DelMar PHARMACEUTICALS

VAL-083

Protocol: DLM-10-001

ASCO Update

June 1, 2015

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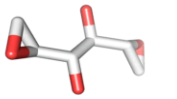
Participants

- **Jeffrey Bacha, BSc MBA**
  - DelMar coFounder, President & Chief Executive Officer
- **Dennis Brown, PhD**
  - DelMar coFounder, Chief Scientific Officer
- **Richard Schwartz, MD**
  - DelMar Chief Medical Officer
- **Kent Shih, MD**
  - Clinical Investigator Sarah Cannon Cancer Research Institute, Nashville TN

VAL-083: First Product Candidate

- Assessed in 42 NCI sponsored Phase 1 and Phase 2 clinical trials
  - Clinically active in multiple cancers including *lung, brain, blood, and cervical*
  - "First-in-class" chemistry
  - Novel alkylating agent
- Unique Anti-cancer Mechanism
  - Inter-strand DNA cross link @ N7 position of guanine
- Pharmacokinetics/Pharmacodynamics
  - Selective for tumor vs. healthy tissue
  - Rapidly crosses blood brain barrier
- Preliminary safety profile established by NCI
  - Dose limiting toxicity: Myelosuppression



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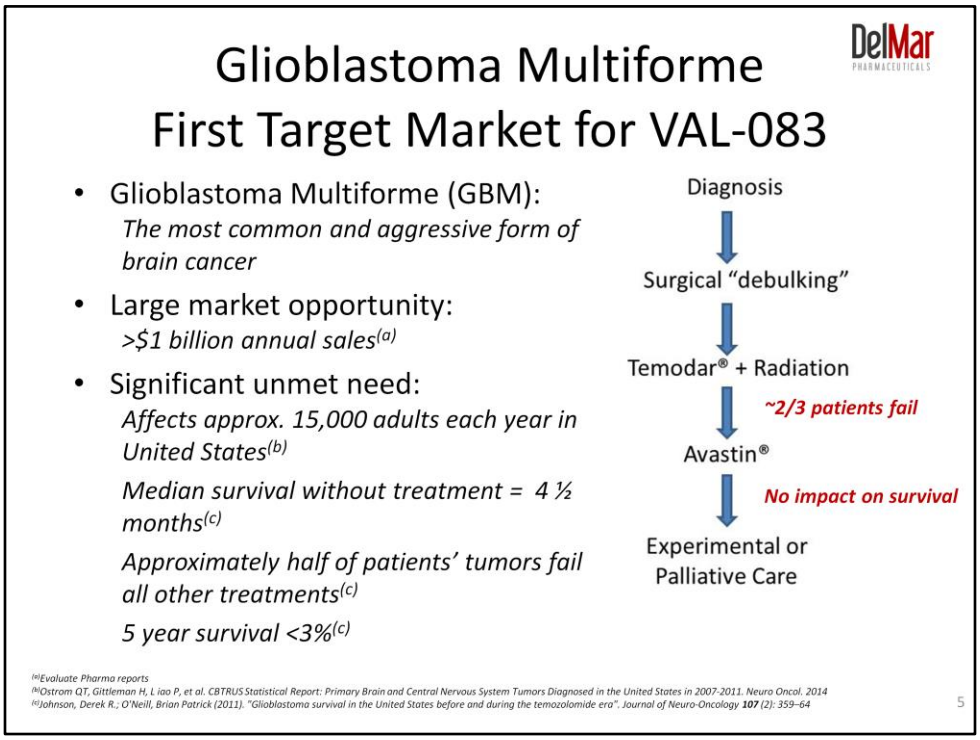
Speaker Notes – J. Bacha: Review and background of VAL-083 program

VAL-083 is a small molecule chemotherapy, which is structurally and mechanistically unique in comparison to other chemotherapies, including those used in the treatment of GBM.

The active moieties are the small "triangles" depicted in the structural diagram. These epoxide groups have an affinity for the N7 position of guanine. In areas where there is a high concentration of guanine on DNA – such as CpG islands found in the promotor region of many genes, VAL-083 is believed to form cross-links at the N7 position of guanine, leading to double-strand DNA breaks and cancer cell death via apoptosis.

The drug was assessed In more than 40 Phase 1 & Phase 2 NCI-sponsored clinical trials, therefore we have a strong understanding of the mechanism, safety profile and pharmacokinetics – and evidence of historical clinical activity in a range of tumor types, including GBM from the literature.

These initial data form the starting-point for our enthusiasm around VAL-083 and the basis for our clinical development program.



Speaker Notes – J. Bacha: Review and background of VAL-083 program

Glioblastoma Multiforme (GBM) is the most common and aggressive form of brain cancer. It is what Ted Kennedy and, more recently, Beau Biden, died from.

This type of cancer affects approximately 15K patients per year in USA – a rare cancer; however, prominent in media coverage due to striking people in the prime of life and the lack of available treatments.

Average life expectancy beyond diagnosis is still well under 2 years – even with the best care.

Surgery is the first step in treatment; however, because this is the brain, the surgeon cannot remove a wide margin of healthy tissue around the tumor to ensure getting all of the cancer – recurrence following surgery is nearly certain.

Standard front-line therapy is chemotherapy with Temodar® + radiation; however, most patients fail this regimen.

We know why they fail: MGMT is a repair mechanism that is responsible for resistance to Temodar.

There is no therapy that has been shown to impact survival for patients failing Temodar. Avastin is approved as second-line therapy in USA but has not been shown to improve survival.

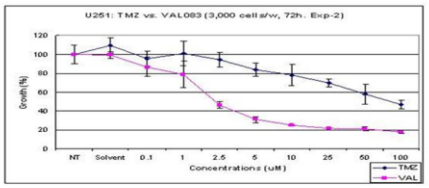
We believe VAL-083 – based on its unique mechanism – has an opportunity to treat these refractory patients, which is a significant (huge) unmet medical need.

VAL-083

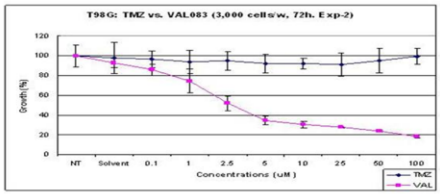
Active Independent of MGMT Resistance Mechanism

- VAL-083 is active independent of MGMT chemo-resistance mechanism *in vitro*[a]
- Measurement of MGMT provides a validated biomarker for patient selection in future clinical trials

U251 cell line
Adult GBM
MGMT negative, TMZ sensitive



T98G
Adult GBM
MGMT positive, TMZ resistant



[a] *Hu et al; AACR 2012*

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Speaker Notes – J. Bacha: Review and background of VAL-083 program

These data were originally presented at AACR in 2012 and are some of the evidence we have developed to show that VAL-083 is active against GBM tumors that are resistant to Temodar.

The top pane shows a tumor that is a LOW expresser of MGMT.

