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Transcript of
Inovio Pharmaceuticals, Inc.
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Participants

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Peter Kies - Chief Financial Officer

Analysts

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Presentation

Operator
Thank you. Greetings, and welcome to the Inovio Third Quarter 2018 Financial Results Conference Call. At this time, all participants are in a listen-only mode. A brief question-and-answer session will follow the formal presentation. [Operator instructions]. As a reminder, this conference is being recorded.

It is now my pleasure to introduce your host Ben Matone, Director of Investor Relations. Thank you, Mr. Matone, you may begin.

Benjamin Matone - Investor Relations
Thank you and good afternoon, everyone, and thank you for joining the Inovio Pharmaceuticals third quarter 2018 corporate earnings conference call. This call is also being webcast live on our website, ir.inovio.com, and a replay will be available as indicated in our press release.

Before we begin, I would like to remind everyone that on this call we will be making certain forward-looking statements that relate to our business, which include our plans to develop our DNA immunotherapy platform, in combination with our proprietary delivery devices, in addition to our capital resources, all of which involve certain assumptions, risks and uncertainties and could cause actual results to differ materially from these statements. These statements are based on the beliefs and expectations of management as of today.

Our actual results may differ materially from our expectations. We assume no obligation to revise or update forward-looking statements, whether as a result of new information, future events, or otherwise. Investors should read carefully the risks and uncertainties described in today's press release which is posted on our website as well as the risk factors included in our filings with the SEC.

Joining us from Inovio are Dr. J. Joseph Kim, President and CEO; and Peter Kies, Chief Financial Officer.

With that, I would now like to turn the call over to Inovio's President and CEO, Dr. Kim.

Joseph Kim - President and Chief Executive Officer

Thank you, Ben, and good afternoon, everyone. For the past quarter and year, I've been extremely pleased with the clinical and R&D progress that the Inovio team has executed. Fueled by our efforts to date, we remain well-positioned to build on our previous successes from publishing results on clinical efficacy data to attracting new grants and partnerships to delivering on multiple advancements, particularly from our immuno-oncology programs, all of which provides Inovio with multiple value-creating catalysts over the coming months.

Our latest clinical results across multiple disease targets continued to generate a positive safety profile and class-leading immune response results. From our early oncology studies such as prostate cancer to infectious disease targets like HIV and MERS, Inovio has been able to consistently demonstrate durable and robust antibody and T cell immune responses.

In a nutshell, that's our core technology which has consistently demonstrated positive results, positive data that I am confident we will continue to generate. These results remain key value drivers and open doors towards new partnerships as well as for new opportunities and grants from our infectious disease platform.

Case in point is our $56 million CEPI funding, which I'll provide some updates on later in the call along with a multi-million dollar grant directly from the Bill & Melinda Gates Foundation that we announced just last month, all providing us with the opportunities to grow and validate our pipeline.

To lead off, I would like to offer an update on our lead asset VGX-3100 and our execution of the Phase 3 program for treating cervical dysplasia. This is a pre-cancer that frequently leads to cervical cancer if left untreated. So you could view this product not only as a treatment for a cervical condition, but as a product to prevent cervical cancer.

Today, REVEAL 1 has opened sites across 19 countries, actively recruiting patients. We continue to expect REVEAL 1 to be fully enrolled by early 2019 when we then anticipate opening recruitments for our confirmatory Phase 3 study REVEAL 2 shortly thereafter. To move as quickly as possible, we plan to utilize many of the same productive sites from our REVEAL 1 study for REVEAL 2. We do this in an effort to expedite recruitment as we continue to take an aggressive stance on getting both the sites up and running, and most importantly to treat patients.

Without getting into the intricacies of the recruitment side paradigm, it is important for me to stress that Inovio approaches this process very proactively, meaning we decide through our intelligence and experience which sites we utilize and keep open for patient recruitments. I believe that Inovio team's experience can deliver on our goal to file our BLA for VGX-3100 in the year 2021.

VGX-3100 is more than a product. It's a pipeline with aspirations to become a franchise in the field of treating all major HPV-related diseases. To remind you, our follow-on HPV trials are well underway for VGX-3100 in Phase 2 studies for vulvar dysplasia or VIN and anal dysplasia or AIN. Excitingly AIN actually has two separate Phase 2 components where we are targeting both HIV-positive and HIV-negative patients.

To recap, Inovio entered into a partnership with the AIDS Malignancy Consortium this year to evaluate VGX-3100 in 75 HIV-positive patients. The AMC will fund and execute a Phase 2 clinical trial to evaluate the efficacy of VGX-3100 in adult men and women with HPV-related high grade anal dysplasia. You can expect to see interim efficacy data from these VIN and AIN Phase 2 studies in 2019.

Now onto Inovio's immuno-oncology programs. We usually report on full studies and it's certainly more compelling to do so. I would like to provide you with what I consider to be a very fascinating case study. A story I would even say has more to be told.

In October, we received an early glimpse into what a T cell-enhancing product coupled with a checkpoint inhibitor could do in a metastatic cancer. It's a sample size of one, but a patient with progressing head and neck cancer, had a full remission, or a complete response, after a treatment with MEDI0457 followed by a PD-1 checkpoint inhibitor. That patient I am excited to share has been in remission for more than two years and counting.

