuticals Collaborators Present Data on the Unique Molecular Mechanisms Responsible for VAL-083 Activity Against Cancer

- Third-party data further validate differentiation of VAL-083 vs. other chemotherapeutic agents -

- VAL-083 activation of immune response pathways may represent a promising personalized medicine approach in the treatment of cancer -

VANCOUVER, British Columbia and MENLO PARK, Calif., Nov. 10, 2015 /PRNewswire/ -- DelMar Pharmaceuticals, Inc. (OTCQX: DMPI) ("DelMar" and the "Company"), a biopharmaceutical company focused on the development and commercialization of new cancer therapies, announced the presentation by its collaborators from the University of British Columbia (UBC) and Vancouver Prostate Centre (VPC) of additional data on the unique molecular signaling events responsible for VAL-083 (*dianhydrogalactitol*) activity against cancer.

"We continue to demonstrate the therapeutic potential of VAL-083 both as a single-agent and in combination with other treatments," stated Jeffrey Bacha, DelMar's president and CEO. "We have previously shown that VAL-083's anti-tumor activity is unaffected by the expression of MGMT, a DNA repair enzyme that is implicated in chemotherapy resistance. To further understand the unique anti-cancer mechanism of VAL-083, we collaborated with researchers from UBC and VPC to study VAL-083's anti-cancer activity on a molecular level."

These new data were presented on November 9th by UBC and VPC researchers in a poster entitled, "*Exploring the Molecular Mechanisms of Dianhydrogalactitol (VAL-083) in Cancer Treatment*," at the 2015 Canadian Cancer Research Conference of the Canadian Cancer Research Alliance (CCRA).

"The study results show a pattern of durability in the DNA lesions caused by VAL-083 which indicate its unique mechanism and potential superiority versus other chemotherapeutic agents. This suggests that VAL-083 is effective at modifying tumor cells and may halt tumor progression by inhibiting natural cellular repair processes," added Mr. Bacha.

"VAL-083's broad anti-cancer activity was established in prior clinical trials sponsored by the U.S. National Cancer Institutes (NCI). Employing modern biological tools to differentiate the mechanisms involved in VAL-083's anti-cancer activity from other chemotherapies provides a basis for future combination treatments as well as guidance for our drug development efforts to concentrate on tumor-types representing significant unmet medical needs," Mr. Bacha stated.

VAL-083 is a bi-functional alkylating agent causing N^7-guanine methylation and interstrand DNA crosslinks and is approved in China as a chemotherapeutic drug for the treatment of chronic myelogenous leukemia and lung cancer. Preclinical studies and clinical trial data suggest that VAL-083 may be active against a range of tumor types via a novel mechanism of action that could provide improved treatment options for patients.

The goal of this study was to further understand the detailed molecular mechanisms mediating VAL-083 sensitivity or resistance in cancer by investigating the signaling events responsible for VAL-083 activity against cancer. The study confirmed a four-fold hypotheses:

1. VAL-083 induces DNA double-strand breaks (DSBs).
2. VAL-083 cytotoxicity is due to cell cycle arrest and apoptosis resulting from DNA cross-linking lesions accumulating in S- and G2-phases of the cell cycle.
3. The antineoplastic effect of VAL-083 is dependent on cancer cells' ability to repair the VAL-083-induced DNA damage.
4. Alterations in DNA damage repair signaling pathway lead to VAL-083 sensitivity or resistance in tumor cells.

Results indicate that treatment of cancer cells by VAL-083 induces phosphorylation of H2AX, a hallmark of double-strand DNA breaks. H2AX is a histone involved in the CHK2 checkpoint activation pathway, a key component of the body's immune response to DNA damage resulting in down-stream signaling ultimately resulting in apoptosis.

"We will continue to explore the signaling pathways involved in VAL-083 for the treatment of cancer," added Mr. Bacha. "The further elucidation of these molecular mechanisms will help to focus our drug development efforts on patients with cancer who would most benefit from VAL-083 treatment. This 'personalized-medicine' approach leverages significant historical clinical data from prior NCI-sponsored studies; which, when juxtaposed against new understanding of VAL-083's biological mechanism, provides an opportunity to accelerate the development of VAL-083 as a new therapy for cancer patients with limited treatment options."

The research presented at the 2015 Cancer Research Conference was funded in part by financial contributions from Canada's National Research Council's Industrial Research Assistance Program (NRC-IRAP) and Mitacs, a Canadian Network of Centres of Excellence, dedicated to supporting scientific and industrial research.

The poster on the molecular mechanisms of VAL-083 may be found on DelMar's website

under http://www.delmarpharma.com/scientific-publications.html.

