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June 6, 2017

DelMar Pharmaceuticals, Inc. (DMPI/NASDAQ CM/$2.15)

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BUY

DelMar Pharmaceuticals is a clinical-stage drug development company targeting orphan and high unmet need cancer indications with 505(b)(2)-reduced risk compounds

SUMMARY AND INVESTMENT HIGHLIGHTS

Current Price $2.15

The incidence rate of all primary malignant brain and central nervous system tumors is 7.4 per 100,000 person-years or equal to about 24,000-26,000 new cases of primary brain malignancies diagnosed annually in the US, according to the National Cancer Institute (NCI). For glioblastoma multiforme (GBM), which constitutes about 70% of malignant gliomas, the 5-year survival rate is 14% for 20-44 year olds and but only 1-3% for 55-64 year old patients, the typical age of diagnosis (Source: University of Maryland Medical Center website). The tremendous unmet need in GBM is substantiated by the glaring lack of treatment progress and improvement in overall survival in this indication over the last 20 years. Only **Avastin®** (bevacizumab-BEV) and CCNU (lomustine) have received FDA approval for treatment of malignant glioma since the 1999 approval of Merck's **Temodar®** (temozolomide or TMZ), the current "gold standard", despite minimal survival benefit gained by adding BEV to TMZ refractory patients. Regardless of treatment, GBM patients universally progress rapidly to death once they have failed TMZ.

The challenges in GBM are great. Novartis alone has tried with 12 different drugs, five of which had their clinical trial programs suspended. Bristol-Myers Squibb just announced in April, its high visibility Phase III **Opdivo®/Yervoy®** combination trial failed to reach its endpoint of survival benefit over **Avastin** in these patients, even though a year earlier the Company was touting what looked like outstanding Phase II results with the combination therapy. The need to protect critical brain tissue, ineffective drug delivery across the blood-brain barrier (BBB) or within the brain itself, very difficult adverse event profiles, and the tendency for gliomal tumor escape due to drug resistance, all contribute to the technical difficulties facing drug

FY Ended June 30 unless otherwise specified Uplisted to NASDAQ July 2016

Estimates	FY2016A	FY2017E	FY2018E
YR Reven	**$0.00**	**$0.00 E**	**$0.00 E**
1Q	$0.00	$0.00	$0.00 E
2Q	$0.00	$0.00	$0.00 E
3Q	$0.00	$0.00	$0.00 E
4Q	$0.00	$0.00	$0.00 E
2018 Preliminary Revenue Estimate			**$0.0** E
YR (loss)$	**($0.81) A**	**($0.67) A**	**($0.69) E**
1Q		($0.23) A	NA
2Q		($0.13) A	NA
3Q		($0.18) A	NA
4Q		($0.13) E	NA
P/E (x)	NA	**NA**	**NA**
2018 Preliminary EPS (loss)			($0.69)
REV/Shr	NA	NA	NA
EV/EBITDA (x)		NA	NA
Stock Data			
52-Week Range			$2.06-$10.87
Shares Outstanding (mil.)			14
Market Capitalization			$30.0 MM
Enterprise Value			$26.3 MM
Current Ratio (3/17)			2.27X
Book Value/Share (3/17)			$0.11
Price/Book			20X
Average Trading Volume (3-Month)			49,500
Insider Ownership			18.9%
Institutional Ownership			13.9%
Short interest (Million shares)			0.06
Dividend / Yield			$0.00/0.0%

*Some numbers may not add due to rounding

developers in this indication. As a result, of rare cancers, brain cancer has seen and is seeing an unusually wide variety of experimental treatments, including radiation combination strategies, gene-based therapeutics, immunotherapy, molecularly-targeted therapies, angiogenesis inhibitors and the use of devices as therapeutics or in combination with drugs—in other words, everything including the "kitchen sink" has and is being tried.

Some 40+ private and public companies, primarily US-based, are pursuing the development of treatments for a variety of neurological cancers, including GBM. A number of these companies will be reporting significant clinical events (primarily early-stage Phase II results) during 2017 and 2018, but only a very few will be reporting later stage trials between now and 2020. DelMar stands out as one of this elite group, who, if the Company's clinical plans stay on track, will be able to complete a Phase III registration trial in GBM in the next two years. Furthermore, DelMar is the only contender with a small molecule heavily studied by NIH researchers, VAL-083, that is showing promise for treating patients who are poor responders (about 50-60% of patients) to current standard of care (SOC) with temolozomide.

Investment Thesis

Today, tumor recurrence occurs in all GBM patients, with recurrent tumor frequently appearing in the same location as the primary disease, but with material molecular differences. No standard of care has been established in recurrent or progressive glioblastoma (Weller et al., 2014) that has shown to meaningfully extend survival, currently at about 3-5 months for these patients, despite numerous clinical trials with a plethora of treatment approaches from both Big Pharma and biotech players. Identifying potentially effective therapies is further complicated by clinical evidence that largely lacks appropriate control arms, has selection bias, small sample sizes and disease heterogeneity among patients (Weller et al., 2013). At the present, the best patients can hope upon recurrence is an incremental improvement in progression free survival (PFS) and perhaps a few weeks' improvement in overall survival (OS) with a "bearable" quality of life. DelMar's small molecule approach with VAL-083 is unique among the current universe of clinical-stage GBM-targeting companies. We consider investment in DelMar warranted at this time for the following reasons:

1. **De-risked asset:** VAL-083, dianhydrogalactitol, is relatively "de-risked" compared to many other innovative GBM treatments as the compound was studied in numerous NIH-sponsored trials during the 1980's. It is well-characterized and its side effect profile, a key limitation in treating GBM, is well-established from use in over 1,000 patients.

2. **Treatment flexibility:** VAL-083 works by a synergistic cell cycle disruption mechanism to temolozomide, the current SOC, but the specificity of its action holds promise in overcoming TMZ resistance mechanisms. Therefore, the drug can be used as an add-on therapy to standard of care in the 30-40% of TMZ responsive patients. But more importantly, because its mechanism is effective independent of resistance mechanisms found in the non-TMZ responder population, VAL-083, has the potential to become a first-line treatment for this patient base.

3. **Phase III program funded:** With the financing that was completed in late April 2017, DelMar now has the financial resources to initiate and complete its planned Phase III VAL-083 trial, which might qualify as a single registration trial for FDA approval, if the statistical results meet expectation.

4. **Relative valuation:** DelMar's stock has reacted poorly since the April financing. After being priced at nearly a 20% discount to the then market, DelMar shares have further slid lower by a further 35+%. At this level, it would appear investors have fully priced in typical "haircuts" concerning the probably of clinical success in GBM in light of BMY's recent failure. As such, we believe the stock is at a very attractive level for new investors.

Company Background and Business Strategy

DelMar Pharmaceuticals, Inc. is a semi-virtual, clinical stage drug development company with a focus on the treatment of rare cancers. Originally formed as a Nevada corporation in June 2009 under the name Berry Only Inc., a reverse acquisition completed in January 2013 with DelMar Pharmaceuticals (BC) Ltd., a British Columbia-based developer of cancer treatment drugs, gave rise to the current corporate entity. Concurrently with the reverse acquisition, Berry changed its name to DelMar Pharmaceuticals, Inc. DelMar Pharmaceuticals, Inc. is the parent company of DelMar (BC), a British Columbia, Canada, corporation and the parent company to Callco and Exchangeco which are British Columbian corporations formed to facilitate the Reverse Acquisition.

DelMar's drug discovery strategy focuses on identifying well-validated clinical and commercial-stage compounds that have a scientific rationale for development in orphan drug indications. Through its relationship with Valent Technologies, LLC (Valent), a company owned by Dr. Dennis Brown, DelMar's Chief Scientific Officer, DelMar is able to utilize Valent's proprietary ChemEstate bioinformatics tools to screen and identify potential promising candidates that are then "validated" by the Company's network of consultants, contract research organizations and academic partners. Based on this strategy, DelMar acquired VAL-083 and its intellectual property and prototype drug product from Valent. Subsequent to the acquisition of VAL-083, DelMar has identified additional drug candidates that may be of interest to in-license or acquire in the future.

Currently, VAL-083 is the Company's primary asset. VAL-083 is a formulation of dianhydrogalactitol (DAG) and is in multiple US clinical trials for recurrent and refractory GBM, the most common form of brain cancer. DelMar is also the exclusive commercial rights holder of VAL-083 in China where it is already approved as a chemotherapeutic agent for the treatment of chronic myelogenous leukemia ("CML") and for lung cancer. DelMar has a collaboration agreement in place with the single VAL-083 manufacturer presently licensed by the China Food and Drug Administration ("CFDA") to produce VAL-083 for the Chinese market. DelMar believes VAL-083 may potentially generate future royalty revenue through product sales or royalties in China. VAL-083 has not received any marketing approvals outside of China.

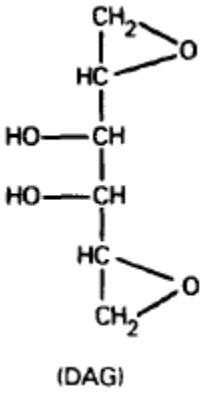
DelMar's business strategy is to remain a semi-virtual drug development company. Upon obtaining an initial regulatory approval for VAL-083, DelMar intends to seek marketing partnerships to generate potential future royalty income and to supplement its own efforts in commercializing VAL-083 for the treatment of GBM and other orphan and niche cancer indications where there is a high unmet patient need. DelMar may also acquire other product candidates or discover them through various research activities.

DelMar currently has 15 employees. The address of the Company's administrative offices is Suite 720 - 999 West Broadway, Vancouver, British Columbia, V5Z 1K5. The primary telephone number is (604) 629-5989. DelMar's clinical operations are located at 3485 Edison Way, Suite R, Menlo Park, California, 94025. The Company's website is www.DelMarpharma.com.

Lead Candidate Background

The Company's lead therapeutic candidate is VAL-083, a "first-in-class" small molecule cancer chemotherapeutic. VAL-083 chemically is a formulation of dianhydrogalatitol (DAG) and belongs to a class of sugar derivatives known as hexitols that were originally synthesized in the late 1950's as potential cytotoxic agents. Three of these hexitols, dibromomannitol (DBM), dibromodulcitol (DBD) and dianhydrogalactitol (DAG) underwent extensive clinical investigation in the United States in the 1970's and 1980's by the NIH and others, prompted by European studies reporting significant anticancer activity by DBM in polycythemia and chronic myelogenous leukemia (CML). Dibromodulcitol, DBD, a stereoisomer of DBM, was subsequently introduced into clinical trials because of its activity in experimental tumor models resistant to DBM. DAG, a

chemical reactant product of DBD in certain conditions, was studied because its activity was somewhat different from the other two compounds.



(DAG)

Source: Chuiten et al, 1981

DAG is the primary conversion reaction product when DBD is exposed to a mild alkaline environment or human serum. Like its sister compounds, DAG alkylates (replaces) a hydrogen with an C_nH_{2n+1} group (such as converting methyl, CH_3, to butyl, C_2H_5 in a hydrocarbon chain). Within a cancer setting, the alkylating agent attaches an alkyl group (C_nH_{2n+1}) to DNA to cause DNA strand breakage. Alkylating agents are commonly used as chemotherapy drugs because of this DNA strand breakage mechanism and in fact, were the first group of compounds used medically for modern cancer chemotherapies, thanks to original research by Goodman, Gilman, and others at Yale University who were studying nitrogen mustards in 1942.

General Mechanism of Action

Alkylating agents such as VAL-083/DAG produce anti-cancer effects by binding to specific locations on the DNA, thus interfering with normal cell cycle (DNA replication) processes. Once normal cell cycle progression becomes dysfunctional, apoptotic signals arise that lead to cell death. Specifically, DAG halts tumor growth by crosslinking guanine (one of the four nucleobases that comprise DNA double-helix strands) across both strands of DNA. This crosslinking prevents the strands from uncoiling and separating as part of the DNA replication process during cell division. As uncoiling the strand is absolutely necessary in DNA replication, the cells can no longer divide. However, these alkylating drugs act nonspecifically across any cell type that is dividing and so these compounds are also toxic to normal cells, particularly those cells that divide frequently, such as those in the gastrointestinal tract, bone marrow, testicles and ovaries. Most alkylating agents can also be carcinogenic in their own right, so controlling specificity of their action and side effects are challenges.

Because of their mechanism of action, DBM, DBD and DAG compounds were assessed in numerous Phase I and early-stage Phase II clinical trials largely sponsored by the NCI in the US as treatments against various cancers including lung, brain, cervical, and ovarian tumors and leukemia during the 1970's and into the mid-1980's. Activity across various cancers differed among the compounds, but DAG demonstrated unique differences from its sister compounds in that it had activity against non-small cell lung cancer and it could cross the blood-brain barrier and accumulate in brain tissue. DAG was also studied in early stage trials as both a monotherapy and in combination studies with radiation and nitrosoureas in brain cancer. However, the results from these limited early human studies were highly variable and issues surrounding myelosuppression, thrombocytopenia and other toxicities following DAG dosages high enough to achieve efficacy tempered medical research enthusiasm for the compound. The compound was largely "forgotten" until DelMar and (Valent's Dr. Dennis Brown) resumed studying its possibilities.

VAL-083/DAG Complements GBM Standard of Care

Third party research and DelMar's own pre-clinical data have shown, however, that the mechanism of action (MOA) of VAL-083, which we discuss in more detail later in this report, is functionally different from other alkylating agents, such as temozolomide and platinum-based drugs commonly used in the treatment of certain cancers. Temozolomide acts by adding a cross-linking methyl group at a two specific positions on guanine to "alkylate" the guanines in a DNA strand. This causes a mismatch of one DNA strand compared to the complementary strand as the DNA enters into replication. The mismatch between the strands leads to double-strand breaks and cell death. However, tumor cells have multiple ways to co-opt or turn-on DNA repair mechanisms that demethyl the crosslinked DNA, thus allowing DNA replication of mutant cells to proceed, disease to progress, and chemical resistance by selective pressure to be established.

VAL-083's mechanism of action avoids mismatch DNA repair mechanisms (MMR). Instead of adding a methyl group on the guanines within a DNA strand, VAL-083 alkylates the same position on guanines on both strands of DNA, thus creating *interstrand* crosslinks that prevent the DNA from uncoiling and separating for replication. This type of cross-link cannot be repaired by typical DNA mismatch repair mechanisms. Thus, in addition to addressing MMR chemo-resistance in GBM, VAL-083 may also be effective in treating other resistant or refractory cancers, such as non-small cell lung cancer where there are alkylating agent-induced MMR mechanisms at play. The drug's primary dose limiting toxicity (DLT) is myelosuppression which is, in today's world, much better managed clinically, than 30-40 years ago.

Recent News

During the past two months, DelMar has presented a number of abstracts and posters at important medical symposia and conferences and completed a needed round of financing to fund a Phase III trial with VAL-083.

1. **Recent Poster presentations:** On May 8th, DelMar announced the presentation of two abstracts and posters at the World Federation of Neuro-Oncology Societies (WFNOS) meeting, held this year in Zurich. The first abstract, entitled *"Distinct Mechanism of Action of dianhydrogalatitol (VAL-083) overcomes chemo-resistance in glioblastoma."* Zhai et al, 2017, builds on growing evidence of VAL-083's ability to circumvent both initial and secondary GBM resistance mechanisms. The second poster, *"Clinical trials of VAL-083 in patients with chemo-resistant glioblastoma."* Bacha, et al, 2017, reviews VAL-083's clinical history. These presentations follow abstracts around similar subjects presented at this year's AACR meeting. DelMar will be presenting the abstract, *"Clinical Trials of VAL-083 in Patients With Chemo-Resistant Glioblastoma"* at ASCO in June.