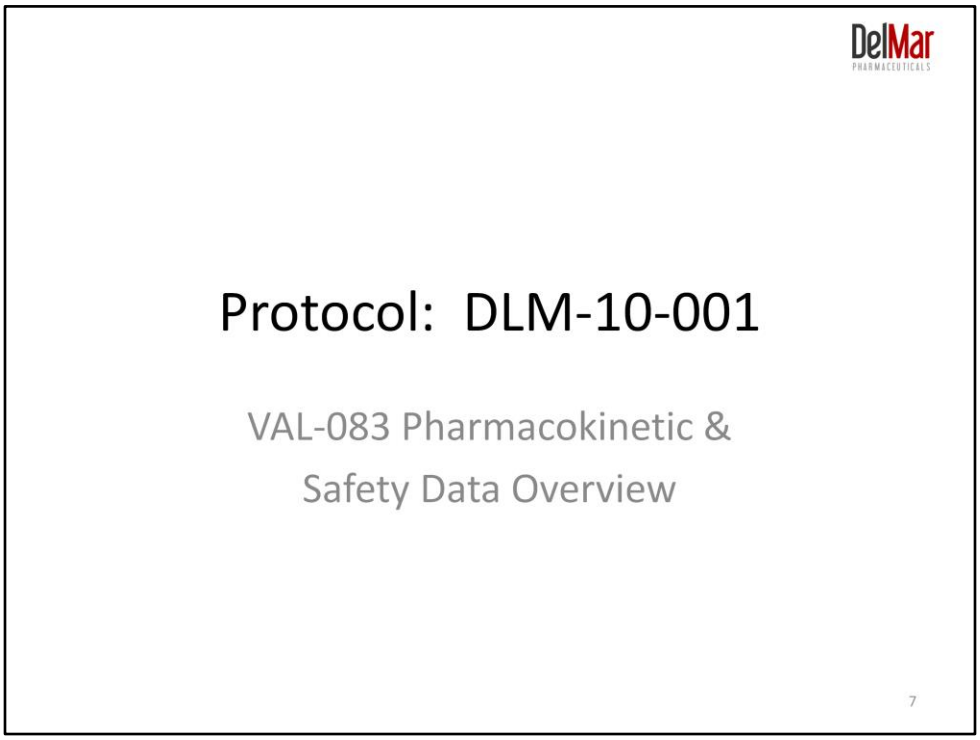
- Both Temodar (blue) and VAL-083 (pink) show a dose response as expected

The bottom pane shows a tumor that is a HIGH expresser of MGMT

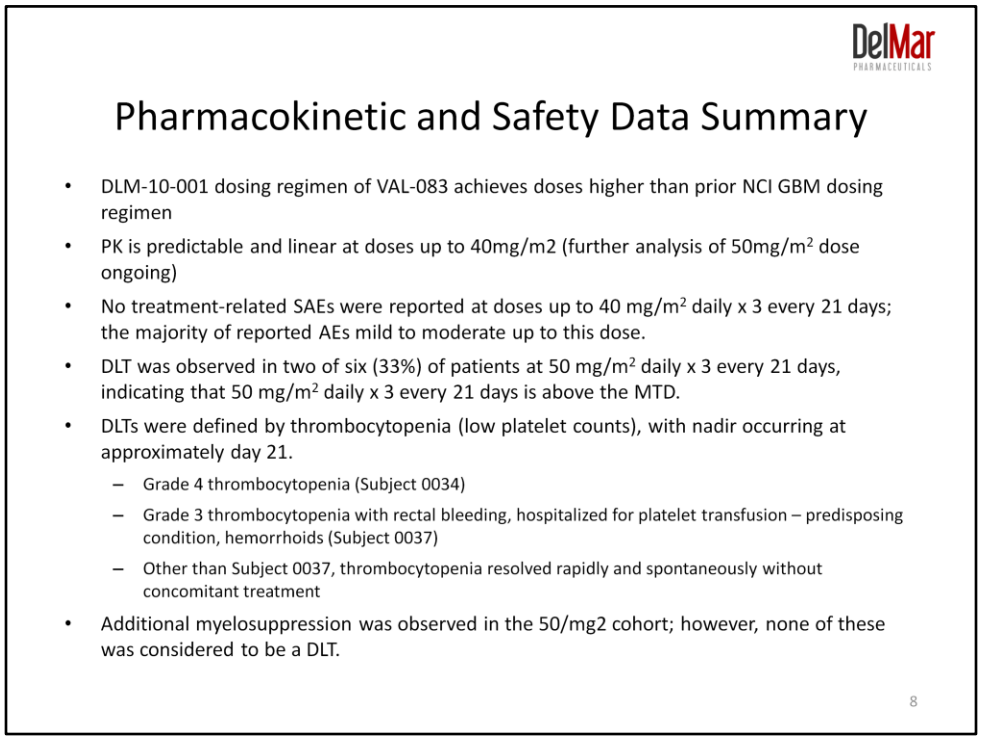
- The blue line is flat – Temodar has no activity – even at 100µM, which is 10x the clinically achievable dose
- VAL-083 looks the same in both settings. The IC50 is ~2.5µM in both settings. Thus VAL-083's cancer killing activity is INDEPENDENT of MGMT

These data - combined with historical clinical activity from the literature - are what drives excitement for doctors because it demonstrates that VAL-083 has a potential to treat GBM patients who fail or whose tumors are unlikely to respond to today's standard of care.

There is no viable treatment for these patients today. We believe VAL-083 offers a potential viable treatment option.



J. Bacha – turn over to R. Schwartz for discussion of ASCO clinical data

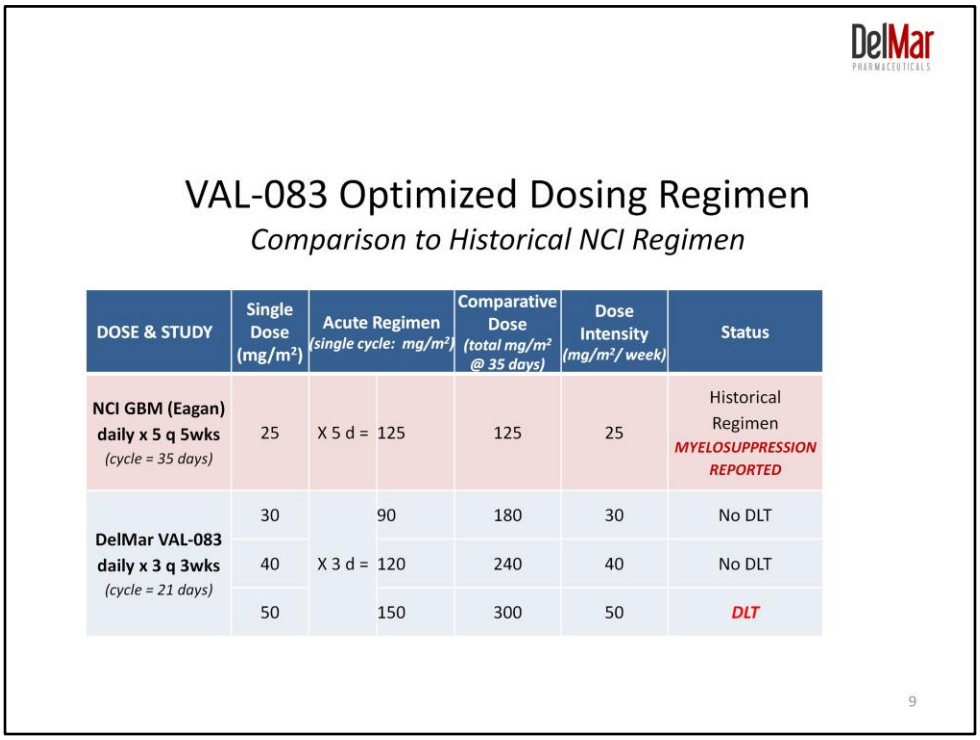


R. Schwartz speaking notes – ASC PK and Safety Data summary

The data in the upcoming slides are an overview of what we presented at ASCO today.

Refer to slide for observations.

