DelMar Pharmaceuticals, Inc.

Edited Transcript of Key Opinion Leader Event

November 22, 2019 at the Annual Meeting of the Society for Neuro-Oncology

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Naureen Quibria, Ph.D., Equity Research Associate, Maxim Group - Moderator

Mr. Zarrabian: Good afternoon everyone. Please be seated and we will start this meeting. Thank you very much for attending tonight. So, to start with, my name is Saiid Zarrabian. I'm president and CEO of DelMar Pharmaceuticals and we set this meeting up in order to be able to tell both the scientific community, but also the investment community, about the achievements of DelMar. Before we start, I would like to ask every member of the panel here to just briefly give their name and their affiliation so everybody in the audience knows what their job titles are. And then we will get going with the presentation. Dennis, we will start with you and Greg and then we will go to the panel.

Dr. Brown: Thanks, Saiid. I would like to thank everyone for coming. All these efforts, they take a long time and need a lot of people to help out to make things happen and I appreciate everyone coming today and seeing where we are and giving some insights on what we should think about for the future. So, I am Dennis Brown. I was co-founder of the company and currently the Chief Scientific Officer.

Mr. Johnson: I am Greg Johnson and I am acting Chief Operating Officer.

Dr. Quibria: I am Naureen Quibria and I am with Maxim Group. I'm an Equity Research Associate.

Dr. Cloughesy: Dr. Tim Cloughesy. I'm a Neuro-Oncologist at UCLA.

Dr. Chen: I am Zhong-ping Chen at Sun Yat-sen University Cancer Center and I am a surgeon, and also the principal investigator on DelMar's clinical trial being conducted in China.

Dr. de Groot: My name is John de Groot. I'm a Neuro-Oncologist at MD Anderson in Houston.

Dr. Butowski: Nick Butowski Neuro-Oncologist at UCSF.

Mr. Zarrabian: Thank you very much. With that I would like to ask Dennis to start with the slide deck, we have a couple of slides that Dennis is going to present first about the compound itself, VAL-083 and then Greg Johnson who is our acting Chief Operating Officer is going to present a high-level view of the data that was presented today through the SNO Posters and 8-K early this morning. Thank you, Dennis.

Dr. Brown: Thanks, Saiid. Well again, thank you very much.

There are a lot of scientists and doctors here trying to help people solve some problems that are very critical for patients with a very difficult disease, GBM. So I've been involved with looking around the world to identify new drugs for about 45 years now and I was, I got to be lucky enough to come out to California about 37 years ago when I had the opportunity to commute from Stanford to UCSF to work with Victor Levin to learn about drug discovery and development. And in those days, I was part of a really remarkable program with the NCI which was really the critical organization that was looking for compounds, testing them, and moving them to clinical trials. Some luminaries are here who have done a lot of work in this area and what I was always impressed about was the level of intellect and the commitment to trying to find new opportunities from the chemistry, the biology, moving things up in decision networks to getting them into the clinic.

And so, over all those years I was always intrigued by how much work had been done and the level of sophistication. But there's always a move to keep moving on so when I was at Stanford in the 80s most of the work that was done was mostly small chemical drug discovery. There was a discovery of cisplatin about that time and that was a game changer. You got a metal that can somehow do something significant in cancer. And there was another series of molecules that were damaging DNA and some of them are the mainstay of the therapy for brain tumors. And those are alkylating agents that attack DNA. And the drugs that the clinicians use today Temozolomide, BCNU CCNU all came out of the development of NCI's primary work and work by thinktanks such as the Southern Research Institute.

But in those days the biology was not as powerful as it should have been so the mechanism of action was always unclear. What exactly was it doing to the cell? And people moved along and everyone felt there would be a next generation product, an antibody or a signal transduction effect or some other vaccine system that would solve this problem of brain tumors. And there has been progress but what was always interesting to me was that there were agents and other opportunities that were left on the side of the road. They were not formally developed, or the biology was not good enough, there was always some other kind of limiting problem. And VAL-083 really represents that, VAL-083 was brought to the United States by Victor Levin who went to Budapest, Hungary and met the chemist that worked on it and he saw this molecule as being somewhat interesting and brought it back to the United States and it became the subject of numerous clinical trials. And what was interesting about the molecule is this. This is the molecule we have been working on. This is really a sugar that has been derivatized, it has epoxide groups at both ends, and they're very reactive but they have a propensity to bind to DNA. And in those

days, they couldn't quite discern the difference between what this compound did and what nitrosoureas did.