2. **Financing:** DelMar completed a registered public offering of an aggregate of 2,769,924 shares of common stock and warrants on April 18th. The stock was priced at $3.25 per share, an approximate 19% discount to the then current trading price. Five-year warrants were priced at $3.50 and were immediately exercisable. DelMar netted approximate net proceeds of $8 million. Gross proceeds were approximately $9 million.

3. **New research collaboration:** DelMar announced on April 25th, the formation of a three-year collaboration with Duke University, one of the leading GBM research institutions in the US, to evaluate VAL-083 in newly diagnosed GBM patients. As most GBM patients are poor responders to TMX standard of care, the aim of this research will be study VAL-083 alone or in combination with other agents to determine activity in a range of GBM subtypes and to see if there are specific characteristics of these GBM subtypes that might be more responsive to VAL-083 than standard of care. DelMar will fund a number of pre-clinical studies geared towards a more "personalized medicine" approach to treating GBM. The research will be led by Dr. Madan Kwatra, Director of the Glioblastoma Drug Discovery Group at Duke.

4. **Phase III Clinical Design and Timing Announced:** DelMar recently announced its upcoming Phase III VAL-083 in **Avastin**-refractory patients. The trial is expected to begin in Q3 of this year and be sufficiently powered to meet FDA guidelines for a single registration trial in this indication. The trial is expected to enroll 180 patients across 25 centers in a two arm, 2:1 randomized study protocol. The three month survival benefit endpoint will be powered at 90% for a hazard ratio equal to 0.54 Management expects the trial to be finished in about 2 years.

Clinical Status

The last two years of have been very productive for DelMar, with the Company completing several important milestones. On the regulatory front, VAL-083 was granted orphan drug designation in both the US and Europe for the treatment of glioma. During 2016, VAL-083 also received orphan drug designation in the US for treatment of ovarian cancer and medulloblastoma. On the clinical side, DelMar presented a number of abstracts

at AACR during the last two years that summarize clinical experience in glioblastoma in Phase Ib/IIa trials as well as presented data further elucidating VAL-083's mechanism of action. Additionally, DelMar presented data at the 2017 AACR meeting showing VAL-083 may have promise both in the treatment of ovarian cancer as a single agent in platinum-resistant tumors or in combination with platinum-based treatments because cisplatin and other members of the platinum anticancer family of drugs act by mechanisms typical of alkylating agents. As such, DelMar believes VAL-083 may offer similar clinical advantages in tumors typically treated with platinum-based agents as those treated with other alkylating drugs such as nitrosoureas. DelMar also has presented non-clinical data supporting VAL-083's differentiated mode of action in non-small cell lung cancer at the 2016 AACR.

Near term clinical milestones

- ✓ H1 2017 -- Initiation of Phase II trial for VAL-083 in GBM patients with first recurrence (MDA study).
- H2 2017 -- Initiation of Phase II trial for VAL-083 in newly diagnosed GBM patients (Japanese study).
- H2 2017 – Initiation of Phase III trial for VAL-083 in GBM patients refractory to TMZ and Avastin.
- H2 2017 – Launch genomics study for planned Phase IV NSCLC trial for VAL-083 in China.
- 2018 – Report interim data from Phase III registration-directed trial for VAL-083 in refractory GBM.

Phase Ib/IIa Trial Results

DelMar completed a two-stage Phase Ib/IIa dose escalation and expansion trial with VAL-083 in 2016. The Phase Ib dose escalation results were reported at AACR in 2016 and the Phase II expansion cohort results were recently reported at AACR 2017. As the following Exhibit 1 indicates, the trial patient base was well representative of the standard of care treatment of radiation followed by TMZ.

Exhibit 1. Patient Profile in Phase Ib/IIa VAL-083 trial

Table 1 Prior Treatment of 48 GBM Patients in DLM-10-001

Temozolomide (TMZ)	48 *(100%)*	Bevacizumab (BEV)	46 *(95.8%)*
Radiotherapy (XRT)	47 *(97.9%)*	Failed TMZ + BEV	46 *(95.8%)*
TMZ + XRT	47 *(97.9%)*	OTHER TREATMENTS	38 *(79.2%)*

In addition, molecular markers, MGMT methylation and IDH-1 mutation, status was available.

Table 2. MGMT and IDH1 mutation status in DLM-10-001

MGMT Status (n = 19)		IDH1 Status (n = 11)		Both Reported (n = 4)
Unmethylated / High MGMT (>67 ng MGMT/mg total protein)	84.2%	Wild Type:	90.9%	Unmethylated & IDH1^{wt}: 100%
Methylated / Low MGMT (<45ng MGMT/mg total protein)	15.8%	Mutant:	9.1%	

Source: DelMar Pharmaceuticals AACR 2017 poster:

In the 34-patient dose escalation phase of the trial, cohorts of 3+3 design were studied beginning at a 1.5 mg/m^2 dose administered on day 1,2,3 of a 21 day cycle and ending at toxicity limited dosage levels at 50/mg/m^2/day. No drug-related adverse events were observed at doses ≤20 mg/m^2 /day. Myelosuppression was mild at doses up to 40 mg/m^2 /day. The Company reported that, as expected, dose limiting toxicities were observed at 50 mg/m^2 /day in 2 of 6 patients, defined as 1 patient with Grade 4 thrombocytopenia and one

patient with Grade 3 thrombocytopenia with hemorrhage. Observations of DLT (G4 thrombocytopenia) were also made in an interim cohort (n=3) at 45 mg/m^2 /day, which was initiated in parallel with a Phase II expansion cohort to further explore the therapeutic window of this dosing regimen. These analyses thereby confirmed a 40 mg/m^2 /day as the maximum tolerated dose (MTD). These results generally comported well with prior reports in the literature from studies conducted with DAG in the 1970's and early 1980's, wherein thrombocytopenia was reported as the dose limiting toxicity in the range of 40 + mg/m^2/day.

Importantly, the trial researchers were able to estimate the VAL-083 concentration in patients' tumors by extrapolating CNS exposure from observed plasma concentrations. VAL-083 CNS tissue concentrations exceeded the IC50 against glioma cell lines seen in *in vitro* studies, thus confirming the agent's ability to effectively cross the blood-brain barrier.

Exhibit 2: Phase Ib/IIa Trial Survival Data

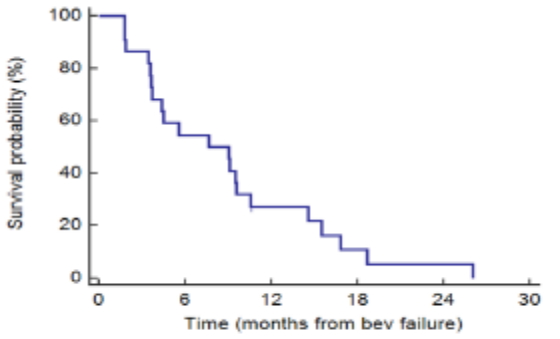


Figure 5: Kaplan Meier plot of patients receiving ≥20mg/m^2 VAL-083

Reference	Post Avastin Salvage Therapy	Median Survival from Bevacizumab Failure
Rahman *(2014)*	nitrosourea	4.3 months
Mikkelson *(2011)*	TMZ + irinotecan	4.5 months
Lu *(2011)*	dasatinib	2.6 months
Reardon (2011)	etoposide	4.7 months
Reardon *(2011)*	TMZ	2.9 months
Iwomoto *(2009)*	various	5.1 months
DLM-10-001	*VAL-083 (n=22)*	8.35 months

Source: DelMar posters

Preliminary analysis shows increasing dose dependent response and a trend towards increased survival in the higher dose cohorts. Median survival was reported at OS=9.2 months at doses ≥30 mg/m^2 /day vs. 5.1 months reported by Iwomoto, the highest median survival reported post-**Avastin** failure.

Phase III Trial Now Funded; DelMar Commits to start STAR-3 Trail in 2H 2017

DelMar has announced its general Phase III trial plan for a registration trial in **Avastin**-failed GBM. The trial is planned to enroll approximately 180 patients, take two years to complete and cost in the range of $12 million. The planned pivotal, randomized controlled Phase III study, is entitled: **S**tudy in **T**emolozomide-**A**vastin **R**ecurrent GBM (**STAR-3**). The trial will measure survival outcomes in patients who have failed **Avastin** compared to a "physician's choice" control. The control arm will consist of a limited number of salvage chemotherapies currently utilized in **Avastin**-failed GBM. If survival outcome measures reach a targeted 3 month survival benefit, this single trial could serve as the basis for a New Drug Application (NDA) submission for VAL-083. With the recent (April 21st) financing putting $8 million into DelMar's coffers, the Company has now begun the process for initiating this trial. If successful in showing the targeted survival benefit, VAL-083 could become a "game-changing" in providing (at least) an incremental path to longer survival for these patients who typically live 2-4 months once refractory to **Avastin**.

DelMar's Clinical Trials Seek to Deliver a New Treatment Paradigm

The standard of care for GBM patients is surgical resection followed by temozolomide and radiation therapy (RT). However, virtually all GBM tumors become resistant to this treatment paradigm and patients are forced into salvage therapy with bevacizumab. It is now appreciated that nearly two-thirds of newly diagnosed GBM

patients may not be good responders to the SOC because they have tumors with a high expression of the nuclear DNA mismatch repair enzyme, O^6 methylguanine-DNA-methyltransferase (MGMT), a dealklyating enzyme. Tumors that possess a unmethylated MGMT ($MGMT^{--}$) promoter, correlate with high levels MGMT mismatch repair (MGMT) enzyme activity, and not only can evade TMZ and other mono-functional alkylating agents such as carmustine (BCNU) and lomustine (CCNU), but also contribute to further tumor pathogenesis. Published studies have documented that expression of MGMT is an important factor in predicting the outcome of GBM. Patients whose tumors exhibit high expression of unmethylated MGMT (MGMT-) promotor have a poor prognosis and significantly shorter progression free survival (PFS) period and lower overall survival (OS) at given time points in comparison to patients with a methylated MGMT promoter and/or low MGMT expression. In a 2011 study of more than 800 GBM patients, those with tumors carrying the unmethylated MGMT promoter had a median overall survival of 14 months versus 21 months for those with a methylated MGMT promoter. The difference in progression-free survival after treatment during was 5.7 and 8.7 months, respectively.

Exhibit 3: Current Treatment Paradigm Compared to Future VAL-083 Paradigm

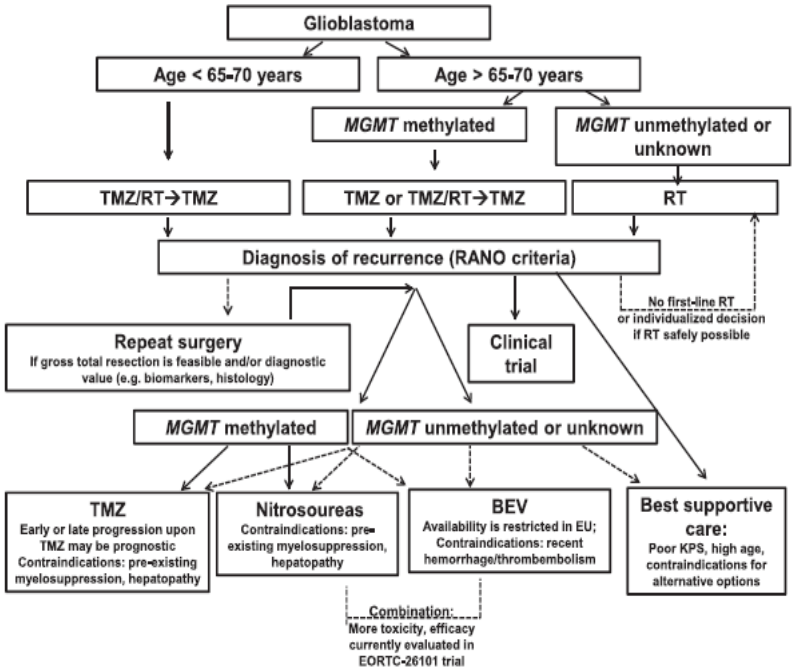
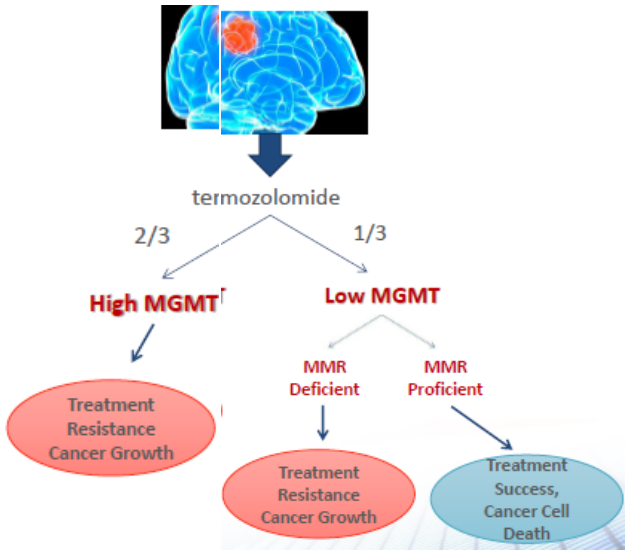
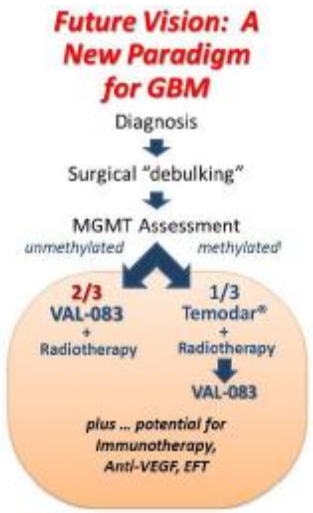


Fig. 1. Approach for individualized treatment decisions in patients with glioblastoma.
Continuous arrows indicate evidence-based current clinical practice. Dashed arrows represent possibilities of individual decision-making which has still to be confirmed.
Abbreviations: CCNU, lomustine; KPS, Karnofsky performance scale; RT, radiotherapy; TMZ, temozolomide; TMZ/RT → TMZ, radiotherapy with concomitant and maintenance TMZ.

Prognosis for patients with high-grade gliomas remains poor. The estimated median survival for patients with GBM is between 12 to 18 months. Recurrence after initial therapy with temozolomide and radiation is nearly universal and in many cases leads to incapacitating damage to normal brain and systemic tissues. Since May 2009, the majority of patients in the US with an initial recurrence of high-grade glioma receive bevacizumab, a monoclonal antibody against vascular endothelial growth factor (VEGF), which is thought to prevent angiogenesis in these highly vascular tumors. BEV has response rates from 32-62% and has improved overall median survival in patients with recurrent high-grade gliomas1. However, the response is short lived, and nearly 100% of patients eventually progress despite salvage treatment with bevacizumab. No chemotherapeutic agent administered following progression through bevacizumab has made a significant impact on survival. Patients progress to death within 1-5 months after resistance develops. Therefore, patients with high-grade gliomas who have progressed through bevacizumab represent a population in dire need of a feasible and tolerable treatment. Unlike many other cancers, there are no 3rd line treatments for GBM.

