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): October 28, 2017

DELMAR PHARMACEUTICALS, INC.

(Exact name of registrant as specified in its charter)

Nevada	**000-54801**	**99-0360497**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification Number)

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

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

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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(17 CFR §230.405) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2).

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 8.01 Other Events.

On October 28, 2017, DelMar Pharmaceuticals, Inc. presented a poster at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics in Philadelphia. A copy of the poster is attached as Exhibit 99.1 hereto.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit Number Description

99.1 Poster

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: October 31, 2017

By: /s/ Jeffrey Bacha
Name: Jeffrey Bacha
Title: Chief Executive Officer

VANCOUVER PROSTATE CENTRE

ABSTRACT # A109

DNA damaging agent dianhydrogalactitol (VAL-083) targets HR repair pathway and suggests combination therapy with topoisomerase and PARP inhibitors

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MD Anderson Cancer Center

DelMar PHARMACEUTICALS

BACKGROUND

DNA damage repair (DDR) describes the network of pathways that are responsible for minimizing the effect of daily DNA damage such as mismatched base pairs, single strand breaks (SSBs) and double strand breaks (DSBs). Multiple DNA repair pathways are known, including mismatch repair (MMR), O6-methylguanine DNA methyltransferase (MGMT), non-homologous end joining (NHEJ) and homologous recombination (HR), which act either by repairing the damage, arresting cell growth or, if necessary, promoting cell death. DDR defects are a hallmark of cancer development, rendering the cancer cells sensitive to targeted DNA damaging agents.

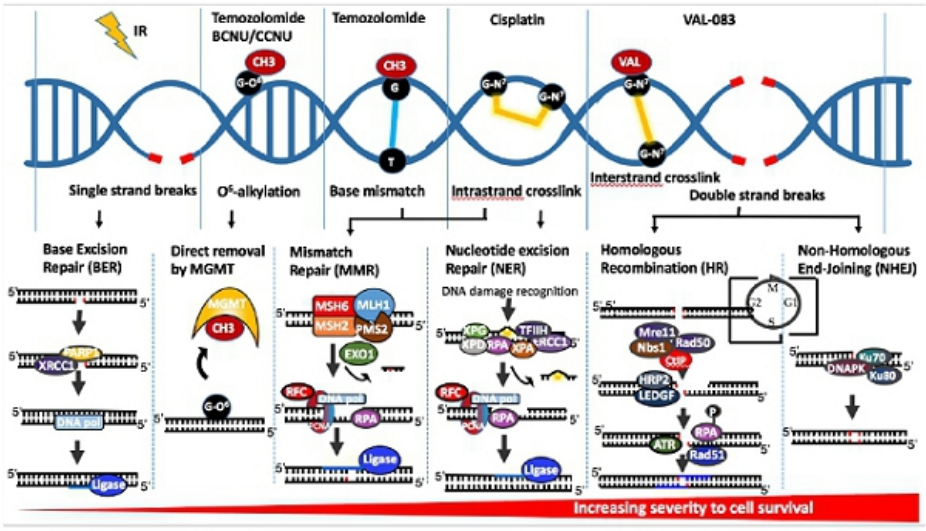


FIGURE 1. *DNA damage and damage repair pathways.*

VAL-083 is a DNA-targeting agent with a unique mechanism of action and established clinical efficacy and safety

VAL-083 (dianhydrogalactitol) is a first-in-class, bi-functional DNA-targeting agent, with a mechanism of action that differs from other DNA-targeting agents.[1] VAL-083 introduces irreversible DNA interstrand crosslinks (ICLs) at the N^7-position of guanine throughout the cell cycle, leading to persistent DNA DSBs and cancer cell death. The DNA DSBs, which form during the S phase, persists for 24-72h after VAL-083 pulse treatment, ultimately leading to S/G2 phase cell cycle arrest and cell death through two parallel pathways: **p53-dependent** and **p53-independent**[2] (Figure 2).