They knew they were binding to DNA causing strand breaks, but they didn't discern if there was anything different in the mechanism. So, the molecule, aside from not only being from Eastern Europe, had a weak patent position on it and people kept moving along. They kept thinking there would be a next generation molecule that would be certainly a lot better to cure brain tumors. About 10 years ago, I came across the drug in China and it was already approved in China for lung cancer and CML. So, I was very intrigued that the Chinese government, and the scientists there felt there was something valuable there. So, we went back to look at it and went to see the companies developing it and brought the compound back to the United States. We started working with UCSF and Nic and Dr. Burris at Sarah Cannon, and ultimately at MD Anderson, to see if there's something interesting.

We went back and did some molecular biology with some really wonderful scientists at UCSF, MD Anderson, and University of British Columbia and we started to discern that there's a difference between Temozolomide's effect on cancer cells and this drug. VAL-083 binds to the N7 of guanine and Temozolomide, which is a monofunctional alkylating agent, causes single alkylation damage at the 06 of Guanine. So Temozolomide which is a pro-drug has to open up, has to get up here, and it typically just has one fist to work with. But what was learned in the last 25 years is that it's readily repaired by an enzyme called MGMT (methylguanine methyltransferase) so that enzyme which is highly expressed in a high percentage of brain tumor patients can quickly mop up that damage and it's as if that patient did not receive any of the drug.

So, what we were able to discern is VAL-083 is a bifunctional alkylating agent, a bifunctional crosslink on DNA is formed which is somewhere, here, up in here and in the N7 position and this is extremely resistant to the effects of MGMT (methylguanine methyltransferase)**.** And that became intriguing, so we started talking to clinicians, we started to do some more experiments. And we started to compare the differential responses in numerous GBM cell lines that were sensitive to Temozolomide. For example, they were methylated, and we looked at differential IC 50's between what our drug would do relative to Temozolomide and you can see we had differences in IC 50's that we would be able to achieve in comparison to temozolomide to kill half of the tumor cells . For VAL-083, IC50's of approximately 2-5 micromolar were observed relative to 10 micromolar with Temozolomide for the methylated cell lines. And when you went into the unmethylated cell lines we had about equal sensitivity between the two different genotypes, but you can see with Temozolomide you would require up to 100 micromolar to achieve equal cell kill. So, this is the type of data that we were able to use to start speaking with clinicians, and to start looking at ideas of how we might move forward. And with that we went back, we started to manufacture the drug, started going back to the FDA, and started the design elements for some clinical trials. What I would like to just finish up is that we've been able to be involved with some really wonderful doctors, and scientists that have helped give us the ideas on where we should consider studying this compound hopefully to registration.

Mr. Johnson: Thanks Dennis. So the rest of these slides are going through the data that's being presented in the two posters at SNO and you can see the title of the two posters here and these are being presented from 7:30 - 9:30 if you want to go by and look at the poster and get additional detail on this. There are a number of data slides in this deck and I'm only going to go through them pretty quickly and hit the high level and really focus on the new PFS and OS data that we're sharing for the first time.

But the data slides are included in the DelMar corporate slide deck which you can access through the DelMar website so if I breeze through something and you wanted to look more carefully at confidence intervals, you can go to the website and follow up on that. So, clinical study DLM14-001 is the newly diagnosed study. Both of these studies are open label studies being conducted in unmethylated GBM patients. The newly diagnosed study is being conducted at Sun Yat-sen University Cancer Centre with Professor Chen as the principle investigator. This started as a dose escalation study and we have landed on 30 mg/m^2 as the dose that we are going to carry forward. That's a dose being done on days 1, 2 & 3 of a 21-day cycle and we standardized on that 30 mg dose across all of the trial arms we are working on. So, for the newly diagnosed, the adjuvant and the recurrent, the dose level that we intend to take to market, that we intend to move forward with is 30mg/m^2.