Source: Seystahl et al, *Thera. Opts in Recurrent GBM*, **Critical Reviews Onc/Hem**, 2016





Source: Left: DMPI corporate presentation Jun 2017, right: DelMar ASCO poster 2016

DelMar's current trial strategy revolves around establishing a new treatment paradigm for GBM, wherein a patient is stratified at the outset according to the MGMT promoter methylation status and other tumor biomarkers. When patients have a positive methylation status, they would move through radiotherapy (RT) + TMZ standard of care and VAL-083 would be administered as a salvage therapy upon recurrence. Since MGMT+ status is found in only about 35-40% of patients, DelMar is seeking to demonstrate that VAL-083 would be an effective first-line treatment in the approximate 60% of patients who, because of their unmethylated ($MGMT^{--}$) status, are weak responders to TMZ. This patient base is currently routinely administered TMZ despite their likely weak response because there are simply no alternatives to offer patients. DelMar is hopeful that because VAL-083 works by a complementary but different mechanism, it might prove to be a viable and effective first-line treatment for this population.

Exhibit 4: VAL-083 Compared to Other Commercial Cytotoxic Agents

ANTI-CANCER DRUG	temozolomide	BCNU/CCNU	Platinum-based chemotherapies	bendamustine	VAL-083
Peak Annual Sales	**$1.1 B** (2013)	**~$100 m** (1980s)	**>$1 billion** (2000s)	**$800 m** (2014)	***TBD***
Cytotoxic Target	O6-guanine	O6-guanine	N7-guanine	N7-guanine	N7-guanine
DNA damage	Base mismatch Single-strand break	Inter-strand crosslinks (G-C) Double-strand break	Intra-strand crosslinks (G-G) Double-strand break	Inter-strand crosslinks (G-G) Double-strand break	Inter-strand crosslinks (G-G), Double-strand break
Cell cycle arrest	G2/M	G2/M	G2	S/G2	S/G2
ATR-Chk1	activated	activated	activated	activated	not activated*
ATM-Chk2	activated	activated	activated	activated	activated
MGMT	dependent	dependent	independent	independent	independent
MMR	dependent	independent	dependent	independent	Independent
p53	dependent	dependent	dependent	Less dependent	Less dependent
Cross blood-brain barrier?	yes	yes	no	no	yes

Source: DelMar Pharmaceuticals

Follow-on Phase II trials to Build on VAL-083's Efficacy in Unmethylated MGMT Patients

Enrollment has begun for the first of multiple Phase II trials that seek to expand support of VAL-083's efficacy in unmethylated MGMT patients. The first additional Phase II trial is a 48-patient trial currently underway in

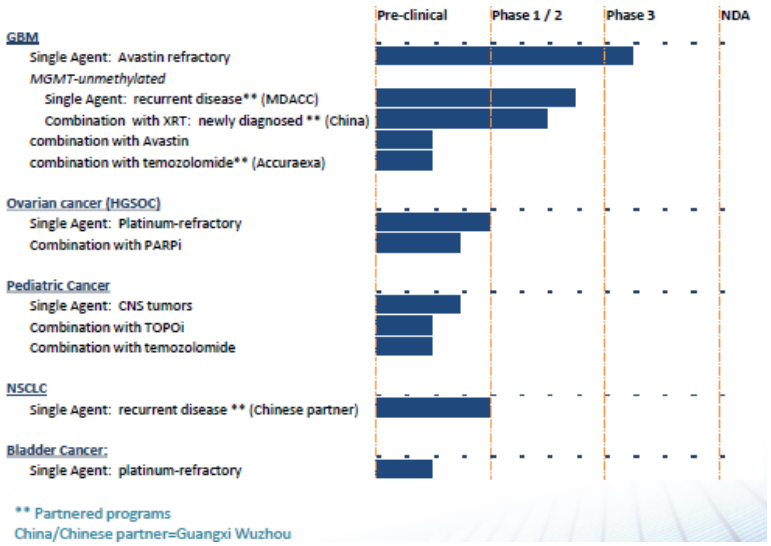
collaboration with MD Anderson trial (MD Anderson trial). The purpose of this phase II, open label, single arm, biomarker-driven study, is to determine if treatment of $MGMT^{--}$ recurrent glioblastoma with VAL-083 improves overall survival (OS), compared to historical controls. This study restricts enrollment to those patients who are BEV-naive upon recurrence. Enrolled patients will receive 40mg/m^2 IV-administered doses of VAL-083 daily for 3 consecutive days at the start of each 21-day cycle. Patients will continue to receive VAL-083 for up to 12, 21-day treatment cycles or until they fulfill one of the criteria for study discontinuation. In patients who demonstrate response or stable disease, and who show treatment tolerability, permission to continue treatment beyond 12 cycles will be considered. Response to VAL-083 treatment will be assessed prior to beginning each next treatment cycle. The first patient in this trial was dosed in February. Final data for the primary endpoint is expected in September of 2020. The trial is expected to be fully completed in June 2021.

DelMar's second open-label Phase II trial is a dose-escalation study in newly diagnosed patients who are verified with $MGMT^{--}$ promoter gene status. The study aims to determine the safety and maximum tolerated dose of VAL-083 as a front-line treatment (instead of TMZ) with SOC radiation therapy, with dosing starting at 20mg/m^2. Patients will begin treatment with VAL-083 within a prescribed time window following surgery or diagnostic biopsy. This trial is being conducted with Japanese partner, Sun Yat-sen University and will enroll a total of 30 patients. The trial is expected to start enrolling in the next few weeks and take two years to complete.

Pipeline

As VAL-083 progresses through the clinic, DelMar is beginning to explore its applicability in other indications based upon the fact that DAG was previously studied in a number of other cancers aside from GBM.

Exhibit 5: Clinical Pipeline Summary for VAL-083



Source: DelMar Pharmaceuticals Corporate Presentation June 2017

Future Indications.

As DelMar researchers continue to expand the knowledge around VAL-083's MOA, cancers with p53-mediated resistance have become an attractive next target for VAL-083 use. These cancers are characterized by high recurrence due to treatment resistance, metastases to the CNS (in the case of lung cancer) and poor options when SOC fails. The p53 gene plays a central role in the protection of the human body from cancer and is responsible for initiating the process of programmed cell death, or apoptosis, which directs a cell to commit suicide if it becomes damaged or cancerous. The p53 pathway is also integral to the cytotoxic activity of many

chemotherapy drugs, most notably, the platinum-based agents. Mutated p53 is a frequent occurrence in non-small cell lung cancer (NSCLC) and p53 mutations are highly correlated with resistance to chemotherapy and poor patient outcomes in NSCLC.

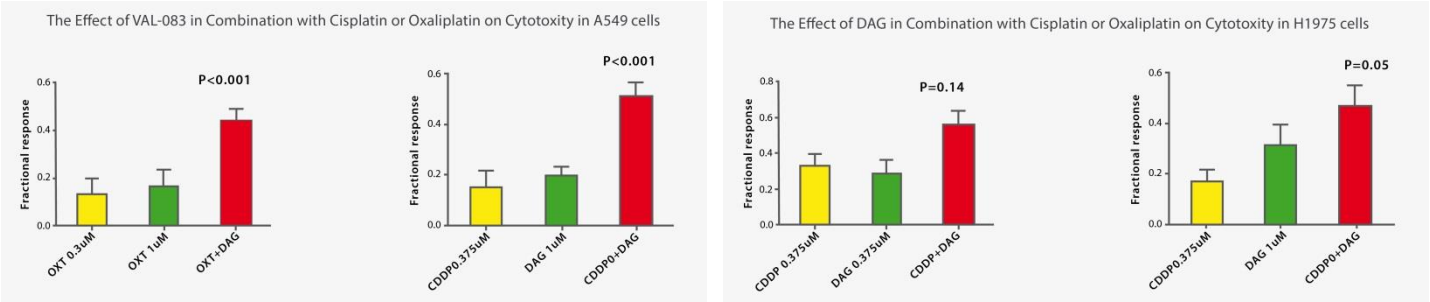
Therefore, VAL-083 may be of therapeutic benefit in combination with platinum-based agents.

- Platinum-based agents cause *intrastrand* crosslinking of guanines at the N7 position, and cell cycle arrest at G2 (replication processes have begun, DNA repair is possible)
- VAL-083 causes *interstrand* crosslinking at the N7 position, but the double stranded breaks cause cell cycle arrest at S phase (before replication, repair unlikely).
- S-phase cycle arrest allows for synergy with other cytotoxic targeted agents such as PARP inhibitors.

DelMar believes NSCLC is a logical next indication for VAL-083 for the following reasons:

- Large market potential: NSCLC accounts for 85% of all lung cancer cases in the United States and approximately 90% of lung cancer cases diagnosed in China. It has a low 5-year survival rate (about 15%) and metastases to the brain and CNS are a primary cause of death.
- Globally, the market for lung cancer treatment may exceed $7 billion by 2019 according to report published by Transparency Market research.
- Platinum-based chemotherapy remains standard-of-care for non-operable NSCLC. Some patients with EGFR-mutations may receive tyrosine kinase (TKI) therapy, as salvage therapy post-failed platinum based chemotherapy.
- Patients with refractory or resistant NSCLC have limited therapeutic options.

Exhibit 6: Pre-clinical Evidence Supports Combination Therapy Approach



Source: DelMar website

Similarly, ovarian cancer presents many of the same challenges as GBM and NSCLC. Recurrence is highly associated with p53 mutations and once patients fail platinum agents, median survival is a very short 6-9 months. New targeted treatments, such as PARP inhibitors, work only in a relatively small subset (BRCA-deficient) tumors, leaving the vast majority of patients who recur with few, if any, salvage treatment options. In addition, encouraging studies with DAG in the 1980's (Stehman, **J. Gyn. Oncol**, 1983) lend support for possible VAL-083 efficacy in the recurrent ovarian cancer setting.

Collaboration with Accurexa for Combination Therapy

It has become widely recognized that holding to a single therapeutic agent approach to successfully treat GBM is almost assuredly futile as virtually all trials with single agents have failed to produce results any better than the current standard of care of surgery, radiation therapy followed by TMZ. Different methods of delivering

current treatments are actively being explored such as delivering cytotoxic agents directly into the surgical site in hopes such local drug delivery will enhance efficacy and reduce systemic side effects. This was the strategy behind the development of **Gliadel®,** a commercialized polymeric wafer infused with BCNU (carmustine) that is implanted into brain at the tumor site. In September 2016, DelMar signed a development collaboration agreement with Accurexa to combine VAL-083 and/or TMZ or BCNU into Accurexa's ACX-31 implantable polymer wafer. The ACX-31 implantable wafer was originally developed by Johns Hopkins University's Del Henry Brem and Harvard's Dr. Robert Langer, who were also the developers of **Gliadel**, which was approved by the FDA in 1996 for glioblastoma. ACX-31 is a next generation wafer capable of delivering multiple drugs. DelMar will supply VAL-083 in exchange for an exclusive option to license or acquire any ensuing products or intellectual property.

VAL-083 License and Other Intellectual Property

DelMar currently holds 7 issued US patents, 8 issued non-US patents and approximately 100 patent application filings across 14 patent families. The Company obtained the intellectual property surrounding VAL-083 via a Patent Assignment Agreement that was entered into with Valent Technologies, LLC. in September 2010. In accordance with the terms of the Assignment Agreement, Valent is entitled to receive a future royalties on revenues derived from the development and commercialization of VAL-083. In the event Valent terminates the agreement, DelMar may be entitled to receive royalties from Valent's subsequent development of VAL-083 depending on the development milestones DelMar achieved prior to the termination of the Assignment Agreement. The Assignment Agreement is considered to be a related-party transaction as Dennis Brown, DelMar's Chief Science Officer and a DelMar director is also a principal of Valent.

Since the transfer of the VAL-083 IP to DelMar, the Company has continued to broaden its intellectual property surrounding VAL-083 with the submission of patent applications claiming novel compositions and methods of use of VAL-083 and related compounds. In additions, intellectual property has been added around synthesis methods and quality control of the manufacturing process of VAL-083. The following lists DelMar's key US-issued patents:

1. **US9,630,938:** Methods of synthesis of substituted hexitols such as dianhydrogalactitol (purification)
2 **US9,085,544**: Method of synthesis of substituted hexitols such as dianhydrogalactitol (purification)
3 **US9,066,918**: Methods to improve the therapeutic benefit of suboptimally administered chemical compounds including substituted hexitols such as dianhydrogalactitol and diacetyldianhydrogalactitol
4 **US9,029,164:** Analytical methods for analyzing and determining impurities in dianhydrogalactitol
5 **US8,921,585**: Method of synthesis of substituted hexitols such as dianhydrogalactitol (purification)
6 **US8,563,758**: Method of synthesis of substituted hexitols such as dianhydrogalactitol

VAL-083 Manufacturing

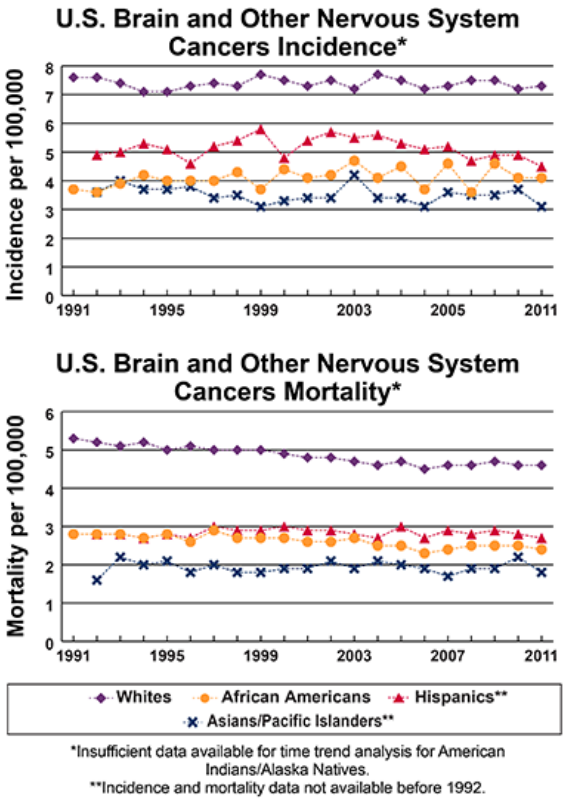
DelMar has an exclusive API purchasing relationship with a Chinese manufacturer of VAL-083 that has enabled the Company to obtain drug product for the human clinical trials being conducted in the United States VAL-083 is produced in accordance with Chinese Pharmacopoeia and the CFDA. The Chinese manufacturer has established a commercial-scale manufacturing process based on the North American process originally developed for the NCI. Approval by the FDA will require VAL-083 to be manufactured in accordance with United States Pharmacopeia ("USP") in accordance with Good Manufacturing Practices ("cGMP") regulations. Over the past year, DelMar has been reviewing North American contract drug manufacturers in anticipation of the transference of manufacturing to a FDA cGMP-compliant CMO for its Phase III clinical trials and potential commercial product.

Rationale Behind DelMar's Clinical Strategy: Developing a Personalized Medicine Approach to Treating GBM, an Especially Complex Cancer

Overview

Glioma is the most common form of solid tumor found in the brain. These tumors arise typically from glial cells, a family of cells supportive of neuronal function that includes astrocytes, oligodendrocytes, ependymal cells and stem/progenitor cells. Recent evidence suggests that neural stem cells may also be a cell of origin in gliomas. Proliferation of abnormal glial and/or neural stem cells in either the brain or spinal cord tissues leads to tumorigenesis. Gliomas account for about 40% of all primary brain and central nervous system (CNS) tumors and 78% of all malignant CNS tumors. Glioblastoma multiforme is the most invasive form of glioma and is 100% fatal.