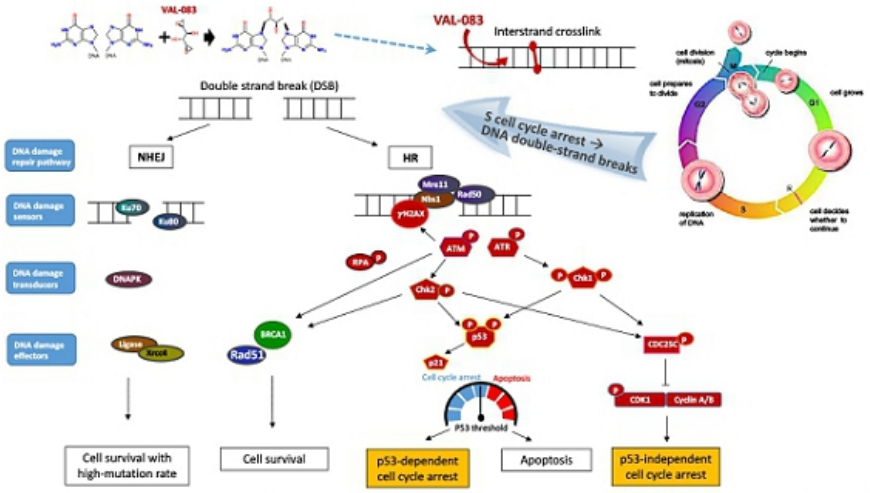


FIGURE 2. *Mechanisms of action of VAL-083 induced chemotherapeutic cytotoxicity. Apoptosis can be induced through either p53 dependent or p53 independent pathways. BRCA1 dysfunction, which is common in ovarian cancer, increases the cytotoxic potential of VAL-083.*

This unique mechanism of action suggests that **VAL-083 may be efficacious in treating patients whose tumors are refractory to current standard of care chemotherapeutics, including Pt-based and alkylating agents**, either as a single agent or as a component of combination therapy regimens.

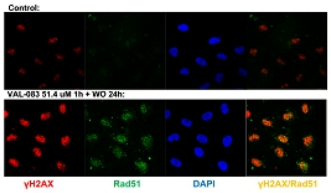
References

1. Zhai B, et al. Cancer Res. July 2017: 77(13), abstract #2483.
2. Peng C, et al. Acta Pharmacol Sin. 2017 Apr;38(4):561-570.
3. Zhai B, et al. EORTC-NCI-AACR Annual Meeting, 2016. #363
4. Bacha J, et al. AACR Ovarian Meeting, Oct 1-4, 2017. #A01

RESULTS

VAL-083 pulse treatment induced increased DNA damage foci formation

A549 lung cancer cells showed increased Rad51, BRCA1, RPA32 and γH2A.X foci formation after VAL-083 pulse treatment suggesting VAL-083-mediated DNA double strand breaks and activation of the HR DNA damage repair system.



A549 cells	γH2A.X +	BRCA1 +	Double positive
Control	21.59 %	10.23 %	10.23 %
VAL 1h + WO 24h	81.72 %	72.84 %	67.74 %

A549 cells	γH2A.X +	Rad51 +	Double positive
Control	23.73 %	1.69 %	0
VAL 1h + WO 24h	87.18 %	74.36 %	67.95 %

A549 cells	γH2A.X +	RPA32 +	Double positive
Control	16.26 %	1.63 %	0.81 %
VAL 1h + WO 24h	87.06 %	83.53 %	80 %

FIGURE 3. Serum-starved A549 lung cancer cells were treated with 51.4 µM VAL-083 for 1h, followed by washout for 24h. Cells were pre-extracted in CSK buffer for 5 min at 4°C, fixed in 4 % paraformaldehyde, washed in PBS and stained with corresponding antibodies.

VAL-083 cytotoxicity is increased in BRCA1 dysfunctional ovarian cancer cells

VAL-083 activity was increased (IC50 decreased) when BRCA1 was impaired. This suggests increased activity in ovarian cancer with dysfunctional BRCA1.

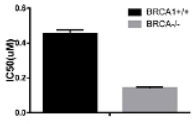


FIGURE 4. VAL-083 cytotoxicity against BRCA1-proficient (BRCA1+/+) and -deficient (BRCA1-/-) ovarian cancer cells A2780.