Key points –
- Safely delivering doses higher than NCI clinical trials;
- Dose limiting toxicity is thrombocytopenia as expected based on published literature.



R. Schwartz speaking notes – ASC PK and Safety Data summary

The table on slide 9 compares the historical NCI dosing regimen to DelMar's optimized dosing regimen.

NCI: 25mg/m2 for 5 consecutive days in a 35 day cycle.

Delmar: 3 consecutive days in a 35 day cycle.

We safely delivered 40mg/m2, which represents a significant increase in single dose, dose intensity *(25 vs. 40 mg/m^2 per week)* and overall exposure compared to NCIs dosing cycle *(125 vs. 240 mg/m^2 over 35 days)*.

Notably, we did observe evidence of dose limiting toxicity at the 50mg/m2 dose suggesting this dose is ABOVE the maximum tolerated dose.

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VAL-083 Pharmacokinetics

Pharmacokinetic analyses show dose-dependent generally linear systemic exposure with a short plasma terminal half-life of about 1 hour; Cmax in cohort 7 (40 mg/m^2) was 1130 ng/mL (7.7 µM). *In vitro* studies indicate µM concentrations of VAL-083 are effective against various glioma cell lines.

Cohort	Dose mg/m^2	Tmax h	Cmax ng/mL	Cmax (uM)	AUC ng*h/mL	t-1/2* h
1	1.5	0.25	16.5	0.11	18.9	2.02
2	3	0.25	46.4	0.32	48.5	0.83
3	5	0.25	80.5	0.55	108.0	1.27
4	10	0.25	172.0	1.18	191.7	1.19
5	20	0.25	265.5	1.82	249.9	1.22
6	30	0.25	458	3.14	704.7	1.06
7	40	0** & 0.25	1130** & 781.5	7.7** & 5.4	997.9	0.90

*Terminal half-life, lambda z. All values are mean of 2-6 patients, except ** which is for one patient (#28).
**For patient #28 PK samples were collected immediately after start of infusion, Cmax was the 1st sample collected soon after start of infusion; for all other patients PK sampling started after the end of the 1 h infusion.

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R. Schwartz speaking notes – ASC PK and Safety Data summary

The table on slide 10 summarizes the pharmacokinetic observations that were presented today at ASCO

The time to reach peak concentration in the plasma is short – as expected with intravenous administration.

- Note – one patient in cohort 7 (40mg/m2 dose) had Cmax measured immediately after infusion, which accounts for the higher observed Cmax for one patient

AUC appears linear – this is presented graphically in a later slide

Half life is 1 – 2 hours in plasma, which is consistent with published literature for VAL-083.

Notably, the half-life in the CSF is reported to be more than 20 hours. Since VAL-083 readily crosses the blood brain barrier, we anticipate good accumulation in the CNS and tumor.

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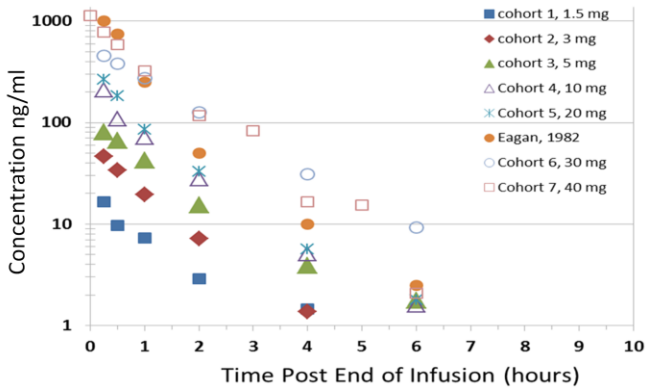
PK by dose cohort & published literature *(thru dose cohort 7)*



PK profile from the literature (Eagan et al 1982) is included to compare current trial data with historical data.

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R. Schwartz speaking notes – ASC PK and Safety Data summary

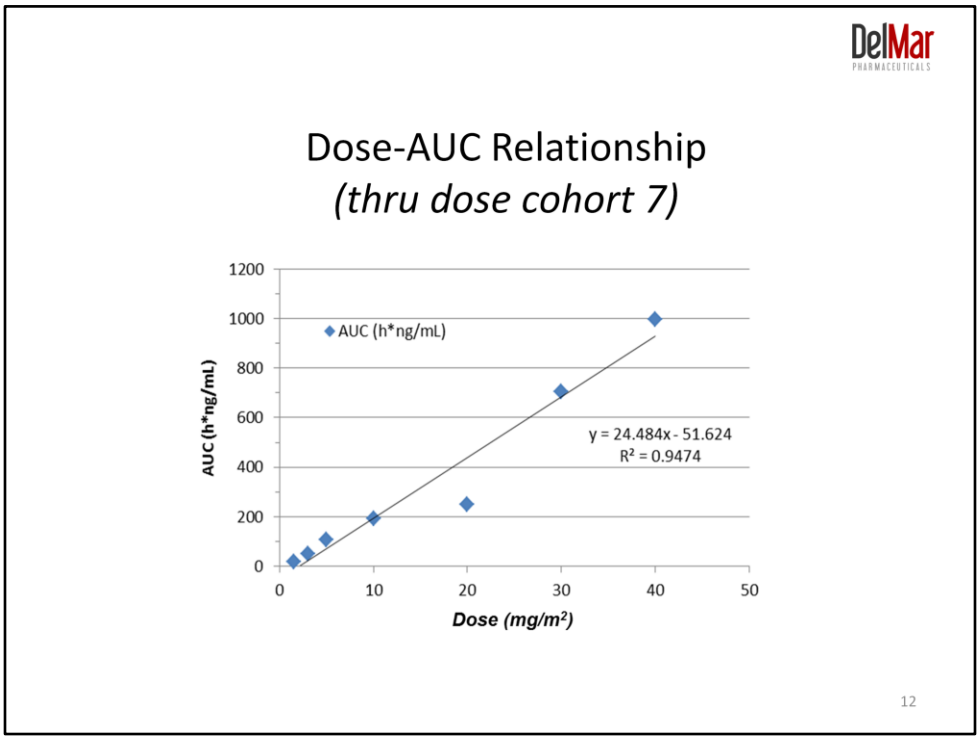
This slide depicts plasma concentration as a function of time.

As noted, Tmax is shortly after infusion with half-life of 1-2 hours.

We observe a nice dose-dependent increase in exposure.

Notably, we can compare this to the expected exposure from the literature and can demonstrate that what we're seeing in our study is consistent with the expected plasma exposure from prior NCI-sponsored research.

NOTE – Eagan study from published literature at 25mg/m2 are represented by the orange values – you can see that they fall neatly between our observed 20mg/m2 and 30mg/m2 dose – as expected.