Exhibit 7: Incidence



Source: NCI Snapshots 2014

- Of the estimated 79,270 cases of brain and other CNS tumors likely diagnosed in the US in 2017, approximately 26,000 will be primary malignant tumors.
- An estimated 16,947 deaths will be attributable to primary malignant brain and CNS tumors in the US in 2017.
- The median age of diagnosis for GBM is 64 years and 45 years for anaplastic gliomas.
- Malignant gliomas are 40% more common in men than women.
- There are more than 100 histologically distinct types of primary brain and CNS tumors. Survival post diagnosis is highly variable patient-to-patient and across tumor subtypes.
- Brain tumors are the most common cancer occurring in ages 0-14, and leading cause of cancer-related death in this age group.

(Source: ABTA; US CBTRUS, 2017)

As reported in the **GLOBOCAN 2012 v1.0** report from the International Agency for Research on Cancer, on a worldwide basis, the incidence rate of primary malignant brain and CNS tumors in 2012 (age-adjusted, world standard population) was 3.4 per 100,000, of which the male incidence rate is estimated at 3.9 per 100,000 and the female incidence rate estimated at 3.0 per 100,000. In 2012, these incidence rates corresponded to an estimated 139, 608 men and 116,605 women being diagnosed with primary brain tumors around the world. The incidence rates are higher in more developed countries (5.1 per 100,000) compared to less developed countries (3.0 per 100,000).

Primary Cause of GBM Still Not Elucidated

Specific underlying causes of GBM have not been identified in the majority of GBM patients, although it is known that malignant transformation of glia cells results from the accumulation of sequential genetic aberrations and growth-factor driven cell signaling dysfunction. Only approximately 5% of patients have a

family history of GBM, some of whom are associated with rare genetic syndromes such as neurofibribomatatosis (NF), Li-Fraumeni syndrome which has germ-line p53 mutations and Turcot's syndrome. Other factors implicated in the development of gliomas are exposure to ionizing radiation and genetic polymorphisms in DNA repair, cellular detoxification and cell-cycle regulation. Oncolytic viruses from the polyomavirus family (BKV, in particular) and human herpesvirus-6, both dsDNA viruses, also have been implicated in a small percentage (around 5%) of gliomas, although a direct causal relationship between viral presence, viral activity and glial cell transformation has not been confirmed. It is interesting to note, however, that these viruses' mechanisms of action, and in particular, their ability to subvert cell cycle control and deactivate/bind tumor suppressor genes, are very similar to molecular dysfunctions seen in GBM.

Patients typically experience a variety of neurological symptoms at diagnosis such as headache, nausea, seizures and progressive deficits in movement, vision and speech. Because of GBM's mechanism for growth disturbs the blood-brain barrier (BBB), patients are also susceptible to vasogenic edema, and increased interstitial pressure and mass effect caused by cerebral edema. More than 60% of GBM patients experience cerebral herniation that causes actual death. (Kamoun et al, **J. Clin. Oncol.**, 2009). Before **Avastin,** corticosteroids were used to temporarily control brain edema, but these themselves caused dose-limiting side effects.

GBM Is Highly Heterogenous and Complex

Gliomas are classified into two broad categories, low-grade and high-grade, and as primary or secondary tumors, according to their cell of origin and certain molecular features. Primary glioblastomas typically occur in older patients (over the age of 50) and show evidence of endothelial growth factor receptor (EGFR) amplification/mutations that cause the overexpression of EGFR, a tyrosine kinase receptor with downstream effects resulting in cell proliferation and invasion. Other characteristics of primary GBM include loss of heterozygosity of chromosome 10q, deletion of phosphatase and tensin homologue (PTEN) on chromosome 10 and p16 deletions. Primary GBMs develop de novo and usually occur in older patients who do not have a prior history of lower grade astrocytoma.

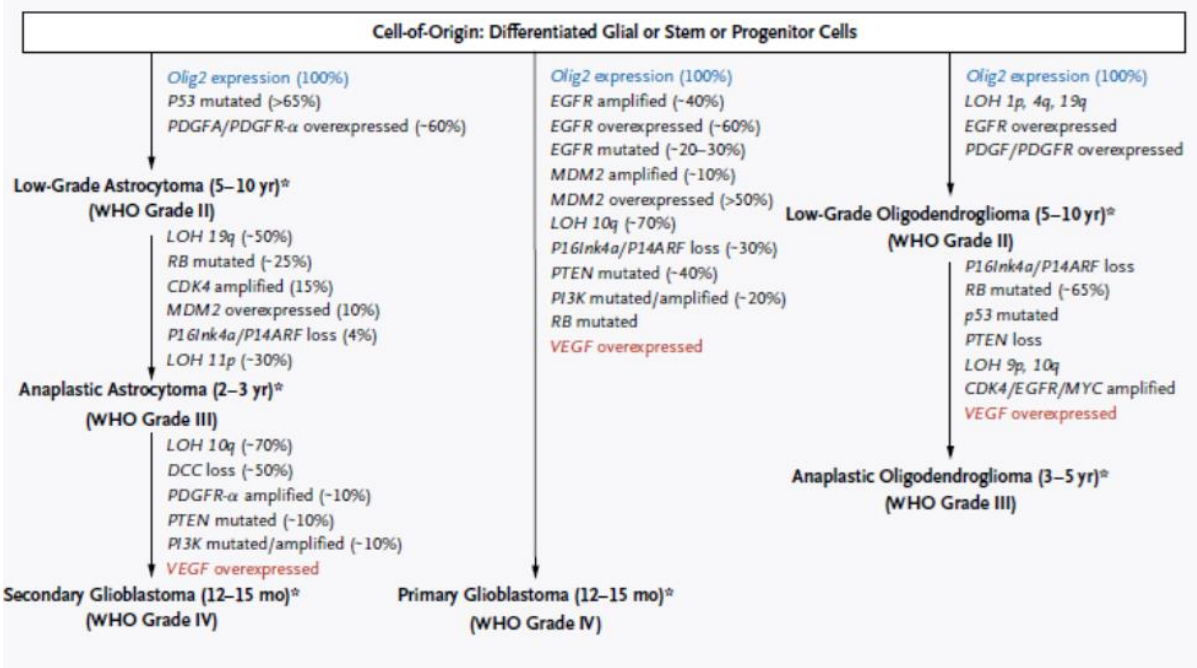
Secondary glioblastomas, typically found in younger patients, likely have transformed over years into glioblastoma from primary low-grade or AA (anaplastic astrocytomas) tumors. These tumors are frequently characterized by a variety of genetic mutations in the p53 tumor suppressor gene and at chromosome 10q coupled with over-expression of the platelet-derived growth factor receptor (PDGFR). Secondary glioblastomas have other transcriptional and DNA copy number abnormalities that are quite different from primary glioblastomas, yet on an morphological basis, secondary glioblastomas are virtually indistinguishable from primary GBM.

The World Health Organization further categorizes gliomas into prognostic grades based upon histologic features. Low-grade gliomas are derived from astrocytes (astrocytoma) and fall into Grade 1 (pilocytic astrocytoma) and Grade II (diffuse astrocytoma) gliomas. Low-grade gliomas are less invasive and patients have a somewhat better prognosis than seen with higher grade gliomas. Grade III gliomas are anaplastic astrocytomas (AA) while glioblastoma multiforme (GBM), the most common and most highly invasive tumors, comprise Grade IV tumors. Hallmark histological evidence of an anaplastic astrocytoma is increased cellularity, nuclear atypia and high mitotic activity while glioblastomas contain areas of microvascular proliferation and necrotic tissue. GBM is uniformly fatal.

GBM is the most highly vascularized of all solid tumors and relies heavily upon angiogenesis as its primary growth driver. As in normal tissues, tumor-associated angiogenesis relies upon the expression of vascular endothelial growth factor (VEGF) to cause new capillaries to sprout from existing blood vessels. In the case of GBM, mutated tumor cells drive a hypoxic microenvironment that drives an overexpression of VEGF and leads

to enhancing an aggressive angiogenic state. Because VEGF also mediates blood vessel wall permeability, the excessive VEGF results in "leaking vessels" and disturbs normal blood-brain barrier (BBB) biology. This fact underlies the use of **Avastin** in recurrent GBM, which serves to slow tumor neovascularization.

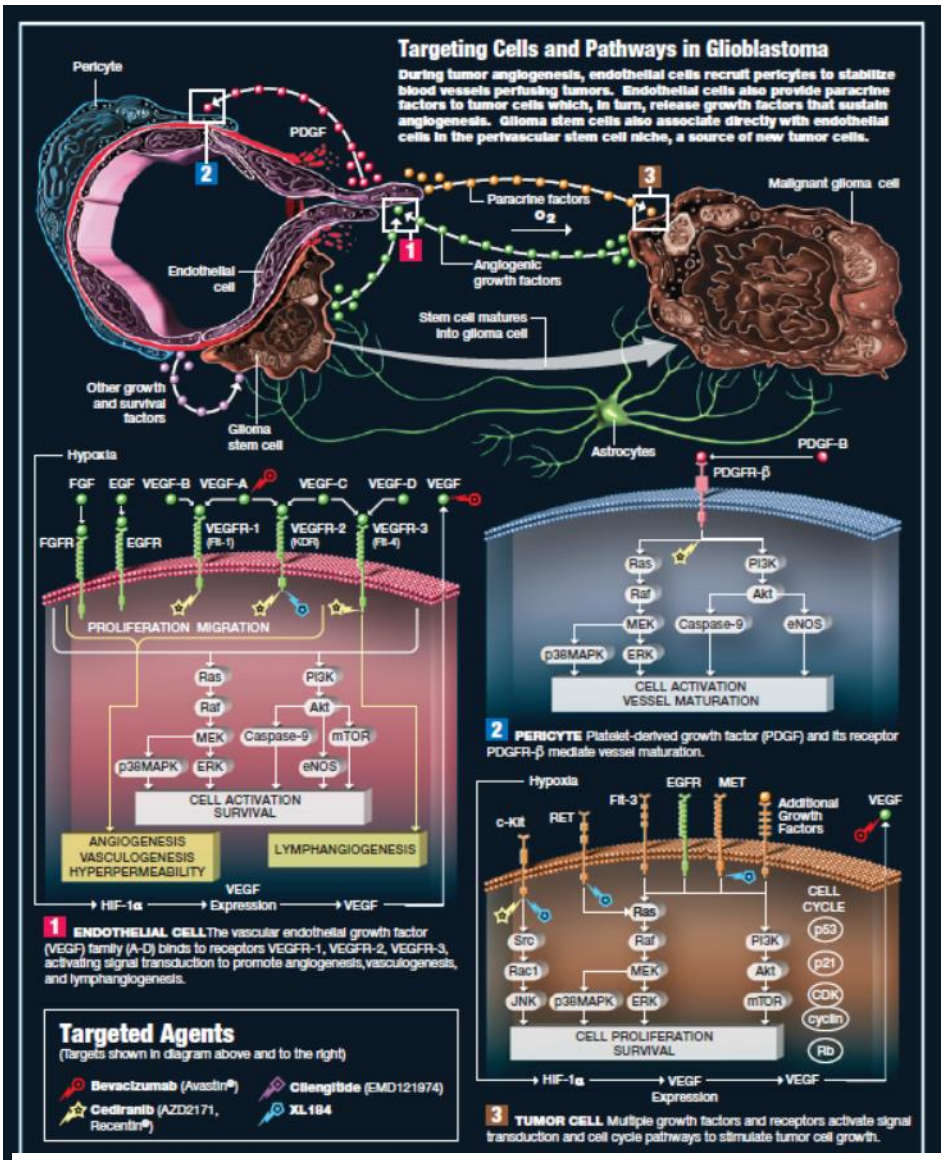
Exhibit 8: Molecular Pathways Involved with Development of Gliomas



Source: Wen et al, *Malignant Gliomas in Adults*, **NEJM**, July 2008

Further complicating the tumor microenvironment are the influences of a number of other growth factors involved in angiogenesis such as basic fibroblast growth factor (bFGF), platelet-derived growth factor (PDGF), angiopoietin-2 (ANG-2), stem cell growth factors and hepatocyte growth factor (HGF). Excess tumor vasculature may also be stimulated by the recruitment of a variety of stem and progenitor cells such as bone-marrow derived progenitor cells and/or mutated cancer stem cells combined with the tumor's capability to exploit alternative cell signally pathways such as intussusception (the opening of fluid-transporting channels within the tumor) and normal vessel co-option. Co-option occurs when mutated glial cells use normal capillaries to further infiltrate brain tissue in generating neovasculature. This process is thought to be one the primary reasons for GBM's hallmark invasiveness.

Exhibit 9: Tumor Angiogenesis



Source: Cloughesy et al, *Update on Antiangiogenic Therapy for Advanced Malignant Glioma*, **The Angiogenesis Foundation**, Summer 2009

Tumor Heterogeneity Confounds GBM Patient Prognosis

Gliomas rarely metastasize beyond the blood-brain barrier. Although this results in a relatively low tumor burden in the body compared to other cancers, gliomas have proven far more challenging to treat as they typically contain both neoplastic and stromal tissues and carry a high mutational burden. Intratumor histologic heterogeneity is further complicated by very highly variable intratumoral genetic make-up. Thus, researchers and clinicians are coming to recognize that GBM is not a single tumor type but rather comprised (and maybe at any given time point) of different molecular subtypes that are associated with distinct genetic and epigenetic signatures resulting in highly variable prognosis.

Genomic profiling is now being used to better define tumor subtypes and to circumvent the limitations of histopathological analysis by using genetic, epigenetic and transcriptomic data as aids to more objectively stratify brain tumors. Multiple studies have utilized these types of genomic data for brain tumor stratification. For example, higher grade gliomas (grades III and IV) are now divided into groups based on their association with clinical outcome. The Cancer Genome Atlas (TCGA) project has further delineated the classification of the GBMs into proneural, neural, mesenchymal and classical gene expression based subtypes.

- **Proneural:** often has high rates of alterations in p53 (TP53), in *PDGFRα*, the gene encoding α-type platelet-derived growth factor receptor, and point mutations in *IDH1*, the gene encoding isocitrate dehydrogenase-1. The classical GBM event, chromosome 7 amplification paired with chromosome 10 loss, is distinctly less prevalent in Proneural subtypes. The Proneural group also shows high expression of oligodendrocytic development genes such as *PDGFRA*, *NKX2-2* and *OLIG2*. High expression of *OLIG2* contributes to downregulation of the tumor suppressor gene p21 (*CDKN1A*), thereby increasing proliferation.
- **Neural:** typified by the expression of neuron markers such as *NEFL*, *GABRA1*, *SYT1* and *SLC12A5*.
- **Mesenchymal:** subtype is characterized by robust angiogenesis and by high rates of mutations or other alterations in *NF1*, the gene encoding Neurofibromin 1 with fewer alterations in the *EGFR* gene and less expression of *EGFR* than other types. Co-mutations of *NF1* and *PTEN*, both intersecting with the AKT pathway, are frequently observed in this subtype as are

other mesenchymal markers known as *YKL40* and *MET* . The combination of higher activity of mesenchymal and astrocytic markers (*CD44*, *MERTK*) is similar to the epithelial-to-mesenchymal transition that has been linked to dedifferentiated and transdifferentiated tumors.