You can read more about this in the October issue of the Journal Clinical Cancer Research. That was the story of one patient from an Inovio-sponsored study of 22 patients with an early stage head and neck squamous cell carcinoma. It was reported that remarkably 91%, or 20 out of 22 patients, showed T cell activity in the blood or tissue after treatment with MEDI0457 in the monotherapy trial.

The evidence that was detailed in this study likely suggests that MEDI0457 primed the immune system and boosted the effects of subsequent anti-PD-1 therapy leading to a durable complete remission. The published data provides us with the optimism to an ongoing MedImmune Phase 2 study that could report interim efficacy data in 2019. In the Phase 2 study being run by MedImmune they are evaluating the anti-tumor activity of MEDI0457 in combination with this checkpoint inhibitor, durvalumab, in 50 patients with recurrent/metastatic HPV 16 or 18 associated head and neck cancer.

Additionally, Medi will open another Phase 2 study in the next several weeks to evaluate the anti-tumor activity of MEDI0457 in combination with durva again, and this time in patients with multiple types of recurrent/metastatic HPV 16 or 18 associated cancers other than head and neck. The initiation of this trial will trigger a milestone payment from MedImmune to Inovio.

I truly see a great future from MEDI0457 and its sister product VGX-3100, one that fulfills our ambition to be the go-to-players for treating all major HPV-related pre-cancers and cancers.

Moving onto INO-5401. Enrollment is going as planned and on target to report interim Phase 2 data for both glioblastoma and bladder cancer studies next year. While recent novel immune therapeutic options have certainly given more patients with bladder cancer and GBM more hope.

The use of checkpoint inhibitors still remains limited and has room for improvement. We are really excited to test the T cell-generating effects of INO-5401 in combination with PD-1 or PD-L1 checkpoint inhibitors separately from Regeneron and Genentech.

INO-5401 is composed of three of our most active SynCon cancer antigens combined into a single product. Our goal is to see if we can replicate in these two INO-5401 Phase 2 studies, the level of T cell responses data and anti-tumor responses observed in one complete responder from the Clinical Cancer Research paper.

And lastly, for INO-5150, we presented prostate cancer data from our Phase 2 study at the International Medical Conference, ESMO 2018 last month. We presented, INO-5150 data demonstrating a slowing of prostate specific antigen doubling time in men with prostate cancer.

The data showed that 86 percent of the patients remain progression free as week 72. Let me repeat that. After treatment with INO-5150, almost nine out of ten patients showed no increase in their tumors, a year-and-a-half after treatment. This data and results from our DNA immunotherapies in combination with checkpoint inhibitors

makes us more attractive as we continue our partnering discussions. Expect to see more development on this in the coming weeks, but Inovio's plan and progress to license out and partner a Phase 2 trial for prostate cancer remains on track.

Before our financial updates, I'd like to run through a couple of our notable platform development program advances. First, PENNVAX-GP, we dosed our first patient in August in a randomized clinical trial that will evaluate PENNVAX-GP's ability to drive remission of HIV infection. Enrollment remains on track and the trial is part of a previously reported multiyear $6.95 million grant from the NIH to develop a single or combination therapy using Inovio's PENNVAX-GP with the goal of attaining long-term HIV remission. We anticipate interim results in 2019.

Second, we dosed our first subject with INO-4700 or GLS-5,300 to prevent infection from the deadly Middle East respiratory syndrome or MERS virus and a Phase 1/2a study. The trial is ongoing in South Korea, sponsored by our Korean development partner GeneOne Life Science, with a full funding from the International Vaccine Institute.

Lastly, regarding our other infectious disease targets, in collaborations and supported with CEPI, we will move our Lassa vaccine into the first human trial early next year. We also expect additional clinical publications from our Ebola vaccine trial and our MERS vaccine trial in the U.S.

With that, I'll turn over the call to our CFO, Peter Kies, who will discuss our third quarter financials. Peter.

Peter Kies - Chief Financial Officer
Thanks, Joseph. For the third quarter ended September 30, 2018, Inovio reported total revenue of $2 million, which compares to $2.6 million for the same period a year ago. Total operating expenses were $28.6 million compared to $31.8 million for the same period in 2017.

While our third quarter 2018 financials and details are outlined within our 10-Q and press release, I would like to provide some additional color as it pertains to the adoption of the accounting standard update surrounding revenue from contracts with customers. Beginning in January 1, 2018, all contributions received from current grant agreements have been recorded as a contra-expense as opposed to revenue on the consolidated statements of operations.

For the three months ended September 30, 2018, $2.6 million was recorded as a contra research and development expense. This amount would have been classified as grant revenue in the prior year. Had this change in presentation not occurred, total revenue would have been $4.6 million for the three months ended September 30, 2018 compared to $2.6 million for the same period in 2017. The same applies to total operating expenses as these would have been $31.2 million compared to $31.8 million for the same period in 2017.