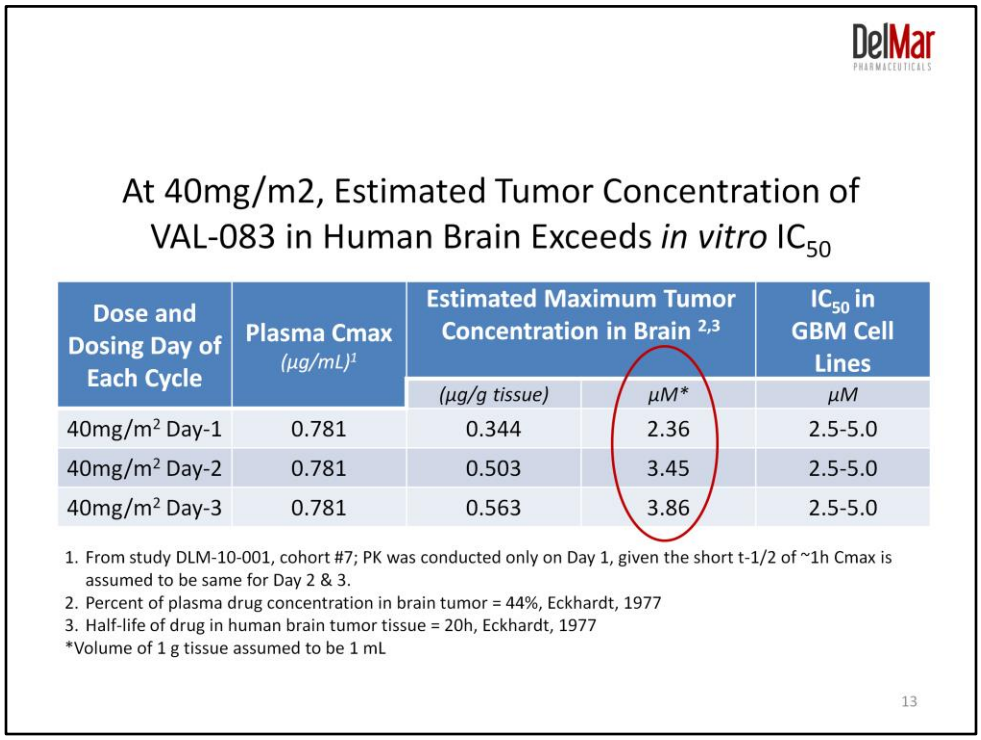


R. Schwartz speaking notes – ASC PK and Safety Data summary

Slide 12 a graphical representation of exposure vs. dose.

As previously noted, our data demonstrate that plasma exposure – as measured by area under the curve (AUC) – is linear and predictable up to 40mg/m^2

(analysis of 50mg/m^2 dose PK ongoing).



R. Schwartz speaking notes – ASC PK and Safety Data summary

Slide 13 is important.

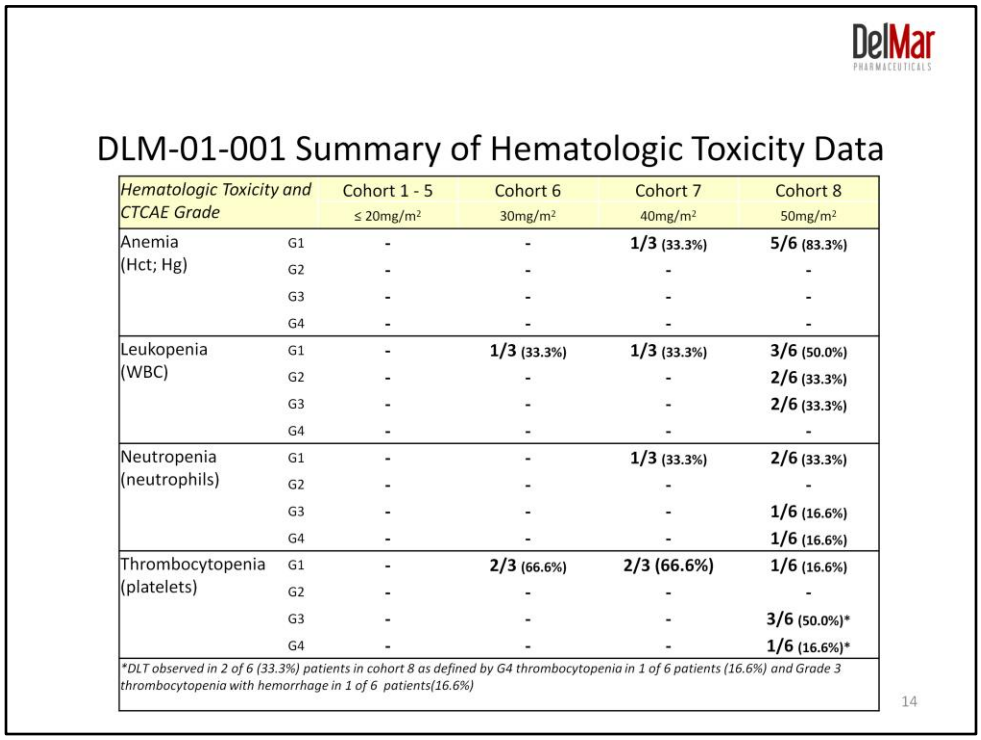
KEY MESSAGE:

What these data suggest is that at the 40mg/m2 dose, we're achieving TUMOR TISSUE concentrations known to be active against multiple GBM cell lines in the laboratory.

Brief explanation of the data & assumptions:

- We have observed plasma Cmax of 0.781 mg/m2 based on a signal infusion.
- The half life in CSF is ~20 hours, so we can expect CSF accumulation with successive daily dosing since VAL-083 readily crosses the blood brain barrier.
- NCI has published previous data showing brain-tumor tissue concentrations following intravenous administration of VAL-083 – we can extrapolate these data to calculate the concentration of VAL-083 that we're achieving at our 40mg/m^2 dose by day 3.
- Based on this – we anticipate that we're achieving concentrations above the IC50 required for GBM cell kill at the 40mg dose.

This is consistent with the clinical activity data that we will discuss later in the presentation.



R. Schwartz speaking notes – ASC PK and Safety Data summary

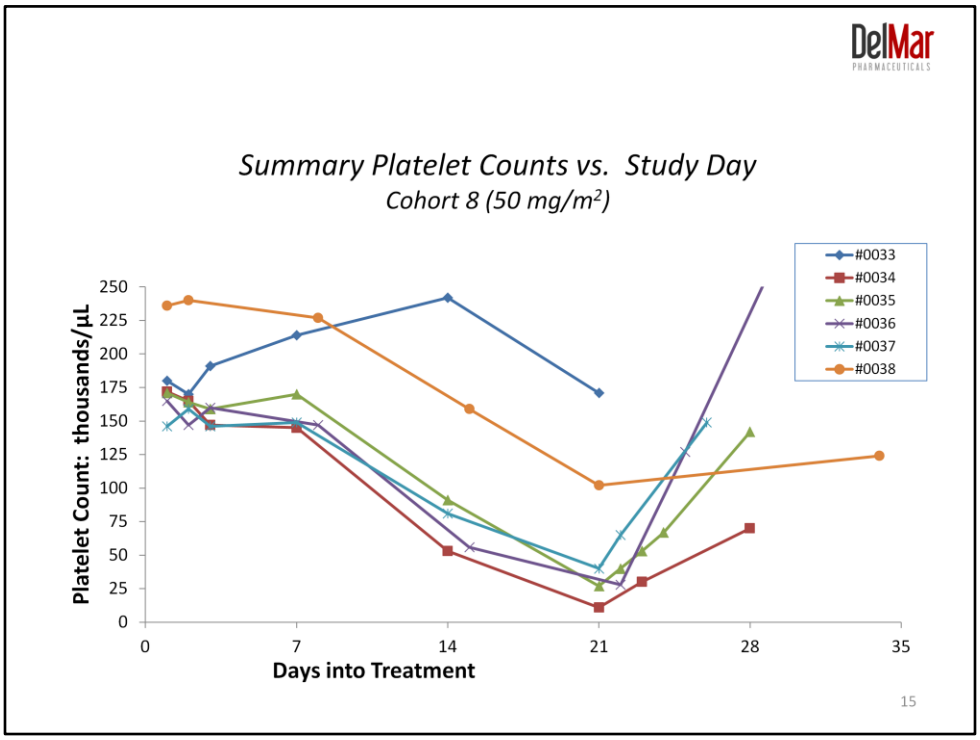
Moving on to safety observations with slide 14

We know from the literature that we expected myelosuppression – namely thrombocytopenia to be the dose limiting toxicity. We reported at AACR that we had observed the first DLT at the 50mg/m2 cohort.