For the newly diagnosed study, there were 9 patients in the dose escalation phase as shown in the table, and we have 14 subjects enrolled so far in the expansion phase giving us 23 subjects to date. The safety on this study as well as the other study, you can see details on the tables that are included in the slide deck and there's additional information in the poster, but the highlight item is we are essentially seeing myelosuppression as the most common adverse effect which is typical of what we saw in earlier work in this molecule. So, progression free survival, this is a snapshot in time obviously for an ongoing phase 2 study that's still enrolling patients. We are working with historical comparative here which is Temozolomide which has shown 6.9 months progression free survival in unmethylated patients, and for the data that's included in the poster includes, for all subjects, we're currently showing 9.9 months progression free survival. And for the 30 mg dose we intend to move forward with we are showing 10.4 months progression free survival against the historical comparator of 6.9 months. This is a snapshot in time and you can see the confidence intervals that are associated with this, but obviously we are happy that this is headed in the direction we would hope to see it maintained as we move forward in this study.

The survival curve, the green line, is the 30 mg dose, so the line of most interest here is the green line in the progression free survival. In terms of tumor response, this is included because this has been what was reported historically in posters for each of these studies as they have been presented at SNO and ASCO and various meetings so for consistency we continue to show best overall response. And for the China study, the newly diagnosed study, investigators assessed 9 out of 19 as complete responders and 10 out of 19 as stable disease so in the overwhelming number of assessments, the disease has been arrested for a period of time which is what you would expect with the survival PFS data that you're seeing.

We've also presented in the poster that we continue to present pharmacokinetic data. Those familiar with the study will remember that we've done CSF PK Data on this study so we've actually been able to demonstrate that the plasma level vs the CSF level - CSF at 2 hours actually has typically a higher concentration of VAL-083 than plasma. There was previous evidence from NCI and earlier DelMar work that it was clear that VAL-083 crossed the blood brain barrier but to see these levels in plasma in CSF validates that and really makes it quite clear that it has an affinity for brain.

The recurrent and adjuvant study being conducted at MD Anderson Cancer Centre, and here the recurrent study arm and the adjuvant study arm are again both treating at 30 mg/m^2 as the study dose. We currently have 62 patients enrolled in the recurrent arm, 35 of those started with the dose of 40 mg before we moved and settled on the 30 mg dose for all trial arms going forward. In the 30 mg arm, 27

out of 48 planned have been enrolled. The adjuvant arm which we announced first patient enrolled in July of this year, we have 5 subjects enrolled today, and again at the same dose as the other trial arms.

In terms of safety, 60 subjects completed at least 1 cycle and are included in the information that is presented here, and myelosuppression is the most commonly observed adverse event. This table gives you additional breakdown on this including the key information that when you compare the 40 mg to the 30 mg dose you see a reduction in the number of subjects who had dose limiting toxicities and importantly in our opinion, fewer treatment delays greater than 3 weeks.

Again, we are treating on 1, 2, & 3 days of a 21 day cycle so we think it's important to be able to get back to the patients on a timely manner for the 21 day cycles and that's why the 30 mg dose we think is showing greater promise in the recurrent arm. In terms of overall survival, this is, the historical comparator for this group is Lomustine which has shown 7.2 months median overall survival for unmethylated patients.

The median OS that we're reporting as a snapshot today is 7.5 months for all subjects and again importantly for the 30 mg dose that we're moving forward with, 10.6 months median overall survival with the confidence intervals that you see. And again, the survival curve here, the blue line is the 30mg starting dose, and the 40 mg dose is the green line.

One thing that we're still discussing and I think is an interesting topic is why is the 30 mg group doing so much better than the 40 mg group? And we've looked at this and there's still additional review being done here. We don't see any apparent relevant differences between the baseline for the patients coming in so they're roughly the same age, same KPS score between the two cohorts so there's no obvious reason to see the 30 mg having bias just because of the baseline of the patients. There's not a significant number of differences in the number of cycles that they're receiving although the timing of the cycles has been a little bit better in the 30 mg group than the 40 mg group, which you would expect with the cycle delays and the DLTs that you saw on the safety table.