The increase in comparable revenue and grant agreement recognition for the third quarter of 2018 compared to 2017 was primarily due to an increase in our MedImmune collaboration and our CEPI grant of $1.5 million and $1.2 million respectively. These increases were offset by a decrease in grant funding recognized from our DARPA Ebola grant of $1.2 million among other variances.

Our net loss for the quarter ended September 30, 2018 was $25 million or $0.27 per share of basic and dilutive, which compares to $34.1 million or $0.39 per share basic and $0.40 per share dilutive for the same period a year-ago. As of September 30, 2018, cash and cash equivalents and short-term investments were $85.5 million compared to $95.6 million as of June 30, 2018.

As a reminder, our third quarter 2018 balance sheet and statement of operations can be found in today's press release or in the Form 10-Q filed with the SEC, as well as on our website under investors and financial reports.

With that, I'll turn the call back over to Joseph. Thanks.

Joseph Kim - President and Chief Executive Officer
Thanks, Peter. Before I get to your questions, I want to keep in the back of your mind an exciting emerging part of our future that offers remarkable upside potential. That is our DNA included Monoclonal Antibody or dMAb program. What kind of upside do I see with this program? Well, please consider traditional monoclonal antibodies account for more than $50 billion in pharmaceutical sales each year and Inovio's dMAb products could provide a new disruptive entry. dMAb may even significantly improve upon this class [ph]. We can build a better monoclonal antibody by using our synthetic design and inpatient production.

Let me explain what I mean by inpatient production. When deliver directly into the body, our DNA encoded plasmids provides the genetic instructions to enable the patient's own cells to become the factory, which manufacturers the therapeutic antibody products. If successful, this would removed the difficult and costly issues during the manufacture of Monoclonal Antibody drugs. We've demonstrated the potential of this platform by publishing several preclinical results of dMAb protecting against deadly infections and eliminating cancers. We also recently published on our own dMAb checkpoint inhibitors with demonstrated anti-cancer effects.

The U.S. patent office just granted the first two patents covering our dMAb technology last quarter and more to come in the future. This platform is advancing at a rapid clip as we plan to initiate our first in human clinical study with a dMAb product in 2019. Speaking of 2019, with the wind at our backs generated by positive 2018 data, 2019 will be a pivotal one for Inovio and our shareholders.

From Inovio, in 2019 you will see additional partnerships, including finalizing a novel cancer combination trial through the Parker Institute as we guided you in the beginning of the year. More collaborative combination trials, especially with checkpoint inhibitors and other molecules, continued progress of our REVEAL Phase 3 studies, the advancement of our global health portfolio of infectious disease vaccines, and most important together with our partners, MedImmune, Genentech and Regeneron, we will report to you on our cancer combination efficacy trials. I look forward to discussing with you Inovio's next giant steps.

Now your questions. Operator, please open the line for the analysts.

Operator
We will now be conducting a question-and-answer session. [Operator instructions]. Our first question comes from Chris Raymond with Piper Jaffray. Please go ahead.

Q: Hi, thanks for taking my questions. Just on the VGX-3100, so I think last quarter you guys gave specific number of site updates. And I think the last we heard at the end of June, you had 70 sites and you were targeting I think 100 by the end of the year. I wonder if you could sort of give any updates as to where you are towards attaining that goal.

And then on 5401, really interesting data, I think you have coming in 2019 with these checkpoint inhibitor combo readouts. Can you just sort of frame for us what to expect? Are we looking for response rate data, PFS data? Any sort of color as to what these readouts are going to look like would be great. Thanks.

Joseph Kim - President and Chief Executive Officer
Yes, absolutely. Thanks, Chris. Thanks for the thoughtful questions. Yes, we have opened altogether more than 90 sites globally for 3100 REVEAL 1 trial. It's a dynamic process. We gave all these sites all the chances to enroll productively the patients. If they fail over several weeks or several months, we close them down, so we don't indefinitely keep them open. So we've opened total of 90-plus sites, almost meeting our targets. But we have 70-plus sites actively enrolling patients very productively across the world.

And then moving onto your 5401 question. Yes, I couldn't be any more excited about the combination PD-1, PD-L1 checkpoint inhibitor study. So for bladder study, we would look for overall response rates PR and CR in both checkpoint-refractory and non-PD-1, PD-L1 experienced bladder cancer patients. These are muscle-invasive metastatic/recurrent bladder cancer patients. And just to remind you, we're dosing INO-5401 in combination with atezo from Genentech, so it will be an ORR-based, along with the immune responses and safety in the bladder cancer trial.

Secondly, the GBM study is slightly differently designed. It's a primary efficacy readout from this study, besides safety and tolerability as well as immune responses, is the overall survival. But in 2019, we'll look forward to having PFS, progression-free survival at six months and also at 12 months in these patients. So although overall survival is our primary efficacy endpoint, we think the PFS numbers will give us a good window into the response anti-tumor effects of INO-5401. So we're very excited about both of those studies.

And then lastly, MEDI0457 is in a metastatic head and neck cancer setting. And I have to reiterate here, MedImmune controls the study and the dissemination of the data and so on. So we're just providing some conjectures here, but we expect based on the Clinical Cancer Research published data of the Phase 1 patients who sequentially received the checkpoint inhibitor, where we saw a one complete responder.

