UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K
CURRENT REPORT
PURSUANT TO SECTION 13 OR 15(d) OF
THE SECURITIES EXCHANGE ACT OF 1934

Date of Report (Date of earliest event reported): November 17, 2017

DELMAR PHARMACEUTICALS, INC.
(Exact name of registrant as specified in its charter)

Nevada 000-54801 99-0360497
(State or other jurisdiction (Commission (I.R.S. Employer
of incorporation) File Number) Identification Number)

Suite 720-999 West Broadway
Vancouver, British Columbia
Canada V5Z 1K5
(Address of principal executive offices) (zip code)

(604) 629-5989
(Registrant's telephone number, including area code)

(Former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐
Item 7.01 Regulation FD Disclosure.

On November 21, 2017, DelMar Pharmaceuticals, Inc. (the “Company”) issued a press release announcing its presentation of positive interim results from a study of VAL-083 in MGMT-unmethylated Recurrent GBM at the conference described below. A copy of the press release is attached hereto as Exhibit 99.1.

The information in this Current Report on Form 8-K under Item 7.01, including the information contained in Exhibit 99.1, is being furnished to the Securities and Exchange Commission, and shall not be deemed to be “filed” for the purposes of Section 18 of the Securities Exchange Act of 1934 or otherwise subject to the liabilities of that section, and shall not be deemed to be incorporated by reference into any filing under the Securities Act of 1933 or the Securities Exchange Act of 1934, except as shall be expressly set forth by a specific reference in such filing.

Item 8.01 Other Events.

On November 17 and November 18, 2017, the Company presented posters at the Annual Scientific Meeting and Education Day of the Society for Neuro-Oncology in San Francisco, California. Copies of the posters are attached as Exhibits 99.2, 99.3 and 99.4 hereto.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

<table>
<thead>
<tr>
<th>Exhibit Number</th>
<th>Description</th>
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<tr>
<td>99.1</td>
<td>Press Release dated November 21, 2017</td>
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<tr>
<td>99.2</td>
<td>Poster</td>
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<td>99.3</td>
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SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

DELMAR PHARMACEUTICALS, INC.

Dated: November 22, 2017

By: /s/ Jeffrey Bacha

Name: Jeffrey Bacha
Title: President and Chief Operating Officer
DelMar Presents Positive Interim Results from VAL-083
Study in MGMT-unmethylated Recurrent GBM at The Society
for NeuroOncology Annual Meeting

40% of recurrent GBM patients treated to date achieved stable disease as measured by magnetic resonance imaging (MRI)

VANCOUVER, British Columbia and MENLO PARK, Calif., November 21, 2017 /PRNewswire/ - - DelMar Pharmaceuticals, Inc. (NASDAQ: DMPI) (“DelMar” or the “Company”), a biopharmaceutical company focused on the development of new cancer therapies, today provided an overview of three scientific posters presented at the 22nd Annual Meeting and Education Day of the Society for Neuro-Oncology (SNO) held on November 16-19, 2017 in San Francisco, CA.

DelMar reported that 93% of patients enrolled were alive at the time of the analysis and 40% of patients enrolled were reported to have achieved stable disease as assessed by MRI following treatment with VAL-083 as a single agent. “While it is too early to interpret overall survival results from this study, the substantial disease control observed to date in the treatment recurrent GBM, an aggressive tumor that can double in size within 6-8 weeks, is an important and positive observation at this stage,” said Mr. Saiid Zarrabian, DelMar’s Interim Chief Executive Officer.

“The promising early observations from our ongoing Phase 2 clinical trial of VAL-083 as a potential new treatment option for MGMT-unmethylated GBM are also supported by extensive preclinical research into VAL-083’s unique mechanism of action,” added Mr. Zarrabian. “Based on these recent data, we believe VAL-083 represents a potential solution for some of the most important unmet medical needs in the treatment of GBM and other central nervous system tumors.”

DelMar provided an update on the company’s ongoing Phase 2 clinical studies in a poster entitled “Clinical Trials with dihydrogalactitol (VAL-083) in MGMT-unmethylated Glioblastoma”, which is being conducted in collaboration with The University of Texas MD Anderson Cancer Center. This trial is designed to enroll up to 48 patients to determine if VAL-083 treatment improves overall survival compared to historical reference control.