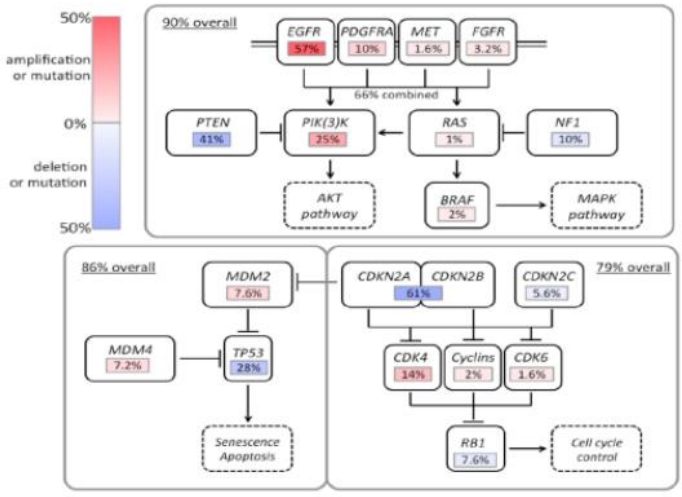
- **Classical**: Ninety-seven percent of tumors in the 'classical' subtype carry extra copies of the epidermal growth factor receptor (*EGFR*) gene, and most have higher than normal expression of epidermal growth factor receptor (*EGFR*), whereas the tumor suppressor gene, p53, often mutated in glioblastoma, is rarely mutated in this subtype. Chromosome 7 amplification paired with chromosome 10 loss is a highly frequent event in GBMs and was seen in 100% of the Classical subtype

(Source: Adapted from Verhaak et al, 2010. Note: Some gene expression classification systems have collapsed these four categories into proneural, proliferative and mesenchymal)

Many other genetic alterations have also been described in glioblastoma with the majority of them clustered in two pathways, the RB and the PI3K/AKT paths. Glioblastomas have alterations in 68-78% and 88% of these pathways, respectively. Some of the other common GBM defects include:

- High rates of defects in the tumor suppressor p53. p53 is believed to sense DNA damage and either pauses the cell in late G_1 for DNA repair or directs the cell to commit suicide through the apoptotic pathway. Altered p53 loses its function and allows cells with genetic damage to survive and enter S phase of the cell cycle, leading to an accumulation of cells with genomic mutations that may promote tumorigenic growth.
- 40-50% of primary GBM patients have an EGFR amplification defect, about 50% of these express a mutated auto-phosphorylated variant known as *EGFRviii*. *NFκ-BIA* is also often deleted but not mutated in glioblastomas; most deletions occur in nonclassical subtypes of the disease. Deletion of *NFκBIA* and amplification of *EGFR* show a pattern of mutual exclusivity.
- Overexpression of PDGF ligand and receptor that creates an autocrine loop of hyper proliferation.
- Growth factor-driven cell signaling transduction pathway defects in the MAP kinase pathway governing proliferation and cell-cycle progression and the PI3 (phosphatidylinositol-3)-K-Akt/mTOR pathway involved with inhibition of apoptosis and cellular proliferation.
- 40-50% of GBM patients have inactivation of PTEN tumor suppressor gene regulation of the PI3-K pathway
- Inactivation of these various cell signaling pathways promote the upregulation of EGFR, PDGFR and VEGFR pathways that promote the proliferation of pluripotent stem cells that can be transformed into glioma stem cells that further promote angiogenesis and resistance.

Exhibit 10: Signal Transduction and Tumor Suppressor Cellular Pathway Alterations in Glioblastoma



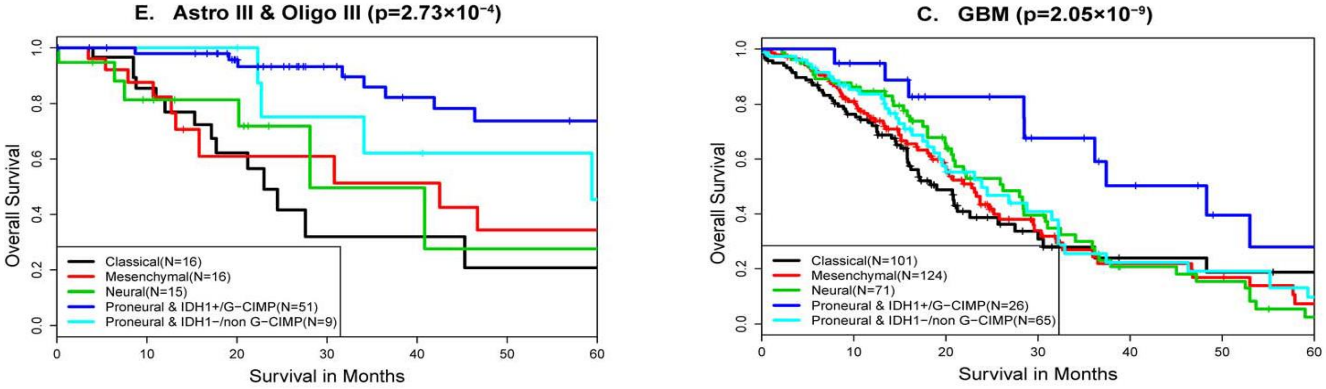
Source: Brennan et al, *The Somatic Genomic Landscape of Glioblastoma*, **Cell**, 2013

Each GBM subtype display a variable prognosis until the tumor reaches neuronal, mesenchymal and classical histologic status, at which time, survival curves closely align and overall survival rates drop to very low levels.

Exhibit 11: Median Survival in GBM by Histologic Group

		Classical (N = 135)	Mesenchymal (N = 149)	Neural (N = 89)	Proneural (IDH1-/ NON G-CIMP) (N = 74)	Proneural (IDH1+/ G-CIMP) (N = 28)
GBM (N = 476)	Average Age*	57.66	55.36	54.36	55.49	43.68
	Survival (p = 2.05e-09)*	19 (15.8, 27.5)	22.8 (19.6, 29.5)	26.2 (20.8, 32.4)	23.9 (19.3, 32.2)	48.3 (28.5, NA)
	Race (White%)&	41(95.3%)	44(95.7%)	39(97.5%)	24(100%)	10(90.9%)
	Gender (Male%)*	64 (56.1 %)	95 (71.4 %)	50 (62.5 %)	43(68.25%)	14(50%)

Kapler-Meier Survival Analysis of Grade III(AA) and Grade IV (GBM) Tumors by Histological Tumor Group and Subtype, Adjusted for age at diagnosis



Source: Adapted from Guan et al, *Molecular Subtypes of Glioblastoma are Relevant to Low-Grade Gliomas* , **PLoS ONE**, March 2014

Researchers have yet to develop standards for reliably assessing patient prognosis because of GBM's very high intratumoral heterogeneity, its high intratumoral molecular diversity and the propensity for progressive mutation of multiple molecular pathways over time. Some evidence has emerged that selected molecular features are correlated with better or worse prognosis. For example, the R132 *IDH1* mutation, which was first discovered in GBM patients, and now been found to be prevalent in lower grade gliomas, is a prognostic marker for better prognosis in both Grade II and III GBMs, especially those with the proneural subtype. Other features such as the deletion and low expression of *NFκBIA* were associated with unfavorable outcomes. Patients whose tumors display *NFκBIA* gene deletions typically have poor outcomes, similar to those

Source: Phillips et al, *Molecular subclasses of high-grade glioma predict prognosis, delineate a pattern of disease progression, and resemble stages in neurogenesis,* **Cancer Cell** , March 2006

Exhibit: 12: Summary of Molecular Markers of GBM

A	Proneural	Proliferative	Mesenchymal
Histological grade	WHO grade III or WHO grade IV with or without necrosis	WHO grade IV with necrosis	WHO grade IV with necrosis
Cellular morphology	Astrocytic or Oligodendroglial	Astrocytic	Astrocytic
Evolution of signature	Arises in 1° tumor, may persist or convert to *Mes*	Arises in 1° tumor, may persist or convert to *Mes*	Arises in 1° tumor or by conversion from other subtype
Patient age	Younger (~40 yrs.)	Older (~50 yrs.)	Older (~50 yrs.)
Prognosis	Longer survival	Short survival	Short survival
Histological Markers	Olig2, DLL3, BCAN	PCNA, TOP2A	CHI3L1/YKL40, CD44, VEGF
Tissue similarities	Adult and Fetal Brain	HSC, lymphoblast	Bone, cartilage, smooth musc, endothelium, dendritic cells
Biological process	Neurogenesis	Proliferation	Angiogenesis
Analogous forebrain cell	Neuroblast	Neural Stem Cell and/or Transit Amplifying Cell	Neural Stem Cell
Chromosome gain/loss	None	Gain of 7 & Loss of 10 or 10q	Gain of 7 & Loss of 10
PTEN locus	PTEN intact	PTEN loss	PTEN loss
EGFR locus	EGFR normal	EGFR amplified or normal	EGFR amplified or normal
Signaling	Notch activation	Akt activation	Akt activation

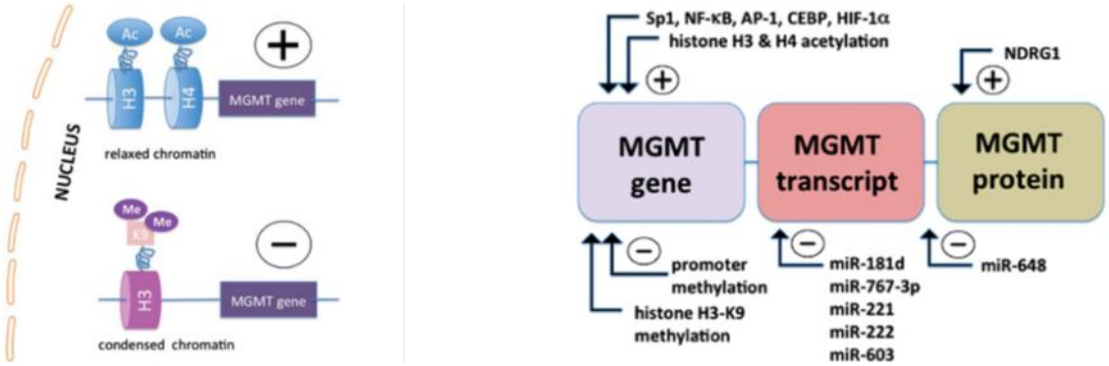
patients with tumors harboring *EGFR* amplification as compared to outcomes in patients with tumors that have normal gene dosages of *NFκBIA* and *EGFR*. A two-gene prognosis predictive model based on expression of *NFκBIA* and O6-methylguanine DNA methyltransferase has also been found to have strong association with the clinical course of the disease. (Bredal, et al *NFKBIA Deletion in Glioblastomas* **NEJM**, Feb 11, 2011).

GBM Treatment Resistance Drives Poor Prognosis

In addition to complex heterogeneity, an additional challenge in the management of gliomas is the nearly universal propensity of GBM neoplasms to contain cells that survive surgery and radiation therapy to form recurrent lesions at or very near the primary site, but with molecular modifications that make the tumor resistant to further treatment. Radioresistance in GBM stem cells generally results from the preferential activation of DNA damage response mechanisms. Upregulated genes in mesenchymal GBM include *YKL40*, CD44, and *STAT3*. *YKL40* presence is reported to predict radioresistance in human tumors (Pelloski et al., 2005) and to promote clonogenic survival, consistent with a relative increase in this gene's expression upon tumor recurrence after treatment. Chemo-resistance results from the over-expression of DNA repair mechanisms and in particular, overexpression of O^6-methylguanine-DNA methyltransferase. MGMT protects glioblastoma tumor cells against alkylating agents including TMZ by removing the alkylating agent's crosslinks from the O^6-position of guanine.

The landmark European Organization for Research on Treatment Cancer (EORTC) 26981 trial, along with a series of confirmatory studies, demonstrated that epigenetic silencing of MGMT gene by promoter methylation was of predictive significance for prolonged survival in patients under 70 years old who were treated with a combination of TMZ and RT. Since an MGMT enzyme can only repair one DNA alkylation due to its suicide repair mechanism, reverse capacity is low and the degree of methylation of the MGMT gene promoter greatly affects DNA-repair capacity. High levels of MGMT methylation is associated with an improved response to alkylating agents, such as TMZ. From various studies, the MGMT locus has been found to be methylated in approximately 40-45% of GBM patients. CpG Island methylator phenotype (G-CIMP) expression enhances the likelihood of MGMT DNA methylation.

Exhibit 13: MGMT Gene Expression



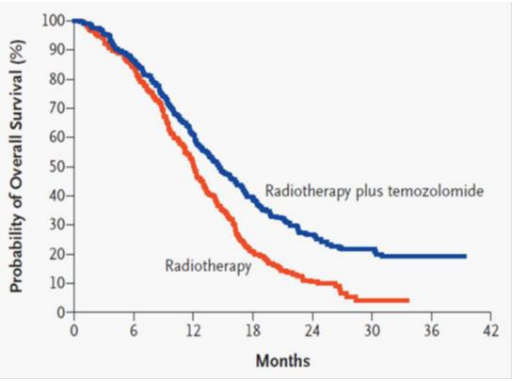
Left: Effect of histone modification on MGMT gene expression. Acetylation of histones H3 and H4 promotes MGMT transcription, whereas di-methylation of lysine 9 on histone H3 represses MGMT transcription.

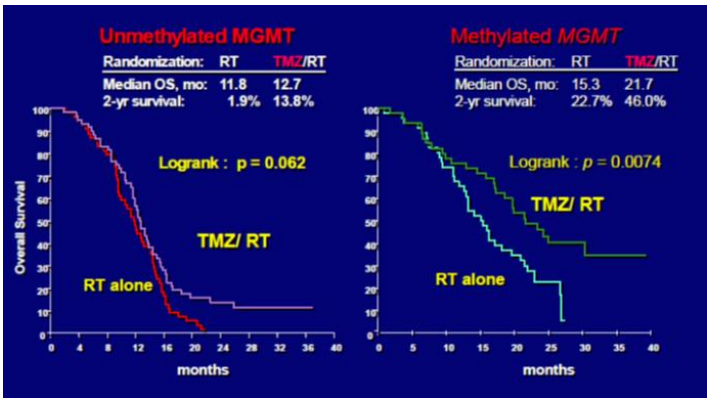
Right: Summary of different known modulators of MGMT gene expression. MGMT expression is increased by different nuclear transcription factors (Sp1, NF-κB, AP-1, CEBP and HIF-1α), together with the acetylation of histones H3 and H4 and the stabilization by binding of N-myc downstream regulated gene 1 (NDRG1) protein. On the contrary, MGMT expression is downregulated by different mechanisms, namely methylation of the CpG islands in the promoter, di-methylation of histone H3K9, degradation of mRNA by miR-181d, -767-3p, -221, -222, -60 and , interference with protein translation by miR-648

Source: Cabrini et al, *Regulation of expression of MGMT and the treatment of glioblastoma (Review),* **Intl. Jrl. Onc.,** 2015

As described in Brennan, et al, *The Somatic Genomic Landscape of Glioblastoma*, **Cell**, 2013, when correlated with outcome, MGMT methylation status distinguished responders from nonresponders among tumor samples classified as classical subtype (n = 96; p = 0.01) but not among samples classified as proneural (n = 66; p = 0.57), mesenchymal (n = 104; p = 0.62) and neural (n = 55; p = 0.12) subtypes. This study provided evidence for MGMT DNA methylation as a predictive biomarker in the GBM classical subtype of GBM, but not other subtypes. Regardless, however, MGMT promoter methylation testing is allowing for a more individualized management of glioblastoma patients.