We expect some additional data from that study as well as MedImmune run Phase 2 study, where they're combining durva plus 457 and really the target to beat is Keytruda, who has achieved overall response rate of 16% across various trials of PR and CR, with 4% CR in metastatic head and neck cancer setting. So we think that's the bar to beat and hopefully MedImmune can demonstrate that.

Q: Okay, thank you.

Joseph Kim - President and Chief Executive Officer
Thank you, Chris.

Operator
Our next question comes from Shawn Egan with Citi. Please go ahead.

Q: Good evening. This is Shawn Egan calling in for Joel Beatty. Thanks for taking my question.

Joseph Kim - President and Chief Executive Officer
Hi, Shawn.

Q: How are you?

Joseph Kim - President and Chief Executive Officer
Good. Thank you.

Q: So today you've generated a lot of really strong immune responses kind of across the board. Looking at it from a high level, have you identified any factors that will help you predict, which tumors could respond clinically to your products immune responses?

Joseph Kim - President and Chief Executive Officer
Well, so I think overall, we've demonstrated strong T cell responses to our hTERT antigen, which anchors the INO-5401 as well as our INO-1400 programs. We've also seen strong T cell responses to PSA and PSMA. Coincidentally PSMA is a second antigen in our INO-5401.

So we're pretty bullish on, obviously the 5401 study has just begun earlier this year, but we think we can generate antigen T cell, particularly CD8 positive T cell immune responses across these antigens. And in terms of the antigen spectrum, we've pretty much tested every top 100 cancer antigen list produced by the National Cancer Institute.

In fact WT1, which is a third component in INO-5401, has been perennially number one cancer antigen rated by the National Cancer Institute. So yes, we have selected INO-5401 antigens through extremely systematic vetting across our preclinical models and now we're beginning to see those translating into the clinical studies.

In terms of the responsiveness, I mean we have to get more data, but what I can tell you is what my feeling is, I don't think it really matters the PD-L1 expression levels of these tumors. I think our delivery of the SynCon antigens into the muscle of the patient, where their muscle cells become the factory of these antigens and their process and presented properly through MHC class I to the T cells, especially the CDA T cells, I think it's the right mechanism of action to maximize the T cell immune responses in these patients. So needless to say, we're very excited about what we may see in our MEDI0457 Phase 2 studies in head and neck as well as the bladder and GBM studies in combination with the checkpoints.

And lastly, as I mentioned, MedImmune isn't stopping at just head and neck. Through their collaborators at MD Anderson. We expect to start a second Phase 2 study with durva plus 457 in all other major HPV-caused cancers other than head and neck, obviously that basis is already covered with the current Phase 2 study. So next several months, we expect to see a lot of clinical data from our studies with Medimmune, with our collaborators from Regeneron and Genentech. So stay tuned, I couldn't be any more excited about these studies.

Q: Great. I look forward to him as well, Joseph. And then just as a brief follow-up. With the advancement of your VIN and AIN studies as well as the advancement of MedImmune studies, how much read through is there from one HPV indication to another and how would success in one indication provide confidence for the other indications?

Joseph Kim - President and Chief Executive Officer
Well, our feeling is that we could expect a good read through, here's the reason why. So especially in HPV-caused diseases, it's the same virus, in particularly the E6 and E7 oncogenes that turns the normal cells, once HPV infects those cells, whether it's in the vulvar area or the cervical area or anal area, from normal cells to a precancerous one and eventually the cancerous one.

So developing an immunotherapy attack against E6 and E7 oncogenes, exactly is what VGX-3100 does, is a great strategy against these cancers. And we've demonstrated with very high p-value, meeting all of the efficacy endpoints for VGX-3100 against cervical dysplasia in a controlled randomized double-blind Phase 2 study. So we think the same mechanism of action, which is the patient's own T cell immunity, should be able to clear the

lesions and the virus in other organs in the body. Of course, we still have to prove that and that's what these three Phase 2 studies for VIN, and AIN, two separate ones for AIN, will help us determine in the next few months.

Q: Great. And then just shifting gears real quick, if I could ask one more question. Regarding the HIV remission study, your interim look, how many patients will that look include and what endpoints will be assessed and how long will patients have been in the study at that point?

Joseph Kim - President and Chief Executive Officer
Yes. It's a total of 70 patients. A lot of it is going to be looking at the HIV reservoirs as well as the immune responses that we've already seen CD8 T cell responses in the HIV infected patients, we expect to be similar in HIV naive patients that we already saw in HVTN 098 study where we saw unprecedented the level of CD8 T cells to a vaccine, HIV vaccine.

So we're very excited about this study because it's really, it's a high risk, high benefit where we all know that no amount of drugs in combination, and Gilead saw this and so did other big pharma companies, when used together can clear HIV infection in humans. That just has not been seen. Chronic HIV infection does not clear using a combination of drugs.

So what the KOLs are thinking is maybe a combination with drugs and an immunotherapy like a vaccine could flush out these viruses hiding away in these reservoirs. And we're hoping that we can demonstrate that in this very ambitious study as a monotherapy with drugs or maybe in combination with a checkpoint inhibitor that could come a later part of this overall study.