Anecdotally the scientists reported for some time now that they simply feel that the 30 mg patients are feeling better, that they're just responding better, and maybe just not taking as large of a beating from the chemotherapeutic as it were and that may be playing a role in this. The study does collect quality of life data through an MD Anderson validated instrument. We have very little of that data in hand and are just starting to analyze it, and I don't know, a doctor in the group may want to comment on this later, I don't know that we will necessarily see anything in the Q of L data that will help us understand the preference for 30 mg vs 40 mg dose but it is something we're still looking at.

In terms of tumor response, reported again in the poster just for consistency, you can see that about 25% or so of the patients have had stable disease in their initial assessment. And then finally for the adjuvant arm, we have 5 subjects who have been enrolled to date, one's been on for 6 cycles, the others have all been on for 1 or 2 cycles at this point. All subjects are still on study in continuing treatment. We haven't had any SAEs or DLTs reported yet for this group and only the first subject has reached their first MRI assessment and they were assessed as stable disease. So again, these slides are on the DelMar website if you want to look at the data in more detail and of course on the posters being presented from 7:30-9:30 and it has this as well as historical data and background on the molecule if you want to dig in a little deeper at the poster session. But I'm going to stop in terms of data presentation now, so

we can move to the Q&A session with the thought leaders which I think is probably the best thing we can do with our time today. Thanks.

Dr. Quibria: Thank you Greg. It's a pleasure and honor to be among this esteemed group of scientists, and physician scientists, from the neuro-oncology field. We just heard a little bit about VAL-083 and the ongoing trials and studies that DelMar has. But I'd really like to take a step back and think about the treatment landscape for GBM and potentially how VAL-083 can fit into the treatment paradigm so if we could start really from the basics. I would really love to hear all of your takes on this, why is GBM, glioblastoma, such a hard to treat tumor type? Perhaps Dr. Cloughesy you might want to speak?

Dr. Cloughesy: I think there's several reasons why GBM is difficult to treat. First, it's in the brain, and the brain is very important to us. So, if we try to resect this tumor it's actually infiltrative, so we actually have to remove some brain. That's obviously a problem so we can't completely resect it.

And then there's always infiltration so whatever we do resect, there's always residual tumor. Radiation for instance, is very effective, but we also can't give such high doses of radiation where we destroy normal brain because again, we have to protect the normal brain. So, we have limitations with regard to what we can give. Many of the drugs then, if we think about surgery, radiation and then the other drugs that we use, many of them we borrow from other settings. We're borrowing them from other cancers and trying to apply them to glioblastoma. And many times, those drugs will not have access to the blood brain barrier. Most drugs, and actually early on almost all chemists were trying to dial out the blood brain barrier because they wanted to keep drugs out of the blood brain barrier, and out of the brain if they were trying to treat systemic disease. I think only now are we are beginning to see efforts moving forward to try to bring drugs into the brain and see what kind of difference that makes. I think it's really intriguing that this drug has good penetration into the CSF and the CSF is not exactly the same as the brain but it's a good surrogate, it's better than not having it there, I think it's a good start for this. Obviously seeing the effects that we're seeing gives us a good sense that it's getting into the brain so that's really interesting. I would say though the last thing is there's all these mechanisms of resistance that's set up within these tumors. Sometimes it has to do with heterogeneity but realistically this tumor isn't more heterogeneous than any other solid tumor. I think we just aren't having the right drugs getting there at the right concentration to have the effect that we want to have.

Dr. Quibria: Dr. Chen, your thoughts? Why is glioblastoma such a hard to treat tumor type?

Dr. Chen: From my understanding, as Dr. Cloughesy said before, as a surgeon it's difficult to remove all of the tumor in the brain, sometimes of course we can almost do it, but of course we still have some infiltration, so for now for treatment of GBM, at least in China, same as in the States, we have surgery, we are doing radiation, but at least half maybe, are resistant, not so sensitive to radiation, so afterwards they grow again. And for the chemo, we traditionally used Temozolomide in China also so even in unmethylated cases, but the response is very limited, but we are still using it because we don't have a new agent yet. So currently we are conducting a phase 2 trial and we get some definite results and most of the cases will respond, maybe 2/3 will respond to this kind of drug. Of course, we still have some non-responders but compared to Temozolomide it's much better so far. We've enrolled about 25 cases, and so far, have analyzed about 20 cases and the results are very interesting up to now. So, I think this is fairly helpful at least in the current stage for GBM in unmethylated cases, which are still difficult cases.