Exhibit 14: MGMT Promoter Methylation and Its Impact on Survival

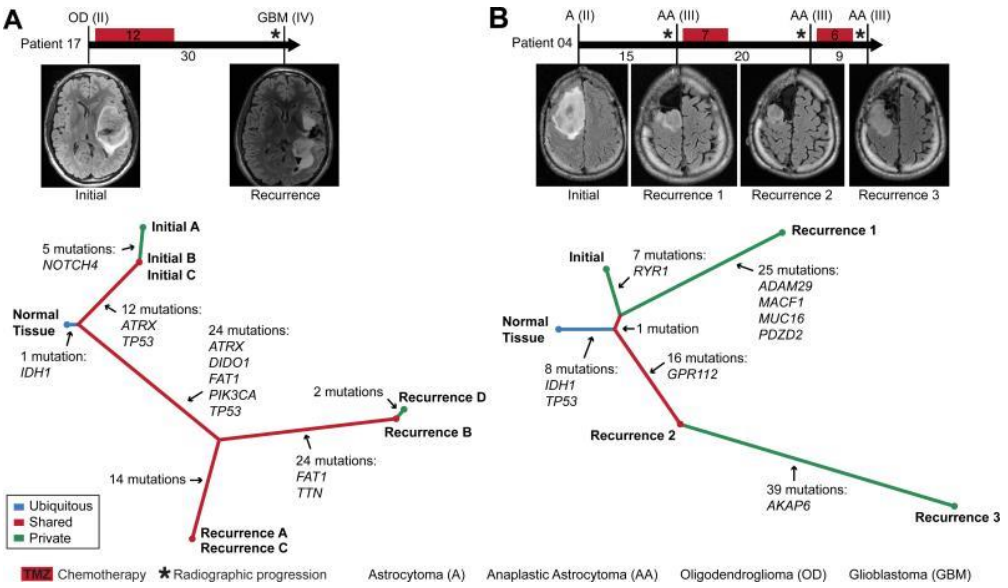




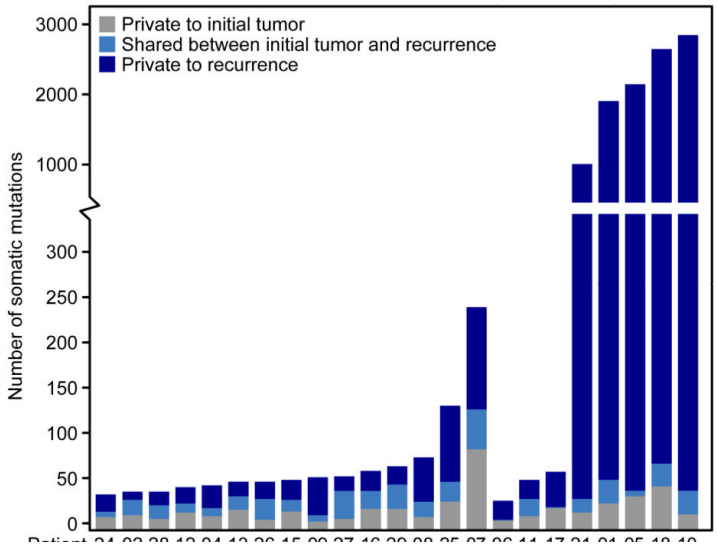
SOC developed 12 years ago with seminal clinical study by Strupp. Showed clear benefit of adding temozolomide to RT. However, in looking at patient responses more closely, it was found that the clinical benefit demonstrated in the trial as a whole was largely due to a significant benefit to the methylated MGMT patient base. As the $MGMT^+$ vs $MGMT^-$ (unmethylated) analysis below shows, the addition of TMZ gave only marginal benefit to patients whose tumors were $MGMT^-$.
Source: Adapted from Strupp by Perry, Life Sci Advisors GBM KOL Perry presentation 3/9/17

Even though MGMT methylation provides a useful marker for potential response to TMZ, this is of a temporary nature as patients invariably become resistant and suffer disease progression. Unfortunately, in part because of the mechanism of action of TMZ, the problem is further compounded by the expression of new mutations that seem to be conferred by TMZ itself. In the Johnson et al study published in **Science** in 2014, this point was highlighted in a review of patients' disease progression over time. Panels A and B depict two separate patients.

Exhibit 15: Genetic landscapes of low-grade gliomas and their patient-matched recurrences



Total number of mutations private to or shared between the initial and first recurrent glioma of 23 patients



Source: Johnson et al, *Mutational Analysis Reveals the Origin and Therapy-driven Evolution of Recurrent Glioma*, **Science**. Jan. 2014

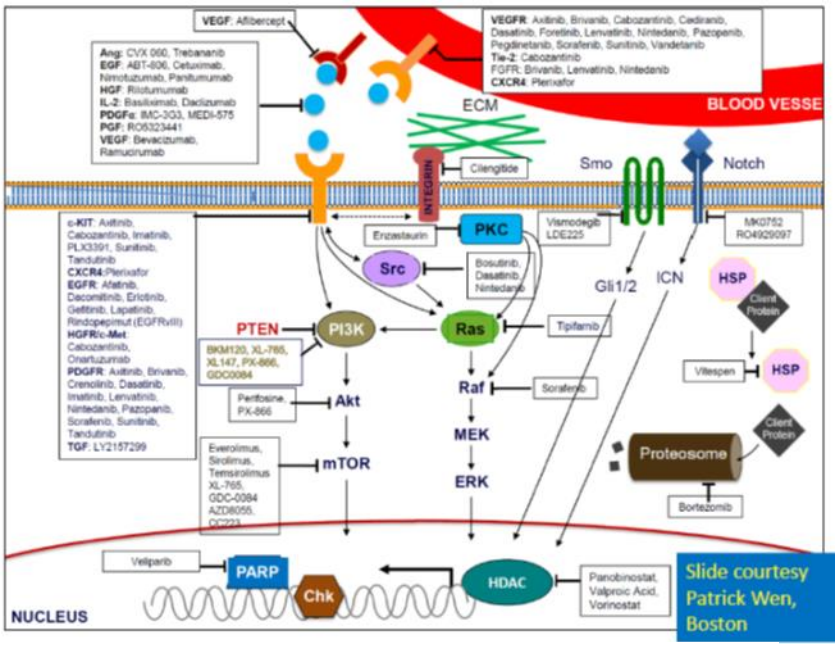
Although, the identification of these molecular markers has provided enticing rationales for targeted therapy development, the association between genome-wide classifiers and the clinical features of patient gliomas is still unclear and to date, virtually all attempts to extend survival by the use of targeted therapeutics have failed.

DelMar believes that VAL-083 can avert MMR driven types of resistance as its mechanism of action does not allow single DNA strand repair and therefore, perhaps will provide a longer window before other types of resistance occur. In addition, because it may be more effective in mitigating resistance, VAL-083 may perhaps retard the evolution of new mutations.

Competition- A Plethora of Therapeutic Approaches has resulted in a Litany of Clinical Failures

The competitive landscape in GBM is very different than most other cancers. The heterogeneity of tumor, coupled with multiple molecular escape mechanisms that lead to rapid treatment resistance, has combined to thwart virtually every new technical approach to treating GBM including various radiotherapy regimens, numerous small molecule alkylating agents, with various dosage and administration regimens, anti-angiogenic compounds, immune therapies, targeted therapies, fusion proteins, gene therapy, stem cells and now CAR-T and checkpoint inhibitors. Many of these approaches showed early success in Phase I and Phase II trials, only to fail in Phase III, either in an outright sense by failing to meet primary survival endpoints, or by failing to even incrementally do any better than the standard of care and TMZ. A recent promising candidate, rindopepimut from Celldex, that targets EFGRviii mutation, failed to demonstrate any additional benefit over the control arm in its 2016 Phase III trial. BMY's

Exhibit 16: Many Attempts, No Winners



Source: Adapted from Life Sci Advisor GBM KOL Perry presentation 3/9/17

mutation, failed to demonstrate any additional benefit over the control arm in its 2016 Phase III trial. BMY's

Opdivo® + **Yervoy®** combination failed Phase III this past April and in Merck AG's CENTRIC 2014 cilengtide trial in a MGMT + patient base, the median overall survival in the treatment group was less than control arm. (ASCO poster 101514, Stupp et al).

Exhibit 17: Partial List of Clinically Investigated GBM Treatments

Type of Treatment	Example
Convection-enhanced surgical delivery of pharmacologic agent	Cintredekin besudotox
Drugs to overcome resistance to TMZ	
Dose-dense TMZ	
MGMT inhibitors	O^6-benzylguanine
PARP inhibitors	BSI-201, ABT-888
New chemotherapies	RTA744, ANG1005
Antiangiogenic therapies	
Anti-$\alpha v\beta 5$ integrins	Cilengitide
Anti-hepatocyte growth factor	AMG-102
Anti-VEGF	Bevacizumab, aflibercept (VEGF-Trap)
Anti-VEGFR	Cediranib, pazopanib sorafenib, sunitinib, vandetinib, vatalanib, XL184, CT-322
Other agents	Thalidomide
Targeted molecular therapies	
Akt	Perifosine
EGFR inhibitors	Erlotinib, gefitinib, lapatinib, BIBW2992, nimotuzumab, cetuximab
FTI inhibitors	Tipifarnib, lonafarnib
HDAC inhibitors	Vorinostat, depsipeptide, LBH589
HSP90 inhibitors	ATI3387
Met	XL184
mTOR inhibitors	Everolimus, sirolimus, temsirolimus, deforolimus
PI3K inhibitors	BEZ235, XL765
PKCβ	Enzastaurin
PDGFR inhibitors	Dasatinib, imatinib, tandutinib
Proteasome	Bortezomib
Raf	Sorafenib
Src	Dasatinib
TGF-β	AP12009
Combination therapies	Erlotinib plus temsirolimus, gefitinib plus everolimus, gefitinib plus sirolimus, sorafenib plus temsirolimus, erlotinib, or tipifarnib, pazopanib plus lapatinib
Immunotherapies	
Dendritic cell and EGFRvIII peptide vaccines	DCVax, CDX-110
Monoclonal antibodies	^{131}I-anti-tenascin antibody
Gene therapy	
Other therapies	^{131}I-TM-601

* Data are from Sathornsumetee et al.,[3] Furnari et al.,[18] Chi and Wen,[20] and Sathornsumetee et al.[21] EGFR denotes epidermal growth factor receptor, FTI farnesyltransferase, HDAC histone deacetylase, HSP90 heat-shock protein 90, MGMT O^6-methylguanine–DNA methyltransferase, mTOR mammalian target of rapamycin, PARP poly (ADP-ribose) polymerase, PDGFR platelet-derived growth factor receptor, PI3K phosphatidylinositol 3-kinase, PKCβ protein kinase C β, TGF transforming growth factor, TMZ temozolomide, and VEGFR vascular endothelial growth factor receptor.

Source: Wen et al, Review, *Malignant Gliomas in Adults*, **NEJM**, 2008

Therefore, competition is really about providing effective complementary and alternative therapies (at this juncture) that contribute incrementally to moving the TMZ + RT survival curve outward and improving or sustaining a quality of life, especially as it relates to mitigating seizures and declines in cognition and motor capabilities. A number of later stage trials being run by companies such as Agenus, Oncoceutics, Bayer and others, will be reading out in the late 2017-2018 timeframe.

The Optune Device

It is worth mentioning, however, that the most recent advancement in the treatment of glioblastoma has been with adding tumor-treating electric fields (the FDA-approved **Optune** medical device, NovoCure Ltd.) to the standard TMZ adjuvant care. Tumor-treating fields are low-intensity electric fields that are alternating with an intermediate electric field frequency of 200kHz. In a randomized Phase III study reported at this year's AACR and led by Roger Stupp, MD, from the Northwestern University (who is credited with conducting the original research that led to the standard use of TMZ following radiation), researchers found the median PFS was 6.7 months for patients treated with TTFields + TMZ compared to 4.0 months with TMZ alone, a highly significant improvement in PFS measures (hazard ratio of 0.63, p=0.00005). A hazard ratio of 0.63 means that these patients had a 37% reduction in risk of death compared to those treated with TMZ alone. Additionally, the median overall survival (OS) was reported as 20.9 months for TTFields vs 16 months for TMZ, also a highly significant differential (hazard ratio of 0.63, p=0.00006).

The two year survival rate comparison was 43% and 31%, respectively (p=0.0008) and five year survival rates were 13% and 5%, respectively (p=0037) for the 695 patient study. Use of TTFields for more than 18 hours per day was associated with significantly higher long term survival rates compared to use of less than 18 hours per day. These survival rates represent perhaps a new benchmark for glioblastoma survival and target for new combination therapies. These results, according to Dr. Stupp, are the first set of trial results in the last 10 years that have shown any improvement in survival for these patients since the addition of TMZ to radiotherapy, which increased two-year the survival rate from 10% to 27%. (AACR press release, 4/2/17). Most encouragingly, TTFields show a beneficial effect in all subgroups of patients treated, including those with the most unfavorable prognosis. Data presented to the FDA at the trial's interim analysis provided the basis for an expanded indication into newly diagnosed GBM from the earlier approved recurrent GBM indication.

Market Opportunity

A **Transparency Market Research** report, *"Pipeline Review of Glioblastoma Treatment Market –Global Industry Analysis, Size, Share, Growth, Trends and Forecast 2014-2022"* published in mid 2015 valued the global GBM drug treatment market at $340 million and estimated the market would reach $900 million in 2022 for a CAGR of 11.4% over the 2014-2022 timeframe. Research and consulting firm **GlobalData** anticipates the glioblastoma treatment market will increase five times from $659 million in 2014 to $3.3 billion by 2024, a 17 percent compound annual growth rate over the 10 year period. Much of the growth incorporated into these projections anticipated the approval of now failed Phase III drugs, (most especially **Opdivo**) leaving the market still dominated by temolozomide (generic as of 2013) and **Avastin**, which lost market exclusivity in April 2017. According to IMS data quoted in various reports, peak sales of branded **Temodar** reached approximately $1.1 billion, but by 2011, sales had slipped to $935 million because of loss of exclusivity in the EU and the pending loss of exclusivity in the US. **Avastin** generated approximately $200 million from GBM treatment that same year. While **Avastin** sales dollars have shown steady growth due to a lack of competition and wider use in refractory GBM, generic temolozomide produced by now several manufacturers has eroded away much of **Temodar**'s franchise. According to IMS Health, the six strengths of generic TMZ capsules had sales of $176.5 million in the US in the 12 months ended May 31, 2016, while Merck reported **Temodar** sales in 2016 of $283 million. Based upon these data points and assuming minor revenue from alternative drugs such as BCNU, we are estimating the current GBM drug treatment market at about $600 million. With the lack of recent drug trials

being successfully converted to approved drugs coupled with **Avastin** now off-patent, we see this market remaining fairly static until a new drug approval occurs. The **Optune** device, priced at some $20,000 per month of treatment, is not likely to materially expand the market. Based upon the **Avastin** experience in the salvage therapy setting, we believe DelMar has a $200-300 million salvage market opportunity if VAL-083 can meet compelling overall survival endpoints.

Taking a more "global" view, GBM is a high-cost medical need. In a study entitled. *"Treatment Patterns, Survival, and Healthcare Costs of Patients with Malignant Gliomas in a Large US Commercially Insured Population,"* (Ray et al, **Am. Heath & Drug Benefits**, 2014), researchers studied costs across SOC (RT + TMZ), RT alone, TMZ alone and no treatment patient cohorts. In a rigorous analysis to purify patient data for GBM and reflective of the Stupp regimen, the total healthcare costs over 1 year per patient were $184,107 for the TMZ+RT group and was sharply higher than the one-year $80,000 cost reported by Kutikova which did not include the cost of TMZ. As these patients nearly universally progress to death, finding treatments that can materially extend life as well as reduce the total burden to the healthcare system (ie. emergency room visits, reduced ancillary drug needs such antiemetics and neutropenia-related drugs, doctor office visits, etc.) is sorely needed.