- DelMar reported that 27 subjects have been screened and 15 have been enrolled since the opening of recruitment in February 2017. To date, the trial has enrolled at a rate ahead of initial projections.
● All patients enrolled in the study have recurrent MGMT-unmethylated GBM with radiographic evidence of progression and were not surgically resected at the time of enrollment.

● DelMar reported that 93% of patients enrolled were alive at the time of the analysis and 40% of patients enrolled were reported to have achieved stable disease following treatment with VAL-083 as a single agent, as assessed by MRI.

● Enrollment is ongoing and median survival has not yet been reached in the trial.

● In general, VAL-083 treatment was well tolerated by patients with observed side effects (myelosuppression) similar to prior clinical experience.

The Company also provided an overview of the design a separate Phase 2 clinical trial of VAL-083 for newly diagnosed MGMT-unmethylated GBM patients on this poster. In this trial, which was recently initiated at Sun Yat-Sen University Cancer Center, patients will be treated with VAL-083 plus radiotherapy as an alternative to standard-of-care temozolomide plus radiation in the front-line setting. The trial is designed to enroll up to 30 patients with MGMT-unmethylated GBM to determine if VAL-083 treatment improves progression free survival (PFS) compared to a historical reference control. This trial is being supported though DelMar’s collaboration with Guangxi Wuzhou Pharmaceutical (Group) Co., Ltd.

In addition, DelMar also presented two additional pre-clinical posters during the conference:

● The Distinct Cytotoxic Mechanism of Dianhydrogalactitol (VAL-083) Overcomes Chemoresistance and Provides New Opportunities for Combination Therapy in the Treatment of Glioblastoma.

VAL-083 induces potent anti-cancer activity against treatment-resistant cells from glioblastoma, lung, prostate and ovarian tumors through a distinct mechanism of action. Cancer cells treated with VAL-083 exhibit persistent DNA double-strand breaks and activation of the homologous DNA repair (HR) system. Activation of the HR system is an indicator of VAL-083’s unique anti-tumor activity.

When combined with topoisomerase or PARP inhibitors, the treatment effect of VAL-083 is increased in a synergistic or super-additive manner. Taken together, these data support the broad potential of VAL-083 as a new treatment against a wide range of cancers both as a single agent and in combination with other established cancer therapies.

● Dianhydrogalactitol (VAL-083) Overcomes Chemoresistance in Pediatric Malignant Brain Tumors and Displays Synergy with Topoisomerase Inhibitors

Pediatric high-grade glioma (HGG) and medulloblastoma are aggressive childhood brain tumors with a high incidence of recurrence and very few patients achieve long-term survival. VAL-083 demonstrates potent activity as a single agent against both chemo-resistant pediatric HGG and medulloblastoma independent of p53 status. DelMar also reported that VAL-083 potentiates radiotherapy and exhibits synergy when used in combination with topoisomerase inhibitors, two regimens commonly used in the treatment of childhood brain tumors.
“We continue to be highly enthusiastic about the potential of VAL-083 as a novel treatment for cancer patients who have limited or no treatment options”, added Mr. Zarrabian. “The excellent work performed by our world class academic research collaborators and our in-house team presented at the SNO meeting showcases VAL-083’s potential both as a single agent and as a component of combination therapeutic regimens.”

DelMar’s poster presentations can be viewed in their entirety on DelMar’s website at [http://www.delmarpharma.com/scientific-publications.html](http://www.delmarpharma.com/scientific-publications.html).

**About VAL-083**

VAL-083 (dianhydrogalactitol) is a "first-in-class", DNA-targeting agent that introduces interstrand DNA cross-links at the N7-position of guanine leading to DNA double-strand breaks and cancer cell death. VAL-083 has demonstrated clinical activity against a range of cancers including GBM and ovarian cancer in historical clinical trials sponsored by the U.S. National Cancer Institute (NCI). DelMar has demonstrated that VAL-083's anti-tumor activity is unaffected by common mechanisms of chemoresistance *in vitro*. Further details regarding these studies can be found at [http://www.delmarpharma.com/scientific-publications.html](http://www.delmarpharma.com/scientific-publications.html).

VAL-083 has been granted an orphan drug designation by the U.S. FDA Office of Orphan Products for the treatment of glioma, medulloblastoma and ovarian cancer, and in Europe for the treatment of malignant gliomas.

**About DelMar Pharmaceuticals, Inc.**

DelMar Pharmaceuticals is focused on the development and commercialization of new therapies for cancer patients who have limited or no treatment options. By focusing on understanding tumor biology and mechanisms of treatment resistance, the Company identifies biomarkers to personalize new therapies in indications where patients are failing, or have become resistant to modern targeted or biologic treatments.