Dr. Quibria: Do the others have any additional comments?

Dr. de Groot: I think Dr. Cloughesy, Dr. Chen, sort of laid it out really nicely. The only other issue is that immunotherapy is on everybody's mind and the brain is a bit of an immune privilege site in that it has a very immunosuppressive tumor micro environment so some of the therapies that have been put forward for other diseases haven't had the same kind of success in the brain so I think that's just one other level of complexity that we are still trying to better understand. Great opportunities for other types of therapies while we try to figure that out.

Dr. Quibria: So how does methylation status play into this? You know the MGMT un-methylated status of some patients is about 60% of the GBM patient population. You know how does it in fact impact your treatment or how the patients are treated?

Dr. Reardon: I can start on that. MGMT methylation status I think we use as a general predictor of how likely our standard of care with Temozolomide may have a benefit for patients. It's not a perfect test in many ways, and it's not a perfect predictor. I have patients with MGMT methylated tumor who seem to have done well and some have better than average disease control and then others with a methylated promotor who progressed within a few months. So it's a general predictor I think that can give us a sense of how likely a patient is to benefit from our current standard of care, but even in the best case scenario about a patient we feel confident and reliable that the test is accurate and demonstrating a methylated promoter, the benefit is still not durable in the patient and they have a better likelihood of benefit but unfortunately the treatments just aren't durable.

Dr. Quibria: So, do you actually stratify patients or is it just a prognostic factor at this point, when you're treating?

Dr. Reardon: Well, in terms of clinical trials, it's certainly an important factor to be looking at in the outcome of clinical trials and stratifying patients based on that. Some of the trials include the MGMT status as an eligibility criterion as well.

Dr. Quibria: So, Dr. Butowski, do you see any advantages, given DelMar's VAL-083 that has a different MOA, in trying to treat these difficult to treat patients in unmethylated population?

Dr. Butowski: I think the obvious advantage would hopefully be that in the unmethylated population where Temodar is not necessarily very helpful at all, that DelMar's drug would be helpful, or hopefully be more efficacious than what we presently have to choose from which of course isn't much of anything. That would be my main hope but it also seems to be effective in the methylated population as well to some degree.

Dr. Quibria: Right. So, you know, for the standard of care for several decades has been Temozolomide for front line therapy but as we have seen, DelMar is running a study in China examining or evaluating VAL-083 in front line. What do you think the rational would be to evaluate VAL-083 in front line?

Dr. Cloughesy: Well first, recurrent disease is a really difficult to treat tumor population and so you have this very high bar. The tumor is moving very fast and you're trying to get these therapies in to slow the tumor down. You may or may not see that effect. It may be that it is slowing the tumor down in some way but as we check the scans it may still be getting larger and we would say that's a failure. Maybe if we treated longer there may be other benefits that would come out of that but it's a really tough population. As a matter of fact, Temozolomide did not show much benefit in a recurrent study that was randomized so if we look at that and we say, "Okay that's a tough population maybe we should be

looking at this up front" and I think that's a great opportunity to allow more time on drug, more opportunity to have an effect, and more opportunity for either additive or synergistic effect with radiation which we now know is a beneficial therapy.

Dr. Quibria: Anybody else have anything add, Dr. Chen perhaps?

Dr. Chen: From my understanding now having of course if worked on the unmethylated patients, this should be very good because the patients didn't receive any other treatment, yet. So, in the first line they are tolerating it very well. Even now at 30 mg it's okay, they can tolerate it. Most of them can even reach 10 cycles. For the recurrent population, sometimes it could be a problem because they have already received an intensive treatment of other drugs.

Dr. de Groot: The only thing I would add to that is that you know we all believe Temodar provides a significant benefit to our patients but we're not particularly satisfied with that end result so if there's a drug that can be used even in the unmethylated patients and it has the opportunity to do better, then I think we would all be willing to move away from Temodar.