Myelosuppressive DLT is expected for any alkylating agent. Fortunately, VAL-083 does not have a history of any other significant toxicity . There's no evidence of cumulative organ damage or severe GI tox in the literature – so as far as chemotherapy goes, it is reasonably well tolerated.

- VAL-083 exhibits a reasonable safety profile at doses below 50mg/m2 for our dosing regimen of 3 daily doses every 21 days.
- No drug-related SAEs were reported at doses below 50mg/m2 and any observed non-hematologic toxicity was minor.
- In the 30mg/m2 and 40mg/m2 dose we noted some evidence of mild myelosuppression , which became more frequent at the 40mg/m2 dose.
- At the 50mg/m2 dose we noted consistent higher-grade myelosuppression, including two DLTs related to thrombocytopenia (low platelets)
    - One patient was grade-4
    - One patient was grade-3 with bleeding related to hemorrhoids; a Grade 3 + bleeding

is considered a DLT according to the protocol



R. Schwartz speaking notes – ASC PK and Safety Data summary

Side 15 depicts the observed platelet counts in the 50mg/m2 cohort

KEY MESSAGE: ***50mg/m2 is above MTD; platelet counts recover rapidly and spontaneously***

As you can see, the platelet counts in patients receiving 50mg/m2 of VAL-083 were severely impacted with grade 3 and grade 4 thrombocytopenia.

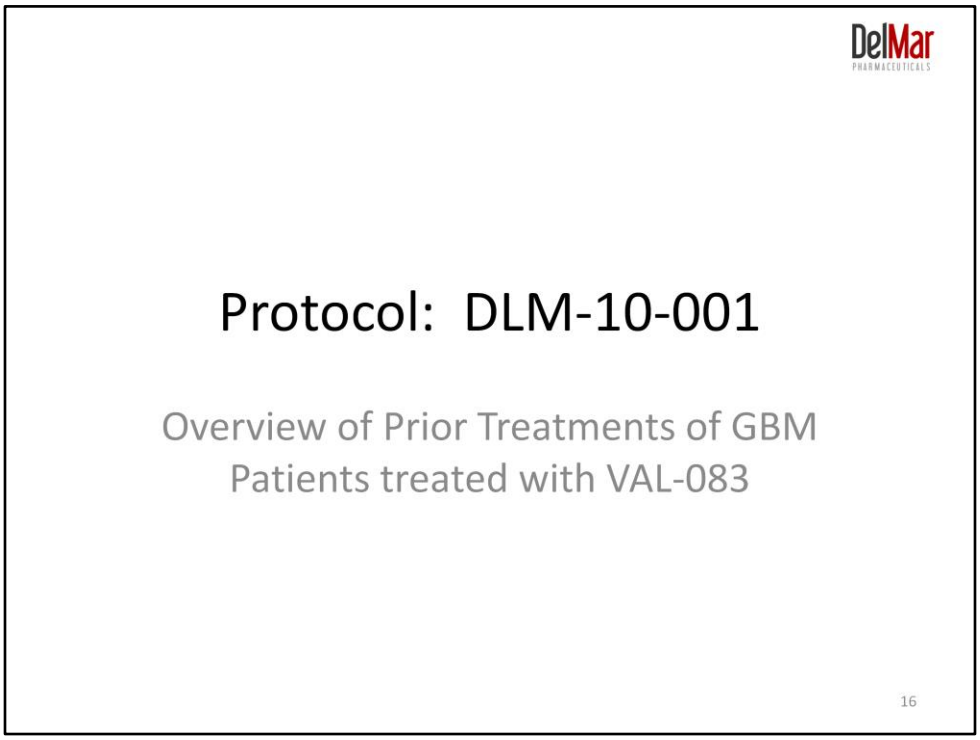
Nadir occurs somewhere around day 21 and the platelet counts recover rapidly from there

Notably, some of the grade 3 thrombocytopenia were close to grade 4 – 27,000 and 28,000 in two separate patients. Based on the fact that we have a "blind spot" in the data since no data were collected between day 14 and day 21, we can look at the slope of the data before and after day 21 and surmise that the NADIR may be before Day 21, suggesting that those patients may actually have had platelet counts below 25,000, which would be grade 4 thrombocytopenia.

Patients with "deep" grade 3 thrombocytopenia also experienced treatment delays, which defeats the purpose of our dosing regimen.

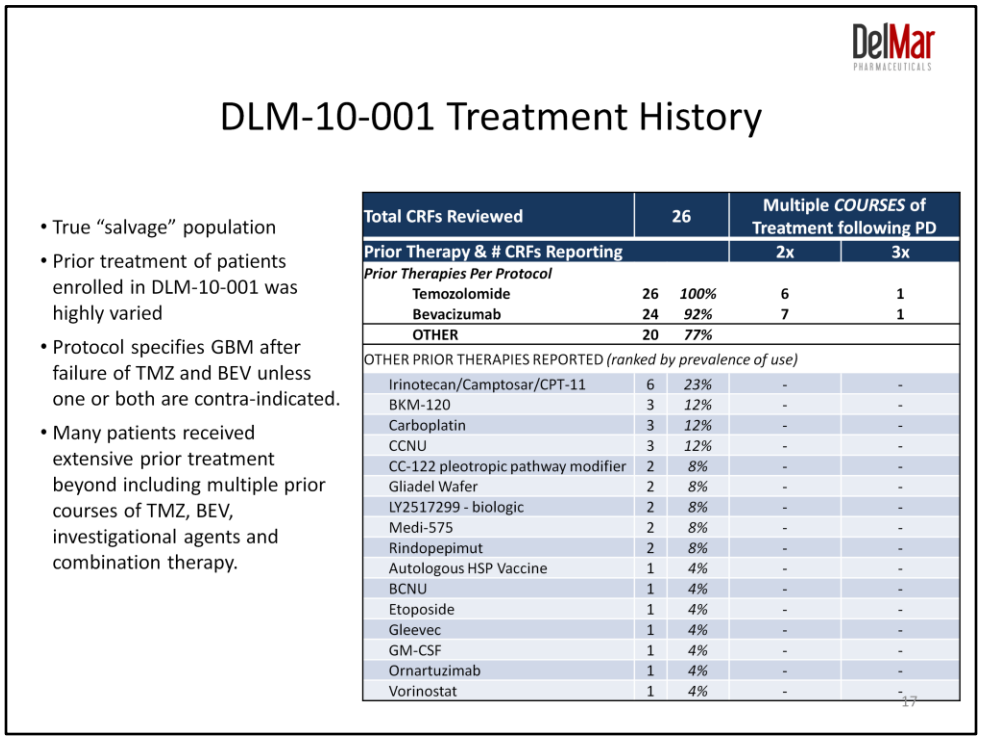
Based on the aggregate of these observations – including two DLTs -- it was determined that 50mg/m2 is above the MTD, so we've determined to move forward with a 40mg/m2 dose into the 14 patient Phase 2 expansion portion of our trial.

We are also exploring an interim 45mg/m2 dose – we'll discuss this later in the presentation.