Q: Thank you for all the color Joseph. Have a good day.

Joseph Kim - President and Chief Executive Officer
Great. Thank you, Sean.

Operator
Thank you. Our next question is from Gregory Renza with RBC Capital Markets. Please proceed with your question.

Q: Hi, guys. Thanks for taking my question.

Joseph Kim - President and Chief Executive Officer
Hi, Greg.

Q: Hi, how are you? Joseph, I just wanted to start more broadly just in regards to your views on just the general partnership environment around oncology and combo assets given that you're certainly in the trenches and certainly in the context of 5150. I'm just curious to get your views on how that's evolved to just generally, certainly willingness to partner the confidence in generally interpreting early stage data and just the assigning valuations. Any views you have in there, it would be great. Thanks.

Joseph Kim - President and Chief Executive Officer
Yes, I think that's a really good question. And answer is going to be really a description of an evolving landscape because immuno-oncology certainly is a very exciting growth area. Checkpoint inhibitors have been doing phenomenally well, PD-1, PD-L1 inhibitors, CTLA-4 previous to that, but that's been plateauing.

So the true effect of PD-1, PD-L1 is going to be a basic level of efficacy that all cancer patients I believe are going to expect, around 20% ORR across the board, minus melanoma perhaps, and perhaps much lower in GBM and pancreas and ovarian. So the landscape has been how do we improve upon those PD-1, PD-L1 effects because in the next five years there will be about 40 different PD-1, PD-L1 inhibitor products that could hit the market, 35 just in China.

So how are we going to improve upon that 20% ceiling? I think Bristol Myers took a big step in partnering with Nektar for their program, with the goal of improving upon broadly for overall responses. I think that was an ambitious deal. And then there are other combination approaches that other vaccines or other immunotherapies with checkpoint inhibitors. I think the jury is still out on all of these.

But what I know for sure and where our Inovio's value proposition lies, is regardless of mechanism of action and regardless of what PD-1, PD-L1 or other immunotherapy agents that you're going to use. One thing for sure, I don't care what the PD-L1 status is, high or low and every company looks at. Merck thinks 1% or more is high PD-L1. That's the only time that I see where 1% is considered high, whereas MedImmune considers 20% high or 25% high, so every group has a different perspective on these. The one thing I know for sure is you need CD8 positive killer T cells to kill tumors. There are other ways to kill tumor cells, but the most efficient way is CD8 positive activated granzyme B, perforin expressing CD8 T cells. So at the end of the day, I think INO-5150, 5401 and MEDI0457 and all of the other follow-on programs that we have are really, we're backing on the power of our patients' own immune system, specifically the CD8 positive professional killer T cells that can dissolve these tumors.

And excitingly, we demonstrated that in one patient in our Clinical Cancer Research Publication, complete response by the fourth dose of PD-1. In this case, it was Opdivo, by fourth dose at month two, you could read that yourself, but what excites the investigators is not the fact that we saw a complete responder in one patient, but the ferocity and the velocity of how fast the complete response was demonstrated. So this is four biweekly doses of nivo and our PIs at UPenn Medical Center had never seen a checkpoint inhibitor alone demonstrating that type of anticancer response that fast.

So I think that really gets us excited and our partners MedImmune and others are very excited about this. And as we build out our database and number of patients with both partial and complete responses in the next several months and years, we'll be able to really demonstrate this thesis and we will be able to turn those into approved products and partners, partnerships and licenses and so on. So that's really a long winded answer to your question, but thank you for that thoughtful question.

Q: Absolutely, very helpful. Thank you. And just a quick follow-up, with respect to your collaboration with MedImmune on 457, how would you characterize that structurally? Is that something of a model where you view that as a benchmark for future partnerships, the way that perhaps arranged or do you see perhaps those deviating in other directions?

Joseph Kim - President and Chief Executive Officer
Well, I think first of all, our relationship with MedImmune is spectacular and gets stronger each time. It's a very close knit relationship. We value it and this is a standard license agreement where we received $27.5 million up front, we'll continue to receive milestone payments as 457 progresses and advances in the pipeline, and I will share in the future upsides of royalties when the 457 is sold in the market. And we like this because I think MedImmune, I could be speaking out of line here, but it's competing for its life against Keytruda and Opdivo as well.

So and there's newcomers like Regeneron's PD-1 semi and like I said, there's like 45 more, new potential entrants in the next several years. So this is a dog-eat-dog world. So how does MedImmune stand out? How does durva compete with Keytruda in head and neck cancer?

Well, the true answer is if you can beat Keytruda 16% ORR in metastatic head and neck cancer setting, let's say you double it to 30%. We think, I mean that's my opinion, that market share can be taken from Merck significantly. So that enhances MedImmune's strategic positioning of durva, compared to Keytruda or Opdivo. And, and we see that model to work very well.