Mr. Zarrabian: In the recurrent setting one of the strategies the company has pursued is improving the median overall survival but also given Lomustine's safety profile as such as it is, you believe that if VAL-083 continues to go down, in the recurrent setting, down the path now where we're extending overall survival and having a better safety profile does this give us a better opportunity in this difficult to treat population than other options?

Dr. Reardon: Yeah, particularly in this disease, balancing out the benefit and the toxicity is critical. I think Lomustine, although it is our historical go-to as a backup salvage therapeutic, our patients really struggle with it. Glioblastoma increases in frequency as we get older, our older patients have diminished bone marrow reserve, most can only tolerate a dose or two of Lomustine even at reduced doses before their blood counts drop and some of them just don't recover. It's a tough drug in our patient population to be able to maintain a dose intensive exposure that would presumably be required for an anti-tumor benefit. It's a drug we all use but even more so than, as John mentioned, temozolomide I think which has a lot of mixed feelings about because we know the potential side effects and harm it can cause, especially accumulatively.

Dr. Butowski: I agree with David, that having your drug that you can presumably stay on longer because it's less toxic over time would result in benefits for the patients, both in terms of survival obviously, but in terms of quality of life as well.

Dr. Quibria: Alright, that's helpful. So, you know, some of you are actually involved in studies with checkpoint and other different types of therapies, as physicians and within the equity market, you might be more attracted to the more techy/technological type of drugs, like CAR-T and ADCs (anti-drug conjugates) and so forth. DelMar has a very promising drug in chemotherapy, how do you think the company can potentially overcome the bias that that everybody might potentially have towards something that could work, but you know it's a chemotherapy and it's not like biospecifics or something?

Dr. Cloughesy: If it works, it will be better. None of those other therapies have worked so if we have a therapy that works that means that's the benefit. It doesn't matter if it's sexy or not, our patients live longer. They get to be sexy on their own, our drug doesn't need to be sexy.

Dr. de Groot: The only other thing I would say is that the drugs that we know have an impact on improving overall survival are other toxic chemotherapy and alkylating agent and Temozolomide. So, I think if we can do something, or give something better than Temozolomide then I think that's what I think our patients would love. I think everyone wold absolutely love it.

Dr. Quibria: So, you know, DelMar has two studies as we mentioned. One that is running in China and that's in the front-line setting. More recently a lot of companies have started doing studies in China and we saw a BTK inhibitor that was recently approved here in the States, and most of those data was derived in China and yet it was approved here. Do you think there is a potential to take the data that is being generated now in China in the setting of phase two for potential of some sort of accelerated approval utilizing that data?

Dr. de Groot: Can you talk a little bit about your experience now working with your international multi center...

Dr. Cloughesy: Yeah. First, I think any single arm study that is done in front line its end point is likely OS and OS is a clinical benefit. I don't think that, cause it's you know, feel, function and survive, right for the FDA. And it needs to be randomized if that's going to happen. The only path forward for accelerated approval is tumor shrinkage in the recurrent setting because it's harder to understand the impact of tumor shrinkage with mixtures of radiation and other things so it makes it harder for regulators to understand what's going on. There's a number of really interesting opportunities in particular, global opportunities creating master protocols that where you have a common control in multiple treatment arms, and the idea that we could use data from multiple countries in that setting and have the ability when a drug hits and is positive to have a new drug application across these various countries, all kind of somewhat simultaneously, I think is a huge value and I think we are all trying to fight glioblastoma. Doesn't matter what country it's coming from, but it's better for us if we engage our friends in China, in Europe, in Canada and the US and if they engage us, so that we could work together to do this more quickly and more expediently and probably cheaper.

Dr. Quibria: That's helpful. Does anybody else have anything else additional? Alright so you know in terms of, you also mentioned in the recurrent setting. Do you think there may be path forward, a quicker path forward, in the recurrent setting? What do you think the bar would be in overall survival?

Dr. Reardon: I think that the accelerated path would be the most straight forward with the significant radiographic response because we know historically other than the antiangiogenic agents that decrease the permeability of blood supply and decrease contrast uptake which gives us a pseudo response, other than that context in the recurrent setting the percentage of patients who historically have had a radiographic response to any therapeutic has been single digit, essentially. So, if, even a single arm setting shows a significant rate of radiographic response, I think that would be a reason for consideration for accelerated approval. If you wanted to look at a landmark OS or PFS, it would need to be a randomized study.