Schwartz speaking notes – Prior treatments

Now we'd like to discuss the prior treatments of patients in our trial



R. Schwartz speaking notes – Prior treatments

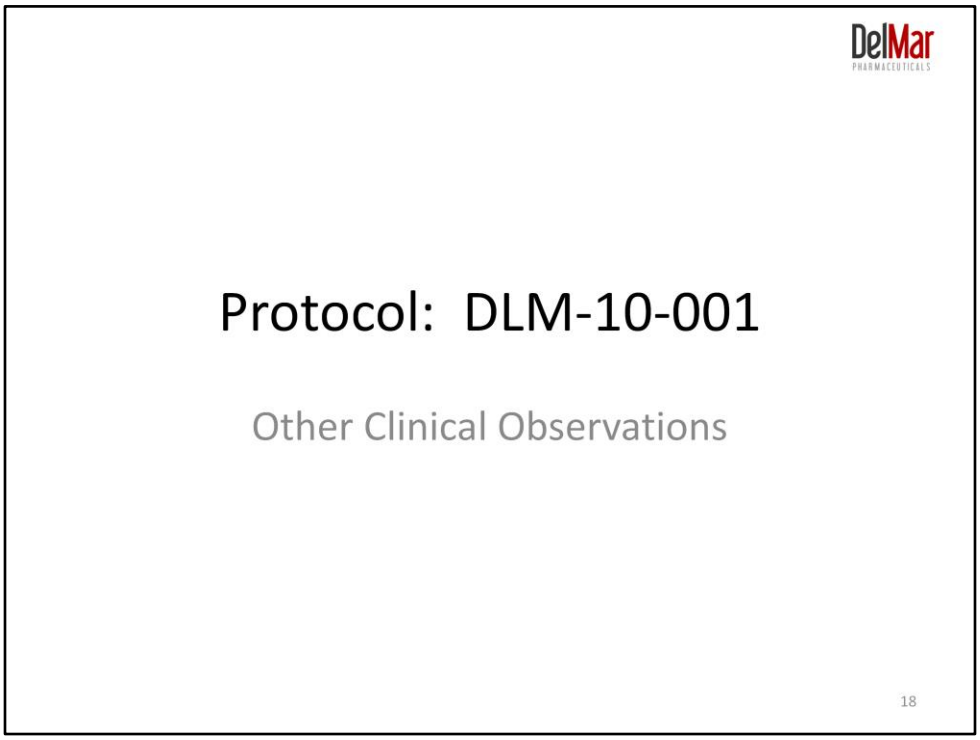
Our protocol requires patients to have failed both temozolomide and bevacizumab unless one or both are contra-indicated.

All patients failed Temodar and most also failed Avastin.

What we didn't necessarily anticipate was the number of prior courses of treatment that some patients had received and the fact that a over three quarters of the patients enrolled had also failed a prior salvage or experimental therapy prior to enrollment.

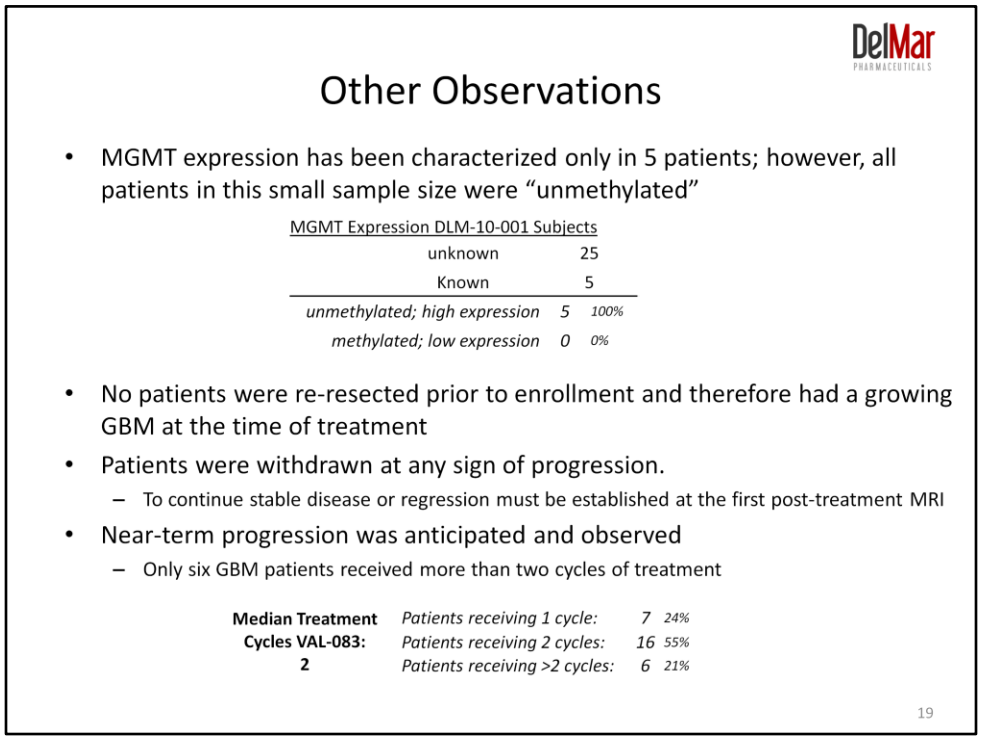
These patients are truly a salvage – or even a "salvage salvage" – population and would be expected to have a very poor prognosis.

Importantly, we achieved a good safety profile following a range of prior therapies, including vaccines, immunotherapies and aggressive chemotherapy regimens.



R. Schwartz speaking notes – Prior treatments

No lets move on to other observations



R. Schwartz speaking notes – Other observations

Slide 19 summarizes what we've seen with respect to MGMT and other observations.

We have only characterized MGMT expression in 5 patients; however, all of them were "unmethylated", which is correlated with high MGMT expression and poor patient outcomes. This is a small sample size, but 100% is an interesting observation.

None of the patients were re-resected in our clinical trial. Since they'd progressed following prior therapy, they had a growing GBM at the time of enrollment.

Thus, we had to expect near-term progression during the dose escalation portion of our clinical trial. In accordance with the protocol, patients were taken off study at any sign of progression, and, as a consequence, most patients only received one or two cycles of therapy.

Median number of cycles = **2** with only six patients receiving more that two cycles of therapy.

With expected near term progression, we also asked the question whether limited treatment could impact survival by slowing tumor growth.

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Summary of DLM-10-001 PFS and Survival Data May 29, 2015

Cohort	Number of GBM patients	VAL-083 Cohort mg/m2	Median PFS after start of BEV (mo)	Median PFS after start of VAL-083(mo)	Median # Cycles of VAL-083	Median survival after start of VAL-083 (mo)
all	29	All	5.9	1.2	2.0	4.6
1 - 3	9	<10	8.8	1.2	2.0	4.4
1 – 5	16	<30	6.0	1.2	2.0	5.6
1 - 7	19	<40	7.0	1.2	2.0	5.7
6	3	30	9.0	1.3	2.0	9.2
7	3	40	4.2	1.4	2.0	8.8
6+7	6	30 and 40	6.6	1.4	2.0	9.0
8	6	50	3.6	1.4	1.0	1.7*(na)

** Three of 7 patients in Cohort #8(including current median) are still alive, two of whom have only recently begun treatment, thus explaining the short median survival in this cohort.*

R. Schwartz speaking notes – Other observations

What we did see – as depicted on slide 20, is some evidence that we may be impacting patient survival at higher doses. - perhaps by slowed tumor growth.