About VAL-083

VAL-083 is a "first-in-class," small-molecule chemotherapeutic. In more than 40 Phase I and II clinical studies sponsored by the U.S. National Cancer Institute, VAL-083 demonstrated clinical activity against a range of cancers including lung, brain, cervical, ovarian tumors and leukemia both as a single-agent and in combination with other treatments. VAL-083 is approved in China for the treatment of chronic myelogenous leukemia (CML) and lung cancer, and has received orphan drug designation in Europe and the U.S. for the treatment of malignant gliomas.

DelMar has demonstrated that VAL-083's anti-tumor activity is unaffected by the expression of MGMT, a DNA repair enzyme that is implicated in chemotherapy resistance and poor outcomes in GBM patients following standard front-line treatment with Temodar® (temozolomide).

DelMar recently announced the completion of enrollment in a Phase II clinical trial of VAL-083 in refractory GBM. Patients have been enrolled at five clinical centers in the United States: Mayo Clinic (Rochester, MN); UCSF (San Francisco, CA) and three centers associated with the Sarah Cannon Cancer Research Institute (Nashville, TN, Sarasota, FL and Denver, CO).

In the Phase I dose-escalation portion of the study, VAL-083 was well tolerated at doses up to 40mg/m^2 using a regimen of daily x 3 every 21 days. Adverse events were typically mild to moderate; no treatment-related serious adverse events reported at doses up to 40 mg/m^2. Dose limiting toxicity (DLT) defined by thrombocytopenia (low platelet counts) was observed in two of six (33%) of patients at 50 mg/m^2. Generally, DLT-related symptoms resolved rapidly and spontaneously without concomitant treatment, although one patient who presented with hemorrhoids received a platelet transfusion as a precautionary measure.

Sub-group analysis of data from the Phase I dose-escalation portion of the study suggested a dose-dependent and clinically meaningful survival benefit following treatment with VAL-083 in GBM patients whose tumors had progressed following standard treatment with temozolomide, radiotherapy, bevacizumab and a range of salvage therapies.

Patients in a low dose (≤5mg/m^2) sub-group had a median survival of approximately five (5) months versus median survival of approximately nine (9) months for patients in the therapeutic dose (30mg/m^2 & 40mg/m^2) sub-group following initiation of VAL-083 treatment. DelMar reported increased survival at 6, 9 and 12 months following initiation of treatment with VAL-083 in the therapeutic dose sub-group compared to the low dose sub-group.

Further details can be found at http://www.delmarpharma.com/scientific-publications.html.

About DelMar Pharmaceuticals, Inc.

DelMar Pharmaceuticals, Inc. was founded to develop and commercialize new cancer therapies in indications where patients are failing or have become intolerable to modern targeted or biologic treatments. The Company's lead drug in development, VAL-083, is

currently undergoing clinical trials in the U.S. as a potential treatment for refractory glioblastoma multiforme. VAL-083 has been extensively studied by U.S. National Cancer Institute, and is currently approved for the treatment of chronic myelogenous leukemia and lung cancer in China. Published pre-clinical and clinical data suggest that VAL-083 may be active against a range of tumor types via a novel mechanism of action that could provide improved treatment options for patients.

For further information, please visit http://delmarpharma.com/; or contact DelMar Pharmaceuticals Investor Relations: ir@delmarpharma.com / (604) 629-5989. Connect with the Company on Twitter, LinkedIn, Facebook, and Google+. Investor Relations Counsel: Amato & Partners LLC.

Safe Harbor Statement

Any statements contained in this press release that do not describe historical facts may constitute forward-looking statements as that term is defined in the Private Securities Litigation Reform Act of 1995. Any forward-looking statements contained herein are based on current expectations, but are subject to a number of risks and uncertainties. The factors that could cause actual future results to differ materially from current expectations include, but are not limited to, risks and uncertainties relating to the Company's ability to develop, market and sell products based on its technology; the expected benefits and efficacy of the Company's products and technology; the availability of substantial additional funding for the Company to continue its operations and to conduct research and development, clinical studies and future product commercialization; and, the Company's business, research, product development, regulatory approval, marketing and distribution plans and strategies. These and other factors are identified and described in more detail in our filings with the SEC, including, our current reports on Form 8-K.

Logo - https://photos.prnewswire.com/prnh/20150909/265198LOGO

To view the original version on PR Newswire, visit:http://www.prnewswire.com/news-releases/delmar-pharmaceuticals-collaborators-present-data-on-the-unique-molecular-mechanisms-responsible-for-val-083-activity-against-cancer-300175682.html

SOURCE DelMar Pharmaceuticals, Inc.