Dr. Cloughesy: I might say that you know there's different opportunities, even in these master protocol platform trials to evaluate simultaneously different populations so you could evaluate recurrent at the same time you're evaluating newly diagnosed and seeing how they come out. These kind of adaptive approaches provide the opportunity where there's a shared control to get an OS signal, which I think David's saying would be the real path forward unless you have this crazy blockbuster that's obviously shrinking tumor, but I think this 10 month survival is really interesting and it would be a shame not to figure out what's going in recurrent and newly diagnosed and if you could do that at the same time that would be really valuable.

Mr. Johnson: I think one of the disconnects that we see sometimes between the scientific community and the investor community with GBM particularly if the investor's not focussed specifically on GBM, is the idea that a three or four month increase in survival benefit, perhaps to the investment community doesn't sound like it's much of a change. Whereas the impression I have always gotten speaking to KOLs in the space is that a 3- or 4-month improvement in overall survival in GBM population would be one of the larger shifts forward we have had in the last decade. Can you address that disconnect at all? What do you think, does a 3- or 4-month improvement in overall survival mean something here?

Dr. Butowski: I would say yes, I definitely think everyone here would take 3 or 4 months in terms of improved survival. I think the disconnect is just not understanding how little has been advanced in the field versus others. But I think it would be widely accepted without difficulty.

Dr. Cloughesy: I was just going to say the other value is not just median survival but a hazard ratio that then incorporates the rest of the curve so you could have a relatively small median survival but still have a hazard ratio because you're taking care of the tail of the curve and that would be of real value.

Dr. Reardon: I think it has to be the totality of the data too that they could take into account the side effect profile. There's a good side effect profile in this patient population and a 3 to 4-month improvement when we know the historical benchmark comparator is approximately 10 months, that's 30 - 40%, that's meaningful. If there's a maintenance of good quality of life and not significant toxicity.

Dr. Quibria: Alright, should we open it up to questions if there's anybody out there that has any questions for our panel while they're still here?

Mr. Zarrabian: I have one last question. You all have been observing DelMar now, being the scientific advisory Board members and Dr. Chen being the PI in our first line trial. What's your overall impression of the data that was presented in the posters today and in regard to both the recurrent study where Greg shared our Kaplan-Meier curve and also in the first line study? We have obviously been drinking from the Kool-Aid inside the company so we get encouraged by most data updates that are positive. So, on a non-biased view, what's your impression as practicing GBM experts of the data today? And I know we have a long way to go, I know we still have to measure overall survival, we have told the market that these trials will complete within the next 12 months and we will have top line results within 12 months, but even with that anticipation of final data, what's your views so far?

Dr. Cloughesy: I haven't treated any patients but looking at the data I think you know the overall survival signals are the one that I like to see. I think we're seeing that. That's the strongest piece that we could use. And then it's nice that we also have these other aspects coming in that there's complete response. I would love to see those because that's really a pretty, again, hard to know how to attribute

in a newly diagnosed setting, but when you're seeing it, it's not a bad thing to see the tumor go away, there's nothing bad about that. Question is, can we completely attribute it to it, but that is associated with a good survival signal with everything like that, I think it's all heading in the right direction.

Dr. Reardon: I agree with Tim and I think that putting it into perspective of what has benefited our patients historically which is a cytotoxic treatment approach. We have had other classes of fancy cancer therapies that unfortunately have been quite disappointing so we have a mechanisms of action that has a proven track record here and a rationale for why it may be superior to what we have had previously to what we have currently to work with. All of those factors that have been associated with why it's been challenging to move the bar in this disease, I think there's some reason to be hopeful that this has a likelihood of further confirming the signal so far.

Dr. Butowski: I'll chime in and summarize to say that I think mechanistically, survival data wise, imaging wise, quality of life and safety wise are all very intriguing and exciting to see complete data and move onto next steps.

Mr. Zarrabian: Thank you everyone, I appreciate it, and we promised to finish on time and I think we're there. Thank you for attending.