What you can see here is that in increasing cohorts, the median PFS on Avastin (BEV) was consistent – this suggests that the prognosis of patients across dose cohorts was similar. We're not seeing "better" patients at the higher dose cohorts.

Median PFS on VAL-083 was consistently short around 1.2 – 1.4 months. We expected this, and as a consequence of protocol design, the median number of treatment cycles was two.

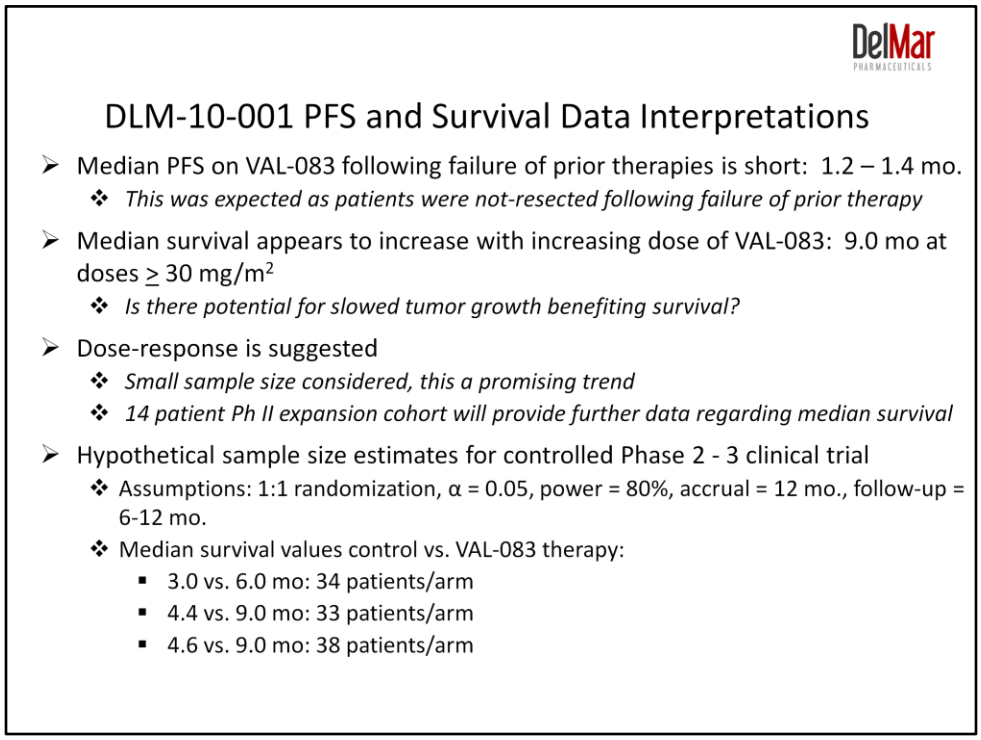
Notably, we see a trend toward increased survival as we go to higher doses.

- Across all cohorts the median survival was 4.6 months following initiation of VAL-083
- In patients receiving <10mg/m^2 the median survival was 4.4 months following initiation of VAL-083
- As we increased dose, the median survival also increased, with patients receiving ≥30mg/m^2 having a median survival of 9 months following initiation of VAL-083.

With the caveat that this is a small number of subjects, this observation is clinically meaningful and promising in this post-Avastin, post-salvage population. This will be explored further in the Phase 2 expansion cohort.

It is worth noting, that patients in the 50mg/m^2 cohort haven't enrolled that long ago, so the data aren't meaningful. Notably, 3 of 7 patients in this cohort – including the current median –

are still alive (although the 50mg/m^2 dose is above MTD and not being considered for further exploration).



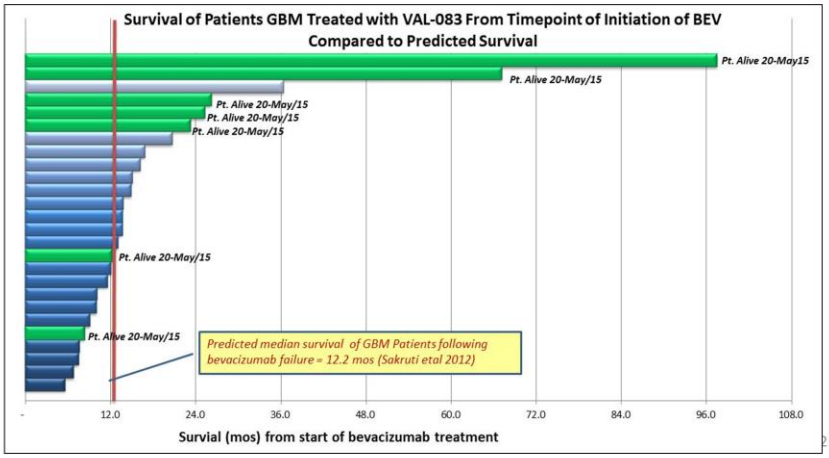
R. Schwartz speaking notes – Other observations

To summarize (refer to slide)

We can also use these observations to estimate the size of a future registration trial, which as you can see is relatively manageable in terms of the number of patients required. (refer to slide)

Other Observations

- Anecdotal Observation:
  - Sakruti et al (2014): Median OS from start of bevacizumab = 12.2 mos (95%CI, 10.0 14.3)
  - To date, 59% of VAL-083 patients have survived longer than 12.2 months



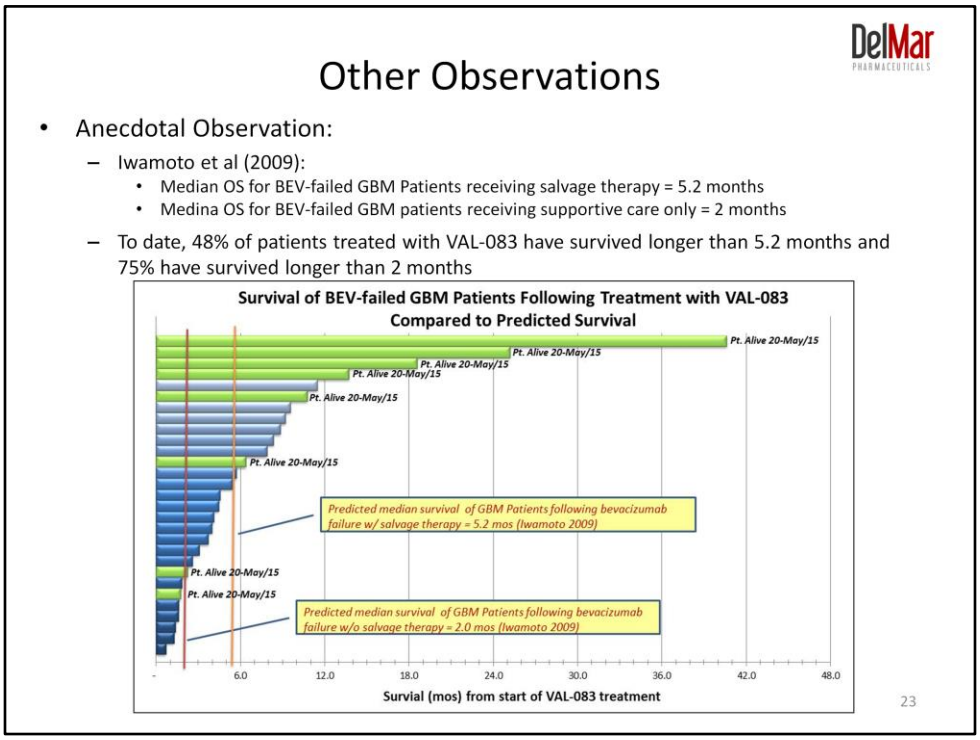
J. Bacha – Other Observations

Another way we looked at the data was to compare to information about GBM outcomes in the context of Avastin therapy from the published literature.