Now, let me just step back and describe INO-5401, 5401 we chose not to do a standard license agreement like we did with MedImmune because 5401 is almost priceless in that it's not just the product that can treat bladder or brain cancer. If we're successful in demonstrating efficacy against these two, we can treat breast cancer, colorectal cancer, lung cancer, pancreas. I mean this is multiple cancer types we can treat with these three universal cancer antigens, hTERT, WT1 and PSMA.

So we strategically did not do a partnership license deal with Genentech or Regeneron. That's not to say we will not do that later after we see efficacy from these trials. But we will still be open to do a deal with Merck or Pfizer or GSK or whoever that wants to bring in such a potentially valuable product like INO-5401. But I don't want to get too far ahead. Let us demonstrate our efficacy first, and anti-tumor activity, and then I think the potential deals are almost limitless for 5401. And thereby the value of our platform also is even bigger because we can pump out another INO-5401 from our platform in a matter of a short time.

So I think we, as you know, Greg, we've been building from foundationally, so we built our platform, we have the technology, we have the patents. Then we established our immune responses capabilities against different cancer targets, different infectious disease agents. Then now we're beginning to demonstrate measurable efficacy from clinical trials, both from the pre-cancer setting and now you're seeing the tip of the iceberg of a cancer setting. And next 12 months or so you're going to see a lot more anti-cancer efficacy data coming about. We will continue to advance our technology and our platform, we expect to have more partnerships to aid in our build-out of these products, so we're very, very excited about where we are now and where we're going to be 12 months from now.

Q: Thanks Joseph. I appreciate all the color.

Joseph Kim - President and Chief Executive Officer
Yes. Thank you.

Operator
Our next question comes from Steve Willey with Stifel. Please go ahead.

Q: Yes, good afternoon. Thanks for taking the questions.

Joseph Kim - President and Chief Executive Officer
Hi, Steve.

Q: Hi, Joseph. Just to follow-up on 5401, can you maybe just talk a little bit about, as you think about the 2019 data presentation, is there a threshold patient number that you're aiming for with respect to a data disclosure or is that really kind of just dictated by the medical conference calendar?

And with respect to the bladder trial specifically, I know that I think the protocol calls for both prescreening and post treatment biopsy samples and just wondering if that's something that we could maybe expect with an initial look at the response data?

Joseph Kim - President and Chief Executive Officer
Yes. Thank you. Great question. Those in the IO feel our lives predicate around those conferences, right? AACR, ASCO, ESMO it's like four seasons in a year, and other minor conferences as well. It's going to be dictated about where we can present and where we publish as well as the weightiness of the sample size and really the quality of the data.

So I can just give you a simple answer, but that's really going to be evolved around our accrual and the quality of the data we're going to get. So I think 2019, I'm not projecting a specific conference but we'll able to titrate that better as we come closer to that. And in terms of the actual data, yes, safety, immune responses as well as the PFS for GBM, obviously objective response rates for bladder and it really depends on the quality of the data and the sample size as well as the level of competition that we're competing against in each of those indications.

Q: Got it. And then just how important is that biopsy data to kind of establish on mechanism, proof of principle? Just given the fact that there's been obviously a lot of debate within the IO space about companies making future development decisions based on somewhat smallish, open label, Phase 2 trials that are really just kind of kicking out response data?

Joseph Kim - President and Chief Executive Officer
Yes, I mean it's we all have such a short memory, the whole field made huge investment decisions based on a handful of PD-1 inhibitor data in the beginning. Couple patients and so on. So I think there are some failures or the inability to replicate small scale data in a larger setting. I think that's making some of the players gun shy, but I think immunotherapy still is the most important field out there in medical science right now.

And certainly we think those biopsies are important, but it's just one measurement. Obviously overall survival is a great endpoint because there's no questions on a patient being alive versus not. Perhaps PFS is also important, but visually, seeing tumors shrink in scans and so on is also very exciting. And those durable responses that immunotherapies create are also important too.

I think the mechanism of action I come back to the biomarker part of what I said earlier. We don't rely on a single biomarker per se, especially in cancer. We look at biopsies, we look at T cell responses in the periphery, we look at the T cell infiltration in the tumors, obviously looking at different radio scans and we take a systematic approach to this. But I'll come back to our fundamental thesis, which is if you can generate activated CD8 T cell responses against the tumor antigen that's relevant, I think we're going to have a great shot at showing antitumor activity, and that's the fundamental scientific basis that we're going after in all of these studies.

So now we're going after HPV antigens and 457 and VGX-3100, we're going after hTERT and PSMA and WT1 for 5401 and so on and so on. And so far we've been able to generate T cell responses in patients setting against all of these important antigens and we aim to build upon that science, and really next several months will help us to decipher whether this thesis is a multibillion dollar payout or not. And, and we'll look forward to that time point.

Q: Got it. And then just respect to the Phase 2 VIN trial, I guess that's still actively enrolling patients, I guess the trials been open now for about maybe 18 months or so, and I think there's about 20 sites accruing. So just wondering if that's indicative of these patients, I guess being a bit harder to find, or is there something specific to

the trial protocol that is triggering a lot of screen failures? Just kind of curious if you could provide some commentary around the pace of enrollment into that trial.