Competitive Landscape

Exhibit 18: Companies with Mid to Late Stage GBM Clinical Trials

Company	Symbol	Trial Phase	Completion	Comments
Acerta Pharma BV	pvt	Phase I/Iia	Complete Jan 2018	ACP-196 1st or 2nd recurrence No information AZ majority owner BTK inhibitor platform
Actelion	atln.vx ALIOF	Phase I	Completed June 2016	Macitentan, dual Endothelian receptor antagonist +SOC oral FDA approved for PAH, abandoned GBM
Activartis Bio	pvt	Phase II	FAILED No PFS impvmt	AVO-113 (Trivax-Dendritic cell vaccine +IL-12) Add on to SOC vs SOC alone (June 2015)
Agenus	AGEN	PI/II	Completed	HSPPC96 autologous vaccine complex
		PII	Completed	HSPPC96 autologous vaccine complex concurrent with TMZ ~46 patients
		PII	Complete July 2017	4yr 90 patient HSPPC96 + Avastin vs Avastin only in recurrent patients 1st end pt: OS and PFS
Agios Pharmaceuticals	AGIO (BN)	Phase I	Complete Aug 2017	AG-120 (ivosidenib-oral targeting IDH-1 mutation)
	AGIO/Celgene	Phase I/II	Completed June 2016	AG-221- oral IDH-1 inhibitor
		Phase I		AG-881 oral mutant IDH1/2 inhibitor
Advantagene	pvt	Phase IIa	Completed Aug 2016	I/O AdV-tk + Valacuclovir prodrug +SOC
AngioChem Inc,		Phase II	Complete July 2017	ANG-1005 -debulking agent add-on to Avastin in recurrent patients
Apogenix GmBH	pvt	Phase II	Completed June 2015	RT + APG101 (CD95+ Fc fusion protein) blocks the CD95 ligand binding of CD95 ligand to its receptor stimulates GBM invasive growth
Cantex Pharmaceuticals/Wash. Universit	pvt	Phase II	Complete Apr 2018	Disulfiram + Cu (CX-02) Add-on at 1st recurrence DSF + Cu sensitizes TMZ-resistant cells
		Phase I	Complete Apr 2020	Disulfiram + Cu pre-conditioning prior to surgery/RT
Washington University		Phase I	Complete Mar 2018	Disulfriam + Cu Add on newly diagnosed
University of Ulm/Reliable Cancer Therapies		Phase II/III	Complete 2020	Disulfiram + Cu Add-on at 1st recurrence
CellDex	CLDX	Phase III	FAILED (Mar. '16)	CDX-110 (Rindopepimut+ KLH) + SOC ACT IV study: OS 20.4mo vs 21.1 mo control HR: 0.99
Cortice Biosciences	pvt	Phase II	TERMINATED	co-admin Avastin + TPI 287, 3rd line -1st 17 pts showed no benefit
		PhasI/Iia	Complete Feb 2018	co-admin Avastin + TPI 287, 2nd line vs Avastin alone 2nd line
CytRx	CYTR	Phase II	Completed Dec 2016	28 patients 2nd line (no Avastin) open label Adloxorubicin (INNO-206)-albumin conjugate NO information
Exelixis/NIH	EXEL	Phase I	Complete Oct 2017	XL-184 (BMS 907351) pan TKI Cabozantinib S-Malate-not recruiting -Abandoned?
Immatics Biotechnologies GmbH		Phase I	Complete July 2018	APVAC1 personalized polypeptide vaccine + immun-stimulators concurrent with 1st line TMZ in newly diagnosed GBM
ImmunoCellular Therapeutics	IMUC	Phase III	Complete Dec 2019	414 patients ICT-107 + SOC cancer vaccine: autologous DC cells pulsed with polyTAAs -HLA-A2 restricted
Inspyr (Genspera)	NSPX	Phase II	Completed Feb 2017	26 patients, open label single arm G-202 mipsagargin prodrug for PSMA targeting SNO 2015 abstract?
Karyopharm Therapeutics	KPTI	Phase II	Complete June 2017-NR*	Selinexor (KPT 330) pre and post surgery in recurrent patients-KING trial
Medicenna Therapeutics	MDNA.V	Phase IIb	Complete Aug 2018	43 patient MDNA55 IL4R targeted infusion via convection-enhanced delivery in recurrent patients, initiated Apr 2017
Merrimack Pharmaceuticals	MACK	Phase I/II	Complete 2020	BrUOG 329 Onyvide + TMZ SOC
Progenics (Merrimack Pharmaceuticals)	PGNX	Phase II	Completed	PSMA prodrug BrUOG 263-FAILED-no effect due to minimal expression of PSMA and Tox
Nektar Pharmaceuticals	NTKR	Phase II	Completed Feb 2015	PEGlyated irinotecan-Abandoned??
Northwest Biotherapeutics	NBIO	PIII	Completed Nov 2016	348 patient DCVax-L(autologous denritic cell vaccine)+ SOC or SOC alone open label expanded access continuing
Oncoceutics	pvt	Phase II	Complete Dec 2017	ONC-201 MGH & Dana Farber sm molecule imipridones target G-protein coupled receptors-oral
OncoScience AG	pvt	Phase III	FAILED	150 patients, SOC + OSAG 101 Theraloc (Mab) FAILED, FAILED EMA approval (YM Biosciences) EGFR-inhibitor
OncoVir	pvt	Phase I/II	FAILED	Poly-ICLC/HiltonolTLR3 cytosol nucleic acid sensor (KLH) Abandoned post EORTC Phase III changed SOC to RT + TMZ
Oryx GmBH		Phase I/IIa	Completed May 2015	oncolytic vaccine ParvOryxO1 (parvovirus) OS6=77.8% Further development??
Stemline Therapeutics	STML	Phase I/IIa	Complete July 2017	GBM tumor antigen, IL-13 vaccine + poly ICLC +/- Avastin immedicately or later --1st recurrence 27% prob of median PFS of 5.6 mo
Symphogen AG	pvt	Phase II	Complete Oct 2018	SYM004 (rEGFR Mab) in Avastin failure vs non-Avastin failure
TRACON Pharmaceuticals	TCON	Phase II	no update	TRC105- anti-endoglin Mab + Avastin
TCON/NIH		Phase II	TERMINATED	lack of efficacy signal
NIH		Phase I/IIa	FAILED (Dec '16)	TRC105- anti-endoglin Mab + Avastin endpoint: 3 mo impvment over 3.45 PFS Avastin historical control
Tocagen	TOCA	Phase II/III	Phase III complete 2019	Toca 5- recurrent GBM or AA, Phase II Toca511 +Toca FC portion complete, topline to report 1H 2018
		Phase Ib	Complete Nov 2019	Toca 6- multiple arm for metastatic cancers +/- checkpoint inhibitors
		Phase I	Complte July 2017- NR	Retroviral replicating expression vector for Toca511 5-FC conversion to 5-FU in recurrent GBM

Competitive Landscape (Continued)

TVAX Biomedical (ELIAS An. Hlth)	pvt	Phase I/IIa	Completed 2015	Autologous primed polyantigen vaccine
Vascular Biogenics Ltd (Vascular Thera.)	VBLT	Phase III	Complete Dec 2017	GLOBE: 252 pts VBL-111 +/- Avastin
Ziopharm Oncology	ZIOP	Phase I/IIa	Completed	prodrug INXN-2001 ligand activated to Ad-IL12 1st/2nd recurrence median OS >12 mos
		Phase I	Complete Dec 2017	prodrug INXN-2001 ligand activated to Ad-IL12 1st/2nd recurrence
Roswell Park Cancer Institute	NA	Phase I	completed	vaccine therapy with montanide ISA-51/survivn peptide vaccine
Bayer		Phase II	completed	Sorafenib (TKI, anti-VEGF +TMZ open label--side effects
		Phase II	Oct 2017, Oct 2018	Regorafenib-(BAY 73-4506)-3rd line post Lomustine-
				Stivagra -follow-on to regorafenib --
Genetech		Phase II	completed	Avastin + ONAR (MET inhibitor) failed. HR: 1.45
				noted biomarker correlation to MET HGF and MGMT methylation status, tumor subtype
Abbvie		Phase I/IIa	Completed	ABT414 (EGFR+mononmethyl auristatin F (MMAF) conjugate)
Amgen		Phase I/Iia		AMG 595 tareting EGFRviii
		Phase I/IIa	Complete May 2017 N/R*	FAILED 1st arm: AMG 386 +/- Avastin in recurrence
Bristol-Myers Squibb	BMY	Phase III	FAILED (Apr. '17)	Opdivo + Yervoy (CheckMate-143 trial)
		Phase II		CheckMate 498

Source: ClinicalTrials.gov, company websites
*NR-Trial still open, not recruiting

Financial Review
Third Fiscal Quarter

DelMar is a drug discovery and development company without revenues. For the third fiscal quarter, research and development expenses increased to $1,086,107 for the three months ended March 31, 2017 from $790,323 for the three months ended March 31, 2016. The increase was largely attributable to increased clinical research and intellectual property-related activities, and partially offset by a reduction in non-cash expenses compared to the prior year. Excluding the impact of non-cash expense, research and development expenses increased to $968,332 during the three months ended March 31, 2017 from $660,857 for the three months ended March 31, 2016. The increase in clinical costs for the 2017 period compared to a year ago was primarily due to protocol development and manufacturing costs associated with the upcoming Phase III study. Intellectual property costs increased in the current period compared to the prior period as the Company continued to expand and advance its patent portfolio.

General and administrative expenses were $698,125 for the three months ended March 31, 2017 compared to $630,226 for the three months ended March 31, 2016. The increase was primarily due to an increase in professional fees, personnel, and office and sundry costs partially offset by a reduction in non-cash expenses, including a reduction of approximately $50,100 in stock option expense and warrants paid for services in 2017 compared to the 2016 period. Excluding the impact of non-cash expenses, general and administrative expenses increased in the three months ended March 31, 2017 to $635,769 from $517,030 for the three months ended March 31, 2016. In addition to normal increases in staff costs, office and sundry costs were higher during the three months ended March 31, 2017 compared to March 31, 2016 due in part to higher stock exchange listing fees as the Company's common stock was listed on NASDAQ in the 2017 period compared to the OTCQX listing in the prior period. In addition, the Company incurred costs to upgrade its web site and participated in additional promotional activities. DelMar succeeded in its uplist in July 2017.

The Company ended the quarter with $2.1 million in cash. Subsequently, in April 2017, DelMar completed a financing that provided approximately $8 million in additional funding. These funds will be used to support the Phase III trial which is anticipated to cost $8-12 million.

Exhibit 19: Consolidated Balance Sheet and Income Statement

Consolidated Condensed Interim Balance Sheets (Unaudited) - USD ($)	Mar. 31, 2017	Jun. 30, 2016
Current assets		
Cash and cash equivalents	$ 2,100,406	$ 6,157,264
Taxes and other receivables	70,561	18,387
Prepaid expenses	119,776	144,131
Deferred costs	25,705	
Assets current	2,316,448	6,319,782
Intangible assets - net	24,443	36,017
Total assets	2,340,891	6,355,799
Current liabilities		
Accounts payable and accrued liabilities	791,805	584,002
Related party payables	70,259	43,444
Current portion of derivative liability	157,145	
Liabilities current	1,019,209	627,446
Stock option liability		175,875
Derivative liability	91,545	693,700
Total liabilities	1,110,754	1,497,021
Stockholders' accumulated equity		
1 special voting share at March 31, 2017 (June 30, 2016 - 1)		
Common stock Authorized 50,000,000 shares, $0.001 par value 11,675,174 issued at March 31, 2017 (June 30, 2016 - 11,187,023)	11,675	11,187
Additional paid-in capital	31,511,379	28,833,105
Warrants	1,618,656	1,658,382
Accumulated deficit	(38,401,763)	(32,237,859)
Accumulated other comprehensive income	21,178	21,178
Stockholders' equity, total	1,230,137	4,858,778
Liabilities and equity	2,340,891	6,355,799
Series A shares		
Stockholders' accumulated equity		
Preferred stock, value	278,530	278,530
Series B Preferred Stock		
Stockholders' accumulated equity		
Preferred stock, value	$ 6,190,482	$ 6,294,255

Source: DelMar SEC filings

Consolidated Statement of Operations and Loss

Consolidated Condensed Interim Statement of Loss and Comprehensive Loss (Unaudited) - USD ($)	Year Ended 6/30/2016	3 Months Ended 3/31/2017	3 Months Ended 3/31/2016	9 Months Ended 3/31/2017	9 Months Ended 3/31/2016	Year Ended 6/30/2017E	Year Ended 6/30/2018E
Expenses							
Research and development	$ 3,360,878	$ 1,086,107	$ 790,323	$ 2,939,746	$ 2,183,355	$ 4,174,439	$ 6,219,915
General and administrative	2,853,140	698,125	630,226	2,586,050	1,994,923	3,219,632	3,927,951
Total Operating expenses	$ 6,214,018	$ 1,784,232	$ 1,420,549	$ 5,525,796	$ 4,178,278	$ 7,394,072	$ 10,147,866
Other loss (income)							
Change in fair value of stock option and derivative liabilities	2,341,660	77,479	(276,584)	(58,501)	943,050	(8,501)	500,000
Change in fair value of derivative liability due to change in warrant terms	295,456		7,000		270,965		
Foreign exchange	(13,838)	6,897	(10,523)	13,726	16,257	18,726	15,000
Interest income	(108)	(148)	(41)	(249)	(71)	(185)	(150)
Other (income) loss	2,650,846	84,228	(280,148)	(45,024)	1,230,201	10,041	514,850
Net and comprehensive loss for the period	$ 8,864,864	$ 1,868,460	$ 1,140,401	$ 5,480,772	$ 5,408,479	$ 7,404,112	$ 10,662,716
Computation of basic loss per share							
Net and comprehensive loss for the period	$ 8,864,864	$ 1,868,460	$ 1,140,401	$ 5,480,772	$ 5,408,479	$ 7,404,112	$ 10,662,716
Series B Preferred stock dividend		209,811		676,865		886,676	886,676
Net and comprehensive loss available to common stockholders	$ 8,864,864	$ 2,078,271	$ 1,140,401	$ 6,157,637	$ 5,408,479	$ 8,290,788	$ 11,549,392
Basic and fully diluted loss per share	$ 0.81	$ 0.18	$ 0.10	$ 0.54	$ 0.50	$ 0.67	$ 0.69
Basic weighted average number of shares	10,948,481	11,574,052	11,077,275	11,432,376	10,896,887	12,288,606	16,843,284

Source: DelMar SEC filings, DJ estimates

Pre-Uplist 2016 Preferred Stock Offering Gave Certain VAL-083 Royalty Rights to Holders

In May 2016, DelMar sold 700,238 Series B Preferred Shares for gross proceeds of $5.6 million. The Series B Preferred Shares are not registered for resale and did not include any anti-dilution provisions or warrant coverage. As an incentive to the investors to retain ownership of the Preferred Shares, DelMar also entered into a Royalty Agreement with the holders whereby DelMar will pay a single-digit royalty based on their pro rata ownership of the Preferred Shares. The royalty shall be payable based on future direct sales of VAL-083 by the Company or for cash received from partnering or licensing arrangements. The royalty is earned by the holder at closing if and only if the holder retains the Preferred Shares and has not converted to common stock in parallel with the offering, DelMar also entered into an agreement with certain warrant holders to amend terms of 2,095,238 warrants by removing and changing certain language in order to remove derivative liability accounting treatment. In consideration of acceptance of amended terms, the warrant term was extended by one year to March 31, 2019. The warrants remain exercisable at $0.78 and are subject to a redemption provision, at the Company's option, if the price of the Company's common shares trades above $1.60 for twenty (20) consecutive trading days. Further information can be found in the Company's SEC filings.