VAL-083 displays synergy/superadditivity with Topoisomerase and PARP inhibitors

VAL-083 induced S/G2 phase cell cycle arrest in all cancer cells tested, including ovarian, prostate, lung and glioma cancer cells. This suggests the potential for synergy with S-phase dependent agents like topoisomerase (Top) and PARP inhibitors (PARPi)

- **As expected, VAL-083 demonstrated synergy with etoposide (Top2 inhibitor) and camptothecin (Top1 inhibitor) in PC3 and A549 cancer cells[3]**
- **VAL-083 demonstrated superadditivity with PARP inhibitors olaparib, talazoparib and veliparib in A2780 ovarian cancer cells[4]**

VAL-083 cytotoxicity is independent of chemo-resistance mechanisms p53 status, MMR and NHEJ, implicated in resistance to platinum-based and PARPi therapy

The IC_{50} for VAL-083 in the cisplatin-resistant cell-lines 2780CP-16, OVCAR-10, Hey and OVCA-433 were 4 to 7-fold greater than for A2780; while the corresponding IC_{50} values for cisplatin in these models were 10 to over 25-fold greater. These results demonstrate that there is only partial cross-resistance between cisplatin and VAL-083, further suggesting distinct modes of action for the two drugs.

To explore the dependence of mismatch repair (MMR) and non-homologous end-joining (NEJH) DNA repair mechanisms, VAL-083 activity was investigated in human cancer cell lines HCT116, LoVo, M059K and M059J. MMR-deficiency is implicated in Pt-resistance, and NHEJ-deficiency is implicated in resistance to PARP inhibitors (PARPi). VAL-083 was equiactive against cancer cells that are proficient and deficient in these DNA-repair mechanisms, suggesting a distinct mechanism and an ability to overcome treatment resistance to Pt-based and PARPi chemotherapy.

TABLE 1: VAL-083 cytotoxic activity in a panel of ovarian cancer cell lines.

Ovarian Cancer Cell Lines	A2780	2780CP	OVCAR-10	HEY	OVCA-433
Histology	Unknown	Unknown	Adeno-carcinoma	HGSOC	HGSOC
p53 mutation	WT	V172F	V172F, G266R	P72R	P72R
Cisplatin sensitivity/resistance	Sensitive	Resistant	Resistant	Less sensitive	Resistant
VAL-083 IC_{50} µM (SE)	0.54 (0.046)	2.2 (0.289)	3.6 (0.173)	2.1 (0.289)	2.3 (0.058)
Cisplatin IC_{50} µM	0.22	12.0	9.0	3.1	10.2

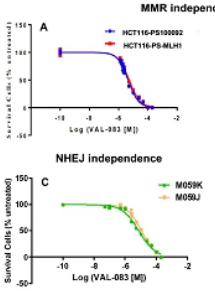
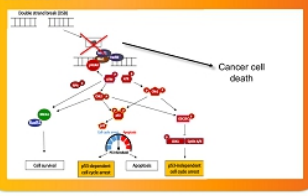


FIGURE 5. *Cytotoxicity of VAL-083 (72 hr) in two pairs of human isogenic colorectal cancer cell lines: (A) MMR-proficient HCT116-PS-MLH1/ MMR-deficient HCT116-PS100092 and (B) MMR-proficient LoVo-PS-MSH2/MMR-deficient LoVo-PS100092, established by lentiviral infection. (C) VAL-083 cytotoxicity (72 hr) in isogenic glioblastoma cell lines: NHEJ-proficient M059K and NHEJ-deficient M059J. N=3.*

IMPACT

VAL-083 may offer an effective treatment alternative against HR-impaired tumors, including high-grade serous ovarian carcinoma



VAL-083 combination treatment with topoisomerase or PARP inhibitors may offer effective treatment alternatives against various solid tumors, including ovarian and CNS tumors

VAL-083 may offer a treatment alternative against tumors with p53-, MMR-, MGMT- or NHEJ-mediated resistance to chemotherapeutic agents, including cisplatin and temozolomide

VAL-083	MGMT		p53	
	high	low	proficient	defective
MMR+	sensitive	sensitive	sensitive	sensitive
MMR-	sensitive	sensitive	sensitive	sensitive
NHEJ+	sensitive	sensitive	sensitive	sensitive
NHEJ-	sensitive	sensitive	sensitive	sensitive

cisplatin	MGMT		p53	
	high	low	proficient	defective
MMR+	sensitive	sensitive	sensitive	resistant
MMR-	resistant	resistant	resistant	resistant
NHEJ+	sensitive	sensitive	sensitive	resistant
NHEJ-	sensitive	sensitive	sensitive	resistant

Temozolomide	MGMT		p53	
	high	low	proficient	defective
MMR+	resistant	sensitive	sensitive	sensitive
MMR-	resistant	resistant	resistant	resistant
NHEJ+	resistant	sensitive	sensitive	sensitive
NHEJ-	resistant	sensitive	sensitive	sensitive