Joseph Kim - President and Chief Executive Officer
Yes. Just to speak about that in detail, yes, we have had that trial open for about 14 months and we are still enrolling. We're more than half enrolled. VIN is a small population indication about 8,000 in the U.S. So it's an orphan [ph] indication. It still is a horrible disease from disfigurement and morbidity standpoint, but our team is doing a great job in recruiting those patients. And I think the proof will be in the pudding, so we expect to have those results in 2019.

And similarly AIN is accruing much quicker than VIN was and I think that speaks to the horrible disease that anal dysplasia is, where the patients are reminded almost daily, sometimes more than several times a day on their illness. So I think there's higher motivation and these patients go through multiple surgeries in a given year, so there is a high demand for that and certainly our accrual is reflecting that status. So yes, we think all of these dysplasias present as a very attractive medical indication and certainly we think they'll translate into attractive commercial indication as well.

Q: Got it. And then just lastly on the first human dMAb product, should we expect that to be an infectious disease? And then, I guess, what's the preclinical data you have as far that compares both, I guess interpatient responses or interpatient variability? I would imagine that there's some component here of transduction efficiency, right, that differs on, perhaps, injection to injection or patient to patient basis that somehow influences systemic antibody titers. So just kind of wondering if you could give some color around kind of the target disease setting and then just the heterogeneity of titers that you would expect to see post transduction. Thank you.

Joseph Kim - President and Chief Executive Officer
Yes, Thanks, Steve. Awesome question and really delving into various deepness of science. So the taking it from the delivery and so we've been able to generate over 100 micrograms per ml expression systemically in small animals.

We're able to translate that in monkeys in the order of 30 micrograms per ml range. We've published these results, some we have extensively against different targets, infectious disease targets first because that's where the funders gave us money to build out this platform, so we've published on pseudomonas flu, HIV and so on. And then we just published some anti-cancer dMAb, PSMA as well as our own version of treme and YERVOY, anti CTLA-4 where we showed very strong anti-tumor responses. We have our own PD-1 inhibitor of Keytruda version and Opdivo version and so on.

In terms of the subject to subject variability there are, but it's not as variable as you might imagine. And we can control that through those things and so on. I can't speak to any person-to-person variation that we may see. We still have to do the first trials and optimize them. But really this was just a pipedream a little over 3.5 years ago. So we've really pushed it from Bill Gates' pipe dream to where we could dose our first patient with the dMAb product.

So we have in terms of the targets, we've been like the mad scientist. So we've published on anti-viral effects of the dMAb. In fact, compared to MedImmune, protein based mAb, we published that dMAb against pseudomonas target is as good or better than a protein mAb that MedImmune and we co-published this last year, and similarly an influenza model as well. So I would venture to say at least in animal models that dMAbs are actually better than protein mAbs because they fold perfectly naturally to the host, whether its mice or monkeys and we hope to show in humans.

And then in terms of potential targets, we have our own version of Humira. We have our own version of PCSK9, all of the checkpoint inhibitors. I'm not saying we're going to be developing these as products. Against all of those, we could again with various partnerships and licenses, but we just want to show the capability and then we'll select the best targets to develop in the clinic.

And we could go after rare disease target where we can get to a commercialization much quicker because there are so many proteins or mAbs where the patients have to dose themselves multiple times in the week and that will be a perfect candidate for the dMAb for our deep protein therapeutic approach.

Q: So with respect to just the first inhuman target indication, is this going to be infectious disease then?

Joseph Kim - President and Chief Executive Officer
Yes, because that's where the funders—it's enabling us to develop out our platform with a targeted funding, so yes.

Q: Got it. Thanks for taking the questions.

Joseph Kim - President and Chief Executive Officer
Yes, thanks, Steve.

Operator
Our next question comes from Ram Selvaraju with H.C. Wainwright. Please proceed with your question.

Q: Hi, thanks so much for taking my questions. Just three very quick ones, could you perhaps comment on given the plethora of combination regimens that are being tried with all these various checkpoint inhibitors, whether in a general sense you're seeing any evidence, both now and in potentially the midterm that it might be challenging to recruit patients in future proof-of-concept studies trying to establish the validity of your combination regimen approaches in patients who have insufficient or inadequate response to checkpoint inhibitors.

Secondly, if you could comment on whether the applicability in sequence is potentially more likely to result in a favorable response as opposed to a true combination. So do you see any evidence that sequential administration is better than simultaneous administration of your regimen plus a checkpoint inhibitor?

And then lastly, I wanted to know this is relative to, I think a question that was asked earlier about the dMAb platform, whether you have clear evidence at this juncture of differential frequency of administration from patient to patient and whether that's likely to be necessary in the clinical development setting for the dMAbs. Thank you.

Joseph Kim - President and Chief Executive Officer
Yes, thanks, Ram. So let me take the last question first, dMAb the patient-to-patient variability, I think we have to dose first in human studies first. That's what we'll focus on with our first Phase 1 study. So it's hard to speak to that now. In mice, they're very consistent and monkeys pretty well, even other larger animals, we have pretty good consistency, but humans are different.