At ASCO last year, a retrospective study of survival from the START of Avastin therapy was presented. The authors noted that the survival was consistent after first, second, or later recurrence – median survival following the START of Avastin therapy was just over 1 year.

At this point, 59% of patients treated with VAL-083 have survived for longer than predicted following the start of Avastin.

This is anecdotal, but interesting.



J. Bacha – Other Observations

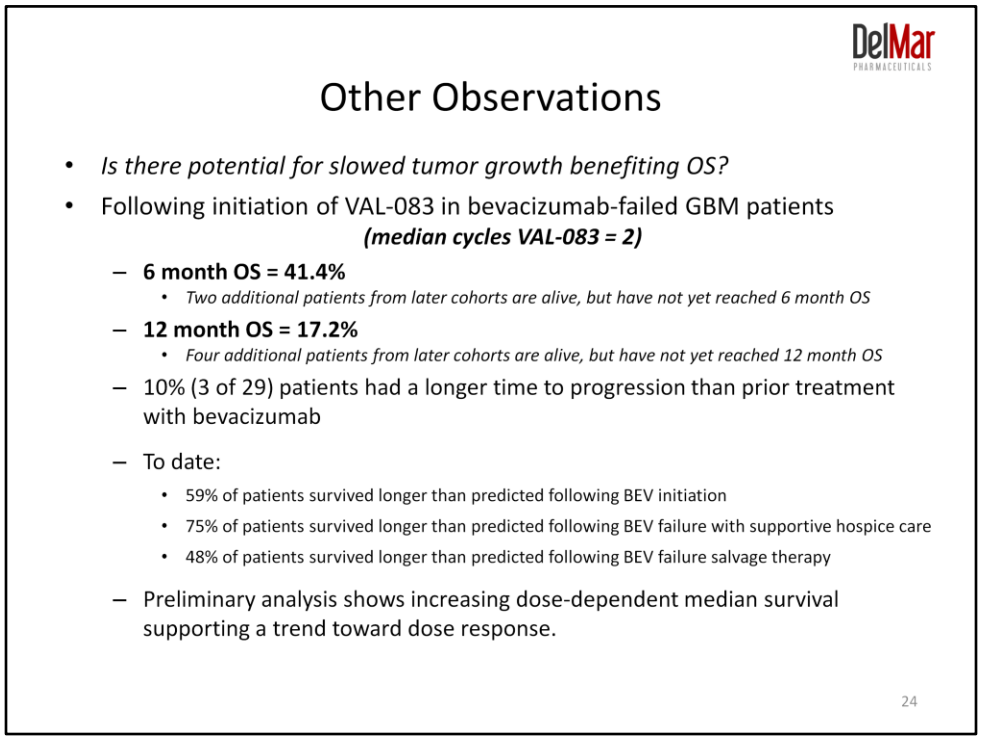
We also looked at predicted survival following FAILURE of Avastin.

Median survival following Avastin FAILURE with hospice care is 2 months. 75% of patients treated with VAL-083 have done better than this.

Median survival following Avastin FAILURE with salvage therapy is 5.2 months. 48% of patients treated with VAL-083 have done better than this, with others tracking toward this milestone.

This is anecdotal, but interesting.

As noted earlier, patients receiving higher doses of VAL-083 had a median survival of 9 months, suggesting a substantial benefit in comparison to salvage therapy.



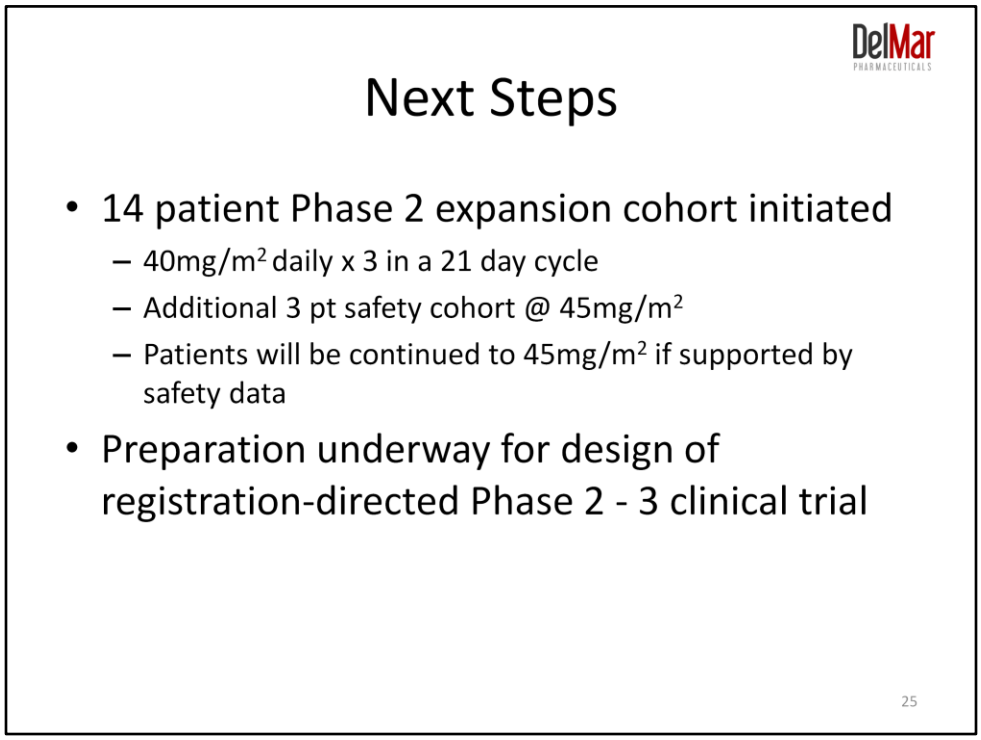
J. Bacha – Other Observations

To summarize:

The 6 month and 12 month survival in an Avastin *and salvage* failed population is 41.4% and 17.2% respectively, with a number or patients still alive and tracking toward these survival milestones.

Patients treated with VAL-083 appear to be doing better than predicted in the context of Avastin-related GBM literature.

Patients at the higher dose cohorts had a median survival of 9 months, suggesting a dose response and the potential for meaningful clinical impact for refractory GBM patients with no viable treatment options.



J. Bacha – Next Steps

Obviously, we find these data to be encouraging and we're excited to be moving forward with the next steps in the development of VAL-083 for refractory GBM.

We have initiated a 14-patient Phase 2 expansion cohort at a 40mg/m2 dose – the first patent has already been enrolled.

Based on preliminary evidence of a dose response, our clinical advisors believe exploring an interim 45mg/m2 is worthwhile. We have received IRB approval at two sites to enroll a small safety cohort at 45mg/m2. If the safety data warrant, we will continue the 14 patient expansion at this higher dose.

Otherwise, our dose for registration-directed Phase 2 - 3 studies will be 40mg/m2.

We anticipate reporting initial data from the 14-patient Phase 2 expansion later this year.

We have also initiated activities related to advancing into pivotal registration-directed Phase 2 - 3 trials with VAL-083 in refractory GBM.

QUESTIONS