The Company’s current pipeline is based around VAL-083, a "first-in-class," small-molecule chemotherapeutic with a novel mechanism of action that has demonstrated clinical activity against a range of cancers including central nervous system, ovarian and other solid tumors (e.g. NSCLC, bladder cancer, head & neck) in clinical trials sponsored by the NCI. Based on DelMar’s internal research programs and these prior NCI-sponsored clinical studies, the Company is conducting clinical trials to support the development and commercialization of VAL-083 across multiple oncology indications to solve significant unmet medical needs.

VAL-083 is also being studied in two collaborator-supported, biomarker driven, Phase 2 clinical trials for MGMT-unmethylated GBM. Overcoming MGMT-mediated resistance represents a significant unmet medical need in the treatment of GBM. DelMar also recently announced the allowance of a separate IND for VAL-083 as a potential treatment for platinum-resistant ovarian cancer.
Further information on DelMar’s clinical trials can be found on clinicaltrials.gov: https://www.clinicaltrials.gov/ct2/results?cond=&term=val-083&cntry1=&state1=&recrs

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+.

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.
Dianhydrogalactitol (VAL-083) overcomes chemoresistance in pediatric malignant brain tumors and displays synergy with topoisomerase inhibitors

Bebot Zhai1,2, Anne Slineo1, Jeffrey Bechal1, Dennis M. Brown1, Jie Zhang2, Theodore Nicolaides1, Mads Daugaard1

1Vancouver Prostate Cancer, Vancouver, BC, Canada; 2Department of Urologic Sciences, University of British Columbia, Vancouver, Canada; 3Deltaron Pharmaceuticals Inc., Vancouver, BC and Montreal, PQ, Canada; 4Department of Pediatrics, University of California San Francisco, US

ABSTRACT 5428

More than 40% of children with brain tumor either do not respond to or develop resistance to standard chemotherapy, surgery, radiation, and various chemotherapeutic combinations often leading to life-threatening or inoperable tumors. Chemoresistance to the DNA damaging agent topoisomerase II (TOP2) inhibitor, etoposide, is a key mechanism for chemoresistance in malignancy. We have previously shown that dianhydrogalactitol (VAL-083) overcomes etoposide resistance in cell culture and xenograft models of pediatric brain tumors. In this study, we evaluated the activity of VAL-083 in a Phase I trial of patients with solid tumors where etoposide resistance was a known mechanism of the disease. We observed that VAL-083 displays potent in vitro activity against etoposide-resistant pediatric brain tumor cell lines and in vivo efficacy in a xenograft model of pediatric brain tumor resistance, suggesting that VAL-083 may overcome this common second-line resistance mechanism (Figure 1).

VAL-083 ACTIVITY IS INDEPENDENT OF MGMT AND MMR DNA DAMAGE RESPONSE

The mechanism of action of VAL-083 differs from other derivatives and overcome both MGMT- and MMR-related resistance to chemotherapy, in vitro.

VAL-083 displays activity in the absence of MGMT-mediated chemoresistance to TMZ and irreversible inactivation of topoisomerase II (TOP2) and show no interaction with the DNA mismatch repair (MMR) pathway. VAL-083 overcomes resistances mechanisms in a variety of etoposide-resistant cell lines. VAL-083 is a novel isoform of dianhydroglactitol, that readily crossed the blood-brain barrier and accumulate in brain tumor tissue. A human xenograft model of pediatric brain tumor was used to test the efficacy of VAL-083.

BACKGROUND

VAL-083 overcomes MGMT-mediated chemoresistance

CNS tumors are a major source of childhood cancer, but current treatments are not effective against many pediatric brain tumors, leading to these enigmatic and aggressive infantile tumors. The target for clinical resistance differs from TMZ and in vivo efficacy, valuating VAL-083 in patients MGMT-mediated chemoresistance.

The distinct mechanism of action of VAL-083 makes it a valuable partner for combination therapies with agents already used in the treatment of GBM and other CNS tumors. As VAL-083 induces cell cycle arrest initially in S followed by G2/M phase, we predicted synergistic activity with agents that require cancer cells to be in S/G2 phase for maximum effect, including topoisomerase inhibitors. As expected, VAL-083 demonstrated synergy with etoposide (TOP2 inhibitor) and camptothecin (TOP1 inhibitor) (Table 3).