So we look forward to getting the data and optimizing then. I'm not claiming our first in human study going to be a pivotal study that's going to go to licensure first, but I think it's just the exciting part of this translational

development of our new application to our platform. But thanks for the great questions. Similar to what Steve Willey asked, very good questions technically.

In terms of the first question, yes, the combination, there was a study that was presented I think at last year's ASCO or one of the other conferences that there aren't enough trial participants to fit all of the IO studies out there. So of course, it's a very competitive space. So for GBM or pancreatic study, we could enroll very, very fast because that's a very desperate cancer.

Bladder and melanoma that's much more competitive because a lot of the companies and investigators are going after more immune responsive tumors. And that's precisely the reason why we position our 5401 study, one for metastatic bladder and second for GBM. We wanted to test out the spectrum of immune responsiveness. So reality where – we have to go where the patients are and it's, it's something that all companies and different investigators have to live with.

And then second question about sequential. Yes, that's great question. Answer is yes. But it's hard to find the right sequence in light of these combinations. Patients aren't like rodents, where you can have different groups and controls and so on. So the reality is you do small sample size trials, usually open-label and a lot of times you're not even controlling properly, but that's the reality of IO, the speed of which where we're competing with everybody else.

All of our Phase 2 studies don't have checkpoint alone comparisons. We're banking on using these well-established PD-1 or PD-L1 inhibitors, so we can compare. But that's not to say that sequential uses of these checkpoints are not important. And we have some studies that we're designing for the future that will test some of that, and I think other groups are as well. So collectively I think the field is going to learn more about the science of these combination studies in the coming months and years.

Q: Great. Thank you very much.

Joseph Kim - President and Chief Executive Officer
Thanks.

Operator
Our next question comes from Jason McCarthy with Maxim Group. Please go ahead.

Q: Hi. This is actually Noreen [ph] on for Jason. Thanks for taking the questions. Actually first nobody mentioned it. So Joseph, I just wanted to say nice job on that op-ed in The Hill by the way.

Joseph Kim - President and Chief Executive Officer
Thanks, Noreen.

Q: So with regards to—my first question relates to 5150. You presented some positive data on the program in prostate cancer at this year's ESMO, and in the Phase 1 clinical trial you had patients that had no history of androgen-deprivation therapy, given that ADT appears to be fairly standard now followed by anti-androgens, which have also moved up the treatment paradigm for prostate cancer with Erleada and Xtandi in the non-metastatic setting. Where do you think 5150 would be positioned if you're moving forward with the program with a partner?

Joseph Kim - President and Chief Executive Officer

Wonderful question. We think just from the reality of where the field is, we'll have to go to later stage patients' [indiscernible]-resistant metastatic setting in combination with established products or maybe even other immunotherapy. So that's really where we can show the biggest bang for the buck, because the field doesn't have the patients or the patience to do these earlier stage studies because it's harder to get approval through these earlier interventions. So I think, we and our potential partners will have to go into the late stage in this case [indiscernible]-resistant setting, aesthetic setting. So that's where we expect the next trial to be.

Q: Sure. Okay, great. Thanks for the clarification. And with regard to your 510 [ph] program in GBM with Regeneron, you sort of touched on this earlier, but I was just wondering if you could give us a sense of what you would view as a positive efficacy signal with this combination given that, if you look at the CheckMate 143 trial with Nivo versus Avast [ph] and the ORR, Nivo was so low at 8% and so that program was terminated prematurely. So what would give you confidence in the program and additionally, would you happen to know the response rates of Regeneron's PD-1, and monotherapy and GBM?

Joseph Kim - President and Chief Executive Officer
The last question, I can't really comment on. But I can comment on the first part. Yes, certainly GBM has been a challenge even for PD-1 inhibitors, so especially in recurrent setting, but also in newly diagnosed setting. So that's precisely the reason why we've chosen to go after GBM as one of our two combination cancer studies for 5401.

Our stated objective is having overall survival benefit of three months when it's all said and done. Are we going to see that next year? No. Next year, so we expect to have that data in 2020, but next year what we could show is PFS progression free survival at month six, as well as month 12, what percentage of our patients have not progressed, and we look at the survival rates from the field. In unmethylated patients overall survival is about 14 months in methylated patients, about 22 months, and we're enrolling both cohorts, both methylated and unmethylated. So ultimately three months' improvement of overall survival, but we expect to see a PFS 6 and 12 of high percentages hopefully from these patients next year, and then we have immune responses and safety, we'll be tracking.

In contrast for bladder, these are the patients that we're focusing on our checkpoint refractory for the most part. And we'll look for these patients will be scanned every two months. So we're definitely looking for anti-tumor effects either PR or CR in those patients, so ORR will be our objective there.

Q: Great. Thanks for the clarification and thanks for taking the questions.

Joseph Kim - President and Chief Executive Officer
Yes, thanks, Noreen.

Operator
There are no further questions at this time. I would now like to turn the floor back over to Joseph Kim for closing comments.

Joseph Kim - President and Chief Executive Officer
Yes, thank you. Thank you for staying really through all of the very important Q&A. We look forward to sharing great advancements in the coming months with you. And thank you for your interest and attention through this call. Thanks a lot.