Peer Group Analysis and Valuation

Company	SYM	Clinical Phase	Market Cap
GBM focused			($MM)
CytRX	CYTR	Phase II	81.4
ImmunoCellular Therapeutics	IMUC	Phase III	4.0
Inspyr (Genspera)	NSPR	Phase II	0.8
Medicenna Therapeutics	MDNA.V	Phase II	57.1
Merrimack Pharmaceuticals	MACK	Phase I/II	186.7
Northwest Biotherapeutics	NWBO	Phase III	39.6
Tocagen	TOCA	Phase II/III	316.1
VBL Therapeutics	VBLT	Phase III	148.0
		Group Average	**104.2**
DelMar Pharmaceuticals	DMPI	Phase II-III	27.64

We have chosen to assess DelMar's valuation in relation to its peer group. Since DelMar has completed only small, narrowly focused open label Phase II trials to date and will not have interim data from a Phase III trial until the latter half of 2018, we believe that basing a valuation upon revenue modeling is premature, especially considering the very high Phase III failure rate of investigational drugs in this indication. The Company's pipeline is also not yet developed sufficiently to reasonably assess any possible revenue stream. With that in mind, it is clear from a review of the Company's peers that DelMar shares are significantly undervalued.

Conclusion

GBM is an extremely challenging indication for which there is a substantial unmet medical need. Many different technical approaches have been explored in attempting to improve the outlook of this patient base. Recent high-profile drug failures have pushed valuations down to where, if there is any positive outcome from any of the trials currently underway, we expect shares in the group to respond enthusiastically. DelMar is unique with its dual functioning, well researched alkylating agent, VAL-083, which has been at least partially de-risked in terms of a well-established side effect profile and evidence of synergy with SOC. Therefore, **we are placing a BUY rating on DelMar shares.** *SG*

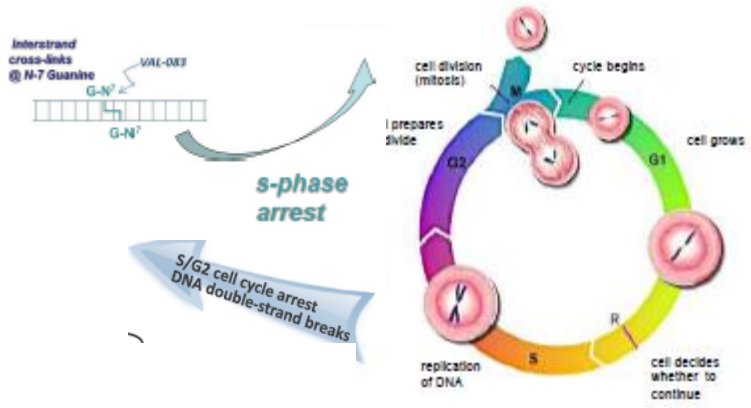
Appendix 1. Technology Background: VAL-083 Mechanism of Action

Alkylating agents keep the cell from reproducing by damaging DNA through crosslinking. Examples of alkylating agents include many well-known cytotoxic cancer agents such as the platinum family (carboplatin, cisplatin, oxalaplatin) and other often used agents, such as carmustin, cyclophosphamide, dacarbazine, lomustine and temozolomide. These drugs work in all phases of the cell cycle and are used to treat many different cancers, including cancers of the lung, breast, and ovary as well as leukemia, lymphoma, Hodgkin disease, multiple myeloma, and sarcoma.

The feature of VAL-083 that distinguishes the molecule from others in the alkylating class is that is a bi-functional alkylating agent in that it creates crosslinks connecting the two strands of DNA as opposed to *intra*strand DNA crosslinks caused by typical alkylating agents such as TMZ. TMZ acts by alkylating (adding a methyl group) to the O^6 and N^7 positions of the guanine DNA nucleotide on one DNA strand. The mismatch , if unrepaired, causes a break in the strand that leads to cell death. However, tumor cells can overcome and repair DNA mis-match through various DNA mis-match repair (MMR) mechanisms, including upregulating the expression of MGMT, a repair mechanism. MGMT demethylates the defective DNA strand to remove the mismatch so that the cell cycle can progress and the cell is replicated.

VAL-083 causes DNA *inter*strand cross-links at the N^7-position of guanine by adding a second alkylating group, DAG, that crosslinks to the N^7 position of the complementary DNA strand. Rather than causing breaks in single strands of DNA as does TMZ and other alkylating agents, VAL-083's cross-link causes the two strands of DNA to break as the cell attempts to "read" through the DNA during the cell cycle. The double stranded DNA break is not easily repairable and the cell becomes "stuck" at the S1/G phase, unable to progress through normal cell cycle. Cell cycle arrest triggers signally towards an apoptotic pathway leading to the cell's death.

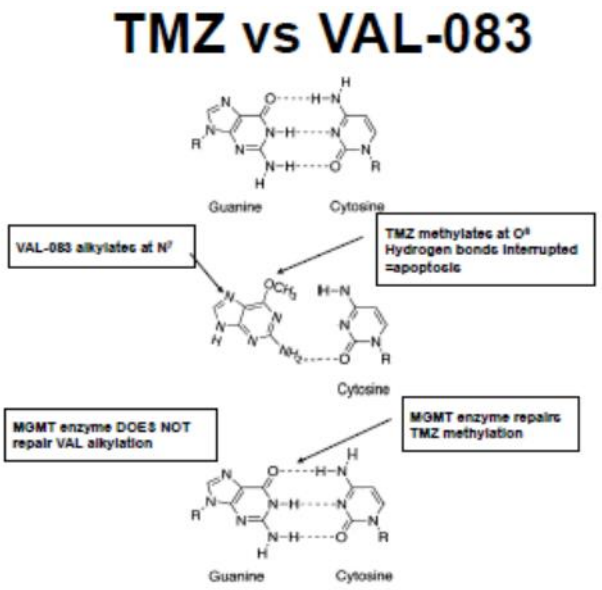
More specifically, VAL-083 induces phosphorylation of H2A.X, a hallmark of double-strand DNA breaks, leading to cell cycle arrest in the late G2/S phase. 4 H2AX is a histone involved in the CHK2 checkpoint activation pathway, a key component of the body's immune response to DNA damage. DelMar has presented posters at key academic meetings demonstrating VAL-083's antineoplastic activity is independent of MGMT expression across multiple GBM cell lines and the compound shows activity against GBM cancer stem cells.



Source: Adapted from DelMar Second Quarter Update; ASCO poster 2017

Further, DelMar has reported that the cytotoxic activity of VAL-083 appears to be less dependent on wild type p53 in comparison to other chemotherapeutic agents. Alteration in p53 has been correlated with poor patient outcomes in GBM. In particular, gain-of-function mutant p53 is strongly associated with a poor prognosis for

overall survival in patients with glioblastoma, potentially by increasing MGMT expression and thereby decreasing chemo sensitivity to TMZ.



Source: DelMar; Society of Neuro-oncology Annual Meeting 2014

Appendix 2. Management

Jeffrey Bacha, BSc, MBA has been Chief Executive Officer and President of DelMar since January 25, 2013, and a director of the Company since February 11, 2013. Mr. Bacha is one of our founders and has been President, Chief Executive Officer and director of DelMar (BC) since inception. Mr. Bacha is a seasoned executive leader with nearly twenty years of life sciences experience in the areas of operations, strategy and finance for public and private companies. From July 2006 to August 2009, Mr. Bacha was Executive Vice President Corporate Affairs and Chief Operating Officer at Clera, Inc. From March 2005 to July 2006 Mr. Bacha was Consultant and held various positions at Clera Inc., Urigen Holdings Inc. and XBiotech, Inc. From 1999 through 2004, Mr. Bacha served as President & CEO of Inimex Pharmaceuticals. Mr. Bacha is also a former Senior Manager and Director of KPMG Health Ventures. Mr. Bacha holds an MBA from the Goizueta Business School at Emory University and a degree in BioPhysics from the University of California, San Diego.

Dr. Dennis Brown, PhD, has been Chief Scientific Officer of the Company since January 25, 2013 and director of the Company since February 11, 2013. Dr. Brown is one of our founders and has served as Chief Scientific Officer and director of DelMar (BC) since inception. Dr. Brown has more than thirty years of drug discovery and development experience. He has served as Chairman of Mountain View Pharmaceutical's board of directors since 2000 and is the President of Valent. In 1999 he founded ChemGenex Therapeutics, which merged with a publicly traded Australian company in 2004 to become ChemGenex Pharmaceuticals (ASX: CXS/NASDAQ: CXSP), of which he served as President and a Director until 2009. He was previously a co-founder of Matrix Pharmaceutical, Inc., where he served as Vice President (VP) of Scientific Affairs from 1985-1995 and as VP, Discovery Research, from 1995-1999. He also previously served as an Assistant Professor of Radiology at Harvard University Medical School and as a Research Associate in Radiology at Stanford University Medical School. He received his B.A. in Biology and Chemistry (1971), M.S. in Cell Biology (1975) and Ph.D. in Radiation and Cancer Biology (1979), all from New York University. Dr. Brown is an inventor of about 34 issued U.S. patents and applications, many with foreign counterparts.

Scott Praill, CPA, BSc. has been Chief Financial Officer of the Company since January 29, 2013 and previously served as a consultant to DelMar (BC). Since 2004, Mr. Praill has been an independent consultant providing accounting and administrative services to companies in the resource industry. Mr. Praill served as CFO of Strata Oil & Gas, Inc. from June 2007 to September 2008. From November 1999 to October 2003 Mr. Praill was Director of Finance at Inflazyme Pharmaceuticals Inc. Mr. Praill completed his articling at Price Waterhouse (now PricewaterhouseCoopers LLP) and obtained his Chartered Professional Accountant designation in 1996. Mr. Praill obtained his Certified Public Accountant (Illinois) designation in 2001. Mr. Praill received a Financial Management Diploma (Honors), from British Columbia Institute of Technology in 1993, and a Bachelor of Science from Simon Fraser University in 1989.

Source: DMPI 10K 2016

Companies Mentioned in this report:
Merck – MRK/NYSE/$65.07/ Not rated
Novartis – NRV/NYSE/$81.83/Not rated
Bristol-Myers Squibb -–BMY/NYSE/$52.36/Not rated
Accurexa -- ACXA-OTC/$0.20/Not rated
Agenus -- AGEN/NASDAQ/$3.40/Not rated
Celldex -- CLDX/NASDAQ/$2.73/Not rated
NovoCure Ltd – NVCR/NASDAQ/$13.80/Not rated

Risk Factors

In addition to normal economic and market risk factors that impact most equities and the common risks shared by DelMar Pharmaceuticals Inc. with other companies in the industry, we believe an investment in DMPI involves the following risks:

- **FDA and regulatory risks** – DelMar is subject to regulatory review for its ongoing research and development activities, commercial marketing approval as well as laboratory facilities, principally with the US Food and Drug Administration, but also potentially with the EMA and other international regulatory agencies if the Company undertakes clinical trials or sells its products in the future outside the US.
- **Need to defend patents, trade secrets and other intellectual property** -- At present, the Company holds a limited number of patents relating to its products, methods and manufacturing and depends in part on trade secrets. The Company may need to defend its intellectual property in the US and overseas in the future. Further, the Company currently has limited patent protection for some of its pipeline product candidates. The Company, or its licensing partners, have made various applications which may never result in effective patents, as there is already an existing array of prior art that may preclude granting of patents.
- **Dependence on Key License** -- the Company is dependent upon license agreements covering its core technology. The licensors may have the right to terminate these license agreements under certain conditions. Such termination would materially adversely affect the value of the product candidate being developed under the license agreement and may result in our having to negotiate new or reinstated licenses with less favorable terms.
- **Need to raise additional capital** -- Although the Company has historically successfully raised funds in the public markets, there can be no guarantee of such success in the future. Currently, the Company has limited cash on hand to fund ongoing research and development programs, ongoing

clinical trials and product commercialization and launch activities. Until such time as cash flows from product sales surmount R&D, clinical and operational activities, the Company will need to seek additional funding. Unforeseen events including potential delays in product sales, clinical programs and regulatory approvals could require the Company to raise additional capital through the sale of equity, therefore potentially diluting current shareholders.

- **Limited stock liquidity** -- Trading volume in DelMar has been comparatively light compared to other stocks in its industry, and as such, news regarding DelMar, its target markets, partners and/or competitors could lead to significant volatility in the stock price.
- **Competitive Markets** -- The Company competes in prescription drug markets with a number of other manufacturers, marketers and service companies, many of whom represent much larger companies with substantial resources. There can be no assurance that the Company will be able to successfully launch new products into these competitive markets in the future. Further, advances in prevention and vaccination rates may limit future market potential of some of the Company's products.
- **CRO and Contract Manufacturer Risk** -- DelMar is pursuing a semi-virtual operating business model and is therefore reliant upon outsourced services for certain key functions, including managing clinical trials and manufacturing. Outsourcing may have associated risks as to CRO expertise in clinical trial enrollments, for project management and clinical monitoring services. In addition to CROs, DelMar relies on a third party manufacturer in China for synthesizing API and lyophilizing the product. The Company has indicated its desire to seek a cGMP FDA inspected contract manufactures which may add risk in terms of product availability, quality control and successfully maintaining FDA and/or EMA certifications.
- **Risks of poor manufacturing processes**, quality control issues and product delays may postpone ultimate production of the drug. Additionally, the Company intends to work with a partner to conduct Phase III trials, take the product through the regulatory process and ultimately market it worldwide. The partner may lack the desire or skill to successfully steer the product(s) through the regulatory process and the partner may have other competing products.
- **Pricing risk** -- The Company could encounter several types of pricing risk. First, at the current high annual costs for antivirals, the drugs may be unaffordable for a broad segment of the population, thus reducing the market size below present expectation of potential and forecast. Price increases may attract new legislation and implement of regulations that limit drug profitability. Governments may impose additional non-price related regulation and disclosure that can increase costs for the for the Company's target indications and industry. With the intent to partner for product commercialization, the Company may not have control over the pricing decision and so revenue expectations may not be met.

Important Disclosures:

Dawson James Securities, Inc. (the "Firm") is a member of the Financial Industry Regulatory Authority ("FINRA") and the Securities Investor Protection Corporation ("SIPC").

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1) **Buy**: the analyst believes the price of the stock will appreciate and produce a total return of at least 20% over the next 12-18 months;
2) **Neutra**l: the analyst believes the price of the stock is fairly valued for the next 12-18 months;
3) **Sel**l: the analyst believes the price of the stock will decline by at least 20% over the next 12-18 months and should be sold.

The following chart reflects the range of current research report ratings for all companies followed by the analysts of the Firm. The chart also reflects the research report ratings relating to those companies for which the Firm has performed investment banking services.

	Company Coverage		Investment Banking	
Ratings Distribution	# of Companies	% of Total	# of Companies	% of Totals
Market Outperform (Buy)	2	33%	1	50%
Market Perform (Neutral)	0	0%	0	0%
Market Underperform (Sell	0	0%	0	0%
Rating Suspensions*	4	67%	4	100%
Total	6	100%	5	83%

*Suspensions are ratings under review for possible change due to unusual market-moving news, and/or analyst departure/change

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