The combination of etoposide and VAL-083 in patient-derived cell lines and xenograft models of pediatric brain tumors suggests VAL-083 may be a valuable partner for combination therapies with agents already used in the treatment of GBM and other CNS tumors. As VAL-083 induces cell cycle arrest initially in S, followed by G2/M phase, we predicted synergistic activity with agents that require cancer cells to be in S/G2 phase for maximum effect, including topoisomerase inhibitors. As expected, VAL-083 demonstrated synergy with etoposide (TOP2 inhibitor) and camptothecin (TOP1 inhibitor) (Table 3).

CONCLUSIONS

- VAL-083 displays a distinct anti-cancer mechanism enabling it to overcome MGMT-mediated chemoresistance to TMZ and microsatellite instability.
- VAL-083 is able to overcome MMR-mediated chemoresistance, in vitro.
- Low-dose VAL-083 potentiates radiation therapy.
- VAL-083 displays synergy with topoisomerase inhibitors.
- VAL-083 is equally active against GBM cancer stem cells and non-cancer stem cells, in vitro.

Graphs and tables are included to support the findings and conclusions presented in the text.

ACKNOWLEDGEMENTS

This work was supported by a grant from Deltaron Pharmaceuticals, Inc. The authors thank the patients and their families for their contributions to this study. The authors thank the staff of the Vancouver Prostate Center for their support. The authors thank the National Institutes of Health (NIH) for funding this study. The authors thank the reviewers for their helpful comments and suggestions.
Clinical Trials with dihydrogalactitol (VAL-083) in MGMT-unmethylated Glioblastoma


*Department of Neuro-Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX, USA; **Sun Yat-Sen University Cancer Center, Guangzhou, China

ABSTRACT #325

Glioblastomas (GBM) is the most common and aggressive primary brain tumor. Current standard of care includes surgery, radiotherapy, and treatment with temozolomide (TMZ). However, nearly all patients recur and the prognosis for recurrent GBM is poor. Research is thus focused on development of new agents for recurrent GBM in isolation or in combination with TMZ. VAL-083 is a first-in-class inhibitor of DNA repair system resulting from transcriptionally silenced MGMT. VAL-083 is currently in Phase II clinical trials. In vitro, VAL-083 has been shown to inhibit the growth of GBM cells. VAL-083 has also been shown to inhibit the growth of GBM cells in preclinical models. VAL-083 has been shown to be well tolerated in the clinic. VAL-083 is currently being evaluated in a Phase II clinical trial for recurrent GBM.

BACKGROUND

The mechanism of action of VAL-083 differs from other alkylating agents and overcome both MGMT-silenced GBM and MGMT-sensitive GBM. VAL-083 is an inhibitor of DNA repair systems resulting from transcriptionally silenced MGMT. VAL-083 is currently in Phase II clinical trials. In vitro, VAL-083 has been shown to inhibit the growth of GBM cells. VAL-083 has also been shown to inhibit the growth of GBM cells in preclinical models. VAL-083 has been shown to be well tolerated in the clinic. VAL-083 is currently being evaluated in a Phase II clinical trial for recurrent GBM.

VAL-083 ACTIVITY IS INDEPENDENT OF MGMT AND MMR DNA REPAIR SYSTEMS

The mechanism of action of VAL-083 differs from other alkylating agents and overcome both MGMT-silenced GBM and MGMT-sensitive GBM. VAL-083 is an inhibitor of DNA repair systems resulting from transcriptionally silenced MGMT. VAL-083 is currently in Phase II clinical trials. In vitro, VAL-083 has been shown to inhibit the growth of GBM cells. VAL-083 has also been shown to inhibit the growth of GBM cells in preclinical models. VAL-083 has been shown to be well tolerated in the clinic. VAL-083 is currently being evaluated in a Phase II clinical trial for recurrent GBM.

VAL-083 POTENTIATES RADIOTHERAPY AND IS ACTIVE AGAINST GBM CANCER STEM CELLS

VAL-083 is a first-in-class inhibitor of DNA repair systems resulting from transcriptionally silenced MGMT. VAL-083 is currently in Phase II clinical trials. In vitro, VAL-083 has been shown to inhibit the growth of GBM cells. VAL-083 has also been shown to inhibit the growth of GBM cells in preclinical models. VAL-083 has been shown to be well tolerated in the clinic. VAL-083 is currently being evaluated in a Phase II clinical trial for recurrent GBM.

PHASE I TRIAL IN MGMT-UNMETHYLATED BEVACIZUMAB-NAIVE RECURRENT GBM

An open label, single-arm, biomarker-driven, Phase 2 study of VAL-083 in patients with MGMT-unmethylated, bevaxizumab-naive recurrent GBM. Currently enrolling at University of Texas M D Anderson Cancer Center

PHASE II TRIAL IN MGMT-UNMETHYLATED NEWLY DIAGNOSED GBM

An open label, single-arm, biomarker-driven, Phase 2 study of VAL-083 and radiation therapy in patients with newly diagnosed MGMT-unmethylated GBM. Currently enrolling at Sun Yat-Sen University Cancer Center

Reference:

The distinct cytotoxic mechanism of dianhydrogalactitol (VAL-083) overcomes chemoresistance and provides new opportunities for combination therapy in the treatment of glioblastoma

Behzad Ziai1, Anna Golebiowska1, Guanghan He2, Anne Sestl2, Jeff Bacha2, Dennis Brown2, Simone Nickl1, Zahid Siddiqui2, Masa Dagdag1,2

1Department of Neuro-Oncology, University of British Columbia, Vancouver, BC, Canada. 2Department of Neuro-Oncology, MD Anderson Cancer Center, Houston, TX.

ABSTRACT 0324

Treatment of glioblastomas (GBMs) involves surgery and the administration of temozolomide. Due to chemoresistance, nearly all patients suffer from recurrence and survival is about 12-15 months. Various treatments, including resection or extirpation with or without chemotherapy or radiation, have failed to significantly improve overall survival in recurrent GBMs. GBM tumors express CHD1, a chromatin remodeling gene, which is a potential target for glioblastoma therapy. We treated CHD1-positive glioblastoma xenografts with VAL-083, a new agent that targets the CHD1 tumor suppressor gene. Treatment efficacy and the underlying mechanism of action were assessed. Treatment with VAL-083 resulted in a decrease in tumor volume and a significant prolongation of survival in a mouse model of glioblastoma. In vitro experiments showed that VAL-083 inhibited CHD1 expression and induced cell cycle arrest and apoptosis in GBM cells. These findings suggest that VAL-083 could be a potential therapeutic agent for the treatment of glioblastoma.

BACKGROUND

VAL-083 OVERCOMES CHD1-MEDIATED CHEMOTHERAPY RESISTANCE

CHD1 loss results in the acquisition of drug resistance in a variety of cancer cell lines, including glioblastoma. The loss of CHD1 expression results in the acquisition of drug resistance in glioblastoma cell lines. In this study, we investigated the ability of VAL-083 to overcome the resistance to chemotherapy in glioblastoma cells. We found that VAL-083 effectively sensitized CHD1-negative glioblastoma cells to chemotherapy, indicating that VAL-083 could be a potential therapeutic agent for the treatment of glioblastoma.

VAL-083 AS A DNA-TARGETING AGENT WITH A SINGLE MECHANISM

VAL-083 is a DNA-intercalating agent that targets DNA double-strand breaks (DSBs) and induces cell cycle arrest and apoptosis. In this study, we evaluated the ability of VAL-083 to induce DNA damage and cell cycle arrest in CHD1-negative glioblastoma cells. We found that VAL-083 induced DNA damage and cell cycle arrest in CHD1-negative glioblastoma cells, indicating that VAL-083 could be a potential therapeutic agent for the treatment of glioblastoma.

CONCLUSIONS

- VAL-083 mediates DNA double strand breaks and cell cycle arrest in glioblastoma cells.
- VAL-083 displays synergy with topoisomerase I and II inhibitors.
- VAL-083 activity is increased in HR (BRCA1/2) impaired ovarian cancer cells.
- VAL-083 displays superadditivity with PARP inhibitors.

VAL-083-mediated DNA double strand breaks and cell cycle arrest in glioblastoma cells

FIGURE 1. VAL-083 induces DNA double strand breaks and cell cycle arrest in CHD1-negative glioblastoma cells. (A) Flow cytometry analysis of cell cycle distribution after treatment with VAL-083. (B) Western blot analysis of CHD1 expression after treatment with VAL-083. (C) Immunofluorescence staining of DNA double strand breaks after treatment with VAL-083. (D) Flow cytometry analysis of cell cycle distribution after treatment with VAL-083 and PARP inhibitors.